Risk Assessment in Oral Health

A Concise Guide for Clinical Application

Iain L. C. Chapple
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Part I

Introduction: Risk Assessment in Modern Healthcare

Introduction

Iain L. C. Chapple

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The world's population is ageing, and with that brings a significant burden from chronic non-communicable diseases (NCDs) to individuals and the healthcare economy. Ageing is a process is that is, in part, genetically determined, but one that can be modified by lifestyle factors and the environment in which individuals find or place themselves. Therefore, individuals can make health and lifestyle choices, but professional advice on those choices requires precise data that pertains to that individual, rather than being based on population norms; hence, the emergence of personalised and precision medicine in twenty-first century healthcare.

Risk assessment and risk monitoring is a process that should be central to any successful business: risks are identified, registered on a scale (low to high), and managed in order to ensure financial stability and growth. However, in healthcare, progress in embedding risk assessment into daily practice has been limited. In 1999, the American Academy of Cardiology stated that "effective primary prevention requires assessment of risk to categorize patients for selection of appropriate interventions". In 2002 the WHO strongly advocated assessment of high-risk individuals in risk reduction strategies and pointed out that the "estimation of the potential impact of a health hazard can never wait until perfect data is available, as that is

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unlikely to occur" [1]. This was tantamount to encouraging the world's governments to "get on with it". In 2004 the WHO observed that "reliable and comparable analysis of risks to health is key for preventing disease" [2]. Indeed it was Galileo Gelilei (1564–1642) who stated "measure what is measurable, and make measurable what is not so", for if we cannot measure things, we cannot improve performance against those measures. Given the digital era, and the potential to analyse big data and employ machine-learning algorithms in order to improve individual risk prediction, the healthcare community is running out of excuses for not implementing a paradigm shift in models of care provision from traditional repair models to risk-guided prevention.

Non-communicable diseases are increasing in prevalence globally and are responsible for 41 million (71% of) deaths; 16 million of those deaths are premature, arising in people under 70 years of age [3]. The United Nations General Assembly in 2011 made a political declaration on NCDs and followed up in 2014 with an outcome document. This informed the development of a WHO monitoring framework in order to measure progress in preventing and controlling the major NCDs: cardiovascular disease (CVD), cancer, chronic obstructive pulmonary diseases (COPD) and diabetes, and of course their key risk factors. Concerned by the limited progress made by governments in reducing NCDs, the WHO released an NCD progress monitor on 18th September 2017 [4].

NCDs exhibit common risk factors, and many are shared with oral diseases such as periodontitis. Therefore, the identification of NCD risk factors and counselling patients and the public on managing those factors traverses the healthcare professions. Current public health systems still employ "repair models" in many developed countries, whereby practitioners are remunerated by physical interventions rather than preventative strategies. The latter require the implementation of "wellness" approaches to healthcare, where individual people are risk assessed for common NCDs when they are healthy (well) and risk management strategies implemented in order to maintain their health. This requires patient sign-up to behaviour change protocols and follows individualised risk assessment, personalised biofeedback on relevant risks (in a patient-friendly format) to enhance self-efficacy and belief, and finally the provision of behavioural counselling to effect change. In dentistry worldwide, remuneration is still driven via individual treatment codes, few of which provide payment for the collection of a history, the examination, risk assessment and diagnosis, or indeed treatment planning. Hence, the dental profession claim not to have the time to implement twenty-first century prevention, or wellness models of care due to the archaic nature of public funding systems; patients are not empowered to take responsibility for their wellness and nothing changes.

In the cardiovascular field, a recent report was published by the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines, on risk-driven care pathways. It recommended that: "adults who are 40-75 years of age and are being evaluated for cardiovascular disease prevention should undergo 10-year atherosclerotic cardiovascular disease (ASCVD) risk estimation and have a clinician–patient risk discussion prior to pharmacological

therapy, such as antihypertensive, therapy, a statin, or aspirin" [5]. In this model, pharmacological intervention is only recommended for higher risk groups and lower risk groups are provided with preventative advice on risk factor control. Such an approach puts the patient at the centre of care, passes some responsibility to the patient in managing their own health, and reduces the national drug bill.

The voluntary NCD targets set by the WHO for 2025 in order to achieve a 25% reduction in premature mortality from NCDs are:

1. 10% reduction in harmful use of alcohol
2. 10% reduction in physical inactivity
3. 30% reduction in salt/sodium intake
4. 30% reduction in tobacco use
5. 25% reduction in hypertension
6. 0% increase in obesity/diabetes

Taken together, it is clear that risk assessment and risk reduction are critical to human health and disease prevention. Oral health is no different, and indeed the increasing links between oral and general health place the dental team in prime position to join the fight against NCDs and premature death by engaging in discussion with their patients on risk factor control and behaviour change.

In this book, we address the above issues in a series of 15 chapters by experts in different fields from across the world.

Part I of the book includes 2 chapters, the first addressing key epidemiological concepts related to establishing causality for risk factors in complex diseases. This is followed by a chapter summarising the impact of risk-driven care pathways employed over the last 50 years in Sweden upon the incidence of periodontitis and dental caries, the two most prevalent human diseases responsible for more years lost to disability than any other human diseases since 2007 [6]. Part II explores the use of risk assessment in both medical and dental conditions, with Chaps. 3 and 4 reporting upon the experiences of the professional cardiovascular and diabetes care communities in reducing adverse cardiovascular events and improving diabetes outcomes. There are many lessons for the oral health community to learn from the medical profession's experience of attempting to address the WHO targets at a population level. Chapters 5, 6, 7, and 8 focus on the use of risk assessment in oral diseases, specifically periodontal disease, dental caries, non-carious tooth surface loss, and oral cancer, and these are followed by a ninth chapter that focusses on the psychology of risk-driven behaviour change for individuals. Part III looks at risk-driven care pathways in the public sector and also via capitation schemes within the independent healthcare arena, as well as health economic factors, largely in the USA. The final part includes three chapters that attempt to address the development of the dental team in risk-orientated prevention, the implementation of risk assessment tools in general practice, and the medico-legal aspects of such an approach. The authors hope that this text stimulates thought and informs a shift in practice, away from twentieth century repair-based models of healthcare provision to prevention-based models that are fit for the twenty-first century, such as wellness models of risk-driven prevention.

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Causal Inference and Assessment of Risk in the Health Sciences

Ryan T. Demmer and Panos N. Papapanou

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The quest for causality in the health sciences has been ongoing since time immemorial and causal concepts are known to have been discussed by several philosophers in ancient Greece including Aristotle [1]. In the last two hundred years, many presumed causes of disease have been identified (e.g., smoking as a cause of cancer and cardiovascular disease, LDL-cholesterol and high blood pressure as causes of cardiovascular disease, *Mycobacterium tuberculosis* as a cause of tuberculosis, *Plasmodium falciparum* as a cause of malaria and, in the context of oral diseases, *Streptococcus mutans* as a cause of dental caries). Causal inferences of this nature, albeit often imperfect, have contributed to major advances in the health sciences towards reducing morbidity and extending life expectancy.

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The discipline of Epidemiology has been central to causal inquiry for health outcomes in humans since the beginning of the nineteenth century. Indeed, most definitions of epidemiology are explicit about the importance of understanding determinants of disease as well as describing disease patterns. However, despite numerous historical examples of causal discovery, a number of surprising and/or inconsistent findings, particularly in regard to complex chronic disease aetiology, have weakened confidence in the causal models that helped to vanquish infectious diseases during the early twentieth century. This crisis of confidence in causal inquiry has inspired a level of criticism of epidemiological methodology at large. For example, in 1995, Gary Taubes published an article in Science discussing the challenges and limits of modern epidemiology [2]. Since then, a shift has occurred in the way that the health science professions, as well as the public at large, appreciate epidemiologic inferences as they relate to causal inquiry. Most scientists engaged in human-orientated research studies are well-aware of common misinterpretations: for example, 'correlation does not equal causation' is a commonly cited refrain, which while true, is an oversimplification of a complex thought process. Indeed, although non-causal correlations are abundant, this does not mean that every correlation is non-causal in nature. It is also increasingly common to encounter findings from human studies cited as 'epidemiological', with some scientific journals even cataloguing manuscripts under a specific 'epidemiology' section. Typically, in these situations, 'epidemiological' is an adjective used to specify the descriptive arm of epidemiology or to distinguish observational from interventional etiological epidemiological study designs. As such, this language is either incorrect or redundant.

To appreciate this debate, it is imperative to review the definition of 'a cause' and to understand the underlying logic and models used to identify causal relationships in the health sciences. With respect to the first point, one popular definition of a cause reads as follows: 'any factor without which the disease event would not have occurred, at least not when it did, given that all other conditions are fixed' [3]. To test causal hypotheses and identify causes, epidemiologists utilize a conceptual approach referred to as a 'potential outcomes' or—synonymously—a 'counterfactual framework'. A counterfactual framework observes the disease experience in a group of individuals exposed to a hypothesized cause and then inquires what the disease experience in that same group would have been, had they—counter to fact—*not* been exposed to the hypothesized cause during the same time period, with all other factors kept unchanged. The observations from a theoretical experiment of this nature would then yield a causal effect, which is defined as the proportion of exposed individuals who develop disease during a given time period, divided by the proportion of the same exposed individuals that would have developed disease, had they been unexposed during the same observation period. While this is a valuable thought experiment, it is untenable in reality. Therefore, a cornerstone of etiologic epidemiological designs is the use of group comparisons. All etiologic epidemiological study designs, including observational designs and randomized interventions, have been developed precisely to enable valid group comparisons that can approximate the counterfactual ideal and estimate causal effects.

What Is Risk and How Is It Measured

The concept of risk has served as a fundamental tool for inquiry regarding the occurrence of human health and disease. In the context of a counterfactual (or potential outcomes) framework, risk is a proportion that is numerically equivalent to the probability of disease occurrence defined as follows: the number of people who develop a condition divided by the number of at-risk individuals in the source population under study. In more precise epidemiological terms, risk is often referred to as cumulative incidence (CI); a visual representation of CI and the explicit formula is presented in Figs. 1 and 2. It is worth noting that this definition of risk explicitly requires the passage of time such that disease develops during a follow-up period among a subset of initially disease-free individuals. In contrast to incidence, prevalence reflects the probability of current disease. Prevalence is defined as a ratio of the number of existing cases at a point in time (or during a specific time period) over the total number of individuals in the population under study. For example, if the prevalence of diabetes is 14% in a particular country, this tells us that the probability of any randomly selected inhabitant having diabetes is 0.14 (or ~1 in 7 people). In contrast, if the cumulative incidence (or risk) of diabetes in 2018 is 14%, this tells us that during the 2018 calendar year, the probability of developing diabetes among the initially diabetes-free population is ~1 in 7. Another commonly used measure of disease occurrence is odds, which is defined as the probability of having the disease over the probability of being disease-free (i.e., 1-probability of disease). To state it another way, the cumulative

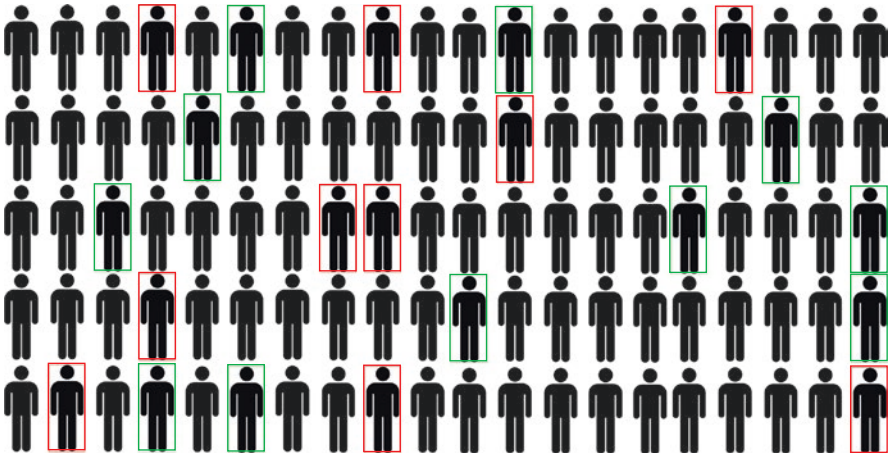
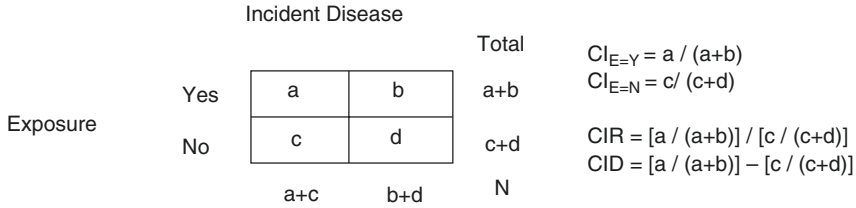


Fig. 1 Measures of disease frequency. n = 100 individuals enrolled into a longitudinal cohort study on January 1st, 2019 and followed for 20 years. Red borders signify disease present at the beginning of the study (baseline), n = 10. Green borders signify disease that developed during follow-up (incident disease), n = 11. Prevalence on January 1st, 2019 = 10/100 = 0.10 or 10%. Cumulative Incidence during the 20 year study period = 11 incident cases/(100 baseline − 10 prevalent cases) = 0.12 or 12%



Epidemiological Measures of Impact

Attributable Risk	$CI_{exposed} - CI_{unexposed}$
Population Attributable Risk	$P_{exposed}(CI_{exposed} - CI_{unexposed})$ or $CI_{all} - CI_{unexposed}$
Attributable Fraction	(CIR –1) / CIR or $(CI_{exposed} - CI_{unexposed}) / CI_{exposed}$
Population Attributable Fraction	$P_{exposed}(CIR -1) / [1 + P_{exposed}(CIR-1)]$ or $(CI - CI_{unexposed}) / CI$

Fig. 2 A 2 × 2 table summarizing the joint distribution of an exposure (i.e., risk factor) and incident disease. Cell a = the number of exposed individuals with disease; b = the number of exposed individuals without disease; c = the number of unexposed individuals with disease; d = the number of unexposed individuals without disease. $CI_{E=Y}$ = cumulative incidence of disease among the exposed. $CI_{E=N}$ = cumulative incidence of disease among the unexposed. CIR = cumulative incidence ratio. CID = cumulative incidence difference

incidence odds (CIO) of disease is simply = [CI/(1−CI)], and prevalence odds would be defined as [prevalence/(1−prevalence)]. Therefore, odds can be calculated and used in the context of both prevalence or incidence. Finally, the concept of incidence rate (or incidence density) is also of central importance to epidemiological inquiry and is closely related to the concept of risk. The incidence rate simply incorporates time explicitly into the denominator as follows: the number of people who develop a condition (incidence) divided by the person time contributed by initially disease-free individuals during the study period. Person time is calculated for each individual as the amount of time that passes between entry into the study and either: (1) the development of disease (or in many study settings disease diagnosis, which often differs from the precise time of disease development); (2) the end of the observation period; or (3) death or loss-to-follow-up. These concepts are demonstrated in Fig. 3.

Risk and Measures of Association

While measures of disease frequency, such as risk (i.e., cumulative incidence), are of value for a number of important reasons, risk is frequently used to assess the evidence for causal associations. This is typically done by comparing risk of disease between two different groups of individuals defined by variation in an ‘exposure’ or hypothesized risk factor. For example, consider the 2 × 2 tables in Figs. 2 and 4 which demonstrate different measures of association derived from risks (or rates) of disease among individuals exposed vs. those unexposed. Figures 2 and 4 define (1)

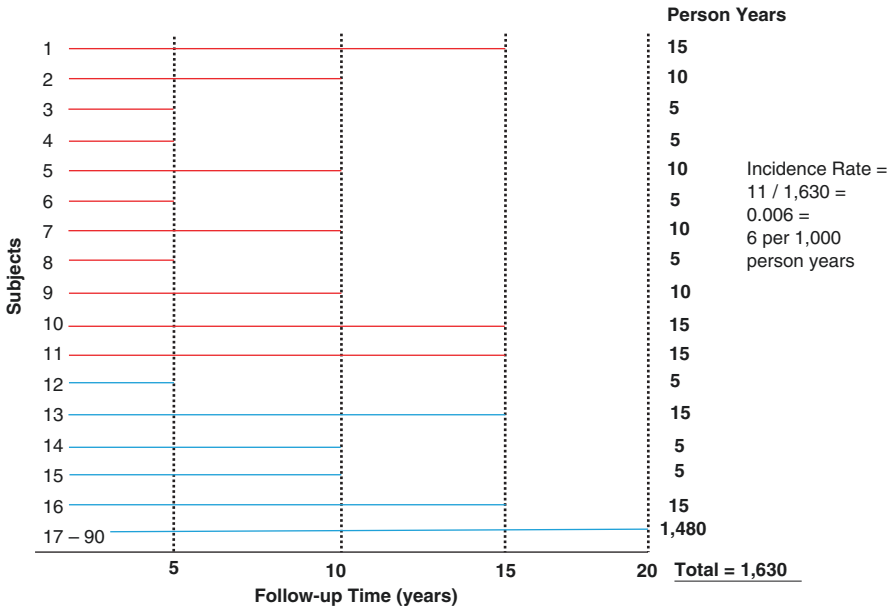


Fig. 3 Visualization of how person time accrues in longitudinal study designs. Red lines signify individuals that develop disease over the observation period, blue lines individuals who remain healthy

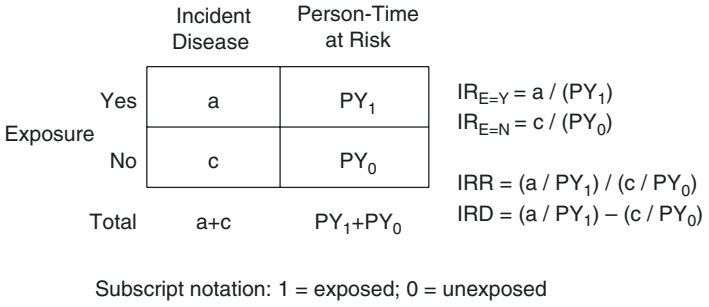


Fig. 4 A 2 × 2 table summarizing the incident disease and person time by exposure (i.e., risk factor) status. Cell a = the number of exposed individuals with disease; PY_1 = the total person time contributed by exposed individuals during the study; c = the number of unexposed individuals with disease; PY_0 = the total person time contributed by unexposed individuals during the study. $IR_{E=Y}$ = Incidence rate among the exposed. $IR_{E=N}$ = Incidence rate among the unexposed. *IRR* incidence rate ratio, *IRD* incidence rate difference

cumulative incidence ratio; (2) cumulative incidence difference; (3) incidence rate; and (4) incidence rate difference.

Based on the aforementioned measures of association, additional measures of impact used in epidemiology can be derived including: (1) attributable risk (AR, synonymous with the cumulative incidence difference—see Fig. 2); (2) population

attributable risk (PAR); (3) attributable fraction (AF); (4) population attributable fraction (PAF). These measures summarize the number of cases of disease that are the result of (i.e., attributable to) the exposure among different populations; the respective populations of interest being the exposed for AR, the total population for PAR, the exposed with disease for AF, and the diseased for PAF, respectively. Formulas for these measures can be found in Fig. 2. Note that the terminology for these measures varies considerably in the literature and one should always take careful note of the underlying formula used when interpreting the meaning of these measures.

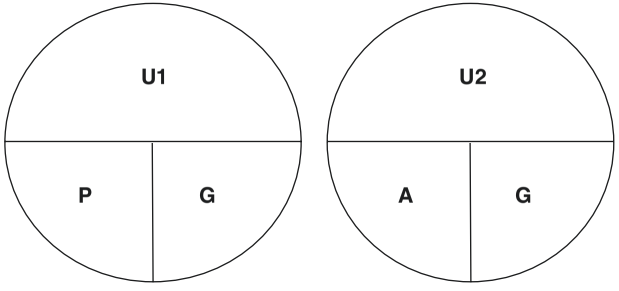
Causal Inference and Causal Models

It is frequently explicitly stated (or intimated) in the literature that observational designs, particularly cross-sectional and case-control designs, cannot be used to infer causality, but can only identify putative causal exposures that require testing in subsequent randomized controlled trials (RCTs) to provide definitive causal estimates. In fact, while experimental designs have the potential to provide less biased and/or confounded causal estimates, observational designs are both capable of and frequently used to inform causal relationships. Some examples follow to demonstrate this point. When exposure status clearly precedes the disease outcome and nature randomizes the exposure, observational designs can be quite powerful. For example, Mendelian randomization embedded in longitudinal observational cohort studies leverages the randomness of the meiotic process to inform whether hypothesized exposures cause disease. Even in a cross-sectional study design, a Mendelian randomization approach could potentially provide strong causal evidence since the genotype clearly precedes the disease outcome (i.e., clearly fulfils the temporality requirement) and the mutation in question was assigned by nature and thus cannot be confounded by events occurring during the life course, such as socio-economic status, access to health care, or health behaviours. Furthermore, in situations, where randomization is unethical, observational designs are generally the only feasible option. The establishment of smoking as a cause of lung cancer and cardiovascular disease using observational designs demonstrates this point. Importantly, a poorly conducted RCT—for example, one in which randomization is not achieved, blinding is not utilized, and/or follow-up rates are low—is prone to all common types of bias and confounding that threaten the validity of observational studies and is, therefore, less likely to enable valid causal inference than a well-conducted observational cohort study. In addition, intervention against a true causative factor in an RCT may still fail to result in lower levels of disease for a variety of reasons, including an immutable or unsuccessfully controlled causative factor, inappropriate timing or intensity of the intervention, and lack of patient compliance. Therefore, the frequently advocated view in recent years that observational designs are of lesser value while a positive RCT outcome is indispensable in the identification of a true causal exposure is misleading and threatens to disregard important scientific progress towards reducing morbidity and mortality in the population.

The use of group comparisons to approximate the ideal counterfactual knowledge under investigation is of critical importance but still fails to provide an explicit causal model linking exposures to disease outcomes. For epidemiological designs to yield meaningful causal inferences, coherent causal models of disease aetiology are necessary, such as the ones employed in studies of infectious disease aetiology (arguably the models that established the discipline of epidemiology). The studies of John Snow on cholera [4] and James Lind on scurvy [1] are classic examples of early epidemiological inquiry. In the context of infectious diseases, causes were generally identified when a microorganism (i.e., the causal factor or 'risk factor') was, or appeared to be, both the necessary and sufficient condition for the disease to occur. In other words, the factor had always to be present in every case of the disease, and the factor alone could produce disease. Accordingly, Koch's postulates were originally developed to provide a framework for establishing a particular microorganism (Mycobacterium) as the cause of tuberculosis. Interestingly, while Koch's postulates were initially quite helpful in elucidating the causal organism of TB, it was realized in retrospect that they were generally less useful in the study of several other infectious diseases. For example, the first postulate posits that a microorganism must be *present in all cases* of disease and *absent in healthy individuals*, a condition which is now known to be false for numerous infectious diseases, including TB. The second postulate states that the microorganism must be isolated from a diseased host and grown in culture, which obviously does not apply to uncultivable microbes or to viruses. The third postulate requires the emergence of disease when a healthy host is inoculated with the causative organism; the existence of asymptomatic carriers for infectious disease (e.g., Typhoid Mary) negates the veracity of this requirement.

As industrialized societies acquired a better understanding of infectious diseases and life expectancy increased during the 1800s and 1900s, the leading causes of death shifted to conditions that are chronic and multifactorial. During this epidemiologic transition, it became apparent that classical causal models were inadequate. Smoking as a cause of lung cancer and cardiovascular disease was a specific and early example of the insufficiency of causal models requiring necessary and sufficient causes of disease. More broadly, causal models that require necessary and sufficient causes are of limited value for all current leading causes of death in the world (e.g., cardiovascular disease, cancer, diabetes, respiratory diseases).

In response to these limitations, a now classic model for causal inference in the context of chronic diseases, that can also be applied to infectious diseases, was proposed by Rothman using a '*sufficient cause*' model of causation [3]. A sufficient cause (SC) is defined as 'a complete causal mechanism that inevitably produces disease'. The SC model visually represents causal hypotheses using causal 'pies' as shown in Figs. 5 and 6. Causal pies are represented as full circles (i.e., sufficient causes) comprised of individual slices termed '*component causes*', each of which is required to assemble in full a sufficient cause and, thus, for disease to occur. According to the main premise of the conceptual model, once all component causes of a sufficient causal pie are in place, disease will inevitably occur. The example in Fig. 5 provides a hypothetical sufficient component causal model for



Sufficient Cause 1 Sufficient Cause 2

Prevalence of U1 and U2 is 100% in population 1 and 2.

Table A. Linking risk factor combinations to periodontitis risk according to sufficient causes 1 and 2

U1	U2	A	P	G	SC	Risk	Population 1	Population 2
1	1	1	1	1	1,2	1	500	500
1	1	1	1	0	None	0	500	500
1	1	1	0	1	2	1	50	350
1	1	1	0	0	None	0	50	350
1	1	0	1	1	1	1	400	100
1	1	0	1	0	None	0	400	100
1	1	0	0	1	None	0	50	50
1	1	0	0	0	None	0	50	50

Table B.
Joint distribution of *P. gingivalis* and periodontitis in population 1

	Periodontitis	No Periodontitis	Total
P. gingivalis present	900	900	1800
P.gingivalis absent	50	150	200

Table C.
Joint distribution of *P. gingivalis* and periodontitis in population 2

	Periodontitis	No Periodontitis	Total
P. gingivalis present	600	600	1200
P. gingivalis absent	350	450	800

Estimates of the causal effect of *P. gingivalis* on periodontitis in two separate populations

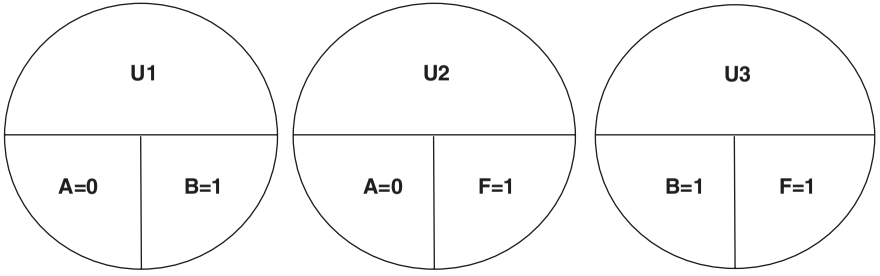
CIR = (900/1800) / (50/200) = 2.0

CID = (900/1800) – (50/200) = 0.25

CIR = (600/1200) / (350/800) = 1.14

CID = (600/1200) – (350/800) = 0.06

Fig. 5 Translating the sufficient component cause model of disease causation into causal estimates of risk in two separate populations, each comprising 2000 individuals. In the upper left, two hypothetical sufficient causes of human periodontitis are described. Sufficient cause (SC) 1 is comprised of the following three component causes: U1 (unknown causal factors assumed to be ubiquitous), P (*Porphyromonas gingivalis* presence), and G (a set of genetic polymorphisms). Sufficient cause (SC) 2 is comprised of the following three component causes: U2 (unknown causal factors assumed to be ubiquitous), A (*Aggregatibacter actinomycetemcomitans* presence), and G (the same set of genetic polymorphisms as in SC 1). The component causes U1, U2, A, P, and G are synonymous with the term 'risk factor' in modern epidemiology. Table A provides all possible risk factor combinations, with 1 = present and 0 = absent, and assuming that U1 and U2 are ubiquitous in both populations. The SC column of Table A indicates which sufficient cause (1 and/or 2 or neither) is completed for each possible risk factor combination. The risk column equals 1 when at least one sufficient cause is completed and 0 if no SC is completed. The population 1 and 2 columns reflect the number of individuals in each population with a given risk factor 1 = present and 0 = absent. Tables B and C reflect the joint distribution of *P. gingivalis* and periodontitis in populations 1 and 2 as derived from Table A. Cumulative incidence ratio (CIR) is defined as the ratio of the proportion of individuals with a certain risk factor that have completed a sufficient cause (i.e., have developed the disease) over the proportion of individuals without the risk factor that have completed a sufficient cause. Cumulative incidence difference (CIDs) is defined as the difference between the above two proportions. Note the difference in the causal effect of *P. gingivalis* on periodontitis in the two populations



Sufficient Cause 1 Sufficient Cause 2 Sufficient Cause 3

Table A. Linking risk factor combinations to diabetes risk according to sufficient causes 1 and 2

A	B	F	SC	Risk	Population 1	Population 2
1	1	1	3	1	900	100
1	1	0	None	0	900	100
1	0	1	None	0	100	900
1	0	0	None	0	100	900
0	1	1	1,2,3	1	100	900
0	1	0	1	1	100	900
0	0	1	2	1	900	100
0	0	0	None	0	900	100

Table B. Joint distribution of *F. nucleatum* and diabetes in population 1

Population 1	Diabetes Present	Diabetes Absent	Total
F. nucleatum present	1900	100	2000
F. Nucleatum absent	100	1900	2000

CIR = (1900/2000) / (100/2000) = 19

CID = (1900/2000) – (100/2000) = 0.9

Table C. Joint distribution of *F. nucleatum* and diabetes in in population 2

Population 2	Diabetes Present	Diabetes Absent	Total
F. nucleatum present	1100	900	2000
F. nucleatum absent	900	1000	2000

CIR = (1100/2000) / (900/2000) = 1.22

CID = (1100/2000) / (900/2000) = 0.1

Fig. 6 Translating the sufficient component cause model of disease causation into causal estimates of risk in two separate populations. In the upper left, three hypothetical sufficient causes of diabetes are described. Sufficient cause (SC) 1 is comprised of the following three component causes: U1 (unknown causal factors assumed to be ubiquitous), A (high dietary fibre consumption), and B (a genetic polymorphism). SC 2 is comprised of the following three component causes: U2 (unknown causal factors assumed to be ubiquitous), A (high dietary fibre consumption), and F (*Fusobacterium nucleatum* colonization in the pancreas). SC 3 is comprised of the following three component causes: U3 (unknown causal factors assumed to be ubiquitous), B (a genetic polymorphism), and F (*Fusobacterium nucleatum* colonization in the pancreas). Table A provides all possible risk factor combinations (assuming U1, U2, and U3 are present in all participants in both populations) in columns A–F (1 = present and 0 = absent). The SC column of Table A indicates which sufficient causes (1, 2, and/or 3 or none) are completed for each possible risk factor combination. The risk column = 1 when at least one sufficient cause is completed and 0 if no SC is completed. The population 1 and 2 columns reflect the number of individuals in each population with a given risk factor distribution. Tables B and C reflect the joint distribution of *F. nucleatum* and diabetes in populations 1 and 2 as derived from Table A. Assuming this causal model is true, the cumulative incidence ratios (CIRs) and cumulative incidence differences (CIDs) have a causal interpretation

the development of human periodontitis in which there are two sufficient causes. In this example, sufficient cause 1 involves the presence of microbial dysbiosis triggered by a particular microorganism (*Porphyromonas gingivalis*) (P), a set of genetic polymorphisms (G) and the additional presence of a number of unknown factors (U1). Sufficient cause 2 is comprised of a different dysbiotic microbial profile, namely dysbiosis triggered by *Aggregatibacter actinomycetemcomitans* (A), the same set of genetic polymorphisms as in SC 1 (G), and another set of unknown factors (U2) which are distinct from U1. In the example visualized in Fig. 5 for periodontitis, G represents a *necessary cause*—i.e., G is a component cause that is present in all sufficient causes of disease and is therefore necessary to be present for periodontitis to occur. However, while G is necessary for the development of periodontitis, G alone is not sufficient to produce periodontitis without the presence of G's causal complements (i.e., P + U1 or A + U2). In contrast, P, A, U1, and U2 represent component causes that are neither sufficient nor necessary to cause periodontitis. If any individual in a hypothetical population completes either SC 1 or SC 2, they will develop periodontitis. A second example (Fig. 6) provides a hypothetical set of sufficient causes positing translocation of *Fusobacterium nucleatum* (F) from the oral cavity to the pancreas as a cause of type 2 diabetes mellitus development. In this example, there are three distinct sufficient causes comprised of six different component causes. This example demonstrates a scenario in which there are no necessary causes.

Two points should be emphasized from the SC model approach presented in Figs. 1 and 2. First, in modern epidemiology, the term 'component cause' is synonymous with the more commonly used term, 'risk factor'. In other words, risk factors are causes of disease that generally work in tandem with other risk factors (i.e., component causes) to produce disease. Note that the term 'risk predictor' is generally used to refer to a variable that predicts risk but for which causality is not assumed (e.g., grey hair is a risk predictor of mortality but not a risk factor). Second, and building on the first point, a somewhat obvious conclusion from the SC model is that there are multiple pathways that lead to the development of a given disease and each pathway involves multiple component causes that work together synergistically. This synergy precisely represents the concept of interaction (or effect measure modification) in statistics and epidemiology. Although we will not discuss interaction in detail here, in the specific context of SC models, when causal factors interact, any one component cause can only cause disease in the presence (or possibly in the absence) of the other component cause(s) in the same SC.

A careful review of the examples in Figs. 5 and 6 demonstrates another important concept that helps us understand why an exposure can cause disease even if the strength of association is weak or varies greatly across different studies (for example, as often observed in a meta-analysis). In the examples presented in Figs. 5 and 6, it is apparent that the cumulative incidence ratio (CIR), i.e., the ratio of the proportion of individuals with a certain risk factor that have completed a sufficient cause (i.e., have developed the disease) over the proportion of individuals without the risk factor that have completed a sufficient cause, and the cumulative incidence

difference (CID), i.e., the difference between the above two proportions, vary across populations in which the distribution of component causes are not equal. This raises a profoundly important point about causal inquiry that is often not appreciated in the health sciences: specifically, the strength of association (using absolute measures) is dependent upon the prevalence of causal complements in the population. The causal complement of a risk factor is defined as the set of all other component causes in all sufficient causes in which a risk factor participates. In the case of Fig. 6, the causal complements of F are A = 0 and U2, or B = 1 and U3. As the prevalence of these causal complements increases, the strength of association between F and diabetes becomes stronger.

So, what are the implications of our causal models for epidemiological research and the ability to identify causes of disease in humans? When we explore risk factors in isolation using reductionist approaches, there can be great variation in the strength of association between a causal factor and a disease outcome across populations. In populations with a low prevalence of causal complements, the strength of association for the main component cause (i.e., risk factor) under investigation will be weak when compared to that in a population with a higher prevalence of causal complements.

In contrast, in disease models where there are multiple sufficient causes in the population, and there is a high prevalence of component causes in sufficient causes where the risk factor of interest does not participate, the observed effect for this particular risk factor will be relatively weak or undetectable. In Fig. 6, note that an increase in the prevalence of individuals with both A = 0 and B = 1 would lead to an increase in the prevalence of individuals susceptible to SC1, yielding weaker associations between F and diabetes because F cannot cause disease in individuals with SC1 already complete (i.e., in individuals that are already 'doomed'). This concept, known as causal redundancy, has been elegantly discussed in a review by Gatto and Campbell [5].

Interestingly, high variability in measures of association across studies conducted in different populations is often taken to suggest lack of evidence for causality. For example, the often-referenced Bradford Hill guidelines for assessing causal evidence [6] include the criteria of 'consistency' and 'strength of association', which imply that inconsistent results across study populations and/or weak associations argue against a causal relationship. While consistently strong associations do increase confidence in a causal hypothesis, lack thereof does not necessarily imply no causality. The examples above clearly demonstrate that under specific causal hypotheses, not dissimilar to the underlying hypotheses of modern chronic disease aetiology, causal effects are *expected* to be inconsistent and at times weak, across different populations, as long as the prevalence of other risk factors varies.

Modern epidemiology is often faced with complex sufficient causal hypotheses, which typically lack a necessary cause. For example, in the cardiovascular disease literature, there has been a long-standing argument as to the usefulness of novel risk factor research [7] because so many risk factors have been identified and nearly all cases of coronary disease have one or several traditional risk factors present [8].

However, while it is evident that almost all individuals with CHD have at least one major risk factor, two facts remain: (1) no one risk factor is always present and (2) a large proportion of CHD-free individuals also tend to have multiple risk factors. Therefore, to date, neither 'necessary' nor 'sufficient' causes of CHD have been identified. Nevertheless, since the classical risk factors are pieces of the causal pie, interventions against one or several of these factors can prevent or delay completion of a causal pie sufficient for disease development in many populations.

There are real-world implications for the aforementioned concepts. It has long been observed that many traditional cardiovascular disease risk factors tend to have weaker associations with clinical outcomes in elderly populations. For example, although the benefits of statin therapy to prevent myocardial infarction have been clearly shown, their use in elderly populations remains debatable. A more recent example involves aspirin use for the primary prevention of cardiovascular events among the elderly, in whom it offers no benefit and may possibly even induce harm, despite long-standing benefits of aspirin use for secondary prevention in younger populations [9].

Using the aforementioned concepts, we can couple a causal model (causal pies) with the counterfactual concept operationalized via real-world study designs and data collection to yield group comparisons that inform causal hypotheses.

Populations Vs. Individuals

The aforementioned examples demonstrating how group comparisons utilized to infer causality inform the long-standing paradox in the health sciences in which research methodologies for identifying causality rely on 'average risk' across the groups being compared (e.g., treatment vs. placebo). From a big picture, public health perspective this concept works well when making policy recommendations regarding prevention and treatment of disease. If a particular intervention reduces disease 'on average', population health improves. However, the paradox arises in clinical situations when treatments, or prevention recommendations, are delivered directly from clinicians to individual patients. This setting is more challenging, given the fact that causality is never certain at the individual level and therefore, one can never know if a particular intervention was successful for a given individual. To paraphrase Jude Pearl [10], risk—along with other measures—can often give profoundly accurate predictions about disease occurrence in populations yet, paradoxically, it has very little accuracy at the individual level. Or stated another way, in most settings, causality can only be determined at the population-level. Reconciling the utility of risk-based measures in populations vs. individuals has been an age-old challenge. While clinical judgement and personalized health care have an important place, the lion's share of treatment decisions with proven efficacy rely on evidence derived from epidemiological designs utilizing group comparisons. The implication of this fact is that treatments will work on average. Underscoring this point is

the often-cited measure of impact in the health sciences, Number Needed to Treat (NNT). The NNT is defined as the inverse of cumulative incidence differenced (Fig. 2) and represents the number of individuals that would need to receive an intervention to prevent one case of disease.

Concluding Remarks: Risk and Causality in the Precision Era

We have entered the era of 'precision-oriented' science, including precision medicine and precision public health [11]. Parenthetically, this popular term seems rather misguided, since the concept of 'precision' relates to measures of reliability (i.e., reproducibility) rather than validity (lack of bias). The term 'personalized medicine' is arguably much better in reflecting a more customized, yet valid, approach to medical care. Irrespective, use of either term may be interpreted to suggest that the pushback against epidemiology is intensifying in favour of other causal models leveraging precision (or more narrowly stated, genomic sciences). However, this is again a misconception. In fact, the 'precision agenda' is merely a refinement of the tools that we use to build sufficient cause models and subsequent study designs. Specifically, the precision era is unlikely to fundamentally change our methods of causal inquiry, although it does offer the potential to clarify causal hypotheses and their underlying sufficient cause models by identifying new, mostly genetic, pieces of the causal pie. In doing so, precision approaches can help to refine inclusion/exclusion criteria for research studies that test hypotheses concerning specific risk factors and/or interventions targeting those risk factors. Refining inclusion/exclusion criteria will enhance future studies by ensuring that 'at-risk' individuals are indeed included in the study samples. Formally speaking, 'at-risk' in the context of an intervention design would be individuals who are about to complete or have completed a sufficient cause that includes the particular risk factor targeted by the intervention. In that setting, the removal of the risk factor would prevent, or in the case of a reversible effect, cure the disease under investigation.

Once new risk factors are identified in more precisely defined populations, it stands to reason that clinical and public health practice will benefit by knowing which treatments to deliver and to whom. If this approach sounds familiar, that is because it is exactly the way causes of disease have been identified and that knowledge translated into evidence-based health policies and treatment guidelines. As such, the current hype surrounding the precision agenda is a refined version of a causal model that has been in place for decades.

In conclusion, causal inference in most contexts relevant to human disease requires group comparison under a counterfactual framework. Because most human disease entities of relevance to public health are complex and likely involve multiple sufficient causes of disease, research designs in humans should be based on well-developed sufficient component cause models to ensure that at-risk individuals are studied in populations where causal redundancy can be minimized.

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Impact of Risk-Based Prevention on Public Oral Health

Examples from Sweden with Long-Term Follow-Up

Ola Norderyd and Åsa Wahlin

Contents

Introduction

Oral diseases have a major impact on public health worldwide. A healthy mouth is a central part of living. It enables us to enjoy life when eating, drinking, and socializing. Both untreated caries and periodontal disease are among the most prevalent chronic diseases of humans [1], causing suffering and even death. In addition, oral diseases generate high costs for both individuals and society [2].

Risk assessment in health sciences is the analysis of the probability that a disease will occur in the future. To formulate risk-based prevention programs,

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knowledge of disease etiology as well as prevalence in different populations is of utmost importance.

In addition to preventive programs, aimed at the whole population, it is necessary to provide individual programs based on assessed risk for individuals. This facilitates the reduction of the number of individuals developing oral disease and also ensures that a high quality of dental care is provided.

To assess the need for preventive and treatment interventions, and also to evaluate the effects of preventive measures in a population, epidemiological studies are performed. Epidemiology can be defined as "the study of the distribution and determinants of health-related states or events in specified populations, and the application of this study to the control of health problems" [3]. Another important aspect is to explore trends and future demands for public health promotion.

The present chapter primarily focuses on the development of dental health in Sweden from the 1970s to the present day. A series of epidemiological studies in Jönköping showed the improvement of the dental health in the population. During this time period, many measures aimed to improve the dental health in the population and preventive programs were implemented for children and adolescents. The Karlstad study evaluated the effect of a preventive program, on individual level, for caries and periodontal disease by controlling the dental biofilm [4].

Impact of Risk-Based Prevention During Four Decades in Jönköping, Sweden

In the beginning of the 1970s, extensive caries, periodontitis, tooth loss, and widespread edentulism were evident in the Jönköping population as well as in the rest of Sweden. In 1974, a New Dental Act was introduced that reduced costs for dental care for the individual but also financed population based preventive care for all children and adults [5]. Since 1973, epidemiological cross-sectional studies have been repeated every decade in the city of Jönköping. These studies were initiated in order to assess oral health changes and trends in the population and to evaluate the impact of the implemented systematic preventive measures over time. A randomized, stratified, representative sample of individuals aged 3–80 years was selected at each time point (no 80 year olds were included in 1973).

All individuals received an extensive oral examination including clinical and radiographic measures. They also answered a questionnaire about dental care habits, knowledge of oral health, attitudes, and diets. The questionnaire has been slightly modernized over the years and more questions and instruments have been included, e.g., validated quality of life instruments.

During the 1970s, preventive dental care programs for all children and adolescents were introduced in Jönköping; child health care services and primary schools were also involved in the programs. Today, all individuals 60 years and younger have been exposed to dental preventive programs during childhood and adolescence. These programs have been evaluated and adjusted in tandem with the changes in oral health. The oral hygiene programs have included education in oral health (diet, hygiene measures, and tobacco use), fluoride application (toothpaste, rinsing, and fluoride varnish), and fissure sealants for all permanent molars.

In addition, more specific risk factors for developing caries among children and adolescents have been identified in the Jönköping population [6]. A cohort of subjects 1 year of age was followed longitudinally up to age 20. Maternal dental anxiety together with tooth brushing with fluoride toothpaste less than twice a day and consumption of caries risk products >3 times per day were associated with caries experience at age 20.

Since the introduction of the preventive programs in the population, the use of fluoride toothpaste has gradually increased. In the last survey in 2013, the majority (85%) of individuals in all age groups brushed their teeth with fluoride toothpaste at least two times a day [7]. Among children and adolescents, individuals who frequently consume soft drinks have been reduced by half over the last decade.

The oral health of the population has improved dramatically. As evidenced by the prevalence of edentulism and increased number of existing teeth (Fig. 1). Edentulous individuals have decreased to approximately 1% and dentate subjects had almost complete dentitions up to 60 years of age in the last survey [8]. One of the World Health Organization's (WHO) goals has been achieved even among the 80 year olds, with the majority of the subjects now having more than 20 remaining teeth [9].

There has been a dramatic increase in completely caries free individuals in the Jönköping population since 1973 (Fig. 2). Additionally, the number of caries-affected

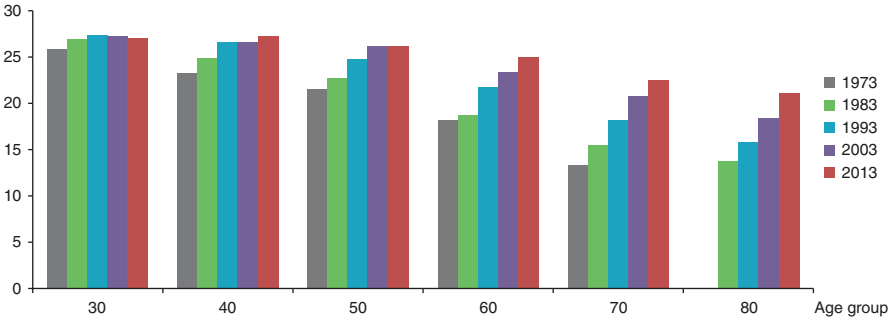

Fig. 1 Mean number of teeth (third molars and edentulous individuals excluded) (Norderyd et al. [7])

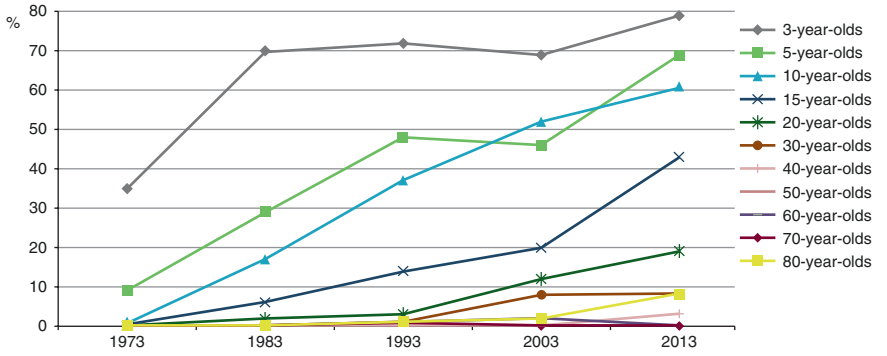

Fig. 2 Frequency (%) of completely caries free individuals in age groups in 1973, 1983, 1993, 2003, 2013 (Norderyd et al. [7])

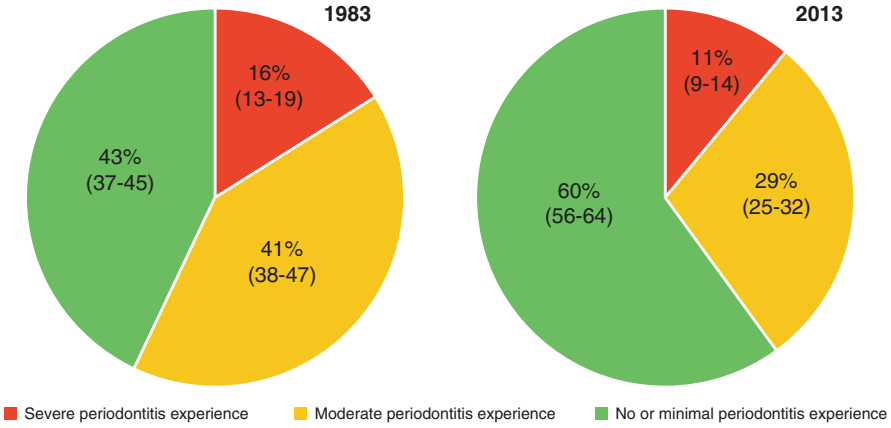

Fig. 3 Distribution of individuals 20–80 years according to periodontitis experience in Jönköping 1983–2013 (Wahlin et al. [10])

surfaces has decreased in all age groups. The only exception was among the 70 and 80 year olds, which may be explained in part by these age groups retaining more teeth over the years. With regard to periodontal health, there has been a decrease in individuals with moderate periodontitis and a gain in periodontally healthy subjects over the 40-year period (Fig. 3) [10]. Over time, there is a non-significant trend showing a decrease in individuals with severe periodontitis and at the same time there was an increase in the number of teeth in the group of subjects with severe periodontitis.

Since the 1970s, the profile of the dental health care has changed in both public and private sectors, with an increase in the proportion of dental hygienists compared to dentists. In all, the number of dental hygienists has increased threefold during the last two decades in both public and private clinics. The population in Jönköping has improved their dental awareness, as can be seen in more regular self-performed oral hygiene of better quality [7]. The majority of the population attend dental care regularly. A decrease in the number of smokers and number of smoked cigarettes can also be seen over time which may have impacted on the prevalence of periodontitis in addition to improved oral hygiene. The continuous improvement in oral health and reduced need for restorative treatment have led to better health, lower costs, and improved life quality in all age groups as well as for the society at large [8].

Impact of Risk-Based Prevention in Karlstad, Sweden, During 30 Years

A well-known, widely reported study from Sweden that commenced in the beginning of the 1970s, evaluated the long-term effect of a systematic professional preventive program [4]. This prospective study covered 30 years at a private practice in Karlstad, Sweden. The test group was maintained and followed for 30 years, while the control group was discontinued after 6 years for ethical reasons. At the start of

the study all participants received detailed information regarding their oral health status and instructions in oral hygiene routines and as a result their awareness of the importance of oral health increased. During the first 2 years, the subjects attended maintenance visits every second month and then every third month for the subsequent 4 years. After 6 years, patients were recalled based on their risk profile: 65% 1× per annum, 30% 2× per annum, and 5% (deemed high risk) 3–6× per annum. Each maintenance visit comprised follow-up of oral hygiene and individually designed oral hygiene instructions and training. The visits also included professional cleaning of the dentition. After the baseline examination follow-up examinations were conducted after 3, 6, 15, and 30 years. Plaque, carious lesions, gingivitis, probing pocket depth, clinical attachment loss (CAL), and community periodontal index of treatment need (CPITN) were registered. Before the start of the study, carious lesions and periodontal pathology were treated and ill-fitting restorations were corrected/replaced. The control group only attended annual recall examinations but after 6 years they were offered maintenance care for ethical reasons.

At the beginning of the study 60% of the tooth surfaces were covered with plaque in contrast to 15% at the final examination after 30 years. The participants lost very few teeth during the 30 years (Table 1). Root fracture was the main cause for losing (non-vital) teeth followed by endodontic reasons. Individuals developed only 1–2 new carious lesions during the study, fewer in the younger group compared to the older group and 80% of the new carious lesions were secondary lesions. No clinical attachment loss (CAL) was detected, except for buccal surfaces among the younger participants; on the contrary, a gain in attachment was detected in all age groups. In comparison, the control group had developed more than 10 carious lesions and had a mean loss of approximately 1 mm clinical attachment at the 6-year examination [11].

Influenced by the Karlstad study, three oral health programs for young adult individuals were compared over 3 years in Jönköping (Table 2) [12]. A randomized,

Table 1 Mean number of teeth present and calculated annual loss of teeth

Group (age)	1972	2002	Difference	Annual rate of tooth loss
20–35	26.7	26.3	0.4	0.01
36–50	25.8	25.1	0.7	0.02
51–65	20.1	18.3	1.8	0.06

Axelsson et al. [4]

Table 2 Effect of three dental health programs on young adult individuals

Group 1—control group No organized prophylactic measures. Recall at 12-month intervals
Group 2 (2_0 and 2_1 2_2)—the "Karlstad Model" Repeated information and oral hygiene instruction at 2-month intervals. Additional professional tooth cleaning (2_1 2_2)
Group 3—individual education Individual basic program, three visits at 2-week intervals the first year. 1-year, 2-year, and 3-year follow-up
Group 4—group education Group based information, three visits at 2-week intervals the first year

Hugoson et al. [12]

blinded, parallel group, controlled evaluation of oral hygiene behavior on plaque and gingivitis was performed. Subjects in all three oral health programs showed significant improvements in gingival health in comparison with individuals in the control group. An additional significant factor for better gingival health was knowledge of the two major oral diseases.

Concluding Remarks

Since the beginning of the 1970s there have been several systematic oral health care preventive programs in Sweden. A few of them have long-term evaluations. The improvement in oral health in the population in Jönköping during more than 40 years can most likely be explained by these preventive measures. Indirectly, this has also been shown in the Karlstad study. They demonstrated that a systematic preventive program with regular recalls 3–4 times a year can prevent caries and periodontitis, and secondly, maintain most of the dentition. It is of utmost importance to continuously operate and develop these programs in the long term to further improve the oral health at a population level. There are a lot of advantages with this approach, both for the individual but also for society, regarding better oral and general health, social well-being, and lower costs. As an effect of this, the improved oral health in children and adolescents has made more resources available for treatment of adults in the Public Dental Service.

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Part II

Risk Assessment in General and Oral Health

Risk Assessment in CVD

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Contents

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Burden of Cardiovascular Disease

Atherosclerotic Cardiovascular Diseases

While age-standardized mortality from cardiovascular disease (CVD) has decreased in many regions of the world, the absolute number of deaths continues to increase [1]. Thus, CVD still represents the most common cause of morbidity and mortality worldwide [2, 3]. The global number of deaths from CVD has increased over the past decade by 12.5%. CVD now accounts for approximately one-third of all deaths and 45% of all non-communicable disease deaths, more than twice that caused by cancer [4]. These changes are mainly driven by population growth and ageing populations with wide inequalities between countries [2].

Over 95% of all CVD deaths are attributable to six conditions: ischemic heart disease, stroke, hypertensive heart disease (resulting in heart failure), cardiomyopathy, rheumatic heart disease, and atrial fibrillation [1, 5]. The two leading contributors to the global CVD burden are ischemic heart disease and stroke. For ischemic heart disease, both the prevalence and mortality increase strongly with age [1]. Likewise, the prevalence of stroke increases with age as well as stroke-related mortality [3].

Accordingly, CVD is very common in the general population, affecting the majority of adults aged 60 years or more. CVD is commonly used to refer to the following atherosclerotic cardiovascular diseases: (1) coronary heart disease (manifested by fatal or nonfatal myocardial infarction), angina pectoris, and/or heart failure, (2) cerebrovascular disease manifested by fatal or nonfatal stroke and transient ischemic attack, (3) peripheral artery disease manifested by intermittent claudication and critical limb ischemia, (4) aortic atherosclerosis and thoracic or abdominal aortic aneurysm.

Other Diseases with Increased Risk of CVD

Apart from atherosclerosis, several other diseases in which infections or non-infectious inflammatory processes play a central role are associated with an increased risk of cardiovascular events such as influenza, psoriasis, rheumatoid arthritis, periodontitis, lupus erythematosus, vascular disease after radiation exposure or vascular disease after transplantation [6]. The presence of these diseases is mostly not reflected in risk estimation systems and therefore needs independent consideration. Moreover, the optimal prevention strategy to reduce the risk for future cardiovascular events in these diseases is not established and randomized clinical studies evaluating prognosis are not available [6].

Key Risk Factors for CVD

Numerous factors are causally related to CVD, including traditional individual level risk factors (e.g., tobacco use, lipids, and elevated blood pressure) and societal level health determinants (e.g., health systems, health policies, and barriers to CVD

prevention and care). Both individual and societal risk factors vary considerably between different regions of the world and economic settings [1]. In addition, atherosclerosis has a genetic background. More than 50 gene variants have been identified which modulate atherogenesis, and environmental risk factors for atherosclerosis are in part also genetically determined. However, the current relevance of these findings for clinical practice is limited due to the small effect sizes of identified risk variants with insufficient discriminatory power and due to a lack of therapeutic options [7].

A substantial body of evidence from a large number of epidemiological studies has shown that environmental factors or lifestyle-related risk factors (also called "*traditional risk factors*") explain a large part of CVD. Approximately 80% of CVDs are related to smoking, high blood pressure as well as lipid and glucose metabolism disturbances (the latter two mediated by an unhealthy diet including high intake of salt, saturated fat, and refined sugar) [8]. The INTERHEART case–control study examined the predominant modifiable risk factors for the occurrence of a first myocardial infarction in 15,152 cases and in 14,820 age- and sex-matched controls in 52 countries. In total nine risk factors could be identified that accounted for >90% of the population attributable risk for myocardial infarction. Six of these risk factors related to increased risk (dyslipidaemia, smoking, diabetes mellitus, hypertension, abdominal obesity, psychosocial factors), while others demonstrated protective effects (healthy diet, physical activity, regular moderate alcohol consumption). Among these factors, dyslipidaemia was the single most important risk factor for first myocardial infarction [9]. Similar results were reported by the interstroke study (13,447 cases, 13,472 age- and sex-matched controls, 32 countries) demonstrating that 91% of stroke burden is attributable to the same nine modifiable risk factors, with the addition of cardiac causes (e.g., atrial fibrillation). The population attributable risk of these ten risk factors was similar for ischemic and haemorrhagic strokes, and hypertension was the predominant risk factor for both types of stroke [10].

For individual CVD risk assessment, the first step is therefore to determine whether one or more of these traditional risk factors for CVD is present. The following paragraphs summarize the contributions of selected risk factors to individual CVD risk:

– Obesity: In contrast to other major risk factors that have demonstrated favourable prevalence developments (e.g., blood cholesterol, blood pressure, and smoking), the average body mass index (BMI) has continuously increased over recent decades across the world. This trend is associated with clinical complications such as increases in blood pressure, dyslipidaemia, insulin resistance, systemic inflammation as well as development of diabetes mellitus and CV events [11]. Both overweight and obesity are associated with an increased risk of all-cause mortality and CVD death [12]. All-cause mortality is lowest with a BMI of 20–25 kg/m^2 [11–13]. Achieving and maintaining a healthy weight has favourable effects on metabolic risk factors (e.g., regarding blood, lipids, glucose tolerance) and total CVD risk [11].

- BMI, as a marker of general obesity, is a good predictor of CVD risk, particularly at higher levels [11–13]. However, there is substantial evidence that individual differences in regional body fat distribution, particularly in visceral adipose tissue and liver fat accumulation, are the main drivers of cardiometabolic risk [12]. This provides an explanation for the heterogeneity in the CVD risk profile observed in overweight subjects which varies depending on the location of adipose deposition.
- Physical inactivity: Physical inactivity is an important component of the noncommunicable disease epidemic worldwide [14]. Based on WHO data collected in 122 countries, it is estimated that one-third of adults are physically inactive (defined as <30 min of moderate-intensity physical activity on at least 5 days each week, <20 min of vigorous-intensity physical activity on at least 3 days each week, and achieving a total of <600 metabolic equivalent-min per week, based on all forms of activity) [1, 15]. Physical inactivity causes 6% of the burden of disease from coronary heart disease, 7% of type 2 diabetes, 10% of breast cancer, and 10% of colon cancer worldwide [16]. It is responsible for 9% of premature mortality, and for more than 5.3 million of the 57 million deaths that occurred worldwide in 2008. It has been estimated that a decrease in inactivity of 25% would prevent more than 1.3 million deaths every year and elimination of the inactivity would increase the life expectancy of the world's population by 0.68 years [16]. The disease burden related to physical inactivity is also responsible for a substantial economic burden worldwide, with estimated costs for health-care systems of $53.8 billion in 2013 [17].
- Smoking: Since 1980, there has been a steady decline in tobacco smoking in most countries, and it is estimated that 31% of men and 6% of women worldwide currently smoke tobacco products daily [18]. However, smoking is still a major risk factor for CVD, linked to a doubling of the 10-year CVD mortality rate in Europe and responsible for approximately 30% of CVD deaths in the USA [43, 47, 51]. Importantly, there seems to be no lower dose limit [52]. Passive smoking is equally harmful and increases CVD risk by up to 60% [12, 53, 54]. There is also growing evidence of adverse cardiovascular effects of electronic cigarettes [1, 19].
- Blood pressure: High blood pressure is a leading risk factor for disease burden globally, accounting for 9.4 million deaths and 7.0% of global disability-adjusted life-years in 2010 [11, 20]. This represents an increase of approximately 2.1 m deaths as a result of high blood pressure compared to 1990 [20]. Overall, the prevalence of hypertension among adults worldwide is around 30–45%, with a strong increase with ageing. Elevated blood pressure is a major risk factor for CVD including coronary artery disease, cerebrovascular disease, peripheral artery disease, heart failure, and atrial fibrillation [11]. Observational studies indicate a graded and linear increase in the risk of death from coronary artery disease or stroke, starting from blood pressure levels as low as 115 mmHg systolic and 75 mmHg diastolic upwards [11, 21].
- Dyslipidaemia: The crucial role of dyslipidaemia, especially hypercholesterolaemia, in the development of CVD has been unequivocally documented by genetic,

pathogenesis, observational, and intervention studies [11]. There is a strong, graded positive association between both total and LDL cholesterol and risk of atherosclerotic CVD. This relationship has been confirmed in men and women as well as in subjects without and with established CVD [22–24].

Low HDL cholesterol is independently associated with higher CVD risk and may even compete with hypercholesterolaemia as a risk factor for coronary artery disease [11, 25, 26]. The causal role of HDL cholesterol in CVD, however, has been questioned by Mendelian randomization studies [27]. Therapeutic options to increase HDL cholesterol levels are limited. This increase can be best achieved by increasing physical activity and addressing other lifestyle factors, rather than with drug treatment [11].

High concentrations of lipoprotein(a) are also associated with an increased risk of coronary artery disease and ischaemic stroke. A causal role in CVD is supported by findings of Mendelian randomization studies. However, there are currently no randomized clinical trials showing that the reduction of lipoprotein(a) levels impacts CVD risk [28].

In addition, hypertriglyceridaemia has been confirmed as an independent CVD risk factor, but the associations are much weaker than those observed for hypercholesterolaemia [11, 29]. There are no randomized trials to provide sufficient evidence to derive target levels for triglycerides.

– Diabetes mellitus: Patients with diabetes mellitus on average have double the risk of CVD compared to those without diabetes mellitus [11, 30]. In addition to the increased risk of ischemic heart disease and stroke, diabetic microvascular complications (e.g., retinopathy, nephropathy, and neuropathy) have a significant negative impact on morbidity. Therefore, patients with diabetes mellitus are considered high-risk subjects and are treated similarly to subjects with already established CVD. Diabetes mellitus is the sixth leading cause of disability worldwide [31]. Mortality and morbidity from diabetes mellitus continue to increase globally, which is a consequence of both demographic changes and a higher burden of risk factors for diabetes mellitus development (e.g., obesity) [1]. Since the 1980s, the worldwide prevalence of diabetes mellitus in adults has increased substantially in both men (from 4% to 9%) and women (from 5% to 8%) [32].

The lifetime risk of CVD varies substantially with the aggregated risk factor burden. This was shown by a meta-analysis at the individual level using data from 18 cohort studies involving a total of 257,384 men and women with measurements of risk factor for CVD at the ages of 45, 55, 65, and 75 years [33]. Participants who were 55 years of age and had an optimal risk factor profile (total cholesterol level <4.7 mmol/L; blood pressure <120 mmHg systolic and 80 mmHg diastolic; nonsmoking status; and nondiabetic status) had substantially lower risk of death from CVD through the age of 80 years than participants with two or more major risk factors (4.7% vs. 29.6% among men, 6.4% vs. 20.5% among women) (Fig. 1). Similar differences within risk-factor strata were likewise observed for other CVD events (fatal CHD or nonfatal myocardial infarction, fatal or nonfatal stroke) as well as across diverse birth cohorts. Accordingly, the lifetime risk of a 55-year-old

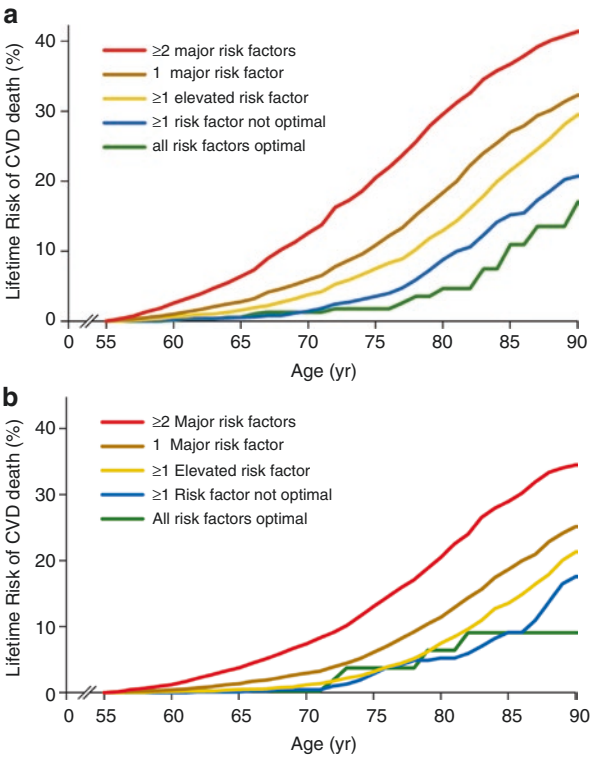

Fig. 1 Lifetime risk of cardiovascular disease death among men (**a**) and women (**b**) at 55 years of age, according to the aggregate risk factor burden, adjusted for competing risks of death (modified according to [33])

subject for any of these CVD events was 14.6% and 10.1% in men and women with an optimal risk factor profile compared to 46.8% and 29.2% in men and women with at least two major risk factors [33].

Purpose and Target Populations of Risk Assessment in CVD

Rationale for Cardiovascular Prevention

Atherosclerosis, the most important cause of CVD, usually develops over a long period, starting during childhood or before. The diseases are often already in an advanced stage when they become symptomatic, or even worse, the first manifestation is a fatal event. Major causes of atherosclerosis are known and a huge body of evidence shows that modifying them reduces risk. Therefore, the aim of the preventive approach is to intervene as early as possible in order to prevent or delay clinical manifestations. Preventive measures typically target middle-aged or older people with established CVD (secondary prevention) or those at high risk of developing a first cardiovascular event because of a combination of risk factors (primary prevention).

Cardiovascular Prevention Strategies

Two principle approaches to CVD prevention have been described over 30 years ago by Geoffrey Rose: the population strategy and the high-risk strategy [34]. These approaches should be regarded as complementary rather than competitive. The aim of the population strategy is to reduce the incidence of CVD at the population level through lifestyle and environmental changes (e.g., banning smoking in public places, reduction of the salt content of food, or promotion of physical activity). Such measures may result in large benefits for the population, but little effect on the individual person, which is also known as the "prevention paradox" [35]. The impact of such an approach on the total number of cardiovascular events may be large, as everyone is targeted and the majority of cardiovascular events occur in the large group of subjects who are at only modest risk. In contrast, the aim of the high-risk approach is to reduce risk factor levels in subjects with the highest cardiovascular risk (i.e., individuals without CVD in the upper part of the total cardiovascular risk distribution or those with established CVD). Although individuals are more likely to benefit from these preventive interventions, the impact at the population level is limited due to the low number of targeted people. A precondition for this approach is the need to identify high-risk individuals through opportunistic or systematic screening. Usually, estimation of total risk is used to identify patients requiring prevention because this reflects a combination of several risk factors that may interact [6, 36]. The focus of the following paragraphs will be on the high-risk strategy of prevention.

Why Assess CVD Risk?

While a general estimate of the relative risk for CVD can be approximated by counting the number of traditional risk factors, a more precise estimation of the absolute risk for a first CVD event is desirable in order to be able to make treatment recommendations for a specific individual [6]. The main purpose of risk assessment is therefore to enable (early) individual recommendations for preventive or treatment measures in order to reduce the risk of CV events. The choice of these measures depends on the risk class an individual person may be attributed to (see Sect. "Preventive Recommendations" for details).

Who Should Undergo CVD Risk Estimation?

Patients with established CVD, diabetes mellitus, very high levels of individual risk factors, and moderate to severe renal impairment are already at high or very high risk of cardiovascular events and need rapid treatment of all modifiable risk factors. Thus, assessment of total CV risk is intended for use in people who are apparently healthy. In line with these principles, current European and US guidelines

recommend periodic CVD risk assessment in individuals without established CVD [11, 37, 38]. CVD risk should be regularly reassessed, because it is not static. For patients at low risk and with no change in clinical status, reassessment is recommended every four to six years. For those at intermediate or higher CVD risk, re-evaluation should be considered more frequently, but the primary focus in these subjects is on risk factor modification [11, 37, 38].

In general, regular risk assessment offers the opportunity to identify CVD risk factors and offer guidance on the appropriate management of specific risk factors (e.g., dietary modifications for hypertension or dyslipidaemia) and overall CVD risk (e.g., maintaining a healthy diet, regular exercise). It is not known, at what age risk assessment should no longer be performed. However, most risk models have only been validated for patients between 40 and 79 years of age and are not applicable to older individuals. For older patients without known CVD, evaluation of risks and discussions about benefits of primary preventive therapies should therefore be considered on an individual basis.

Strategies of CVD Risk Assessment

Estimation of Total Cardiovascular Risk

The term "total cardiovascular risk" describes the likelihood of a subject to develop an atherosclerotic cardiovascular event over a certain period of time [6]. "Total risk" is the risk estimated by considering the effects of the major risk factors of age, gender, smoking, blood pressure, and lipid levels [6]. Accordingly, identification of these traditional CVD risk factors is the first step in risk evaluation. It is generally recommended to use this data to calculate an estimated 10-year CVD risk using one of the existing CVD risk calculators.

Usage of CVD Risk Calculators

Several multivariate risk models have been developed for estimating the risk of initial CVD events in apparently healthy individuals using information on individual characteristics and risk factors. All available risk estimators have their advantages and disadvantages and also have certain significant differences with respect to the risk factors and patient characteristics that are included as well as differences resulting from the population they have been developed for (e.g., regarding age ranges and ethnicity). A potential limitation of many risk calculators is that some major risk factors (e.g., physical inactivity, diabetes mellitus, glucose intolerance) as well as other risk factors are often not included into the risk calculation models. The real risk may also be higher than the estimated risk in sedentary individuals, individuals with abdominal obesity, individuals with a positive family history of CVD, socially deprived groups, ethnic minorities, those with pre-clinical atherosclerosis (e.g., carotid plaques or increased intima-media thickness, low HDL

cholesterol, increased triglycerides, increased fibrinogen, increased apolipoprotein B, increased lipoprotein(a), or impaired renal function) [6]. Moreover, non-atherosclerotic diseases that are also related to an increased risk for CVD (e.g., systemic inflammatory or autoimmune diseases; see also Sect. "Other Diseases with Increased Risk of CVD") are usually not considered for the calculation of individual CVD risk.

No single risk model is appropriate for all patients and a specific tool for CVD risk assessment has to be chosen based on patient-specific characteristics (e.g., age, gender, ethnicity) [39–41]. Moreover, the available risk calculators use different outcomes to define CVD risk. The risk models that predict "*hard*" CVD events (i.e., death, myocardial infarction, stroke) are preferred over those that include other endpoints (i.e., coronary revascularization).

The currently preferred risk calculators differ between different regions around the world:

- SCORE or JBS3 risk estimator in Western Europe [11, 38, 42].
- JBS3 risk estimator in the UK [38].
- ACC/AHA ASCVD risk calculator and Framingham risk score in the USA and Canada [37, 43, 44].
- China-PAR CVD risk calculator in China [45].

In addition, several other tools are available for assessing total cardiovascular risk, including ASSIGN [46], PROCAM [47], WHO/ISH [48], and the Reynolds score [49]. Some of these risk calculators have been comprehensively reviewed [50, 51]. The following risk estimation tools are among the ones most used in Europe and America and will therefore be described in more detail:

- *The SCORE risk assessment model* (https://www.escardio.org/Education/Practice-Tools/CVD-prevention-toolbox/SCORE-Risk-Charts) [11]: The Systematic COronary Risk Evaluation (SCORE) assessment model estimates the 10-year risk of fatal CVD for women and men aged 40–65 years by gender, age, systolic blood pressure, total cholesterol, and smoking status. The interactive, electronic version additionally allows to adjust for HDL cholesterol (www.heartscore.org). SCORE should not be used in subjects at high or very high risk such as those with known CVD, type 2 or type 1 diabetes with target organ damage, and those with moderate to severe chronic kidney disease (all considered at very high risk), or with individuals with markedly elevated single risk factors (considered at high risk). These individuals require immediate attention to all risk factors. The *SCORE* has been developed based on a large European dataset (12 European cohort studies, 250,000 patients, million person-years of observation, 7000 fatal CV events). Different risk charts are available for high-risk and low-risk regions of Europe, respectively (Fig. 2). The *SCORE* risk function can be calibrated to each country's national mortality statistics; adjusted versions are available for some countries (i.e., Belgium, Germany, Greece, The Netherlands, Spain, Sweden, and Poland). The risk

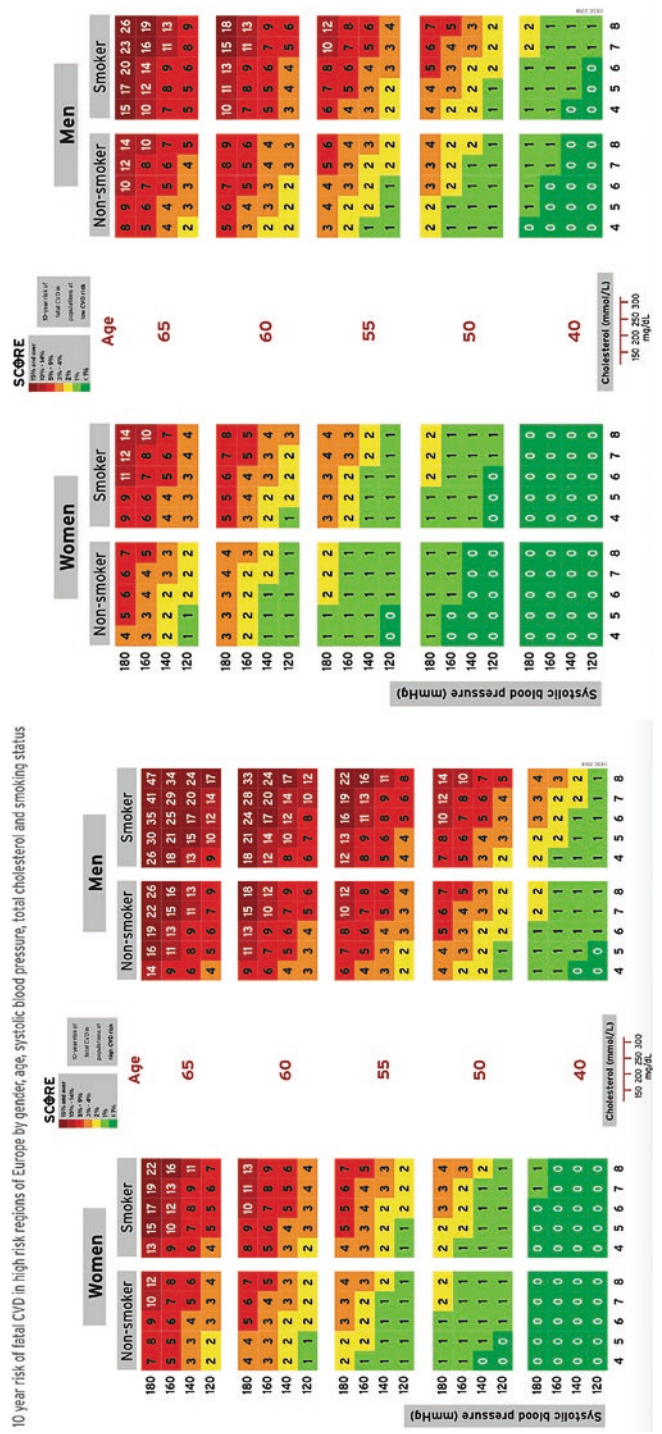

Fig. 2 European SCORE risk charts for low-risk regions (left; low-risk countries: Andorra, Austria, Belgium, Cyprus, Denmark, Finland, France, Germany, Greece, Iceland, Ireland, Israel, Italy, Luxembourg, Malta, Monaco, The Netherlands, Norway, Portugal, San Marino, Slovenia, Spain, Sweden, Switzerland, and the UK) and high-risk regions (right; high-risk countries; other European countries; some countries are at very high risk and the charts may underestimate risk: Armenia, Azerbaijan, Belarus, Bulgaria, Georgia, Kazakhstan, Kyrgyzstan, Latvia, Lithuania, Macedonia FYR, Moldova, Russia, Ukraine, and Uzbekistan) (modified according to [54])

charts are the core of the SCORE risk estimation system (Fig. 2). Moreover, calculation of an individual's "*risk age*" (according to the individual risk profile) is possible [52, 53], which is an intuitive and easily understood method for communicating about risk, particularly in younger patients, aiming to facilitate lifestyle change.

– *Joint British Societies' (JBS3) risk calculator* (http://www.jbs3risk.com) [38]: This estimates the 10-year risk of CVD (i.e., myocardial infarction and stroke) in individuals aged between 30 and 84 years without existing CVD or familial hypercholesterolemia. The JBS3 calculator is based on the QRISK lifetime CV risk calculator and combines many of the same variables from the original QRISK and QRISK2 scores (i.e., gender, age, ethnicity, body mass index, systolic blood pressure, smoking status, total and HDL cholesterol, social status, family history of coronary heart disease in first degree relatives aged <60 years), previously developed in the UK. In addition, the presence of certain high-risk diseases (i.e., diabetes mellitus, severe or treated hypertension, chronic kidney disease stages 3–5, atrial fibrillation, rheumatoid arthritis) is considered in the estimations, although caution should be exercised in these patient groups because of potential underestimation of their individual risk. The JBS3 calculator extends the assessment of risk beyond the 10-year window of most prior risk estimators and estimates the "*heart age*" (i.e., a comparison to a person of the same age, gender, and ethnicity with an optimal risk factor profile). Moreover, it calculates the CVD risk over longer time intervals (i.e., the average expected age of an individual without myocardial infarction or stroke). The tool additionally gives an estimation of the impact of interventions such as the lowering of blood pressure or lipid levels by calculating average gained event free years without myocardial infarction or stroke.
– *ACC/AHA ASCVD risk calculator* (http://tools.acc.org/ASCVD-Risk-Estimator-Plus/#!/calculate/estimate/) [37, 43]: The American College of Cardiology/American Heart Association (ACC/AHA) Pooled Cohort Equations CV risk calculator assesses the 10-year risk of "*hard*" atherosclerotic cardiovascular disease (ASCVD) events that may be reduced by statin therapy (i.e., the first occurrence of nonfatal myocardial infarction, coronary heart disease death, or fatal or nonfatal stroke) in individuals without previous CVD aged 40–79 years. Variables that are considered by this score are: age, total cholesterol, HDL cholesterol, systolic blood pressure (including treated or untreated status), diabetes mellitus (yes or no), and current smoking status. In addition, the newest version of the tool can adjust the estimation with respect to current statin or aspirin use. This risk calculator is based on data from non-Hispanic whites and African Americans in the USA.
– *Framingham risk score* (https://www.framinghamheartstudy.org/fhs-risk-functions/) [44]: The Framingham risk calculators for men and women are based on a population-based sample from the USA, participants of the Framingham Heart Study aged 30–74 years at baseline. Sex-specific multivariable risk functions were derived that incorporated variables such as age, total and HDL cholesterol, systolic blood pressure, treatment for hypertension, smoking status, and diabetes

status. Various Framingham calculators are available for the prediction of the individual 10-year risk of cardiovascular events such as CVD (coronary death, myocardial infarction, coronary insufficiency, angina, ischemic stroke, haemorrhagic stroke, transient ischemic attack, peripheral artery disease, heart failure), coronary heart disease, hard coronary heart disease (myocardial infarction or coronary death), and stroke. Moreover, the 30-year risk of CVD (as defined above) can be calculated based on these data. Additional calculators have been developed based on data from the Framingham study for estimating the risk of further cardiac diseases, which are not proven to be reduced by statin therapy such as heart failure and atrial fibrillation, as well as for intermittent claudication, which may be less important to patients than hard clinical events such as death, MI, and stroke.

The use of CVD risk calculators may be problematic in some specific populations: Thus, for *patients younger than 40 years* there are no robust data for atherosclerotic CVD risk, although generally the incidence is low in this population. Virtually all younger subjects are classified as low risk by all available risk calculators, regardless of risk factor burden. Nevertheless, subjects with a positive family history for CVD in a first degree relative or those with familial hypercholesterolemia as well as individuals with pathological findings in imaging studies (e.g., coronary artery calcium scoring) may present for counsel. In these patients, discussion of the long-term or lifetime risk may be considered, since the 10-year risk is highly likely to underestimate the individual risk substantially. Likewise, the use of risk calculators in patients older than 79 years is problematic for most risk calculators. In these persons, individual counselling regarding the risks and benefits of primary preventive therapies and shared decision-making should be the preferred choice.

Lifetime Risk

Since the strongest determinant of risk is age, a substantial proportion of individuals with a low calculated 10-year CVD risk nevertheless have a high lifetime risk of CVD. This was elegantly illustrated in a study of 4064 individuals less than 50 years of age from two independent studies (the Coronary Artery Risk Development in Young Adults—CARDIA and the Multi-Ethnic Study of Atherosclerosis—MESA) [55]. This study showed that individuals with a low 10-year risk but a high lifetime risk had a greater subclinical disease burden (carotid intima-media thickness or coronary artery calcium score) and a greater incidence of atherosclerotic progression compared with individuals with low 10-year and low lifetime risk, even at younger ages. Therefore, in people with a low or very low 10-year risk, particularly in younger subjects with pathological findings from imaging examinations, calculation of the lifetime CVD risk may be helpful [37].

Preventive Recommendations

The estimation of an individual's CVD risk can have implications for the patient himself (i.e., the need for lifestyle changes and/or initiation of preventive medications) as well as for his family members (i.e., the need for CVD risk screening). Individuals are usually subdivided into risk groups as shown for the SCORE system in Table 1.

The choice of preventive measures depends on the risk class an individual person is attributed to:

- Subjects with a low or very low 10-year CVD risk should be encouraged to maintain their healthy lifestyles (e.g., maintain body weight, regular exercise, healthy diet).
- In subjects with moderate 10-year CVD risk, discussions about possible lifestyle changes and/or primary preventive therapies should be initiated, and additional screening may be considered (e.g., coronary calcium scoring).
- In subjects with a high 10-year CVD, appropriate primary preventive therapies should be started (e.g., lipid-lowering or antihypertensive medication).
- First degree relatives of patients with high CVD risk may be counselled regarding undergoing CVD risk assessment.

The main areas targeted for primary prevention of CVD are summarized within the following paragraphs regarding guideline recommendations (predominantly for European guidelines of the European Society of Cardiology [ESC], the Joint British

Table 1 CVD risk groups according to the SCORE assessment model

Very high risk	– SCORE ≥10% Or subjects with any of the following: – documented CVD – type 2 or type 1 diabetes and target organ damage (e.g., microalbuminuria) – moderate to severe chronic kidney disease (GFR <60 ml/min/1.73 m)
High risk	– SCORE ≥5% and <10% Or markedly elevated single risk factors – familial dyslipidaemia – severe hypertension
Moderate risk	– SCORE ≥1% and <5% further modulated by – family history of premature CAD – abdominal obesity – low physical activity levels – low HDL cholesterol – elevated triglycerides – elevated hs-CRP – social class
Low risk	SCORE<1% and free of above listed qualifiers

GFR Glomerular filtration rate, *CAD* coronary artery disease, *hs-CRP* high-sensitivity CRP. Table modified from [56]

Societies' consensus recommendations [JBS3], and the National Institute for Health and Care Excellence [NICE] as well as for the US guidelines of the American Heart Association [AHA], and the American College of Cardiology [ACC]) and potential treatment effects that may be expected.

Lifestyle Modifications

Exercise and Increase of Physical Inactivity

Regular physical activity is a core of CV prevention. Increased physical activity is positively related to improvements in cardiorespiratory fitness and mental health and has a positive impact on the majority of health outcomes including cardiovascular morbidity and mortality [11]. A meta-analysis of 33 studies with a total of 102,980 participants nicely showed an association of better cardiorespiratory fitness with lower risk of all-cause mortality (13% lower mortality per metabolic equivalent task [MET]) as well as with a lower mortality due to coronary heart disease and CVD [57]. This was also confirmed by a recent study in 122,007 patients demonstrating that cardiorespiratory fitness was inversely associated with long-term mortality during a 10-year follow-up. Interestingly, in this investigation no upper limit of benefit was observed, meaning that extremely high aerobic fitness was associated with the greatest survival. This was also confirmed in older patients and those with hypertension [58]. Likewise, an increase in fitness of one MET was associated with a 15% lower risk for coronary artery disease events (diagnosis or death from coronary heart disease, or coronary revascularization) after 8.8 years in a population comprising 4527 low-risk adults with no previous history of CVD from the Norwegian HUNT3 study [59], underlining that increasing cardiorespiratory fitness may have substantial benefits in reducing the burden of CVD.

Guidelines from the ESC and AHA/ACC give class 1A recommendations with almost identical prescriptions that are also in line with the NICE recommendations [11, 60, 61]. The European guidelines, for example, recommend performing at least 150 min a week of moderate intensity or 75 min a week of vigorous-intensity aerobic physical activity for healthy adults of all ages [11]. The guidelines all agree that any form of exercise provides CVD risk reduction, with those newly starting exercise achieving the greatest benefits. Sedentary subjects should be encouraged to start light-intensity aerobic physical activity [11, 60, 61].

Weight Reduction

CVD risk has a continuous positive relationship with BMI and other measures of body fat. Therefore, international prevention guidelines advise maintenance or achievement of a healthy weight (i.e., a BMI between 20 and 25 kg/m^2) for reduction of major risk factors and improvement of CVD risk. Because all-cause mortality also appears to increase with a BMI <20 kg/m^2 [12, 13], such low BMI levels are not recommended as treatment goals [11]. In addition, there is evidence that optimal weight in the elderly is higher than in the young and middle-aged [13]. No specific intervention is recommended for weight reduction in overweight and obese

subjects, although diet, exercise, and behaviour modifications are the main therapies listed. Recent data suggest that medical therapy with orlistat and/or bariatric surgery might be complementary options. Meta-analyses indicate that bariatric surgery may significantly reduce the risk of myocardial infarction, stroke, CV events, and mortality compared with controls [11, 38, 62, 63].

As outlined above individual differences in regional body fat distribution, particularly in visceral adipose tissue, are the main drivers of the cardiometabolic risk [12]. Therefore, reduction in waist circumference as a proxy of visceral adiposity has become an important alternative target for improvement of CVD risk. In Europe, the WHO thresholds for waist circumference are the most widely accepted. Based on these thresholds, two action levels are recommended: (1) a waist circumference ≥94 cm in men and ≥80 cm in women represents the threshold at which no further weight should be gained and (2) a waist circumference ≥102 cm in men and ≥88 cm in women represents the threshold at which weight reduction should be advised [11].

Smoking Cessation

Stopping smoking is the single most cost-effective intervention in CVD prevention, with short- and long-term benefits seen irrespective of length or intensity of smoking habits [11, 38, 60]. A systematic review and meta-analysis showed reductions in myocardial infarctions and in the composite endpoints of death and myocardial infarction of between 43% and 26% compared with continued smoking [11, 64]. Randomized trials also support advice for smoking cessation, with the risk of CVD approaching (but never equalling) the risk of never smokers within 10–15 years [11]. A growing body of evidence links the introduction of smoke-free legislation with a reduction in hospital admissions for myocardial infarction and other acute coronary event of between 27% and 40%, while larger studies have reported more modest reductions of between 8% and 17% [65–67].

Therefore, all international guidelines recommend smoking cessation [11, 38, 60, 61] and suggest offering follow-up support, nicotine replacement therapies, or other pharmacological agents (e.g., varenicline, bupropion) which may help to improve abstinence rates by 50–70% [68, 69].

Dietary Advice

Diet is thought to play a significant role in CVD risk but the evidence regarding its use is not clear, nor are the guidelines consensual [70]. The AHA/ACC guidelines recommend the DASH diet (Dietary Approaches to Stop Hypertension) consisting of low proportions of sugars and saturated fats in combination with high amounts of vegetables, fruits, and whole grains [60]. The NICE guidelines recommend reducing saturated fat intake, increasing monounsaturated fatty acids and five portions of fruit and vegetables per day as well as a high fibre diet and two portions of fish per week [61]. The ESC recommends changing saturated versus polyunsaturated fatty acids, an increase in fibre, fruit, vegetable, and fish intake as well as adherence to a Mediterranean type diet [11]. Moreover, the ESC recommends limiting the energy intake to the amount of energy that is needed to maintain a BMI >20 kg/m^2 but <25 kg/m^2 [11].

Regarding the evidence for beneficial CVD effects of dietary changes, one larger study in 7447 participants aged 55–80 years at high cardiovascular risk but without existing CVD, the PREDIMED trial, showed a reduction of major cardiovascular events (myocardial infarction, stroke, or death from cardiovascular causes) after a median follow-up of 4.8 years among those assigned to a Mediterranean diet supplemented with extra-virgin olive oil or nuts compared to those assigned to a reduced-fat diet [71]. The event reduction was 31% for the Mediterranean diet with extra-virgin olive oil and 28% for a Mediterranean diet with nuts, versus the control diet.

There is also good evidence that industrially produced transfats are causally linked to coronary heart disease [72] and these are specifically proscribed in ESC and NICE guidelines [11, 61].

In summary, the evidence underlying dietary advice is rather weak and diverse. International guidelines reference different studies, some of which have been conducted many years or even decades ago when dietary patterns were substantially different. Nevertheless, there seems to be a good rationale for recommending diets high in fibre, fruit and vegetable intake, and low in simple sugars and salt. Adherence to a Mediterranean style diet also appears to be cardioprotective.

Preventive Medication

Lipid-Lowering Drugs

The primary mechanism of action of statins is the lowering of serum cholesterol through inhibiting hepatic cholesterol biosynthesis, thereby upregulating the hepatic LDL receptors and increasing the clearance of LDL cholesterol [73]. The use of statins is highly efficacious in preventing myocardial infarction, stroke, and cardiac death [74]. Overwhelming evidence confirms the beneficial effect of statins in individuals with a high risk of CVD events, such as patients with known coronary disease. The evidence that reducing plasma LDL cholesterol reduces CVD risk is unequivocal; the results of epidemiological studies and trials with and without statins using angiographic or clinical endpoints confirm that the reduction of LDL cholesterol is of main concern in the prevention of CVD [11, 24]. Large meta-analyses of statin trials consistently show a dose-dependent relative reduction in CVD with LDL cholesterol lowering. Thus, every 1.0 mmol/L reduction in LDL cholesterol is associated with a 20–25% reduction in CVD mortality and nonfatal myocardial infarction [75].

However, the effects of statin therapy are not only due to the reduction of LDL cholesterol levels, but can also be attributed to many beneficial, pleiotropic effects statin therapy has on various inflammatory mechanisms in atherosclerotic disease, e.g., the reduction of levels of adhesion molecules, proinflammatory cytokines, and reactive oxygen species as well as improvement of endothelial dysfunction [73, 76].

While previous guidelines recommended using statins to target-specific cholesterol and LDL concentrations, recent European guidelines emphasize using CVD risk to guide statin use and downplay the importance of treating cholesterol to a

target value [11, 43]. A lipid-lowering medication with statins is recommended for most patients with a 10-year risk of 7.5–10% or greater in accordance with many international guidelines [11, 38, 60, 61]. Importantly, there seems to be no cholesterol threshold at which there is no benefit to the use of statins [77]. Recent evidence from both trials with statins and PCSK9 (proprotein convertase subtilisin/kexin type 9) inhibitors indicates that even with very low initial LDL levels (of 1.8 mmol/L), lowering LDL further reduces CVD risk [75, 77].

Aspirin

The role of aspirin as a primary prevention measure in subjects without existing CVD has been questioned by several older and recent studies in diverse patient populations without existing CVD (i.e., patients with diabetes mellitus aged ≥40 years, individuals with a 10-year CVD risk of 10–20%, persons of older age) [78–80]. These studies found a moderate or no reduction in cardiovascular events and increased risks of bleeding. A recent systematic review and meta-analysis of the 13 largest randomized clinical trials between 1998 and 2018 were conducted comparing the use of aspirin against no use [81]. These involved a total number of 164,225 participants with no known CVD. The net benefit was evaluated regarding a composite efficacy outcome (cardiovascular mortality, nonfatal myocardial infarction, and nonfatal stroke) and a bleeding outcome (any major bleeding as defined by the individual studies). The main finding of this meta-analysis was that aspirin use was associated with a lower risk of cardiovascular events (hazard ratio [HR] 0.89; absolute risk reduction 0.38%) and an increased risk of major bleeding (HR 1.43; absolute risk increase 0.47%) [81]. Another meta-analysis using data of 33,679 patients from 10 randomized controlled trials even found that the use of aspirin for primary prevention of CVD in patients with diabetes mellitus increased the risk of total bleeding markedly (risk ratio [RR] 1.29, 95% CI 1.07–1.55, $P = 0.01$) without reducing the risk of major adverse cardiovascular outcomes (RR 0.93, 95% CI 0.87–1.00, $P = 0.06$) [82]. In view of these recent findings, general use of aspirin in the primary prevention setting may be questioned and should be reserved for high-risk patients after discussing the pros and cons.

In contrast to primary prevention, evidence substantiates a clear net benefit of aspirin usage in secondary prevention in patients with acute coronary syndromes and stroke with reductions of future atherosclerotic CV events clearly outweighing the increased bleeding risk.

Antihypertensive Medication

The decision to start antihypertensive treatment depends on blood pressure levels and total CV risk. Treatment benefits of drugs are mainly driven by blood pressure reduction per se, not by the drug type used [11]. Blood pressure reduction in patients with stage 1 hypertension (140–159/90–99 mmHg) or worse has been shown to be effective in preventing stroke, coronary heart disease, and heart failure [74, 83]. For example, a reduction in systolic blood pressure by 10 mmHg would be expected to reduce stroke risk by 41% and coronary heart disease risk by 22% independent of other risk factors [84].

Most participants with hypertension need at least two drugs to achieve adequate blood pressure control. Increasing evidence suggests that starting treatments with two or more drugs is the most efficient and effective strategy to lower high blood pressure [11, 85, 86].

The optimal target of antihypertensive treatment is still intensively debated. Treatment targets vary with respect to the individual risk and age of the affected persons, and international societies give different recommendations. Lifestyle measures (i.e., weight control, increase of physical activity, alcohol moderation, sodium restriction, and increased consumption of fruits, vegetables, and low-fat dairy products) are recommended by all guidelines in all patients with hypertension stage 1 or higher as well as in individuals with high normal blood pressure values (130–139/85–89 mmHg) [11, 60, 86, 87].

The European guidelines recommend lowering blood pressure to values <140/90 mmHg in all patients, and to target values of 130/80 mmHg in most patients, and even a goal of 120–129 mmHg for the systolic values in patients ≤65 years of age, provided the treatment is well tolerated [86]. These guidelines furthermore recommend a timely initiation of blood pressure lowering drug treatment in patients with grade 2 or 3 hypertension (≥160/≥100 mmHg) at any level of CV risk [86]. Drug treatment is recommended in older patients (>65 years but not >80 years) with grade 1 hypertension (140–159/90–99 mmHg). In addition, it is advised to consider drug therapy already in those with high–normal blood pressure values (130–139/85–89 mmHg) and an established very high risk of CVD [86].

The US guidelines recommend a blood pressure target of less than 130/80 mmHg in adults with confirmed hypertension and known CVD or 10-year ASCVD event risk of 10% or higher. Additionally, the same target values are recommended for adults with confirmed hypertension, but without additional markers of increased CVD risk [87]. For primary prevention of CVD, the US guidelines recommend blood pressure lowering medication in adults with no history of CVD and with an estimated 10-year ASCVD risk of 10% or higher and an average systolic blood pressure of 130 mmHg or higher or an average diastolic blood pressure of 80 mmHg or higher. For secondary prevention, they recommend use of blood pressure lowering drugs in patients with clinical CVD and an average systolic blood pressure of 130 mmHg or higher or an average diastolic blood pressure of 80 mmHg or higher [87].

Treatment of Diabetes Mellitus

A multifactorial approach is the corner stone of a successful therapy in patients with type 2 diabetes mellitus. Thus, lifestyle management aims to aid weight control by sustainable dietary changes and increased physical activity levels. Moreover, intensive management of hyperglycaemia has been shown to reduce the risk of microvascular complications and, to a smaller extent, the risk of CVD [11]. Intensive lowering of blood pressure with a target of 140 mmHg systolic further reduces the risk of macrovascular and microvascular outcomes. A lower target of 130 mmHg further minimizes the risks of stroke, retinopathy, and albuminuria and is recommended in selected patients. Furthermore, lipid-lowering therapy is a key

mechanism to lower CVD risk in both type 2 and type 1 diabetes mellitus and is recommended in all patients >40 years of age as well as in selected younger patients at elevated risk [11].

Effects of CVD Assessment in Primary Prevention

Although many well validated CVD risk estimation tools are available, their efficacy in improving clinical outcomes in the primary prevention setting is currently not clear. A recent review of 41 randomized controlled trials involving 194,035 participants provided heterogeneous findings, and conclusions were largely problematic due to low-quality evidence [88]. Specifically, it was reported that evidence suggests that CVD risk assessment using risk score calculators may have little or no effect on CVD events (5.4% versus 5.3%; RR 1.01, 95%-CI 0.95–1.08) but may reduce CVD risk factor levels by a small amount compared with usual care. Thus, for example, the observed mean differences were −0.10 mmol/L (95%-CI −0.20 to 0.00) for cholesterol, and −2.77 mmHg (95%-CI −4.16 to −1.38) for systolic blood pressure, respectively. Moreover, providing CVD risk scores may reduce adverse events compared with usual care, but results were imprecise (1.9% versus 2.7%; RR 0.72, 95%-CI 0.49–1.04). CVD risk score usage may also increase new or intensified lipid-lowering medications (15.7% versus 10.7%; RR 1.47, 95%-CI 1.15–1.87) and increase new or increased antihypertensive medications (17.2% versus 11.4%; RR 1.51, 95%-CI 1.08–2.11). The overall conclusion of this review was that uncertainty remains whether current strategies for providing CVD risk scores affect CVD events, but that this strategy may slightly reduce CVD risk factor levels and increase preventive medication prescribing in higher-risk people without evidence of harm [88]. Another systemic review ultimately also concluded that there is still lack of evidence that total CVD risk assessment may reduce CVD events and mortality [89].

Summary

- Cardiovascular disease (CVD) is common in the general population, affecting the majority of older adults. While a general estimate of the relative risk for CVD can be approximated by counting the number of traditional risk factors of an individual, a more precise estimation of the absolute risk for a first CVD event is desirable when making treatment recommendations for a specific individual.
- CVD risk assessment should be performed regularly (i.e., every 4–6 years) in all subjects aged 40–79 years without known CVD or diabetes mellitus. In subjects at intermediate or higher CVD risk, assessment should be repeated more frequently, although the primary focus should be on early risk factor modification.
- Individuals of any age with existing CVD or CVD risk equivalents (e.g., diabetes mellitus) are classified as having a high risk of recurrent CV events and should be treated with appropriate secondary prevention measures.

- For all individuals without CVD, the first step in assessing CVD risk is to determine whether traditional risk factors for CVD are present (e.g., obesity, hypertension, cigarette smoking, diabetes mellitus, premature family history of CVD), usually by a baseline lipid profile and blood pressure measurements. All patients aged 40–79 years of age without known CVD should then be assessed for CVD risk using a validated CVD risk calculator in order to estimate the individual 10-year risk of CVD.
- The strongest risk factor for CVD is age. Therefore, a large proportion of individuals with a low calculated 10-year CVD risk, particularly younger subjects, still have a high lifetime risk. For these people, the lifetime CVD risk should be taken into consideration.
- Preventive recommendations are based on the individual's risk level. A general strategy is the improvement of all modifiable risk factors using both lifestyle modifications (e.g., promotion of physical activity, weight reduction, smoking cessation, dietary advises) and preventive medication (e.g., lipid-lowering drugs, antihypertensive medication).
- Evidence that total CVD risk assessment may help to reduce CVD events and mortality is still lacking, although it has been shown that this approach may slightly reduce CVD risk factor levels and increase preventive medication prescribing in higher-risk people without evidence of harm.

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Risk Assessment for Diabetes

Laura J. Gray

Contents

Introduction

Type 2 diabetes (T2DM) is a serious chronic condition characterised by high blood glucose (hyperglycaemia) caused by either reduced insulin production, reduced insulin sensitivity or a combination of these two. There are many types of diabetes, with T2DM accounting for around 90% of cases. Prolonged hyperglycaemia is associated with many serious complications. Those with T2DM have a reduced quality of life, increased risk of microvascular (problems with eyes, kidneys and feet) and macrovascular complications (stroke and myocardial infarction) and on average have a life expectancy 10 years shorter than those without T2DM [1]. Worldwide the prevalence of T2DM is rising. The International Diabetes Federation (IDF) estimated that 425 million people worldwide have diabetes; this equates to

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one in every 11 adults [2]. In the UK, the current prevalence of T2DM is 5.9%, this is expected to rise to 6.5% by 2045 [2].

T2DM has a long asymptomatic phase, it has been estimated that this may last up to 12 years [3]. Therefore, around 30% of people with T2DM have complications at diagnosis. Worldwide it is estimated that one in two cases of diabetes is undiagnosed [2], whilst in the UK it is estimated that 18.5% of cases are undiagnosed [4]. Earlier detection of T2DM whilst still in the preclinical phase means that treatments to reduce hyperglycaemia and prevent cardiovascular disease can begin sooner which may reduce complications [5].

T2DM is diagnosed using HbA1c, with values of 6.5% or more indicating T2DM [6]. There is a high-risk intermediate state, where HbA1c is raised but lower than the diagnostic threshold (6.0–6.4%). This intermediate state has been afforded many different terms including: prediabetes, borderline diabetes, high risk of diabetes and non-diabetic hyperglycaemia; here, we will use non-diabetic hyperglycaemia (NDH). Like T2DM, having HbA1c within this range will not be associated with any symptoms in the majority of cases and therefore many people will not know they have elevated blood glucose. Those with NDH are at high risk of developing T2DM [7]. It is estimated that there are five million people in England with NDH [8]. Identification of NDH is important, as there is a strong evidence base that T2DM can be prevented or delayed in this group through lifestyle modifications [9].

T2DM is associated with high health care expenditure. The IDF estimates that 12% of global health expenditure is on diabetes, equating to $727 billion [2]. In the UK in 2010/11 it was estimated that T2DM costs the NHS £8.8 billion in direct costs and £13 billion in indirect costs—representing 10% of the total health resource expenditure [10]. This was projected to rise to 17% by 2035/36 [11]. Given that two-thirds of people worldwide with diabetes are of working age, there are also societal costs which need to be taken into account [2, 12] as well as increased social care costs [13]. Health care costs are also increased in those with NDH compared to those with normoglycaemia [14, 15].

This chapter will consider how best to identify those at risk of diabetes (including those with undiagnosed T2DM, NDH or high T2DM risk) so that either treatment can commence earlier than it would have through routine diagnosis, or those with NDH can be referred to a diabetes prevention programme.

Methods of Risk Assessment

There are a number of different approaches which can be considered for identifying people at risk of diabetes. Some of these approaches involve a single stage, others combine a number of assessment methods.

Universal Blood Tests

A single stage risk assessment would involve undertaking a blood test in everyone, so, for example, testing HbA1c. Although HbA1c can be used to diagnose T2DM; here, it would be used in people not presenting with symptoms—i.e., as a screening

test. There are a number of different blood tests which can be used to assess hyperglycaemia, two of which can also be used to diagnose T2DM: HbA1c and the oral glucose tolerance test (OGTT). Universal screening is not currently recommended by the National Screening Committee in the UK, with one of the reasons for this decision being that there is no perfect screening test for T2DM [16].

The OGTT is highly inconvenient and time consuming with low uptake, although for many years it was seen as the gold standard test for the diagnosis of T2DM. The test involves a fasting blood test, followed by ingesting a fixed glucose load with a 2 h wait before another blood test is taken. The advantage of conducting an OGTT over other tests was the 2 h post-challenge result, which has been shown to be a risk indicator for cardiovascular disease [17]. The diagnostic cut-off for the 2 h test ($\geq$11.1 mmol/l) was chosen due to the increased risk of diabetes complications seen beyond this point [18]. Assessing fasting glucose alone is unsuitable for identifying T2DM as up to a third of people with diabetes who would have been identified using the full test would be missed [19].

In 2011, the World Health Organization recommended that HbA1c could also be used to diagnose T2DM, with a value of 6.5% or more signifying the diagnostic range [6]. In contrast to the OGTT, HbA1c can be tested non-fasted and the rise of point of care testing means it is also highly convenient. There are disadvantages associated with the use of HbA1c which include misleading results in those with various haemoglobinopathies, iron deficiency, haemolytic anaemias, and severe hepatic and renal disease which makes HbA1c unsuitable for screening in these groups [20]. There is also data to suggest that HbA1c is systematically higher in particular ethnicities and that it can increase with age which may also affect its interpretation [21]. For example, HbA1c has been shown to be 0.2% higher in South Asians compared to White Europeans independent of age and sex [22].

The OGTT and HbA1c identify different, overlapping groups with T2DM. A study which used data from a screening study conducted in Leicester where both OGTT and HbA1c had been measured on all participants found using HbA1c increased the number of people identified with T2DM (3.3% increased to 5.8%). Of those with T2DM using the OGTT, 1.2% had an HbA1c less than 6.5% [23].

In terms of identifying those with NDH, both HbA1c and fasting plasma glucose can be used, with HbA1c levels of 42–47 mmol/mol [6.0–6.4%] or fasting plasma glucose of 5.5–6.9 mmol/l indicating NDH.

Non-Invasive Risk Assessment

Non-invasive risk assessments employ information about risk factors to make an assessment of an individual's risk of a particular outcome. Those at the highest risk can then be referred for follow-on testing. Such risk assessments can be either diagnostic or prognostic. A diagnostic risk assessment gives the risk of currently having an undiagnosed condition, such as undiagnosed T2DM or NDH. Prognostic risk assessments predict the risk of future events, such as an individual's risk of developing T2DM over the next 10 years. Non-invasive risk assessments can also have different intended users and applications, for example, some may be developed for use

by members of the public, and therefore can only include risk factors which would be known by the public (so, for example, the results of a blood test would not be appropriate), alternatively some may make use of the data routinely stored in electronic medical records and are intended to be used by health care professionals. Some risk assessments are designed to be calculated by a piece of software and therefore have quite a sophisticated algorithm for calculating an individual's risk, whereas others might be developed to be completed by hand in a community setting, therefore requiring a very simple calculation method. Risk assessments can also be invasive in nature, i.e., require information from biomarkers and genetic risk factors, although these have been shown to offer little advantage in terms of performance over the non-invasive tools [24].

Using a non-invasive risk assessment reduces the number of people requiring a blood test by targeting those at highest risk. This reduces the costs associated with screening [25]. Also using such tools engages people with their risk factors. A blood test will demonstrate risk but not explain what has led to that increased risk, whereas a non-invasive risk assessment can instigate a conversation about reducing risk.

Many risk assessments have been developed for detecting the risk of diabetes related outcomes. These have been summarised and critiqued in a number of systematic reviews [26–29]. Below are some examples of assessments with different intended uses: we first focus on the first, and most widely researched internationally, non-invasive risk assessment—The FINDRISC—and then on two assessments developed and used within the UK (Leicester Self-Assessment Score and the QDiabetes score).

Existing Non-Invasive Risk Assessments for Diabetes Related Outcomes

International Example: The Finnish Diabetes Risk Score

Internationally, one of the first risk assessments to be developed for a diabetes outcome was the Finnish Diabetes Risk Score (FINDRISC) [30]. This score was developed using data from a random population sample of 35–64 year olds who were followed up for 10 years. Those who had developed T2DM during this time were identified via a national drug registry. This data was used to develop a score which calculated the 10 year risk of developing drug-treated T2DM. The following risk factors were included in the score: age, BMI, waist circumference, antihypertensive therapy, high blood glucose, physical activity and consumption of fruits, berries and vegetables. The score was designed to be completed by hand and therefore each of the included risk factors is categorised with a score given to each category, and the total score across all risk factors is then calculated. Higher scores represent higher risk. A score of nine or more points is used in practice to identify those at increased risk of developing T2DM [30].

In the original development paper, the score was validated using an independent sample with 5 years follow-up [30]. Using a cut point of greater than or equal to nine gave a sensitivity of 0.81 (95% confidence interval (CI) 0.69–0.89) and a specificity of 0.76 (95% CI 0.74–0.77) for predicting development of drug-treated T2DM

over a 5 year period. The score has also been evaluated using cross-sectional data to test whether it can be used to identify those with existing undiagnosed disease and NDH [31, 32]. In a study using cross-sectional screening data from Finland, a cut point of 11 gave sensitivity of 66.1 (95% CI 58.3–73.8) in men and 70.0 (60.6–79.5) in women for detecting those with undiagnosed T2DM [31].

Since this score's publication in 2003, a plethora of studies validating the score in other countries and settings followed. Many of these showed that when the score was applied to other populations the performance seen in the original paper was not replicated. One such study used a cohort from Oman to compare a number of risk assessments—one which had been specifically developed for use in Oman—as well as the FINDRISC and risk assessments developed for use in Thailand, Denmark and the Netherlands. They found that the risk assessment developed for use in Oman outperformed all the other scores. For example, the area under the receiver operating curve ((AUROC) a measure of discriminatory performance) for the Oman score was 0.83 (95% CI 0.82–0.84), compared to 0.67 (0.64–0.69) for the FINDRISC [33]. This has been a common finding in the literature [34, 35]: although the risk factors associated with developing/having diabetes are similar between populations, the relative weightings of risk factors seem to differ. Therefore it cannot be assumed that a score developed for a particular population will work as well when used in another—highlighting the need for validation studies and potential recalibration before tools are used in practice.

UK Examples: The Leicester Self-Assessment Score and The QDiabetes Risk Score

To date four risk assessments have been developed and validated for use in the UK (Cambridge risk score [36], Leicester self-assessment score [37], Leicester practice risk score [38] and QDiabetes risk score [39, 40]). Of these two are used in practice. These are described in detail below.

The Leicester self-assessment risk score (see Figs. 1 and 2) was developed to identify those at high risk of having either undiagnosed T2DM or NDH [37]. The intended users are members of the public, so a very simple score was developed which does not require input for medical professionals to complete. The score was developed using cross-sectional data from a population based screening study called ADDITION which took place in Leicester and Leicestershire [41]. The score contains seven questions about age, sex, ethnicity, family history of diabetes, high blood pressure, body mass index and waist circumference. All continuous risk factors were categorised and scores were allocated to each category—similar to the FINDRISC score. With a maximum score of 37, a score of 16 or more points was defined as the cut point for further testing. When testing the score using a dataset from another screening study, this cut point was associated with a sensitivity of 0.81 (95% CI 0.78–0.88) and a specificity of 0.45 (95% CI 0.43–0.47). This score has been validated in a number of different populations showing good discriminatory performance, these include those attending a faith centre [42], young South Asians [43] and those with learning disabilities [44]. The score has also been tested in a national longitudinal dataset (English Longitudinal Study of Ageing) to assess its

Fig. 1 The Leicester self-assessment score

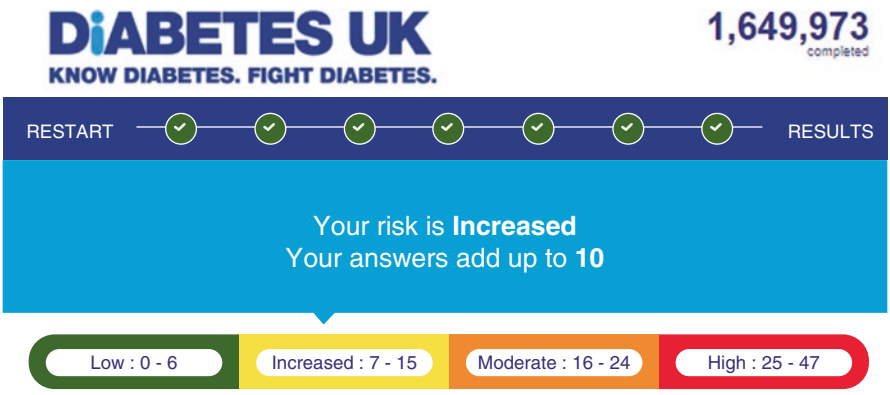
QUESTIONNAIRE: Do you want to know your risk of Type 2 diabetes? For each question, tick one box.

1 . Which age group are you in?					
49 years and younger		0	60 - 69 years		9
50- 59 years		5	70 years and older		13
2. Are you male or female?					
Male		1	Female		0
3. How would you describe your ethnicity?					
White European		0	Any other ethnic group		6
4. Do you have a parent, brother, sister and/ or child with Type 1 or Type 2 diabetes? (Do not count step-relatives)					
Yes		5	No		0
5. Which waist size group are you in? (See instructions)					
Less than 90 cm (Less than 35 inches)		0	100 - 109 cm (39 - 42 inches)		6
90 - 99 cm (35 - 38 inches)		4	110 cm (43 inches) & above		9
6. Which Body Mass Index (BMI) group are you in? (See explanation and instructions)					
Less than 25		0	30- 34		5
25- 29		3	35+		8
7. Have you ever been told by a doctor or nurse that you have high blood pressure?					
Yes		5	No		0
To get your risk score add up the numbers in the blue boxes next to the seven boxes that you have ticked. Write the total number here – **This is your risk score:** To find out what this means go to page 6					

performance in detecting those who go onto develop T2DM over the next 10 years, again showing good predictive performance for this outcome [45].

This score is recommended by NICE for opportunistic screening [46] and has been implemented across England by the national charity Diabetes UK. The score is available for completion on the Diabetes UK website where it has been completed over 1.7 million times since its launch in July 2011 (https://riskscore.diabetes.org.uk/). The risk score leaflet has been translated into a number of Indian languages to increase uptake in hard to reach ethnic groups [47].

The QDiabetes risk score in contrast was developed to be used by health care professionals to identify those at high risk of developing T2DM over the next 10 years. The score was originally published in 2009 [39] and has recently been updated [40]. Both the original and updated versions of the score were developed using data from electronic medical records data. The original score included the following risk factors: age, ethnicity, deprivation, body mass index, smoking status, family history of diabetes, cardiovascular disease, treated hypertension and prescription of corticosteroids. In the updated version, a contemporary dataset was used to update the weightings of the existing risk factors in the score and a number of additional risk factors were included. These additional risk factors were diagnosis of



1 in 10 people with your risk will get Type 2 diabetes in the next 10 years.

You can't change your age or your genes. But if your risk is partly due to your lifestyle, a few small changes can make a big difference. See below for where you're scoring points and to see if you can make a big difference. See below for where you're scoring points and to see if you can make any changes.

Your risk explained

These are the risk factors that you can't change, so focus on the things that you can change or maintain.

	Points
Age: 52	
49 or younger	0
50 - 59	5
60 - 69	9
70 or older	13
Gender	
Male	1
Female	0
Ethnicity	
Only white European	0
Other ethnic group	6
Relatives with diabetes	
Yes	5
No	0

These are the risk factors that you can change. Even small changes can help reduce your risk.

	Points
Waist measurement: 76.2cm	
Less than 90cm (35.5in)	0
90 - 99.9cm (35.5 - 39.3in)	4
100 - 109.9cm (39.4 - 43.3in)	6
110cm (43.4in) or above	9
BMI: 21.5	
Less than 25	0
25 - 29.9	3
30 - 34.9	5
35 or above	8
High blood pressure	
Yes	5
No	0

Fig. 2 Output from the Leicester self-assessment calculator on the Diabetes UK website. Screenshot taken 6/3/2019

schizophrenia or bipolar affective disorder, learning disabilities, diagnosis of gestational diabetes or polycystic ovary syndrome, prescribed antipsychotics, prescribed statins, fasting blood glucose level and HbA1c value. The inclusion of these additional risk factors was informed by NICE guidance and the evolving evidence base for novel risk factors for T2DM. Given the score is designed to be calculated by a piece of software, a more sophisticated algorithm for the calculation of the score can be used. So instead of using a crude scoring system, QDiabetes uses the regression

equation from the Cox proportional hazards model used to develop the score to give an individual probability of developing T2DM. Also given the score is calculated from a person's medical record data automatically without additional input from the health professional, the number of risk factors included is irrelevant. Three models were developed: one which excludes the glucose variables, one which includes HbA1c and one which includes fasting glucose. All models had high levels of calibration and discrimination when tested using a separate validation cohort. The original models have also undergone a number of external validations using other databases of electronic medical records which again showed good performance [48, 49].

The QDiabetes scores have been integrated into some of the leading GP computer systems and are therefore used in routine clinical practice. The score can also be calculated online (www.qdiabetes.org) and is recommended for use by NICE [46].

In 2015 Public Health England undertook a comparison of the four UK risk scores using data from the Health Survey for England. They assessed each scores' performance in terms of detecting NDH and found comparable performance across the four scores [50]. This suggests that the choice of which score to use should depend on the setting in which the score is going to be used. For screening within a primary care setting the QDiabetes score is the most applicable, whereas for opportunistic screening in community settings the Leicester self-assessment score should be used.

National Guidance for Identifying Those at Risk of Diabetes

Currently the National Screening Committee in the UK does not recommend universal screening for diabetes. The reasons for this are: (1) limited evidence for early detection improving outcomes compared to standard diagnosis; (2) concerns over the available screening test, for example, the preferred method of testing could miss up to 20% of people with undiagnosed T2DM; (3) improvements in the care and treatment for those with diabetes. They recommend that although universal screening is not appropriate, there is a case for selective screening as part of an overall vascular check (i.e., the NHS Health Check programme) [19].

The NHS Health Check programme is a mid-life health check offered to existing cardiovascular disease free individuals after their 40th birthday and at five yearly intervals after that. The aim of these checks is primary prevention of cardiovascular disease, with diabetes risk assessed as part of this. A review of the first four years of the programme found that of those eligible only 12.8% were recorded as having attended a health check [51]. This highlights the needs for opportunistic screening for diabetes risk to assess those unlikely to access these routine screening services. Although the programme has limited attendance, over this four year period 934 new cases of diabetes (1 new case for every 110 checks) were detected [51]. However there has been concerns raised over the diabetes filter used within the programme and the potential for missing cases of undiagnosed T2DM [52].

In July 2012, NICE published guidance on type 2 diabetes: prevention in people at high risk. This included a section on how to identify those at high risk (i.e., those with NDH). The latest update of the guidance, on which this summary is based was published in September 2017 [53].

NICE recommended a two stage process for risk identification, firstly assessing risk using a risk score followed by a blood test in those found to be at high risk. They state that the following groups should be encouraged to have a risk assessment:

- All eligible adults aged 40 and above, except pregnant women.
- People aged 25–39 of South Asian, Chinese, African-Caribbean, black African and other high-risk black and minority ethnic groups, except pregnant women.
- Adults with conditions that increase the risk of T2DM.

They recommend using a computer based score if the risk assessment is undertaken in a setting which enables this and otherwise use a validated self-assessment score. The recommendations encourage risk assessments to be undertaken in a range of places in addition to primary care and by a range of different professionals including pharmacists, opticians, occupational health nurses and community leaders. Those found to be at high risk should be referred for further testing using either fasting plasma glucose or HbA1c. This will identify those with current NDH (fasting plasma glucose of 5.5–6.9 mmol/l or an HbA1c level of 42–47 mmol/mol [6.0–6.4%]) or undiagnosed T2DM (using WHO 2012 diagnostic criteria [6]). GPs are recommended to keep a register of those at risk which can be used to contact and invite people for regular review. Those at low risk are recommended to have a five yearly review—in line with the NHS Health Check programme. For people identified as high risk on their risk assessment, but with blood values in the normal range, re-testing every three years is recommended. Those with NDH should have a review annually.

We outline the benefits of early detection of undiagnosed T2DM and NDH in the following section.

Benefits of Risk Identification for Diabetes

This section briefly outlines the potential benefits of risk assessing for diabetes, focussing on the early detection of T2DM and the prevention of T2DM in those with NDH.

Early Treatment for T2DM

Given that many people with T2DM have complications present at diagnosis, screening may reduce this by giving people access to treatment for hyperglycaemia at an earlier stage. The ADDITION-Europe study aimed to assess whether

screening for T2DM followed by intensive management in those with screen-detected T2DM led to improved outcomes. The study was conducted across UK, Denmark and the Netherlands and enrolled people without known diabetes from 343 general practices [5]. To identify people with screen-detected T2DM, centres used a variety of screening methods including non-invasive risk scores, capillary blood tests or OGTT.

Overall, 76,308 people were screened, resulting in 3057 people with screen-detected T2DM being recruited into the trial [54]. The trial cluster randomised general practices to either provide multifactorial intensive risk factor management to those identified with screen-detected T2DM or usual care [5]. The intervention included intensive target and guideline driven management of hyperglycaemia, blood pressure and cholesterol levels by medical treatment and promotion of healthy lifestyles. After an average follow-up of 5.3 years no difference was found between the intensively managed and usual care groups. The incidence of the composite cardiovascular outcome was 7.2% in the intensively managed group and 8.5% in the routine care group, hazard ratio 0.83, 95% CI 0.65–1.05 [5]. Additionally, intensive management of screen-detected patients was not cost-effective compared to standard care in the UK [55]. In both groups, the number of participants meeting targets for hyperglycaemia, blood pressure and cholesterol levels increased over the study period—suggesting that there were improvements in care during this time independent of the trial. There was also no benefit in terms of microvascular complications at five years [56].

The ADDITION trial did not compare screening versus no screening, but rather whether intensive management of those found with screen-detected diabetes improved outcomes compared to standard management. A secondary analysis of the ADDITION group tried to address this and used the Michigan Model for T2DM to simulate a hypothetical trial of screening and intensive treatment, screening and routine treatment and no screening with a 3- or 6-year delay in the diagnosis and routine treatment of diabetes and cardiovascular risk factors. They assessed the progression of diabetes and its complications, comorbidities, quality of life and costs; they estimated the absolute risk of cardiovascular outcomes and the relative risk reduction associated with screening and intensive treatment, screening and routine treatment and no screening with a 3- or 6-year delay in the diagnosis and routine treatment of diabetes and cardiovascular risk factors. This modelling study found major benefits associated with early diagnosis and treatment of T2DM [57]. Although there are limitations with any analysis based on simulated data, this study does suggest that efforts should be made to try to reduce the time taken to diagnose T2DM and initiate treatment.

Prevention of T2DM in those with NDH

Historically there have been a number of pivotal trials which have assessed intensive diabetes prevention programmes in those with impaired glucose tolerance (a specific type of NDH where raised 2 h glucose levels are found on an OGTT but below the diagnostic threshold for T2DM) [58]. These programmes on average reduced the risk of progressing to T2DM by 49% compared to standard care.

Whilst these results were promising, the programmes were expensive to run and not compatible with the NHS. For example, the trial run in the USA gave participants in the intervention group 16 one-to-one sessions in the first 24 weeks, monthly contact thereafter (with in-person contact at least every two months) and group-based consultations quarterly [59]. Therefore researchers attempted to take the learnings of these intensive interventions and develop and test pragmatic interventions, delivered in group-based sessions which would be suitable for implementation in the NHS. The Let's Prevent Diabetes programme was one example of such a programme. This is a 6 h programme delivered to groups in a single day, followed by two annual refresher 3 h sessions. The development and content of the intervention has been described in detail elsewhere [60]. When the intervention was tested in a cluster randomised controlled trial compared to standard care, a non-significant 26% reduction in the progression to T2DM was seen in the Let's Prevent Diabetes arm compared to standard care [61]. When assessing only those who attended all sessions this was increased to a highly significant 88% reduction—showing the importance of attendance on outcome [62]. The study also showed important improvements in step count, sedentary behaviour, diet quality and some biomarkers and was shown to be cost-effective [63]. Since the completion of this study, the NHS has launched a national diabetes prevention programme which is now delivered across England.

Evidence Gaps

In terms of risk assessment for diabetes, a lot of research has been completed and the methods for identifying adults at risk of T2DM are well established. Areas of uncertainty still exist and the evidence base continues to grow. Briefly outlined below are two key areas for which further work is required.

Historically T2DM was associated with older age, but cases of T2DM are now being seen in children, adolescents and young adults [64]. A study assessing the prevalence of T2DM in people aged 10–19 years in the USA found a 30% increase in prevalence between 2001 and 2009 [65]. Similar increases have been reported in the UK [66, 67]. Those developing T2DM at an earlier age live with hyperglycaemia for long durations and therefore have early onset of complications [68]. To date there is very little data regarding undiagnosed T2DM or the number of children, adolescents and young adults at risk of developing diabetes. One UK-based clinical trial recruited overweight and obese 18–40 year olds. Of the 193 participants recruited, 5% had undiagnosed T2DM and 18% had elevated glucose levels putting them at risk of developing T2DM [69]. Risk assessment for diabetes has to date concentrated on adults, with the currently available scores tending to predict risk in those aged 40 years and above. It is not clear whether such risk assessments are suitable for use in children, adolescents and young adults. A recent editorial published in the Lancet stated "Approaches to screen and diagnose adults who are at risk have not been thoroughly validated in young people. There is an urgent need to include young patients in future research to develop and inform strategies targeted at prevention and treatment of type 2 diabetes" [70].

Uptake to routine vascular screening is low, some have suggested that this may be due to low perceived personal risk of T2DM and a low perceived seriousness of the condition [71]. Therefore finding ways to engage hard to reach groups in risk assessment—either routinely provided or opportunistic—is vital. Research into alternative settings for screening has shown promising results, for example, within pharmacies [72, 73], or accident and emergency departments [74–76], as well as alternative community settings such as faith centres [42] but none of these is standard practice.

Dental settings offer a prime setting for undertaking diabetes risk assessment for a number of reasons: (1) people regularly attend dental appointments for check-ups when well, between 2015 and 2017 51% of the adult population in England reporting attending a dental check-up [77]; (2) there is an association between T2DM and poor dental health. Those with diabetes have a three-fold higher risk of periodontitis than those without, and periodontitis has also been shown to negatively affect glucose control [78]. Therefore those with periodontal conditions may represent a high-risk group for T2DM. A number of studies have assessed the feasibility of assessing T2DM risk in dental settings [79–81], and on average these show fairly high yields of undiagnosed T2DM and NDH. Risk assessment in dental settings is also acceptable to patients and dental teams [82].

Since robust risk identification methods exist for both use in primary care and the community, efforts should now focus on implementing these successfully in practice. This includes understanding how completers act on the information they are provided with, for example, when someone completes the Leicester self-assessment score using the Diabetes UK website, what will increase their chances of seeking diabetes testing when faced with a high-risk result? We also need to further understand how to ensure engagement with prevention programmes in those found with NDH and to encourage retention—as research shows engagement and retention are key to delaying/preventing T2DM in this group [62].

Summary

In summary, risk assessment for diabetes not only detects those with undiagnosed T2DM but also those with NDH. There is a strong evidence base that T2DM can be delayed or even prevented in those with NDH if they are enrolled onto a diabetes prevention programme. Picking up undiagnosed T2DM may reduce complications, but the evidence base for this is less compelling. Non-invasive risk assessments offer a cost-effective way to identify high-risk people for further testing. The two way relationship between diabetes and poor dental health highlights the potential for T2DM screening in dental settings.

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Risk Assessment in Periodontal Care: The Principles

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Contents

Introduction

Periodontal disease is a generic term that encompasses a wide spectrum of diseases and conditions, some of which are plaque-induced, whereas others arise independently of the dental plaque biofilm and may or may not be influenced by its accumulation at and below the gingival margin. The 2017 *World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions* is the most contemporary and evidence-based classification system, which attempts to combine research from both biological and clinical studies into a unified system that, for the first time, defines periodontal health, but also creates a staging and grading system for plaque-induced periodontitis. The system embeds risk assessment as a concept throughout and importantly recognises the patient as an individual, paving the way

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for precision approaches to personalised dental medicine [1–6]. Whilst the 2017 classification system embeds risk assessment in the historical assessment of a patient's disease experience to date, consideration also needs to be made of their current and future risk, which may differ from their historical risk due to treatment interventions and lifestyle changes. For this reason, a task force from the British Society of Periodontology (BSP) subsequently developed an implementation plan for the 2017 classification system for clinical practice, in which risk factor assessment, whilst pivotal to disease staging and grading (a measure of historical disease experience at a patient's first presentation), also informs future prognosis and management and thus sits alongside the diagnosis as an essential component of a holistic patient assessment and care pathway [7].

This chapter aims to outline the core principles of risk assessment for patients with inflammatory periodontal diseases, i.e. plaque-induced gingivitis and periodontitis. Gingivitis is a necessary pre-requisite for periodontitis [8] and also an important risk factor for the development of periodontitis; indeed managing gingivitis is now regarded as a key primary preventive strategy for periodontitis [9]. Specific risk assessment tools and their implementation in clinical periodontal care are discussed in Chap. 15 by Trombelli & Farina.

The Burden of Periodontitis and Individual Susceptibility

The global population is both growing and ageing, with 841 million people currently aged 60 years or over, and this is predicted to rise to 2 billion by 2050 [10]. Older people are retaining more teeth and for longer, increasing the prevalence of oral diseases like caries and periodontitis and their impact upon human suffering and the economy.

Inflammatory periodontitis, now referred to as simply "periodontitis" [1], is highly prevalent. Approximately 40–50% of the world's population have experienced or currently exhibit some periodontitis (stages I–IV) [11, 12] and with 11.2% estimated to suffer from severe periodontitis (stage III or IV), it is regarded as one of the most prevalent inflammatory diseases of humans. Indeed, the last 25 years has seen a 67% increase in *severe periodontitis*, the latter impacting 743 million people globally as the 6th most prevalent human disease [13].

Periodontitis is a major cause of adult tooth loss and impacts negatively on speech, nutrition, self-confidence/esteem and quality of life. Periodontitis is also independently associated with mortality and several non-communicable diseases of ageing [14].

Such statistics pose two major questions:

1. What is the burden of periodontitis to the global economy?
2. Why do some individuals suffer from this disease and others not?

The economic impact of oral disease is estimated at US$442 billion or 4.6% of global healthcare spend [15] and in the UK, £105 million is lost in sick days due to

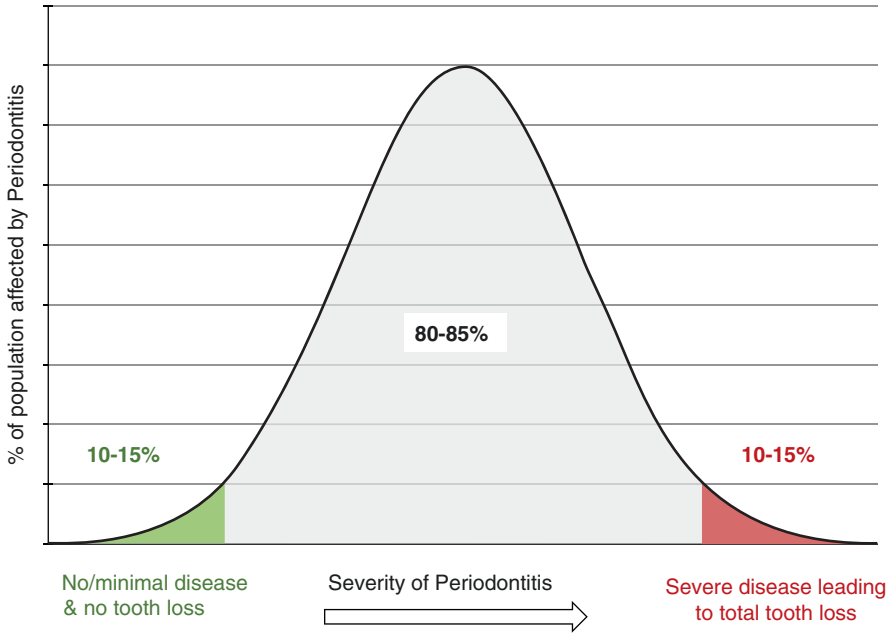

Fig. 1 Gaussian distribution of periodontitis based upon the natural history studies of Löe and colleagues [16]

oral diseases. Periodontitis peaks in incidence between 30 and 50 years of age, yet whilst 11–12% of people are highly susceptible to periodontitis, a proportion of the population also appear resistant to developing the disease [16], and the remainder vary in their susceptibility or "risk" along a normal Gaussian distribution curve (Fig. 1). The classical natural history study of Löe et al. in Sri Lanka [16] represented a powerful demonstration of variations in individual susceptibility to periodontitis. The study involved the examination of tea plantation workers in Sri Lanka aged 14–46 years of age in 1970, and repeat examinations were undertaken up to 1985. The tea workers were an ideal group to study the natural history of periodontitis as they did not undertake any conventional plaque-control measures and as a result demonstrated consistently heavy aggregates of plaque, calculus and tooth staining. Gingival inflammation was present at almost 100% of sites throughout their mouths, and yet their experience of periodontal bone loss, recession and tooth loss varied substantially. Rates of interproximal attachment loss and tooth loss clustered into three groups or sub-populations:

- High risk—or those with rapid progression of periodontal disease (8% of the population);
- Medium risk—or those with moderate progression (81%), and within which there was large variability in disease experience;

- Low risk—or those with no progression of their periodontitis beyond gingivitis (approximately 11%).

The mean attachment loss at 25 years of age in the rapidly progressing group was approximately 9 mm, whereas in the moderately progressing group it was 4 mm, and the disease resistant group experienced less than 1 mm loss of attachment. At the age of 45 years the attachment loss figures were 13 mm (high risk group) and 7 mm (moderate risk group). The researchers were also able to calculate annual rates of periodontal destruction, which were 0.1–1.0 mm in the rapidly progressing/high risk group, 0.05–0.5 mm in the medium progressing/risk group and 0.05–0.09 mm in the "no progression/risk" group. Due to their lifestyles, the tea workers were largely caries free and it was safe to assume that all teeth had been lost to periodontitis. Tooth loss was evident in 1970 at 20 years of age in the high risk group and increased up to the end of the study in 1985. Tooth loss at 35 years of age averaged 12 teeth, 20 teeth were missing at 40 years of age and by 45 years these people were edentulous. In the medium risk group, tooth loss started at 30 years of age and increased to 7 teeth at 40 years of age, whereas in the low (or "no") risk group, no teeth were lost. Several studies have followed this classical report and consistently reinforce the statistics illustrated in Fig. 1, of approximately 10–15% high risk, 10–15% disease resistant and 80–85% variable risk across a normal distribution curve.

Importantly, periodontitis is preventable, but dental care funding systems have encouraged a "repair when it is broken" philosophy by employing a "fee per item of treatment" payment process, rather than a "wellness" approach that is based upon risk assessment when people are "well" and disease free in order to inform and tailor prevention programmes according to risk. Employing a wellness approach results in the replacement of the traditional "treatment plan" with a "care plan", embedding prevention and risk factor control into the overall care of the individual. Public funding models for dental care are discussed in a subsequent chapter by Rooney; however, the recognition of individual susceptibility to disease, alongside the implementation of contemporary healthcare practice that embraces precision and/or personalised medicine, points towards an inevitable change in care pathways for managing periodontitis, with risk assessment forming a keystone underpinning such a transition.

Periodontitis as a Complex Disease

Periodontitis is an example of a complex disease, because unlike diseases such as, for example, tuberculosis (*Mycobacterium tuberculosis*), it does not have a single cause. Complex diseases have multiple component causes or "exposures", which are essentially synonymous with risk factors, and the presence of and interactions between those risk factors ultimately determines the expression of disease. Different individuals may possess or have been exposed to different component causes, and in different combinations, ultimately creating a large spectrum of disease that if expressed varies in severity (stage of disease) and rate of progression (grade of

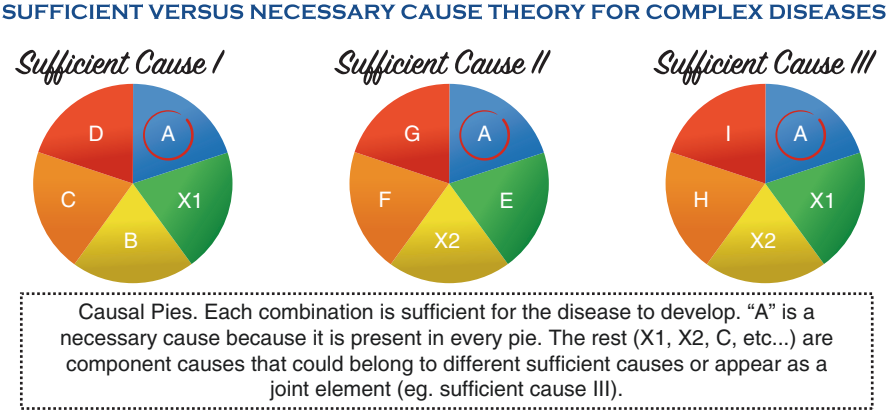

Fig. 2 Causal pie model for complex diseases [17]. Reproduced from Chapple et al. [18]

disease). Rothman [17] described a model for complex diseases referred to as "causal pies" (Fig. 2), in which disease only manifests if there is a "sufficient cause". Here, each component cause represents a slice in the pie and their combination ultimately drives whether a full pie (sufficient cause) develops or not. Slice "A" would represent plaque biofilm accumulation and appears in every complete pie because this is a necessary component cause for periodontitis to develop; on its own however it is insufficient for disease expression, otherwise 100% of humans would exhibit periodontitis. Plaque accumulation is the initiating factor, the key that starts the ignition to the car, whereas what drives the car is the accelerator pedal, i.e. the combination of component causes, principally the host immune-inflammatory response. In this driving analogy, if the ignition is turned off the car stops, thus if all plaque is completely removed the periodontitis will stop. However, whilst plaque is present (i.e. the ignition is turned on), those factors that control the accelerator are multiple and include features built into the engine (non-modifiable or effectively genetic factors), as well as modifiable or human factors controlled by the driver (e.g. how hard the accelerator pedal is pressed, how long for and how frequently). The host response has been estimated to explain 85% of periodontitis expression [19] and in itself is governed by systemic risk factors, some of which are modifiable lifestyle or behavioural factors, whereas others are not, such as genetic factors.

Pathogenesis of Periodontitis

Understanding the pathogenesis of a complex disease like periodontitis helps us to understand the role of various risk factors and human behaviours on the development of the disease. Figure 3 is a model that attempts to describe the transition from periodontal health to gingivitis and ultimately to periodontitis in those individuals at risk of periodontitis. Health is associated with a health promoting plaque biofilm, which can be maintained by regular and thorough disruption of the biofilm by tooth

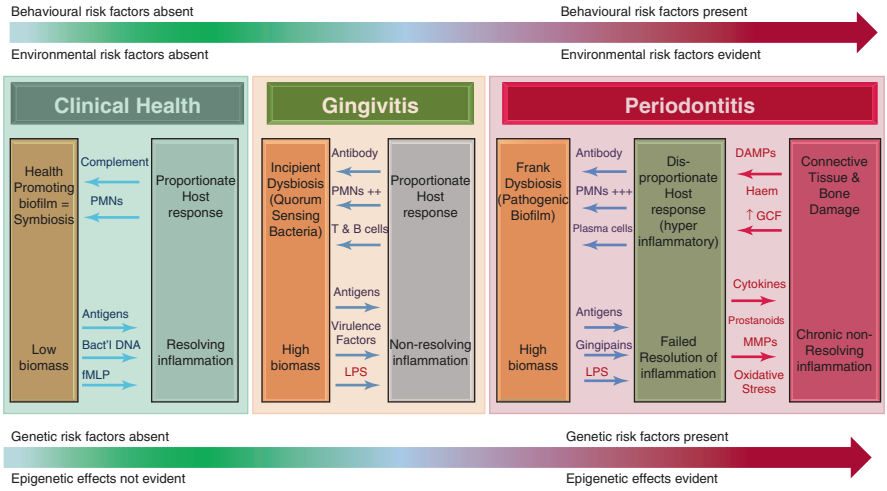

Fig. 3 Pathogenic model describing the relationship between periodontal health, gingivitis and periodontitis, and the role of various risk factors in disease expression. The biofilm initiates a host response, which in health is proportionate and symbiotic. In gingivitis continued biofilm accumulation ensures the inflammation fails to resolve and in high risk individuals a damaging inflammatory-immune response provides a source of nutrition for pathogenic bacteria and a dysbiosis ensues. Figure reproduced with permission from Wiley Publishers [20]

brushing, in particular interproximal brushing. This attains and maintains a low biomass and the immune surveillance mechanisms of the host remain proportionate and in balance with the biofilm. However, if the biofilm is allowed to accumulate, the inflammatory-immune response becomes more active in an attempt to control and contain the bacteria within the plaque biofilm. It is at this point that gingival inflammation increases and, depending upon individual susceptibility determined by specific risk exposures, the inflammation either succeeds in preventing the emergence of pathogenic bacteria (called "dysbiosis") or it fails to resolve and the inflammation becomes chronic [20]. Again, dependent upon an individual's risk profile, the chronic gingivitis may remain localised to the gingival tissues, or it may move apically and develop sufficient impetus to start destroying the alveolar bone and connective tissue attachment to the root surface. Under such circumstances, the supply of iron from haemoglobin is sufficient to allow pathogenic bacteria like *P. gingivalis* to thrive and release virulence factors, which further subvert and frustrate the host's immune-inflammatory response. The latter becomes exaggerated and poorly targeted and effectively destroys the periodontal tissues. The ultimate outcome is tooth loss, which one could argue was a natural defence reaction aimed at the body eliminating an infected structure (the tooth) that threatens the integrity of the internal organs of the body. Equally, one could argue that such an outcome was unnecessary and extreme and out with the risk to systemic health posed by the periodontal infection and associated inflammation. Whatever the reason, understanding how risk factors such as smoking and hyperglycaemia as component causes or slices

of the pie help to drive an exaggerated inflammatory response provides strategies for preventing periodontitis and/or managing the disease through the control of relevant risk factors.

Multi-level Risk Assessment

Risk factors for periodontitis have broadly been categorised into:

1. Systemic risk factors, also known as "modifying factors" [2]: such as smoking, hyperglycaemia, stress, nutrition (high in refined sugars and low in antioxidant micronutrients) or defects in neutrophil function;
2. Local risk factors, also known as "predisposing factors" [2]: largely factors that retain plaque at the gingival margin (e.g. ledges on restorations, anatomical factors or reduced saliva flow).

Systemic risk factors may be modifiable (e.g. smoking) or non-modifiable (e.g. genetic make-up and its impact upon immune cell function). Such factors essentially modify the immune-inflammatory response to a pathogenic biofilm. Local risk factors, also referred to as "predisposing factors" impact upon plaque accumulation and therefore the magnitude of biofilm challenge to the immune system. In this respect, both modifying and predisposing factors can impact upon disease expression directly, but they also interact with each other (Fig. 4).

This realisation led to the concept of continuous multi-level risk assessment [21], whereby risk assessment is performed in layers, dependent upon the level of insight into a patient's risk status. Figure 5 illustrates the concept. Here, systemic risk factors are assessed as part of the collection of a patient's medical, family and social history. When the mouth is examined, additional "mouth level" risk factors may become apparent, such as oral dryness or severe tooth imbrication which may inhibit plaque removal. More detailed examination during periodontal charting will reveal tooth-level risk factors which may require correction as part of the initial "cause related" phase of periodontal therapy. Finally, the completion of initial therapy

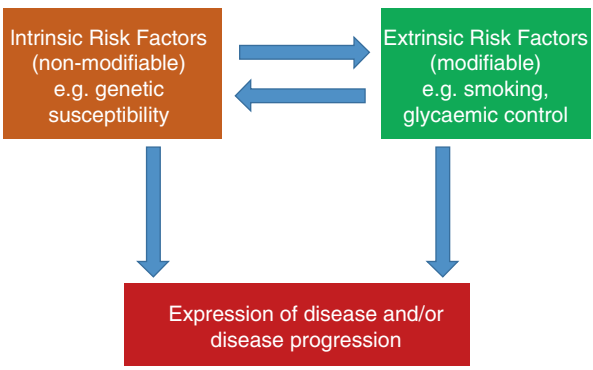

Fig. 4 Interaction of intrinsic and extrinsic risk factors in determining disease expression

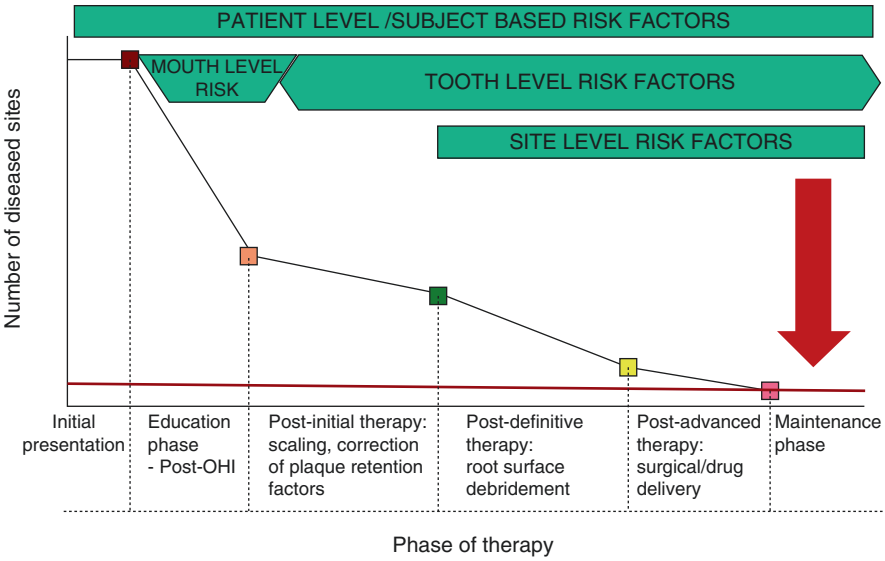

Fig. 5 Multi-level risk assessment mapped to stage of patient diagnosis, treatment and management. Initial assessment during history taking identifies patient-level risk factors via a medical and social history. As treatment commences, risk assessment can start to accommodate local risk factors at the whole mouth level; however, as treatment progresses, the knowledge of the clinician and patient becomes more granular and tooth and site-specific risk analysis can be performed. Based on concept of continuous multi-level risk assessment by Lang and Tonetti [21] and diagram with kind permission from Quintessence Publishing [22]

triggers detailed site-specific probing at about 3–6 months following completion of instrumentation, and this level of detail helps to identify non-responder sites, which may then reveal site-specific factors such as local anatomical grooves or plaque-retention areas, or areas that are missed in a patient's daily plaque-control regime, due to accessibility or incorrect technique.

Continuous risk assessment is a critically important concept, because risk changes throughout the life course and therefore a patient with no apparent disease experience may suffer a major life event, which contributes the final slice in the causal pie to create a "sufficient cause". This could involve, for example, the stress that results from the loss of a loved one or a divorce, which may directly impact upon known neuroendocrine pathways that drive inflammation or indirectly may result in the neglect of oral hygiene practices and consequently an increased microbial challenge. Kye and colleagues [23] pointed out that, unfortunately, traditional clinical parameters of periodontal disease such as bone and attachment loss are simply cumulative measures of past disease experience and do not necessarily help predict future disease activity or progression. Essentially, risk and disease are distinct concepts, because a high risk patient may have no disease at a given time point, but that does not mean that they will not develop disease when, for example, they age and immune senescence (a lower efficiency in immune function that arises with ageing) starts to emerge as a slice in the causal pie. Nevertheless, there is good

evidence that the cumulative disease experience of a patient at presentation (now determined by staging of periodontitis) is a strong indicator of its future risk of progression, in the absence of clinical interventions aimed at risk factor control, behaviour change and clinical treatment. For this reason, the British Society of Periodontology implementation strategy for the 2017 classification system recommends that periodontitis is staged and graded, prior to determining current disease status (via probing pocket depths and bleeding on probing) and careful documentation of current risk factors as a critical third stage [7]. Moreover, the diagnosis should be documented as a "diagnostic statement" that records the disease type and extent, its stage and grade and the current activity status (stable, remission or unstable), and immediately beneath the diagnosis, but part of the diagnostic statement is listed the relevant risk factors. For example, a diagnostic statement may appear as:

Diagnosis: Localised periodontitis, stage III, grade B, currently stable

Risk Status and Risk Factors: High risk; smoking >10 cigarettes/day, high refined sugar intake.

The risk assessment, whether that be performed using an anecdotal "high", "medium", "low" annotation, or more accurately using an objective computer-based tool (see Chap. 14), is thus embedded in the diagnostic statement.

Risk Assessment for Behaviour Change

A frequently overlooked and understated purpose to formal risk assessment is its power as a biofeedback tool to stimulate behaviour change. This was demonstrated in 2005 by Barnfather and colleagues, who conducted a randomised controlled clinical trial in a general dental practice, to determine the impact of feeding back personal results from a near patient saliva test that measured levels of saliva cotinine (nicotine exposure) as part of a brief smoking cessation intervention [24]. The change in saliva colour (grades of yellow to brown) indicated the amount of nicotine exposure and volunteers who were shown their results demonstrated a higher smoking quit rate (23%) versus controls who had the standard brief intervention alone (7%; $P = 0.039$). Overall tobacco use also decreased (68% cases v 28% controls; $P < 0.001$), demonstrating a general as well as an oral health impact from a risk-based biofeedback approach.

Asimakopoulou and colleagues examined the inclusion of personalised risk scores as personalised biofeedback as part of a behaviour change intervention in periodontitis patients, during a randomised controlled trial that evaluated the impact of using individualised risk communication using a computer-based system (PreViser) versus a routine consultation on patients' cognitions and emotions about periodontal disease. They demonstrated that presenting patients with their risk scores from the computer-based tool resulted in significantly improved psychological outcomes, such as taking their disease more seriously, understanding their susceptibility and experiencing greater self-efficacy (belief in being able to positively impact their disease course). Moreover, they had more positive thoughts about their treatment and their intentions to adhere to the prescribed regimes. The authors

concluded that individualised periodontal risk communication has a positive impact on underpinning variables for periodontal adherence [25].

Positively impacting psychological beliefs and intentions is one thing; however, its translation into improvements in clinical measures of disease is more challenging to demonstrate. The same authors however in a further randomised controlled trial in a general dental practice setting demonstrated that using the same computer-based system for biofeedback and motivation, in the absence of traditional treatment interventions for periodontitis, significantly improved gingival bleeding scores, plaque scores and interdental cleaning habits at 3 months [26]. This was the first time that the use of individualised risk score feedback had been demonstrated to be capable of positively influencing clinical periodontal outcomes in the absence of professional instrumentation and validates the importance of patient-led self-care in improving periodontal outcomes. Indeed, as illustrated in Fig. 5, numerous studies have demonstrated that the greatest reduction in number of sites with active disease (pocket depth and bleeding on probing) is achieved by those patients who conscientiously follow the educational/behaviour change phase of management in which oral hygiene and its purpose and benefits are taught.

Recommendations

Given the ageing global population and increased rates of tooth retention and periodontitis, it is time that practitioners ceased paying "lip service" to risk assessment and started to embed it at the centre of individualised patient care plans. Whilst historical disease experience reflects a patient's previous risk for future disease, it is necessary to determine current risk status and to document current risk factors and behaviours as part of a diagnostic statement.

Conclusions

In conclusion, there is now sufficient evidence that risk assessment should form a critical component of patient-centred periodontal care. Personalised biofeedback of risk scores and factors, as part of a behaviour change regime, appears not only effective in improving patients' psychological approach to and beliefs in their ability to manage their periodontal disease, but also appears to lead to improvements in clinical measures of periodontitis, even in the absence of physical root surface instrumentation. The wellness paradigm appears to offer a far more cost-effective approach embedded within a preventive care programme than traditional repair models of care provision.

Competing Interests

Professor Chapple acts as a consultant to Oral Health Innovations Ltd., who hold the licence for PreViser and DEPPA risk assessment technology in the UK and Ireland.

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Caries Risk Assessment

Svante Twetman and Avijit Banerjee

Contents

Introduction

In spite of decades of a significant decline in incidence, dental caries remains a global public health burden with statistics indicating approximately 44% of all people worldwide suffering from untreated caries in their primary and/or permanent teeth [1, 2]. It is therefore obvious that the focus and efforts on effective caries prevention and minimally invasive operative carious lesion management must be intensified. In this context, a caries risk/susceptibility assessment (CRA) of

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populations, groups and individuals is thought to be a cornerstone of preventive dentistry in order to allocate time and resources to those with the greatest need. Even though CRA is implicit in the daily delivery of good quality oral health care by many oral health care practitioners, questions still remain unanswered as to whether or not formalized, documented evidence-based CRA is possible, feasible or even desired in the environment of general oral healthcare practice. The aim of this chapter is to summarize the science and quality of evidence that exists for CRA and discuss some of the complexities associated with its use.

Caries Risk Assessment

Caries risk assessment (CRA) can be defined as "the clinical process of establishing the probability of an individual patient to develop carious lesions over a certain period of time or the likelihood that there will be a change in size or activity of lesions already present" [3, 4]. This sounds fairly simple and straightforward in principle, but needs further elaboration:

1. First of all, dental caries is classified by the World Health Organization (WHO) as a plaque (biofilm)-mediated, non-communicable disease (NCD), with a complex network of biological, genetic, behavioural, socioeconomic and lifestyle-related risk factors in common with other NCDs, for example, obesity and diabetes [2]. This means that one cannot expect one single risk factor alone, such as bacterial load, sugar intake or salivary secretion rate, to be individually useful in order to predict future caries incidence.
2. The second issue is "probability over a certain period of time". To establish this probability, any risk factor, or combination of risk factors, must be tested and validated in prospective trials in which defined cut-off points, or threshold values, are related to the true caries increment. The calculated probability for developing caries is often expressed in terms of sensitivity, specificity, receiver operating characteristics and/or area under the curve, terms that may not be easily digested by the clinician, and even less so by the patient. It should also be observed that the results are only valid for the particular age group and population in which the study was conducted. This means that the external validity may be limited and that the findings can seldom be generalized meaningfully to all patient groups or populations.
3. Prospective risk assessment clinical trials are associated with an ethical dilemma since they must be performed without any form of intervention in order to truly reflect their predictive ability. For example, if a targeted preventive treatment based on the individual caries risk proved to be highly effective against caries process progression, the baseline "prediction" of caries risk would turn out to be incorrect. One can therefore say that the academic research discipline of caries prediction is far from the caries risk assessment procedure that takes place in the oral healthcare practitioners' clinic. The latter is a subjective process, often intuitive, in which the clinician weighs factors for and against caries tailored to the

individual, to describe the risk of future disease in categories, such as low, moderate or high caries risk. This assessment is then linked ideally to informed and appropriate care planning decisions on preventive and restorative care, as well as the periodicity of future recall consultations.

4. The fourth issue deals with the methodology of caries detection. Caries development is not an "on/off" process but a continuum with a slow progression rate in most individuals [2]. The traditional DMFS/dmfs at a tooth/surface level is thereby far too blunt an instrument, as it does not include early initial lesions or distinguish between the active and inactive stages of disease. It is therefore necessary to adopt a system that enables early detection, activity scoring and staging of lesions. One such example is the International Caries Detection and Assessment System (ICDAS) that can be linked to a management protocol, International Caries Classification and Management System (ICCMS). The use of such, or other similar systems, offers a harmonized approach for education, epidemiology and oral healthcare practice that may facilitate the understanding of primary, secondary and tertiary prevention to promote oral health [2, 5].
5. A final point is regarding the common terminology used. It may be considered a semantic point, but all dentate patients are subjected equally to the associated risk factors for caries process instigation—a suitable tooth surface, a dysbiotic cariogenic stagnating plaque biofilm containing a diverse microbiota working collectively to drive the caries process when conditions allow, a carbohydrate source for bacterial metabolism and time. However, not all people are equally *susceptible* to the caries process and it is these other factors highlighted in this chapter that the oral healthcare workforce attempt to elucidate and effectively manage to reduce future caries risk in their patients, on a daily basis.

Methods and Models for CRA

Most oral healthcare professionals seem to perform an informal and intuitive caries risk assessment ("educated best guess") when taking a case history and examining their patients. The most commonly used single variables for individuals are past caries experience and level of oral hygiene, while the level of fluoride exposure seems to be considered less important [6]. The CRA methods that combine several factors can be divided into reasoning-based checklists or algorithm-based computer models. The technologies used most commonly are shown in Table 1 [7–12]. Most checklists, as well as software programs, are available for downloading from the internet free of charge. The reasoning-based models are based on a number of age-related background factors (biological, behavioural, socioeconomic) with demonstrated associations with caries activity. These forms can be completed by oral healthcare professionals together with their patients, or parents/custodians, and the outcome is often categorized into 2–5 risk groups, ranging from low to very high (extreme) caries risk [13]. The computer-based models work in the same way but the input factors are pre-weighted against each other to establish a caries risk profile and/or classify the risk for future disease. These algorithm models can be used

Table 1 Examples of caries risk assessment models intended for clinical practice

Model	Factors[a]	Method	Endpoint/categories
Reasoning-based checklist models			
CAT	12	Manual charting	Low to high caries risk/3–5 levels[b]
CAMBRA[c]	14	Manual charting	Low to extreme caries risk/4 levels
DCRAM[d]	9	Manual charting	Caries risk, yes or no/2 levels
Computer-based models			
Cariogram	9	Software	% chance of caries/5 levels
NUS-CRA[b]	11	Software	% chance of caries/5 levels
PreViser (OHIS)	8–18[b]	On-line (cloud)	Tooth decay risk score/5 levels

CAT Caries Risk Assessment Tool [7], *CAMBRA* Caries Management by Risk Assessment [8], *DCRAM* Dundee Caries Risk Assessment Model [9], Cariogram [10], *NUS-CRA* National University of Singapore Caries Risk Assessment [11], *PreViser OHIS* Oral Health Information Suite [12]

[a]Most comprehensive model; reduced models are available for screening purpose

[b]Depending on age

[c]CAMBRA is recently available as an algorithm-driven app for mobile devices called MyCAMBRA

[d]Validated for young children only

interactively to communicate with the patient in order to increase case acceptance and adherence to subsequent care and behaviour modification. There are manual charts/checklists as well as computer-based models adapted for infants, children and adults but none is tailored specifically for root caries risk [14]. The Cariogram™ has indeed been investigated for root caries prediction but without any modifications for the specific biological, clinical and behaviour-associated conditions that are related to lesion development on root surfaces [15].

Accuracy of CRA

As stated previously, the accuracy of any caries risk assessment can only be established at a group level following prospective trials without targeted interventions. A troublesome concern is that few CRA models are validated longitudinally and a systematic review, based on 18 publications, was unable to identify studies with low risk of bias, without methodological limitations concerning study design, test technology and/or reporting [16]. Consequently, the quality of evidence on the validity of the methods used for caries risk assessment must be graded as low. Some general conclusions can be drawn however. In general, multivariate models seem to perform better than single predictors and the accuracy is higher among pre-school children compared to later in life [4, 17]. Single predictors, or even prediction models, rarely exhibit high sensitivity and specificity values (close to 1.0), so using any single factor individually is not appropriate. The precision and accuracy is expressed commonly as the sum of sensitivity and specificity and may be graded into three levels: good $\geq$ 1.5; limited = 1.3–1.5; poor = <1.3 [17], as illustrated in Fig. 1. High sensitivity is desirable for severe diseases or malignant conditions where an overlooked diagnosis may lead to under-treatment with significant, even fatal consequences. For dental caries, a high specificity may be preferred in order to avoid

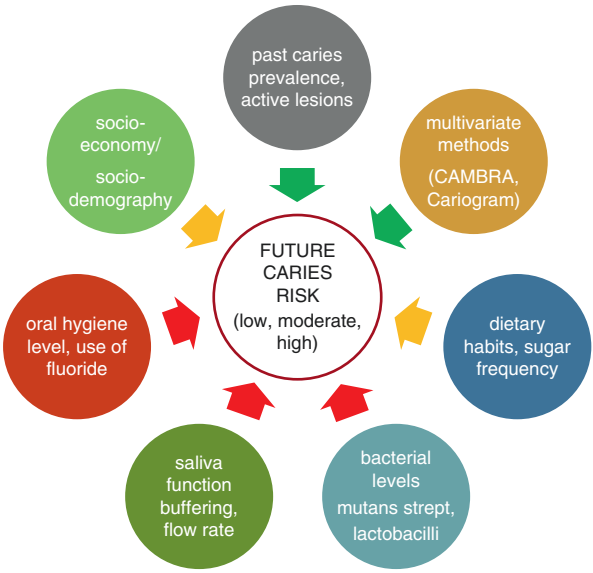

Fig. 1 Accuracy of various methods to predict future caries in children. Based on a systematic review [17], green arrows for good accuracy; amber coloured arrows for limited accuracy; and red arrows for poor accuracy

over-treatment. Another way to express CRA accuracy in a clinically meaningful way is using likelihood ratios (LR+/−). The positive value describes how many times more likely it is for a person with a defined condition ("positive test") to develop caries in comparison with a person without the same condition.

Single Predictors

From the literature it is clear that the most powerful single factor in caries prediction for all age groups is "previous caries experience". The sensitivity ranges from 0.21 to 0.94 and the specificity from 0.20 to 1.0 [8, 16, 17]. In many studies, the estimate of specificity was found to be higher than sensitivity indicating that it may be possible to identify individuals with low risk of developing caries with more certainty. The accuracy was regarded as "good" for pre-school children and "limited" for school children, while for adults there remains a knowledge gap [17]. The pooled positive likelihood for a pre-school child with caries in the primary dentition to have caries in the permanent dentition was 3.2 times higher compared with those that had caries-free primary teeth [16]. It may however be argued that using the past presence of carious lesions as a single predictor is nonsense in conducting a future risk assessment since the patient is already proven to be susceptible. Furthermore, the identification of multiple risk and protective factors involved in the disease process will actually guide the clinician in tailoring an effective care plan and help the patient understand what needs to be done [18].

Another frequently used single predictor for caries is the mutans streptococci count in plaque or saliva, assessed commonly with simple chair-side test kits with a critical threshold value of $\geq 10^5$ colony forming units (CFU) per mL. The theory is

that elevated bacterial numbers are a "biomarker" for the low-pH environment that favours growth of aciduric microorganisms involved in carious lesion formation. However, the tests display in general a low sensitivity and a relatively high specificity, resulting in numerous false negative diagnoses [17]. The pooled positive likelihood ratio (LR+) for mutans streptococci counts exceeding 10^5 CFU/mL has been estimated at 4.0 [16]. The use of salivary lactobacilli counts, salivary flow rate and buffering capacity for caries prediction have been proven to be of limited value as single variables [17]. Likewise, the accuracy of socio-demographic variables, such as education level, annual income and immigration status, when used as a single predictor, is low.

Multivariate Models

Multivariate caries prediction models perform better than single predictors but it must be stressed that these models, with few exceptions, are validated among children and adolescents only [17]. For pre-school and school children, the adoption of a comprehensive model such as NUS-CRA (National University of Singapore Caries Risk Assessment) and Cariogram™ has obtained pooled sensitivity and specificity values >0.80 each (combined >1.60), but the accuracy and precision appear to decrease during adolescence [4, 11, 17]. Using the Cariogram™ CRA tool, the likelihood for a child assessed with a high risk to develop caries is approximately five times higher than for those assessed with low risk. The use of CAMBRA for children aged under 6 years has also yielded high sensitivity (84%) but lower (55%) specificity values [19]. In the pre-school CAMBRA model, the items "existing cavities", "dental plaque" and "frequent snacking" were associated independently with future caries. A side-by-side comparison of four CRA systems has been carried out among kindergarten children in Hong Kong [11]. The results indicated that the computer-based models (NUS-CRA, Cariogram™) had a higher accuracy than those based on manual checklists (CAT—Caries Risk Assessment Tool, American Academy of Paediatric Dentistry and CAMBRA—Caries Management by Risk Assessment). Again, the manual charts (CAT and CAMBRA) displayed an excellent sensitivity (>93%) but suffered from impaired specificity, 5% and 44%, respectively.

For coronal caries in adults, the CAMBRA model based on a combination of indicators of disease, risk/pathological factors and protective factors has been validated in a university setting [8, 20]. The model is described as an excellent tool to distinguish between low/moderate caries risk vs. high/extreme caries risk but the traditional measures of accuracy are not reported. It was shown that 24% of the patients in the low risk group had new cavities at follow-up after 18 months and the corresponding figure in the high risk and extreme risk groups was 70% and 88%, respectively. The validations were however carried out in an educational environment with selected patients and the project suffered from a large attrition rate, which may challenge the external validity for its use in real-life practice. An important finding was that the "unsophisticated" clinical variables such as recent disease

history, frequent snacking, inadequate oral hygiene practices and reduced salivary flow differed sharply between the risk categories [8]. This certainly suggests that CRA can easily be adopted into everyday general oral healthcare practice at low costs without advanced technologies.

Clinical Applications of CRA

One may be intrigued by data from practice-based research indicating that 30–40% of the general dentists do not carry out any formal caries risk assessment of their child or adult patients [21, 22]. However, the important question is if there is evidence that caries risk assessment actually results in improved care for patients? Field trials have indicated that assigned risk categories are not always followed by the appropriate patient-focused preventive actions [22]. Even worse, findings from a Swedish study showed that those considered at low risk were provided with greater preventive measures than those with higher risk [23]. This is most likely not due to ignorance among the oral healthcare professionals, but may be attributed to the "inverse care law". A well-known situation in healthcare is that patients with the highest need tend to be the least likely to attend for treatment and so underutilize any preventive care that might be offered [24]. There is however evidence that the adoption of the CAMBRA model in routine practice can increase the use of preventive measures and reduce the caries increment by up to 20% in adults [20, 25]. In Australia, a long-term evaluation of a trial with a caries management system based on three risk categories has shown that adult patients continued to benefit from a reduced risk of caries and therefore experienced lower needs for restorative treatment [26]. Likewise, the participation in regular preventive programs based on caries risk levels has been shown to reduce the onset of new lesions in Japanese adults [27]. It has also been demonstrated that patients can be classified at a lower risk level after being provided with tailored risk-based preventive measures [28]. As the level of evidence concerning patients' perception and benefits of CRA still must be graded as low, there is a need for more research on the value of caries risk assessment for the various stakeholders including patients and dental practitioners.

Discussion

The ideal method for caries risk assessment in everyday practice should be quick, simple, inexpensive, reliable and easy to understand for both the professional and patient or their parents alike. One problem is that there is no consensus, or definition, of moderate/high caries risk categories. This makes comparisons between different studies difficult and the term "high risk groups" problematic. If the proportion of high risk patients in a population is high and close to 50%, the CRA procedure becomes less meaningful. The fact that existing CRA methods are unable to predict the future disease occurrence with perfect accuracy is a shared dilemma with many other diagnostic/predictive tools used in general healthcare and it is unlikely that

future research will find the perfect model. The shortcomings are certainly not an excuse to skip risk assessment but it is important that oral healthcare providers understand and consider the strengths and limitations of such CRAs. A low validity of a particular model/tool leads inevitably to misclassifications; patients with increased risk are not being identified, while others are falsely identified as being at risk. A high number of false-positive assessments may drive over-treatment in contrast to false-negatives that may result in treatment neglect. Since the accuracy of CRA in most prospective studies ranges between 65% and 85%, it is clear that the use of any multivariate models is better than "guessing" (50%). Although there is no evidence to favour one model or technology over another, the clinician should select and stick to one of them, calibrate and carry out CRA periodically throughout life [4]. This is important because susceptibility can vary with time; for example, a study in school children has shown that 50% change their caries risk category over a 2-year period, for better or for worse [29]. Methods that rely on reasoning-based checklists or computer-based algorithms may not necessarily improve accuracy but their routine use adds to treatment consistency, transparency and patient motivation. The patients' appreciation and understanding that CRA is guiding the individualized care decisions and recall intervals is likely to increase both their motivation to maintain their personal oral health and adhere to behaviour change advice. Indeed, it has been shown that improved CRA documentation and communication can increase patients' adherence to their individualized care protocols [30].

Although risk assessment is a recommended procedure in contemporary caries management, some negative aspects cannot be ignored. Apart from obvious misclassifications leading to suboptimal care, little is known about the patients' reactions of being stigmatized as a "high risk individual". One qualitative study among adolescents has displayed three emotional subcategories [31]:

1. a positive attitude and clear self-confidence that improved health will be achieved
2. a passive attitude that everything will be all right and fixed by the dentist
3. a negative attitude characterized by frustration and a tendency to give up

The latter subcategory must not be overlooked or underestimated. To be pointed out as "almost sick", or a vulnerable person, may affect self-confidence negatively and increase feelings of hopelessness. It is therefore important that oral healthcare providers are able to assist those persons by offering empowerment and supporting self-efficacy in terms of robust, reinforced advice. Indeed, an interesting concept that could be instigated locally at practice level would be to have a patient-facing risk assessment scale with multiple, narrower bands/levels (say, 10–100). The local, regularly case-calibrated team can then engage with their patients and as the patient's behaviour changes, they can be more easily and regularly "rewarded" by movement up and down the scale. This dynamic feedback would be akin to a league table strategy which might engage the competitive aspects of different patients' personalities while benefiting ultimately their oral health. With the advent/development of intra-oral monitoring technology in the future, this information could be updated with online submitted clinical data to help provide a clearer longitudinal

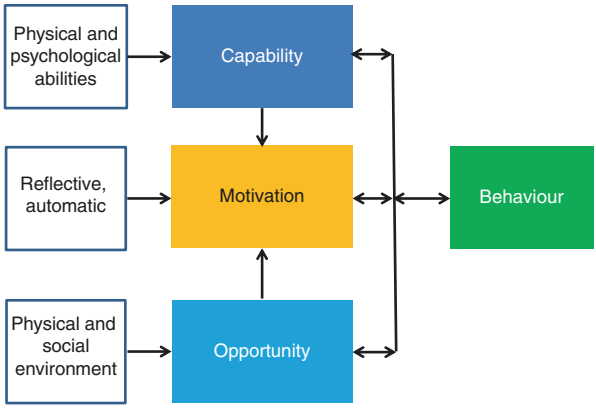

Fig. 2 The COM-B system illustrating that motivation alone does not necessarily induce behaviour change after caries risk assessment. Capability and opportunities may enhance and facilitate changes to a more favourable oral health behaviour. Adapted from Michie et al. [32]

"real-time" susceptibility trend for patients and their oral healthcare providers. An appraisal of behaviour management approaches highlights the effectiveness of the COM-B [32] model ("Capability", "Opportunity", "Motivation" linking to "Behaviour") as illustrated in Fig. 2. Here, the oral healthcare team members help to engage with and enable capabilities, opportunities and motivation together within patients to cope with and alter their caries risk behaviours and emotional maturity [33]. A similar alienation in adulthood is to our knowledge not documented, but cannot be excluded. Another possible, but less likely, negative consequence of caries risk assessment on population and community levels is displacement. Such effects may occur when high risk patients are crowding out regular patients with less extensive needs due to limited resources. This may also appear the other way around in dentistry based on capitation payment models [34]. Patients assessed with low risk are more likely to select a fixed yearly fee plan than patients assessed as being high risk. In the long run, this can contribute to widening the inequality gap in oral health within a society. However, in summary, it seems clear that the beneficial effects of caries risk assessment outweigh the possible disadvantages.

Recommendations

The GRADE system (Grading of Recommendations, Assessment, Development and Evaluation) [35] offers two grades of recommendations; when the desirable effects of an action clearly outweigh the undesirable effects, or clearly does not, a "strong" recommendation can be given. However, when the balance of desirable versus undesirable effects is less certain, because of low-quality evidence or because evidence suggests that desirable and undesirable effects are more evenly balanced, the recommendation is described as "weak". The current quality of evidence concerning CRA is summarized in Table 2. There is a paucity of clinical trials with low risk of bias, calling for improvements and standardizations in study design, performance and reporting of outcomes. In particular, the use of caries risk assessments in adults and older adults remains under-investigated as well as studies on cost

Table 2 Clinical recommendations for caries risk assessment in general dental practice

Statement	Quality of evidence[a]	Recommendation
Desirable effects of CRA outweigh the undesirable	Moderate	Strong
CRA should be carried out at the first visit	Very low	Weak
Reassessments should be done periodically throughout life	Very low	Weak
Multivariate models more accurate than single predictors	Low	Weak
Manual checklists and computer-based algorithms improve accuracy	Low	Weak
CRA results in tailored prevention and reduced caries increment	Low	Weak

[a]According to GRADE [35]

effectiveness and cost utility. This is of equal importance in countries or societies where oral healthcare care is funded through a state- or private-based capitation systems or traditional fee-per-item services. Further qualitative studies are also needed to explore how much the patients value a caries risk assessment and how much they are ready to pay for it. A large step towards better implementation of CRA in daily practice would be to integrate the process in the various electronic patient record systems that are available today.

Conclusions

Although the quality of evidence to support caries risk assessment as an integrated component in effective caries management is low, it is concluded that the benefits outweigh harm. There is currently no single tool or method with perfect accuracy, but multivariate models in general perform better than single predictors, with an accuracy exceeding 80%. The ability to predict future caries susceptibility is greater among pre-school and school children when compared to older patients. Models for root caries prediction are lacking. Emerging evidence suggest that caries risk assessment in general practice assists clinical decision-making and increases patients' understanding and adherence to preventive care regimes.

Competing Interests The authors have no conflicts of interests to declare.

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Risk Assessment: Tooth Wear

David Bartlett and Saoirse O'Toole

Contents

Risk Assessing Each Component of Tooth Wear

Tooth wear is a multifactorial, complex process involving erosion, attrition, abrasion [1]. Erosion is defined as the chemical dissolution of hard tissues due to acids of non-bacterial origin. Attrition is defined as the mechanical removal of dental hard tissues through tooth-to-tooth contact and abrasion is defined as the mechanical removal of dental hard tissues with anything else. Erosive tooth wear has recently

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been defined as the chemical–mechanical process resulting in a cumulative loss of hard dental tissue not caused by bacteria [1]. It is a relatively new term used to highlight that an acidic component often underlies severe tooth wear and knowledge of this is essential to proper risk assessment of tooth wear.

Epidemiological evidence suggests that the prevalence of erosive tooth wear is increasing, particularly in younger age groups [2]. The reason for this is unknown but the change in snacking habits of the population, anxiety and stress levels and the prevalence of gastro-oesophageal reflux and vomiting eating disorders may be influencing factors [3]. A trans-European study on over 3000 young adults aged 18–35 years observed the prevalence of moderate to severe wear to range from 17 to 54%. The outcome of this research suggests that erosive tooth wear is common [3].

Risk Assessing Erosion

A patient with erosive tooth wear may be unaware of their condition, particularly in the early stages. Initially, erosion produces an altered surface texture on the tooth as the higher mineral content of enamel makes it more susceptible to acids. Incisors lose their perikymata and molars the defined morphology of their ridges and the cusp tips become rounded. As wear progresses, distinct defects may be observed. On the occlusal surfaces of molars, this results in small cupped or cratered lesions often where the cusp tips used to be. On the buccal surfaces, the smooth surface may become slightly uneven. Ridges or grooves may start to form and there may be a distinct step in the hard tissue adjacent to the gingival margin, possibly from the protection of the crevicular fluid. As the wear progresses, these defects may grow in size or link up until they affect the entire surface of the tooth and the crown shortens. Once this occurs the changes become visible to patients and they complain of thinning, shorter and "translucent" incisal edges.

Reflux Related Erosive Tooth Wear

The most common medical conditions resulting in erosive tooth wear from stomach acid are gastro-oesophageal reflux disease (GORD) and vomiting-associated eating disorders affecting roughly 10% [4] and <1–2% [5] of the global population, respectively. Both conditions are the most common causes of gastric-related erosive tooth wear. However, rumination habits, whereby the patient voluntarily regurgitates their food in order to rechew can exist in both members of the general public and those with learning difficulties but is comparatively rare. Pregnancy may cause vomiting, particularly during the first trimester and hyperemesis gravidarum is a condition, affecting 0.3–2% of the population whereby vomiting starts early in the pregnancy and may last the duration of the entire pregnancy [6]. In addition, pregnant women are more prone to reflux.

Certain medications may have a central emetic effect such as chemotherapeutic drugs, opioids, digitalis and some oestrogens. Alcoholism may also predispose to

both vomiting and reflux disease. With all these conditions, short periods of acid exposure are unlikely to result in severe pathological tooth wear. However, if they become chronic and uncontrolled, severe erosive tooth wear may result.

Risk Assessing Gastro-Oesophageal Reflux

A certain degree of post-prandial reflux is normal and is physiologically managed by peristaltic action and neutralisation of gastric acids by saliva and swallowing. The pathological state, gastro-oesophageal reflux disease or GORD, is classified as two or more heartburn episodes per week which adversely affect an individual's well-being [7]. The duration of GORD, the frequency of episodes, intraoral signs and whether the reflux occurs during the day or the evening will impact on the severity of erosive tooth wear. Furthermore, reflux passing into the mouth is relatively rare as most suffer symptoms restricted to the distal oesophagus. When reaching the mouth, the potential exists for erosion of the teeth and this can be a sign to assist with the diagnosis of reflux disease. The reflux disease questionnaire (Fig. 1) has been shown to have a sensitivity of 67% and may be useful as a primary care screening tool.

A risk assessment for erosive tooth wear should identify the frequency of reflux, either on a weekly basis (high risk), less than weekly basis (medium risk) or whether it is controlled and there are signs of acid inactivity such as staining on the teeth (low risk). Then assess if the refluxate reaches the mouth. An absence of any symptoms does not necessarily mean that reflux is not involved with the condition. Silent reflux is recognised as a chronic long-term condition which is symptomless but the effects of the gastric acid reflux remain present.

Risk Assessing a Vomiting Eating Disorder

Eating disorders with an ongoing vomiting component can be challenging to assess during a patient history, given the unfortunate stigma attached to mental health issues. If a patient feels ready, an honest conversation about activity of the condition and how they are managing their recovery should provide enough information for an erosive tooth wear risk assessment. Emphasising a supporting role during active disease is part of overall care. Elucidating information regarding the frequency of vomiting, their oral hygiene procedures before and after vomiting and their diet will facilitate practical risk management.

Difficulty arises if an eating disorder is suspected but not diagnosed, particularly as those with eating disorders may consume excessive amounts of diet drinks during bulimic phases, confounding the diagnosis between extrinsic and intrinsic erosive tooth wear. The most commonly used screening tool in primary medical care is a five-part questionnaire with the acronym SCOFF (Fig. 2, [8]). Although this questionnaire has been criticised for having both low sensitivity and specificity in a general population, it may be useful when attempting to broach the subject with an

Reflux Disease Questionnaire

*Please answer each question by ticking **one** box per row.*

1. Thinking about your symptoms over the past 7 days, how often did you have the following?

	Did not have	Less than 1 day a week	1 day a week	2-3 days a week	4-6 days a week	Daily
a. A burning feeling behind your breastbone	☐	☐	☐	☐	☐	☐
b. Pain behind your breastbone	☐	☐	☐	☐	☐	☐
c. A burning feeling in the centre of the upper stomach	☐	☐	☐	☐	☐	☐
d. A pain in the centre of the upper stomach	☐	☐	☐	☐	☐	☐
e. An acid taste in your mouth	☐	☐	☐	☐	☐	☐
f. Unpleasant movement of material upwards from the stomach	☐	☐	☐	☐	☐	☐

2. Thinking about your symptoms over the past 7 days, how would you rate the following?

	Did not have	Very mild	Mild	Moderate	Moderately severe	Severe
a. A burning feeling behind your breastbone	☐	☐	☐	☐	☐	☐
b. Pain behind your breastbone	☐	☐	☐	☐	☐	☐
c. A burning feeling in the centre of the upper stomach	☐	☐	☐	☐	☐	☐
d. A pain in the centre of the upper stomach	☐	☐	☐	☐	☐	☐
e. An acid taste in your mouth	☐	☐	☐	☐	☐	☐
f. Unpleasant movement of material upwards from the stomach	☐	☐	☐	☐	☐	☐

Fig. 1 The reflux disease questionnaire or RDQ which is one of the screening tools available to general medical practitioners

The SCOFF questions*

Do you make yourself **S**ick because you feel uncomfortably full?

Do you worry you have lost **C**ontrol over how much you eat?

Have you recently lost more than **O**ne stone in a 3 month period?

Do you believe yourself to be **F**at when others say you are too thin?

Would you say that **F**ood dominates your life?

*One point for every "yes"; a score of ≥ 2 indicates a likely case of anorexia nervosa or bulimia

Fig. 2 Scoff questionnaire [8]: a potential diagnostic aid for the screening of eating disorders

individual patient. But sensitivity with questions is important as this group of patients have needs which are beyond the scope of dentists. If there are suspicions that an eating disorder is present, the dentist should discuss it with the patient and only if they agree, refer to a medical practitioner.

If the condition is reflux related and the sufferer has symptoms that interfere with their quality of life, it is appropriate to refer to their general medical practitioner or a gastro-enterologist. Management involves life style changes and anti-reflux medication that reduces the acidic nature of the refluxate. Reflux related causes of erosive tooth wear are relatively uncommon but are often responsible for the most severe examples.

Clinically an extra-oral examination may reveal enlarged parotid glands when a disease is very active. There may be soft tissue scarring on the backs of fingers where vomiting is forced with the hands. Intraorally, there may be signs of bruising or soft tissue damage on the hard and soft palate. Erosive tooth wear may be visible on the palatal surfaces of the maxillary arch but may also affect all surfaces or not at all. This is particularly true if the patient had a habit of aggressive brushing after a vomiting episode, where all surfaces will be at increased risk of wear.

If the patient is suffering from an uncontrolled vomiting eating disorder which is active on a weekly basis, they are at higher risk of erosive tooth wear progression. If it is relatively well managed and active less than weekly, they can be classified as medium risk. If the disease is controlled and the worn teeth become stained, it may indicate the condition is controlled or inactive and so can be classified as low risk.

Risk Assessing an Erosive Diet

Dietary erosive tooth wear risk factors are becoming increasingly better characterised. The frequency of dietary acid intake has been shown as the most significant predictor of dietary erosive wear [9, 10]. A recent case–control study reported

that less than one dietary acid intake per day was associated with a negligible risk of wear [9]. However, risk increased when dietary acids were consumed more than once daily. Three daily intakes or greater was associated with a 13-fold increase in the risk of severe erosive tooth wear [9]. Common diet foods include fruits, fizzy drinks (excluding plain sparkling water), energy drinks, juices and smoothies. However, it also includes lesser known dietary acids such as fruit teas, fruit additions or flavourings in drinks, e.g., a slice of lemon or cordial/squash, sports drinks, fruit-flavoured lozenges or sweets, and some medications, particularly effervescent vitamin C tablets. A patient who takes an effervescent multivitamin drink in the morning has an apple as their mid-morning snack, takes a juice at lunchtime and then has a fruit tea that evening has had four acid attacks that day. The risk of developing erosion is negligible if the acids are taken at meal times and this supports current advice on balanced and healthy diets. Consuming fruit with meals is not associated with an increased risk of erosive wear progression compared to those who snack on fruit between meals. Similarly, those who drank acidic drinks with meals were half as likely to have severe erosive tooth wear than those who consumed the same frequency of acidic drinks between meals [9].

We also know that drinking habits such as sipping, swishing or holding drinks in the mouth prior to swallowing increases the risk of having tooth wear [11]. The reason for this is twofold: not only does it increase the activity of the interaction between the acid and the dental surface, but it also replenishes the acid supply to the surface. A habit may also increase the duration of the acid challenge. Habits and increased time spent consuming acids have both been associated with increased erosive tooth wear and increased dental hypersensitivity [12].

There are rare occupational/recreational histories that might contribute to an increased risk of erosive wear. Wine tasters have classically been observed to have severe levels of erosive tooth wear, and possibly dental hypersensitivity, as a result of swishing wine in their mouths multiple times a day [13]. Athletes need consistent rehydration and some have been known to continually drink sports or other acidic drinks [14]. Alcohol abuse places a patient at risk not only of extrinsic erosive tooth wear from drinking acidic drinks frequently over prolonged time periods outside of meals, but also from intrinsic sources from vomiting and/or GORD [15]. The diets of those with occupations or recreational habits associated with increased intake of caffeinated drinks or soft drinks such as long-distance driving, night shift working or even video-gaming should also be risk assessed.

Risk assessment can be categorised according to the patient's daily habitual behaviour. If they consume three or greater dietary acids per day or greater than two dietary acids per day between meals, then they should be categorised as high risk. Daily acidic drink intake with meals would categorise them as medium risk and less than daily acid intake or daily fruit intake with meals places them in a low risk category. Patients should also be categorised as high risk if they have a habit of holding things in their mouth/cheeks prior to swallowing, sipping acidic drinks slowly or rinsing drinks around their mouth.

Risk Assessing Attrition

Bruxism is defined as an oral habit consisting of involuntary rhythmic or spasmodic non-functional gnashing, grinding or clenching of teeth, other than chewing movements of the mandible, which may lead to occlusal trauma [16]. The prevalence of bruxism tends to decrease with increasing age with a prevalence of 10–13% in 18–29 year olds, decreasing to 3% in those over the age of 60 with no observed differences between the sexes [17]. They can further be classified into diurnal habit or a nocturnal habit and the loads generated during sleep are substantially greater than those generated during the day. Clenching the jaws can create significantly greater loads due to the combined action of the temporalis and masseter. However, as there is reduced movement, clenching alone rarely results in severe tooth wear. For this reason, this risk assessment should focus on sleep bruxism.

The aetiology of bruxism is unknown. Until recently, some members of the dental profession believed that local dental factors such as a high restoration or an occlusal interference provoked bruxism with the underlying theory that the body would attempt to self-correct the interference until it was gone. This resulted in a body of opinion that proposed occlusal equilibration. However, more recently this theory has been discarded following evidence that occlusal equilibration did not stop bruxism and bruxism was not induced by the introduction of occlusal interferences.

The two current theories for the aetiology of bruxism are psychological (stress and/or anxiety) or alterations in the central nervous system (CNS) neurotransmission. Stress and anxiety are significant risk factors, particularly for diurnal bruxism. Sleep bruxism is related to micro-arousals prior to rapid eye movement sleep (REM) which can be repeated 8–14 times per hour of sleep. There is also evidence that central stimulation of the central nervous system (CNS) causes bruxism as stimulating the oesophagus with acids has been shown to induce bruxism [18]. Indeed, a pilot study observed a reduction in the frequency of bruxing episodes when proton pump inhibitors were administered [19]. A recent systematic review of sleep bruxism reported childhood and gastro-oesophageal reflux disease were the primary risk indicators for the condition [20]. Several classes of psychotropic drugs have also been observed to interfere with CNS activity and are associated with bruxism. These include stimulants (e.g., amphetamines, methylenedioxy-methamphetamine (MDMA) and cocaine) and anti-depressives, particularly selective serotonin reuptake inhibitors (SSRIs), e.g., Prozac (fluoxetine), Zoloft (sertraline) and Paxil (paroxetine). Anti-histaminergic drugs may also induce bruxism due to their disinhibitory effect on the serotonergic system [15, 21]. Other suggested aetiological factors for bruxism require further research and include upper airway resistance, such as that seen in snoring and sleep apnoea, which has been hypothesised to stimulate rhythmic masticatory muscle activity, and a genetic predisposition [20, 22].

Diagnosis of bruxism in its early stages can be difficult as many patients are unaware of their habit. A comprehensive pain history involving the location, duration, precipitating factors, severity of the pain, relieving factors and where it radiates

to can assist in the diagnosis of muscular or joint pain. Jaw muscle discomfort, fatigue, pain and jaw lock (particularly upon awakening) have been reported by the American Academy of Sleep Medicine as being indicative of sleep bruxism. Often a sleep partner may be able to give evidence that the patient audibly or visibly bruxes at night. The patient may give a history of chipping or fracturing restorations, cusps of teeth or both. In severe parafunction, occlusal loading can result in dental hypersensitivity. Occupation and lifestyle should be assessed as elevators of stress levels, and current or historical recreational drug use should also be investigated. The medical history should be checked for GORD symptoms, sleep apnoea or medications which may cause bruxism.

The clinical assessment of bruxism initially consists of an extra-oral examination which may reveal masseteric hypertrophy or a reduced facial height if there is loss of occlusal vertical dimension (OVD). Extra-oral palpation of the muscles may elicit tenderness but in most cases no symptoms are present. The temporalis muscle, involved in the positioning of the mandible, may be tender when a clenching habit is present and the masseter muscle may be tender from either clenching or grinding. The medial pterygoid muscle can be palpated intraorally but is probably not necessary to confirm the diagnosis and the action of palpating is painful in its own right. In severe cases there may be a reduced range of motion, particularly in the morning, and clicking of the joints.

Intraorally, there can be soft tissue signs such as petechial haemorrhages, white lines of keratinisation on the buccal occlusal line, crenations on the tongue, broken or chipped restorations or cusps. Dental wear facets which interdigitate with the opposing arch may also be visible. In severe bruxism cases the occlusal surfaces are flat. In addition to history taking and clinical examination, specialised devices have been used to attempt to detect a bruxism habit. These include wear on occlusal splints, sensors attached to occlusal splints and home muscle activity tests. More advanced methods include electromyographic (EMG) recording of masticatory muscle activity in addition to the gold standard of polysomnography during sleep clinics.

Although the presence of attritional tooth wear is a confirmatory diagnosis, several studies have shown that the severity of bruxism is not related to the severity of tooth wear which perhaps indicates that other tooth wear aetiological factors may be at play. However, if severe attritional wear is evident, particularly at a young age, the patient should be categorised as high risk.

Risk Assessing Abrasion

Abrasion is defined as an abnormal wearing away of the tooth substance by causes other than mastication [16]. Any foreign body misused or overused in the mouth has potential to be an aetiological factor. This includes chewing on pens, biting finger nails or any other foreign object, holding things between the teeth on a habitual basis, oral piercings and using items such as toothpicks. However, the most common associated cause of abrasion is from oral hygiene procedures [23]. Toothpaste

abrasiveness is measured by a value known as the radioactive enamel abrasivity (REA) or radioactive dentine abrasivity (RDA) value. As dentine is more susceptible to abrasion due to its lower mineral content, the RDA is the widely used measure of abrasivity. An RDA over 100 is classified as high abrasivity and anything over 150, predominantly found in whitening toothpastes can be classified as harmful. Unfortunately, RDA testing is mandatory only in the USA and not in the UK/Europe. Therefore, many toothpastes on the market do not have a published RDA value. Toothpastes and toothbrushing are unlikely to cause abrasion if used with normal pressure. But aggressive brushing either prolonged or with high pressure can cause wear.

Studies have observed a relationship between the use of a hard toothbrush and an increase in tooth wear [10]. However, it is a difficult topic for clinical research. People who choose hard toothbrushes may be more likely to brush more aggressively and with a more abrasive toothpaste to get a clean feeling. Laboratory studies, where you can examine each element separately, have shown that an increased amount of tooth wear is caused with increased toothpaste abrasivity and increased force but not bristle stiffness. Several studies have shown that increased tooth wear was observed with a soft toothbrush. This was hypothesised to be due to a result of flexing of the bristles allowing retention of more of the abrasive toothpaste. The combination of a medium bristled toothbrush and a low abrasivity toothpaste has been reported to show the least tooth wear on both enamel and dentine in the laboratory setting [24].

A history of aggressive toothbrushing with a soft toothbrush may place patients at high risk of erosion. Dentine is more susceptible to mechanical forces than enamel and multiple studies have shown a relationship between gingival recession and tooth wear [25, 26]. Any exposed dentine will place patients in a higher risk category for a combined acid/mechanical wear challenge.

The use of a toothbrush and toothpaste for tooth cleaning is not universal. Chewing on bark, sticks or using cloth with powders or salts is employed in several countries to clean teeth and there is some evidence for their efficacy in plaque removal. However, these can be very abrasive and have also been associated with increased tooth wear [27]. It is important to recognise that a patient may be more comfortable using these oral hygiene procedures and in the absence of a daily acid source they may be categorised only as medium risk.

There remains controversy about abfractional wear, which has been defined as wear through eccentric, excessive loading along the long axis of the tooth resulting in a non-carious cervical lesion. Some laboratory studies suggest that non-carious cervical lesions are more likely to be caused by abrasive or erosive–abrasive wear, while other papers report eccentric contacts and excessive loading to be an aetiological factor in non-carious cervical lesions. There is good evidence that occlusal equilibration is ineffective at reducing non-carious cervical lesion progression [28] and thus occlusal interferences will not be covered in this risk assessment. Abfraction remains a hypothesis as there have not been any clinical studies to show its action. The most likely cause of cervical tooth wear is a combination of acid and abrasion.

The Protective Role of Fluoride in Tooth Wear

Fluoride plays a protective role in tooth wear, particularly in a non-aggressive environment. All fluorides have been shown to reduce erosive tooth wear in vitro and using in situ models.

Remineralisation with fluoride can be effective if the damage to the underlying tissue matrix does not involve tissue loss and fluorapatite is more resistant to erosion and mechanical forces than hydroxyapatite. One epidemiological study reported that supplemental fluoride mouthwashes offered a protective effect on erosive tooth wear in children [29] but further longitudinal studies are needed to substantiate such data. The protective effect will likely be limited in an aggressive acidic environment such as that seen in a severe reflux episode or multiple dietary acid challenges daily where the fluoride ions get depleted rapidly [30]. However, the evidence would suggest that fluoride does have a protective effect and a risk assessment should establish whether the patient incorporates fluoride into their oral care regime.

Timing of Toothbrushing in Relation to an Acid Challenge

Brushing in a neutral environment with a low abrasivity toothpaste removes a negligible amount of tooth structure. However, if acid damage has occurred, toothbrushing may remove demineralised enamel. For this reason, it has been recommended to avoid brushing immediately after an acid challenge. However, recent laboratory studies have suggested that full remineralisation of eroded tissue is difficult to achieve, even after 2–4 h of waiting before you brush. This was also reported in epidemiological studies where no increased risk of tooth wear was observed when participants brushed immediately after an acid challenge, controlling for their acid intake. For the purposes of risk assessment, the acid challenge, rather than brushing after an acid challenge, is seen as the risk factor and there appears to be no significant advantage in delaying toothbrushing after an acid challenge.

The Role of Dry Mouth in Risk Assessment

Saliva plays a protective role in erosive tooth wear. In addition to diluting, buffering and helping to clear the acid, the salivary pellicle acts as protective barrier against acid and as a possible lubricant during attrition. Hence patients with dry mouth may also complain of dentine hypersensitivity or directly avoid dietary acids as they may cause dentine hypersensitivity. If a patient has a condition which causes dry mouth, is on medication that causes reduced saliva flow or has had treatments in the past that damage the salivary gland (radiotherapy to head and neck or chemotherapy), their risk of erosive tooth wear increases. Care should be taken to avoid risk factors before sleeping, such as drinking fruit-flavoured water during the night, when the

salivary flow is decreased. However, it is also not uncommon to find no evidence of tooth wear with dry mouths because suffers avoid acidic foods as they cause sensitivity.

BEWE—Screening Wear Already Present

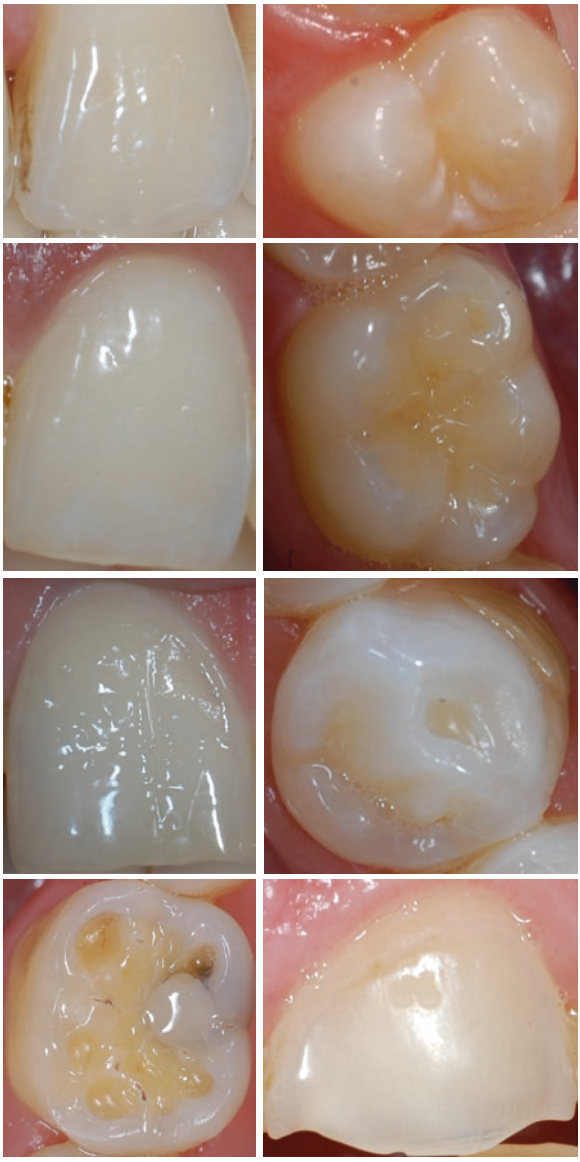
Interestingly, the same risk factors will cause wear in some patients, while others will remain unaffected. This is likely to be due to a combination of individual variation in the protective effect of saliva, the individual forces generated during mechanical wear and the frequency and severity of acid exposure. There is evidence to suggest that the presence of tooth wear is a predictor of future tooth wear, although very few interventional studies have been performed. Therefore, it is necessary to screen and grade tooth wear, not only to document it but also as part of your risk assessment. A useful tool for this is the basic erosive wear examination (BEWE), developed through international consensus [31], as a tool for screening erosive tooth wear in general practice. It grades the exposed surface based upon the percentage of the tooth surface affected and can be seen in Fig. 3.

Similar to a Basic Periodontal Examination (BPE), it is not necessary to record every surface. The worst score in every sextant is recorded to provide a total score. An image of this can be seen in Fig. 4. Conveniently, it can be assessed at the same time as the BPE as both use the same protocol.

The sum score (in the above example 16 out of a total of 18) is a representation of the tooth wear in the dentition. However, care must be taken with evaluating the total sum as the final measure. If severe, but localised wear is present, then this will present with an overall low score. Difficulties also arise in risk assessing using an overall score in a partially dentate arch. A useful method modification therefore is to combine the maximum sextant score with a total score. A maximum BEWE sextant score of 3 but a total score lower than 8 indicates that severe wear is present but is localised. A maximum BEWE sextant score of 3 but a total score greater than 12 indicates that there is severe generalised wear. A maximum score of 2 indicates moderate wear and a moderate risk, whereas a maximum BEWE sextant score of 1 indicates low risk (Fig. 5).

It is important to consider the age of the patient when risk assessing for tooth wear. Physiological wear and tear is normal and it would be unusual to see a sextant BEWE score of 0 in a patient over the age of 30. It would be equally as unusual to not see several sextant scores of 2 in a patient over the age of 55. The defining factor in risk assessment should be the presence of a BEWE score of 3. This is a sign of advanced wear at any age and preventive advice should be given.

It is also important to appreciate that tooth wear can become inactive. As tooth wear is irreversible, during an initial visit there is no way of assessing whether the existing wear occurred 20 years ago or whether it has occurred in the last 6 months. There are some indicators of current activity. For erosive aetiological factors, the presence of staining and a lack of dentine hypersensitivity may indicate an inactive phase. For bruxism, the lack of soft tissue signs or tender muscles may



Grade 0: No wear

Grade 1: Early wear
Initial loss of surface texture and dental characteristics such as perikymata, cusp ridges

Grade 2: Defect <50% of the surface
Distinct defect, with hard tissue loss <50% of the surface area. Dentine may be exposed but it is not necessary

Grade 3: Defect ≥ 50% of the surface
Hard tissue loss ≥ 50% of the surface area. Dentine may be exposed but it is not necessary

Fig. 3 BEWE scoring of the buccal surfaces of the central incisors and occlusal surfaces of first molars—two of the most common surfaces affected by erosive tooth wear. Images sourced from N. Schlüter, J. Klimek, C. Granß, BEWE —A tool for the assessment of treatment needs of dental erosion, Oralprophylaxe Und Kinderzahnheilkunde. 3 (2011) 120–129

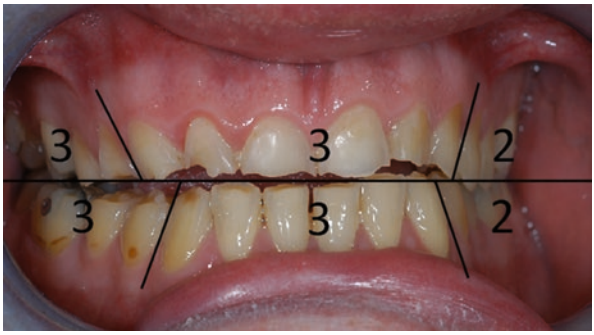


Fig. 4 Sextant BEWE scoring of a patient with severe erosive tooth wear

	High Risk Characteristics (Red)	Medium Risk Characteristics (Amber)	Low Risk Characteristics (Green)
Maximum BEWE Score	3	2	1/0
Total BEWE Score	13 or greater	Between 7-13 depending on age	6 or lower

Fig. 5 Risk assessment according to clinical presentation using the BEWE index

be an indicator that any wear is largely historical. A combination of accurate history taking, patient input and clinical examination should assist you in your diagnosis.

Summarising the Risk Assessment

After a thorough history and examination, including the BEWE, it may be useful to examine the risk characteristics of tooth wear progression separately. Figure 6 summarises the characteristics outlined in the previous section according to high (red), medium (amber) or low (green) characteristics.

Is the Risk Being Managed?

Analysis of the management of risk factors is as important as diagnosing the presence of the risk factors themselves. For each risk characteristic, the operators should attempt to assess if the exposures are being controlled. Figure 7 summarises the overall categorisation for erosive tooth wear.

	High Risk Characteristics (Red)	Medium Risk Characteristics (Amber)	Low Risk Characteristics (Green)
Erosion	Gastric Reflux Symptoms on a weekly basis or poorly controlled GORD. An active vomiting eating disorder (weekly basis) Total dietary acid intake 3+ per day or 2+ per day in between meals An occupation which encourages frequent consumption of acidic drinks Drinking an acid daily with a habit such as slow sipping, rinsing or swishing or holding the drink in the mouth prior to swallowing Spending >10 minutes eating a single portion of fruit every day	Infrequent GORD symptoms or well managed symptoms Managing eating disorder and vomiting episodes occur infrequently Daily dietary acid intake but less than 2 per day	No history or symptoms of GORD. No history of an eating disorder or previous history of an eating disorder but is inactive. Less than daily acidic drink intake. No sipping, swishing or holding drinks in mouth. Fruit intake only with meals. Consumes dietary acids quickly in less than 10 minutes. No intrinsic sources of acid
Attrition	Flattened teeth already present and active soft tissue signs/symptoms. High stress occupation and aware of grinding but unable to wear mouthguard	Currently showing soft tissue signs/symptoms of bruxism but wearing mouthguard	No history of parafunction with no intraoral signs of parafunction
Abrasion	Gingival recession and exposed dentine combined with aggressive brushing and interdental habits (3+ brushing per day and the use of a high RDA toothpaste)	High RDA toothpaste Gingival recession and exposed dentine but non-aggressive brushing and interdental routine Oral piercing Alternate form of oral hygiene procedures	Brushes twice a day with low abrasive toothpaste Non-aggressive interdental cleaning routine. No other sources of mechanical damage such as pen or nail biting.

Fig. 6 Risk characteristics for erosion, attrition and abrasion categorised into high, medium and low risk

Overall risk characterisation for Erosive Tooth Wear	
Red Reassess every 6-12 months until confident that the risk is no longer present	Presence of one aetiological factor in the red category Daily exposure to a combination of acid and mechanical aetiological factors which for which management is not optimal
Amber Reassess every 12 months until confident that the risk is no longer present	Presence of one aetiological factor in the amber category Multiple amber aetiological factors are present but are being managed to some degree of success.
Green Reassess every 2-3 years until change in risk becomes evident	No daily risk of any component If characteristics are present they are being managed effectively

Fig. 7 A summary of the overall categorisation for erosive tooth wear

Gastro-Oesophageal Reflux Disease

Is the patient taking medication to control refluxate entering the mouth? Or are they controlling it with diet and avoiding trigger factors?

Vomiting Eating Disorders

Is the patient attending therapy and how do they feel they are managing the frequency of vomiting episodes?

Dietary Acid Intake

There is limited evidence that dietary advice alone is effective. Making a plan with the patient to address ways to reduce dietary acid intake may be more effective. Can the patient make an easy substitution for a non-acidic alternative that is equally as attractive for them? A useful technique to prompt action is to use the format "**If** I want X, **then** I will have Y", known as an "if-then" plan. Discuss ways of making their environment more amenable to enable the change such as not buying acidic drinks for the home environment or buying non-acidic drinks for work.

Bruxism

If bruxism has been diagnosed, is the patient wearing a mouthguard? There is limited evidence to suggest that any one mouthguard is superior to another, provided they cover all the occluding teeth. Compliance with mouthguards is low so the mouthguard

which the patient is most comfortable in and is more likely to wear will offer the greatest protection. Hard acrylic splints can be adjusted to achieve a balanced occlusal scheme and will last longer but are more expensive and complicated to fabricate.

Abrasion
Can the aetiological factor be removed? Is a less aggressive oral hygiene regime acceptable to the patient? Will they change to a low abrasivity toothpaste? If a piercing is in place, is the patient willing to remove it? Are they willing to attempt to change a habit involving use of a foreign object (pen chewing, nail biting) in their mouth?

Co-morbidities may be present in tooth wear. The relationship between gastro-oesophageal reflux disease and sleep bruxism is now established and many of those people with vomiting eating disorders may also have anxiety and/or depression. Many of the anxiolytic drugs and anti-depressive drugs have the effect of reducing saliva levels/flows and may cause bruxism. The dry mouth may cause the patient to drink more fluids, some of which may be acidic. Having taken the presence, interaction and management of all risk characteristics into consideration, the patient can then be categorised into a red, amber or green risk for tooth wear progression.

Conclusion

For all patients in the amber and red categories, monitoring with study models or clinical photography is indicated. As digital methods of assessing wear improve, this will improve our diagnostic capacity. Active prevention methods such as effective preventive advice, fluoride application measures and mouthguards should be a continuous intervention. Restorative intervention is rarely indicated until the diagnosis of all aetiological factors is confirmed. Monitoring the patient while you are determining the activity of the aetiological factors and risk assessing is not supervised neglect. A lack of restorative intervention for 1–2 years will rarely leave you with a poorer disease outcome, but it may lead you to an improved risk assessment and diagnosis.

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Risk Assessment in Oral Cancer

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Introduction

Global trends for oral cancer have changed in many respects over the past 3 decades. Not only has the number of cases reported each year been rising in many countries, but the age standardised incidence has also increased in many parts of the globe. Taken together with oropharyngeal cancer, this is now the sixth most common cancer in the world. The Global Cancer Observatory (Globocan) has estimated that close to half a million people are affected each year and there are close to 150,000 deaths per annum [1]. In addition to rising trends there are changes in demographic factors and the recognition of emerging risk factors. Oral cancer used to be a neoplasm that affected older people but more recent statistics from cancer registries worldwide indicate that 6–10% of cases diagnosed with oral cancer may be under the age of 45 years. Oral cancer has become a deadlier disease with about 50% dying with or from this disease within 5 years of diagnosis in most centres. This is largely due to late diagnosis resulting in treatment failure of oral cancer patients presenting in tertiary centres with advanced stages of disease.

The Harvard Cancer Risk Index provides a broad classification of cancer risk for several major cancers [2]. So far, approaches to develop risk models for oral cancer have been limited [3, 4]. This chapter examines methods of identifying people at high risk of developing oral cancer to set out a high-risk strategy to individualise approaches to early detection and prevention.

Risk Stratification

In many European countries, health policy on prevention of non-communicable diseases, e.g., cardiovascular disease, is targeted at high-risk individuals rather than population strategies [5]. This risk-driven approach sets out to identify individuals at high risk of disease and then target preventive measures at the point of care delivery. To adopt such a risk-targeted approach, it is important to first identify a population at risk, and then filter down to individuals at risk and detect high-risk lesions that could transform to cancer. For those already diagnosed with cancer, risk assessment could also help in selecting appropriate options for treatment. Risk stratification at each of these levels needs careful consideration with good research evidence from the published literature. Early detection of oral cancer could significantly improve mortality and morbidity. Reported five year survival rates for stage 1 oral cancers can be up to 76% compared to only 37% for stage 4 [6].

Populations at High Risk

Epidemiological reviews have reported a 20-fold difference in oral cancer incidence between low incidence and high incidence countries [7]. This is reflected in the Globocan data, which estimates that two-thirds of the global incidence of oral cancer is found in developing regions in the world, mainly in south and east Asia

and some pacific islands [1]. Geographical distribution of cancers of the lip, oral cavity and oropharynx differs widely based on lifestyles.

Lip Cancer

Lip cancer is more common in Australia, parts of Canada, Southern Spain, Greece, Israel, Serbia and the Ukraine. Lip cancer is relatively uncommon in non-white populations, while white Caucasoid populations with fairer skin are at a heightened risk. In a population living in Western Australia the annual incidence of lip cancer from 1982 to 2006 was 8.9 in 100,000 per annum [8]. It is interesting to compare the Australian data with the Globocan data; the age standardised incidence rate (ASR) in 2012 was 0.4 per 100,000. Australian males may have 22 fold higher risk of developing lip cancer compared with the average rates for age-matched males in the rest of the globe. Rural dwellers, those with high sun exposure and those in outdoor occupations have higher standardised incidence rates when compared to people living in metropolitan areas. The higher rates of lip cancer in the aforementioned populations are attributable to the susceptibility of fair-skinned populations to exposure to UV light as a risk factor.

Oral Cancer

Oral cancer is relatively common in South and East Asia and in some Pacific Islands in the continent of Oceania. According to Globocan [1] two-thirds of all oral cancers are reported from these regions of the world. In the Indian subcontinent, oral cancer could be the most common cancer in men and the third most common in women. The five countries with the highest ASRs in the world are Papua New Guinea, Taiwan, Maldives, Sri Lanka and Pakistan. The rates in these countries are 10–20 fold higher than countries with the lowest incidence [7] and oral cancer is associated primarily with the betel quid chewing habit, referred to later in section "Areca Nut with Betel Quid". Outside South Asia, oral cancer rates are also high among some parts of Western Europe (e.g., North West France and Portugal) and Eastern Europe (e.g., Hungary, Slovakia and Slovenia), parts of Latin America and the Caribbean (e.g., Brazil, Uruguay and Puerto Rico) and some former French colonies e.g., French–la Reunion. Thus any risk assessment for oral cancer should take into account the geographical location and lifestyles, and preventive and awareness programmes should be specifically directed to citizens from these nations.

Oropharyngeal Cancer

There has been a marked increase in the incidence of tongue and oropharyngeal cancer among young people in some high-income countries [9]. In Europe, France has recorded the highest incidence of human papillomavirus (HPV)-associated

oropharyngeal cancer. The incidence is also high in North America. This is largely attributable to the fact that oral HPV infection is common among US men [10]. The majority of oral HPV-infections are acquired by oral sex [11].

Any risk assessment for lip, oral and oropharyngeal cancers should take account of the unequal distributions and excess risk of these three cancers among different populations.

Individuals at High Risk

Major risk factors for oral cancer are tobacco use, high alcohol intake and betel quid chewing [12]. These three agents are among the four most commonly used substances in the world, the fourth being caffeine. Any individual who regularly uses these three substances is at increased risk, but in common with many carcinogens, the effect is dose-dependent. There is a huge volume of literature reporting on the relative risks (or odds ratios) for oral cancer, based on case–control and cohort studies that have examined the carcinogenicity of these agents. These individual studies are collated in several monographs published by the International Agency for Research on Cancer (IARC), the latest being Volume 100 E published in 2012 [12]. Based on the scientific evaluation by the IARC, tobacco, alcohol and areca nut in betel quid are class 1 carcinogens to humans and the oral cavity is a recognised target organ for cancer development in people who consume these agents. Based on this evidence it is therefore possible to identify individuals who are at high risk for oral cancer. It is important to assess the magnitude of effect of combined risk habits when assessing the risk of individuals.

Tobacco

Most cancers of the oral cavity are attributable to the use of tobacco products in any form (cigarettes, cigars, bidis, pipes and smokeless tobacco). Risk for current smokers is about tenfold that of never smokers and as said before is dose-related. Ex-smokers reduce their risk following cessation [13] and after 10–20 years of quitting reach the risk status of never smokers [14].

Cigarettes and cigars are reported to have similar risks, and bidis, a tobacco product smoked in Asia, is reported to have higher risks [15]. Reverse smoking, a peculiar habit of smoking with the lighted end of a cigar, chutta (an Indian smoking product) or cigarette placed inside the mouth also has significantly increased risks [16]. This habit is prevalent in parts of India, the Caribbean Islands, Colombia, Panama, Venezuela, Jamaica, Sardinia and the Philippines.

There is still limited evidence on other types of smoking, e.g., e-cigarettes [17], water pipe smoking [18] or on passive smoking. Recent reports have suggested that e-cigarettes can help improve the success of quitting smoking, but there is controversial evidence as to whether e cigarettes are a gateway to smoking. Some toxic substances included in the e-liquid can be found in the e-cigarette smoke and some adverse effects are reported such as mucosal irritation. However, the main concern is the lack of long-term follow-up studies on the impact of this device on cancer causation.

Smokeless tobacco (ST) is available in a variety of forms and there are nearly 350 million people using ST. Of these, nearly 80% are in the South-East Asia Region (SEAR); highest prevalence of ST use is among men in India, Bangladesh and Myanmar. ST is used along with other products, especially alkalising agents like slaked lime to increase release of alkaloids and areca nut is a group 1 carcinogen [12, 19]. ST users carry a significant cancer risk of oral cancer [20, 21] but many may not be aware of these risks.

Alcohol Use

Alcohol use is an independent risk factor for the development of oral cancer. Its effects are independent of those of tobacco use. The magnitude of effect is reported to be lower than the risk associated with tobacco use. Risk of oral cancer is approximately five-fold for heavy alcohol drinkers compared with non-drinkers or irregular consumers [22]. The effect is heavily dose-dependent. Alcohol on absorption is rapidly metabolised to acetaldehyde which is a class 1 carcinogen and acetaldehyde adducts can be found in oral cancer tissues. Elevated risks among binge drinkers remain unreported.

Tobacco and Alcohol

As already stated, tobacco and alcohol use represents independent risk factors. When combined they have an exponential synergistic effect, with users being 38 times more likely to develop oral cancer when compared with abstainers from both products [23, 24].

Areca Nut with Betel Quid

Betel quid (BQ) with or without tobacco was classified as a class 1 carcinogen by the IARC in their evaluation in 2002. Areca nut is the principle substance in betel quid. The magnitude of effect of betel quid has been documented in many studies from India, Sri Lanka, Taiwan and among migrant populations living in South Africa [19, 25]. A meta-analysis [26] reported that people chewing betel quid containing tobacco in the Indian subcontinent had a relative risk of 2.56 (95%CI, 2.00–3.28; 15 studies) for BQ without tobacco and a relative risk of 7.74 (95%CI, 5.38–11.13; 31 studies) for BQ with tobacco. The meta-relative risk was much higher in women (mRR, 14.56; 95%CI, 7.63–27.76) than in men in India. In Taiwan, where the habit is more prevalent in men, the meta-relative risk for BQ without tobacco was 10.98 (95%CI, 4.86–24.84; 13 studies), probably due to a higher daily frequency of chewing. Taiwanese do not add tobacco to BQ. These data strongly reconfirm that habit of betel quid chewing, primarily areca nut use should be taken into account in assessing the cancer risk of Asian, Taiwanese people and Pacific Islanders for development of oral cancer.

Human Papillomavirus Infection

Infection with the human papillomavirus (HPV)—first known to cause cervical cancer—is now also strongly associated with oropharyngeal cancer, particularly of the tonsil. It is reported that about 70% of tonsillar cancers are infected with HPV, particularly high-risk HPV type 16. In those with HPV positive cancers, a strong association was noted with seropositivity for the HPV-16 L1 capsid protein or an oral HPV-16 infection [27]. Most studies suggest that oral HPV infection is sexually acquired but the method of transmission is not confirmed. However, persons who have had multiple sexual partners (>6) are considered to be at risk of oropharyngeal cancer and to a lesser extent of oral cancer. The National Health and Nutrition Examination Survey (NHANES) 2009–2010 in the USA reported a 6.9% prevalence of oral HPV infection (in oral rinses) among men and women aged 14–69 years [10]. A lower prevalence of oral HPV (3%) (in rinse samples) was recently reported from Argentina [28]. Oral rinse or whole mouth saliva are not confirmed methods for assessing risk for HPV, but behavioural risk assessment of a history of multiple sexual partners or past history of oral sex has been proposed. HPV serology (HPV 16 E6 antibodies) is being tested in pilot studies to assess the potential future risk to individuals of developing oropharyngeal cancer [29]. Currently there is no official recommendation for screening for HPV infection of the oral cavity.

Age, Sex and Socioeconomic Status

Oral cancer affects more men than women, generally in a ratio of 2:1. Mean age is reported to be around 60 years. However, this is not to say that younger people are not diagnosed with the condition and oral cancer has been increasing in people under the age of 45 years [30]. In the USA, for 2018 the mean age of most people diagnosed with these cancers was 62 years, but a little more than one-quarter occurred in patients younger than 55 [31]. Conway et al. [32] in a meta-analysis reported that low socioeconomic status was an independent risk factor for oral cancer. However, about a quarter of cases particularly among the young are now reported in professional classes [33, 34]. We cannot therefore stereotype anymore those who may be affected by oral cancer in terms of age, sex or socioeconomic status.

Risk Factor Models Based on Lifestyles

Few risk factor models based on social histories (lifestyles) have been developed to assess oral cancer risk in the general population. Diajil and Thomson [4] compiled a model based on published studies. Using 12 publications specifically investigating patient risks, they delineated a consistent “high-risk” patient profile for oral

carcinogenesis, while Amarasinghe et al. [3] reported a model suitable for Asian subjects primarily consuming betel quid. These models need further evaluation on large population based studies, with long-term follow-up. Once a model that accurately predicts cancer risk has been developed, it could be utilised for selecting individuals for oral cancer screening.

Lesions at Risk: Oral Potentially Malignant Disorders

People diagnosed with oral potentially malignant disorders (OPMDs) are at increased risk of developing oral cancer. OPMDs are conditions that precede the appearance of invasive cancers of the oral cavity [35, 36]. The term embraces both precancerous lesions and conditions. The spectrum of OPMDs includes: oral leukoplakia, erythroplakia, erythroleukoplakia, proliferative verrucous leukoplakia, oral submucous fibrosis, palatal lesions in reverse smokers, oral lichen planus, oral lichenoid reactions, graft vs host disease, oral lupus erythematosus and some hereditary conditions such as dyskeratosis congenita and epidermolysis bullosa. Actinic cheilitis of the lower lip is also associated with an increased risk of lip cancer. Of the many OPMDs leukoplakia is the most common presentation seen and is defined by the WHO as a "white plaque of questionable risk having excluded (other) known diseases or disorders that carry no increased risk for cancer" [35].

The prevalence of these OPMDs may vary in different populations mostly based on high-risk habits described earlier. A recent systematic review [37] reported the pooled global prevalence of OPMDs (based on 15 studies) was 2.97% (95%CI = 1.09–5.72). However, the prevalence of the different types of OPMDs varied. The two common OPMDs reported were oral submucous fibrosis among betel quid chewers in Asian people estimated at 3.42% (95%CI = 1.76–5.60) and oral leukoplakia in the US and European studies at 3.26% (95%CI = 1.0–6.8).

Malignant transformation rates of these OPMDs also vary. Non-homogenous and red lesions are much higher risk than homogenous white patches [38]. Those exhibiting moderate or severe dysplasia are considered as high risk [38]. Long-term follow-up studies on patients with OPMDs—with or without necessary interventions—allow us to estimate the risk of malignant transformation of these conditions [39, 40]. A systematic review on oral leukoplakia estimated an average malignant transformation rate of 3.5% with a wide range between 0.13% and 34.0% [41], while malignant transformation of erythroplakia was reported to be close to 50% [42].

Oral lichen planus affects about 1% of the world population. Observational studies by long-term follow-up of this condition have provided some evidence that people affected by oral lichen planus are at increased risk of developing oral cancer. A systematic review found 16 eligible papers published since 1988. Among the total number of subjects with OLP (n = 7806), 85 cases developed SCC, while 4 out of 125 OLL patients developed SCC. The overall malignant transformation of OLP was 1.09% [43].

Risk Assessment of OPMDs

Several approaches have been employed for quantitative stratification of cancer risk of OPMDs, mostly focussed on oral leukoplakia and based on clinic-pathological correlates [44]. Use of chair-side adjuncts to detect and evaluate OPMDs has received considerable attention in the past two decades due to several commercial agencies advertising these detection systems, and claiming that they may assist in oral cancer detection. These include vital dyes (e.g., toluidine blue) and optical systems (e.g., VelScope, Vizilite) [45]. Several authors have tested these in secondary care facilities. These studies have reported good sensitivity in their performance to detect OPMDs [46, 47] and their ability to select a suitable biopsy site. However, there is no evidence yet to suggest that these adjunctive aids are able to distinguish high-risk lesions from those with low risk. The high rate of false positives observed in clinical studies was highlighted in a report of the American Dental Association [48]. One exception is *in vivo* microscopy technology that one US group has shown as a promising avenue to help clinicians identify high-risk lesions [49].

Biopsy of a representative area is still considered the gold standard for assessing the risk status of OPMDs. A biopsy allows the grading of epithelial dysplasia, based on the sum of various architectural disturbances and individual cellular features called epithelial atypia seen microscopically. Dysplasia is graded as mild, moderate, severe or in two categories as low or high risk. While it is fraught with some subjectivity in reporting [50] it remains the most widely used tool for risk assessment and planning any surgical treatment. Patients with high-grade dysplasia (moderate or severe) generally have a higher chance for malignant transformation than those with lower-grade dysplasias [40].

Figure 1 illustrates how currently available information on lifestyle risk factors combined with the presence or absence of a potentially malignant disorder could be utilised for chair-side analysis and characterisation of an individual's risk and an approach for risk management based on these individual factors.

Genetic Susceptibility and the Use of Biomarkers

Genetic Polymorphisms

Polymorphisms of genes involved in carcinogen metabolism, cell cycle control, DNA regulation and repair have been shown to be related to elevated risk of cancer development. Compared with common cancers (breast, prostate, colon and lung) comparatively fewer studies have been reported in oral cancer. One of the best-studied cancer polymorphisms to date is the effect of the codon 72 polymorphisms in the p53 gene on susceptibility to a wide variety of cancers, including oral cancer. The relationship between the polymorphism in codon 72 (Arg72Pro) and oral cancer has been studied but results are inconsistent [51]. Genes encoding one of the phase-I detoxification enzymes, microsomal epoxide hydrolase (mEH) and phase-II metabolising enzymes could influence cancer susceptibility.

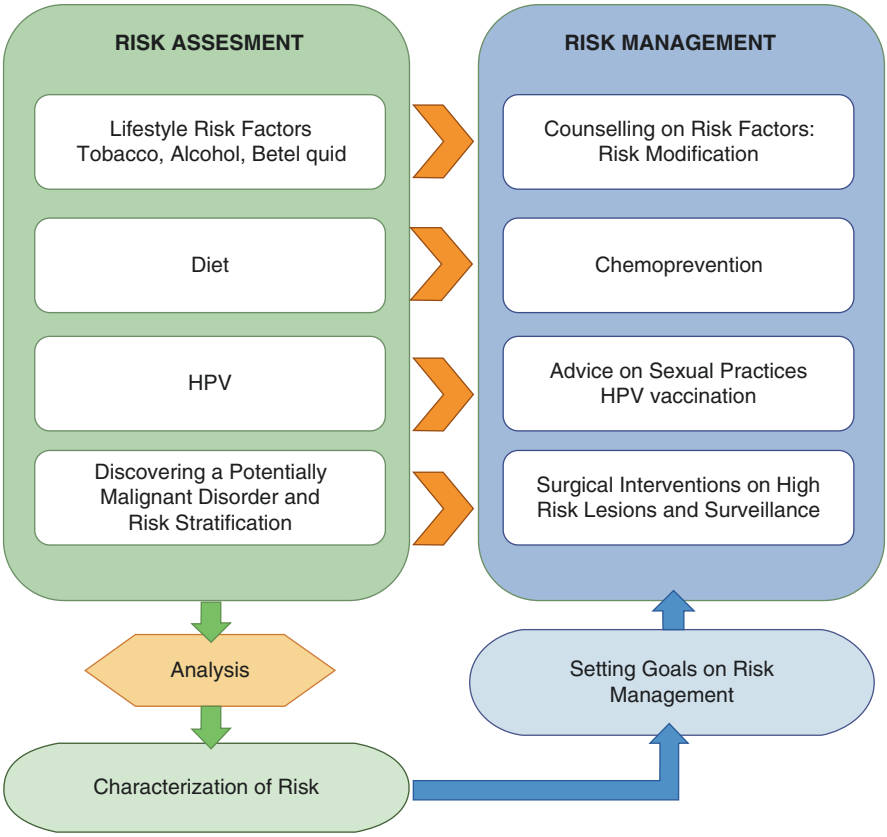

Fig. 1 An approach to risk characterisation based on lifestyle risk factors and risk lesions. Knowledge of these factors can help the clinician in setting goals for risk management on a personalised basis

Glutathione-S-transferase Mu 1 (GSTM1) and glutathione-S-transferase theta 1 (GSTT1) are two phase-II enzymes involved in the biotransformation and elimination of several carcinogens, including the metabolites polycyclic aromatic hydrocarbons from tobacco smoke. An individual with genetic polymorphisms of either of these GST enzymes would thus be expected to have an impaired ability to detoxify carcinogens and have an increased risk of cancer. In a meta-analysis of 50 studies, GSTM1 gene, null genotype appeared to be a risk factor for oral cancer [52].

Gross Genomic Studies

A ploidy analysis is a technique that could be used to measure the amount of DNA present in cells harvested from a biopsy. Tissues with abnormal DNA content are termed DNA aneuploidy, while those with DNA content equal to normal cells are termed DNA diploid. A meta-analysis of five studies demonstrated that aneuploidy is a useful marker of malignant transformation in OPMD [53]. In 2000, Rosin's

group proposed a loss of heterozygosity (LOH) risk model in a retrospective study. The group subsequently validated their previously reported LOH profile models as risk predictors and developed a refined model that involved a prospective cohort of 296 patients with mild/moderate oral dysplasia. The prospective cohort validated that the high-risk lesions (3p and/or 9p LOH) had a 22.6-fold increase in risk ($P = 0.002$) compared with low-risk lesions (3p and 9p retention). Addition of another 2 markers (loci on 4q/17p) further improved the risk prediction [54].

Biomarkers

Over the past two decades there has been a tremendous enthusiasm of using molecular markers (both genetic and epigenetic) for cancer risk stratification [55–58]. A comprehensive list of these potential biomarkers was recently published by Nikitakis [57]. Studies have demonstrated that the gene expression profile can significantly improve the prediction of OSCC development over clinical and histological variables in oral leukoplakia patients [59].

Future Research

These different prediction tools highlighted in this chapter need to be validated in future prospective studies, and a combination of profiles may serve as biomarkers to classify individuals and particularly those with OPMDs for oral cancer risk in routine clinical practice. It is important to extend this approach from the laboratory to the clinic to identify individuals at risk so that high-risk individuals may receive appropriate individualised care to prevent oral cancer.

Concluding Remarks

- White Caucasoid males in outdoor occupations exposed to UV light are at risk of lip cancer.
- Those who consume tobacco, excess alcohol or chew betel quid have an increased risk of tongue and oral cancer.
- Identifying at-risk individuals enables them to take appropriate steps to lower their risk of developing oral cancer.
- It is important to promote evidence-based cessation strategies to help smokers to quit tobacco use.
- Persistent HPV infection may lead to oropharyngeal cancer.
- Prediction of oral cancer risk is an important component of oral health risk appraisals in primary care.
- Detection of high-risk lesions that have the potential to transform to cancer provides opportunities to intervene.
- Stratification of OPMDs as “high risk” or “low risk” by chair-side adjunctive techniques and by cytology/histopathology needs to be tested in multicentre prospective studies.

- There are currently no models to predict an individual's risk by combining various lifestyle risk factors and the presence or absence of an OPMD. The development of a risk prediction model would help to identify individuals that would benefit from oral cancer screening.
- There is an urgent need to develop evidence-based strategies to translate available knowledge to practice.
- Future research should attempt to identify novel biomarkers in order to identify those at risk.

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Risk Assessment for Behaviour Change

Koula Asimakopoulou

Contents

Introduction

Humans are invited to assess and make decisions around risk all the time. From the minute we are born to quite later on in life, our whole purpose of being is shaped by assessments to do with risks and benefits. Such assessments and the ability to take calculated risks have been important for human survival; it is because our hunter-gatherer ancestors were quite adept at working out various risks around them and then successfully changed their behaviour to control or eliminate the risks that they managed to survive and pass on their genetic material to future generations. They, in turn, were further able to assess and then communicate successfully the various risks around them, thus ensuring their and their social group genes survived. Simple though it sounds, such a risk assessment process was central to survival; for example, in calculating the risk of various routes to food in presence of a predator, taking

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the route that would yield the most food with the least amount of risk was a process that our ancestors would have had to perform a fair number of times; those that were not very good at working out such risk correctly were less likely to survive whilst those that were better, lived, mated and passed on their genes. So, the ability to calculate risks effectively has been central to the evolution and survival of the human species. It has been proposed that the development of language would have supported this process [1].

In a similar effort to enhance their attempts to survive against predators, humans have continually resorted to a variety of methods to try and predict risk. Going back to the ancient world, stories about Pythia the Oracle of Delphi are rich in detail showing how the ancient Greeks dealt with the need to predict risks of future behaviour by obtaining practical counsel over the future; in those times, the prediction of future risks was such a high value exercise that it required someone with superior, magical powers, sitting at the foothill of a mountain to be able to see into future hazards and give trustworthy advice, albeit in a riddling fashion. Such advice shaped a lot of the geo-political ancient world. Decisions such as whether to go to war and in what way were arrived at as a result of these mysterious, oracular pronouncements. So, human beings' keen interest in predicting future risks has been with us for some time.

Two thousand years later modern society still engages in risk assessment about future behaviours, albeit in more sophisticated, reliable and hopefully, valid ways. From risk assessments for conditions that might impact future health and behaviour whilst a baby is still in the womb, all the way into adulthood, risk assessment to predict and modify people's behaviour is central to everyday life. From offering people information on the risks of medication side effects and medical procedures such as general anaesthesia, to risks of novel treatments, to discussing personalised risks of common diseases such as coronary heart disease, diabetes or having a stroke, risk assessment features in most areas of life.

Given how long human beings have had an interest in risk and its assessment, and the inconceivably large number of such assessments that must have taken place since our hunter-gatherer ancestors started engaging in this sort of behaviour, one would expect that people might be quite proficient at understanding the concept of risk, communicating risks and taking sensible action when faced with such risks. This chapter explores some of the evidence currently available that shows how well-placed we are to calculate, understand and communicate such risks; it however argues that humans, despite having had a fair amount of practice in working out risks in our past, are not particularly credible assessors or communicators of such risks when left to our own devices. For this reason, the chapter argues that where risk estimation has to be reliable and valid and where such an assessment is carried out with the view of supporting people to change their behaviour towards making decisions that are beneficial for their health, such risk estimation should follow some standardised, validated protocol in an attempt to manage human error. Medicine and dentistry are two areas of human life that provide a good vehicle for understanding how the human mind works around risk.

Clinician and Patient Understanding of Risk in Medicine

Communication of risk is an important and potentially difficult aspect of medical practitioners' and dentists' clinical practice. The medical literature has suggested that communication of risk should be about telling patients what is the probability of the risk occurring, explaining the adverse event characteristics that might occur and finally, being open and honest about the effect of the adverse event on the patient [2].

Risk communication has ethical, practical and behavioural angles to it. Firstly, from an ethical point of view, patients are expected to be informed about the risks of medical and dental procedures so they might give, genuinely informed, consent to undertaking them. It is not just an exercise in delivering person-centred care to ensure that personalised risks of procedures are accurately communicated; it is simply the right and ethically appropriate thing to do to ensure that patients are making decisions about their care that they themselves have some ownership of [3]. In line with this idea the General Dental Council (GDC), for example, clearly state in their standards the need for healthcare teams to obtain valid consent before treatment where the risks and potential benefits of treatment have been fully explained [4]. From a practical viewpoint, risk assessment is routinely undertaken to gauge whether, given any known associated moderators/mediating factors, the patient is likely to be affected by any given disease and secondly, if disease is present, to formulate prognoses and decide whether it is clinically appropriate to go through with a given procedure [5]. So over and above ethical considerations, the accurate assessment and communication of risk is at the heart of most medical and dental procedures whereby it provides invaluable information in underpinning treatment plans, options and eventually choices. Finally, where the success of most of these procedures partly rests on patients' preparedness to adhere to instructions given by the medical or dental team, patients' understanding of the risks associated with non-adherence with clinical recommendations might be arguably be a benefit or hindrance to such behaviours. A classic case where patients' misunderstanding of risk affecting behavioural choices is the now widely reported problem of patients erroneously believing that colds/viruses are treatable by antibiotics or their creative non-adherence in the taking of such drugs where they might fail to complete the course as prescribed as soon as their symptoms have subsided [6].

Given the importance of risk understanding and communication in healthcare settings, researchers have been studying how best to communicate health risks for a long time. Risk communication can be a risky business! Telling a patient that a procedure 'has a 90% chance of success' is likely to yield a different assessment of the procedure than saying that the procedure 'fails 10% of the time'. Equally, telling someone that their risk is 'high' or 'low' will mean different things to different people depending on how much risk they read into such qualitative descriptors. Thinking of an example from orthodontics, some evidence suggests that orthodontists routinely make qualitative assessments of periodontal health [7]. In the absence of a clear method to assess and report on periodontal risks, clinicians have been using the descriptor 'Oral hygiene- poor +++' in clinical notes where oral hygiene

has been used as a proxy to describe patients' adherence with orthodontic treatment. It is obvious that such a descriptor will get interpreted differently by different clinicians and such variations in interpretation will lead to different clinician actions.

Having reviewed the literature on risk communication, researchers in the field have reported the apparent conclusion that risk communication is undermined by what has been termed as '*Collective statistical illiteracy*'—a situation where most adults (clinicians and patients alike) have difficulty understanding basic statistical information [8]. The basis for this conclusion lies in the fact that risk information rests on basic understanding of the concept of probability and frequency estimation, processes that people often find difficult to grasp. A classic example of difficulties in this domain is demonstrated by a US study of undergraduates who were allocated to one of two conditions; in one condition they were told that cancer '*kills 1286 out of 10,000 people*'. In another, they were told that cancer '*kills 24.14 out of 100 people*'. They were then asked to give estimates of how risky cancer was. The study found that the participants' estimates were higher in the first condition rather than the second [9]. In another study reporting on a sample of 1000 German participants, over a third of the sample failed to give the correct answer when they were asked what 40% meant and were presented with a choice of (i) 1 in 4 (ii) 4 out of every 10 and (iii) every 40th person! [10]. There is quite a substantial body of research showing that even in highly educated people, there is an observable difficulty with interpreting simple risk questions. This is even for simple problems such as deciding which of 1%, 5% or 10% represents the highest risk [11].

One would hope that these difficulties do not apply in adults working in healthcare who are invited to make life and treatment decisions and where, given the circumstances they find themselves in, one might expect them to fare much better. In a study of experienced physicians, participants were asked to estimate the probability that a patient had colorectal cancer, if they tested positive on a faecal occult blood test (FOBT) known to have a sensitivity of 50%, a false positive rate of 3% and where the prevalence of this cancer was 0.3%. A variety of experienced clinicians were sampled who produced a range of answers to this question. When assessing the answers provided it was shown that their estimates of the patient having colorectal cancer ranged from 1% to 99% with most answers being around the 50% mark [12]. The correct answer is 5%. The need for clinicians to be supported in their interpretation of risk estimates has been called for in response to these and other similarly alarming data suggesting difficulty understanding and responding to statistical information pertaining to risk [8].

Clinicians are not alone in misunderstanding risk information. Patients seem to hold inaccurate beliefs about risks that may well influence lifestyle choices and the uptake (or not) of healthy behaviours. For example, in a randomised controlled trial of people with Type 2 diabetes, we asked patients to report what they thought was their risk of developing coronary heart disease (CHD) or having a stroke as a result of their diabetes. We did this by randomly allocating patients to consider these risks in either the next 1, 5 or 10 years. We then used the United Kingdom Prospective Diabetes Study (UKPDS) Risk engine to calculate what the respondents' *actual* risks of developing CHD or having a stroke were, using objective clinical data and

the validated UKPDS Risk Engine. There was a large discrepancy between patient perceived and actual risks of CHD and stroke, with patients dramatically *overestimating* their risk of CHD and stroke by 3.5 and 5 times their actual risk, respectively, for these conditions [13]. At the same consultation session, we used the UKPDS Risk engine software output (a series of colour coded bars indicating risk of disease for the patient's age, gender, cholesterol and blood pressure profile) to show patients these actual risks of disease. We further supplemented this explanation using smiley faces and pie charts. At the end of this explanation we invited patients to tell us and then write down what they believed their actual risks of CHD and stroke were, having received this personalised, software-output-framed, correction of risk. The data showed that in all three time frames we were successful in reducing patients' originally inflated risk estimates so that by the end of the consultation all patients had an accurate perception of their future risk of CHD and stroke. The 10-year group, however, were the most resistant to correction. Six weeks later we contacted patients in an unexpected telephone call and asked them to tell us whether they remembered what their risk of CHD and stroke was. We found that memory for these risks for those who had been given 10-year risk estimates had regressed back into their originally inaccurate, inflated, risk perceptions of CHD and stroke whilst those patients who had engaged in thinking about risks of CHD and stroke in the shorter term (1 or 5 years) had retained accurate recall of their corrected estimates [14].

There are two noteworthy findings in this study. Firstly, we found that in contrast to anecdotally held diabetes clinicians' beliefs that people with diabetes do not follow clinician diabetes recommendations because '*they don't understand the risks…. so they just underestimate how dangerous diabetes is*' our sample were, in fact, doing the opposite. We called this phenomenon *unrealistic pessimism* and warned against using fear campaigns to try and warn patients of health risks in an attempt to support them to engage in healthy behaviours [15]. Secondly, and perhaps more importantly, we found that using a standardised, validated risk calculator to calculate risk and a standardised communication script was sufficient to correct these inaccurate beliefs patients held and start discussing behaviour change on the basis of more accurate perceptions of future threat.

It would thus appear that whilst risk assessment and communication may be processes that take place routinely in healthcare settings, a lot of the time it is the case that, left to their own assessment (clinicians) or perceptions (patients) they can both be prone to errors. In this case, using a reliable/valid objective risk calculator may be a useful tool that could support the accurate understanding and communication of potential health risks.

Risk Assessment in Oral Health

The reliable assessment of risk of disease is central to effective decision-making both in medicine and in dentistry. Yet, there are numerous research studies to show that treatment decisions and planning can lack consistency, in medicine as well as

dentistry where clinicians rely solely on clinical opinion to make these judgments [16]. At the same time, there seems to be a strong belief that the correct identification of disease risk factors could predict and lower the incidence of, e.g., periodontal disease and in doing so, such information may be readily used by dental teams in order to change patient behaviour [17]. In particular, in this focus-group study of US dental teams, participants were found to be strong advocates of using a risk assessment tool to educate patients and, building on this, using the tool as a means to support patient behaviour change. The view that was voiced here was that the research community should move onto supporting dental teams to use these tools during patient consultations, explicitly with the view of helping patients engage in the lifestyle changes necessary to arrest the development of oral health disease. In doing so, work from the behavioural sciences explicitly looking into supporting behaviour change through the use of objective risk assessment may have the answers that dental teams reported they needed. This work is reviewed next.

Theoretical Attempts to Use Risk to Change Behaviour

Psychologists are interested in systematically predicting behaviour and risk as a tool to do these features in several models of health behaviour change. Behind these models rests the assumption that giving people information is not enough in itself to change behaviour, but risk information supplemented with other behavioural variables might be a successful combination [3]. Two psychological models that propose risk understanding as one of several factors that might support behaviour change in people are summarised below. Whilst neither of these has been developed explicitly for use in dentistry, some of the insights they provide may be helpful.

The Health Belief Model

The Health Belief Model [18] (HBM), has been studied extensively in various health settings, patients and health conditions. Researchers have used it to predict behaviour change in the uptake of diverse behaviours, from genetic screening, to the uptake of influenza vaccinations, to adhering with diabetes, hypertension and renal disease regimens, contraceptive use, smoking and drinking [19, 20]. It is a comprehensive model aiming to break down the building blocks of undertaking a new health behaviour and to identify the inter-relationships between those components.

The model suggests that patient beliefs about a health behaviour are likely to influence whether they engage in the behaviour. Risk here takes the form of patient beliefs in terms of how *susceptible* they believe they are to an illness and how severe they perceive that illness to be; at the same time, patient beliefs about the benefits and barriers associated with taking corrective action in the light of disease risk will influence whether patients engage in a health behaviour or not.

So how might the HBM and its principles be used in a healthcare setting? Taking the dental clinic and interdental cleaning as an example, the model would propose

that the chances of a patient engaging in this behaviour would be influenced by a series of beliefs, as follows; a person would need to believe that they were at some high risk of developing periodontal disease (susceptibility to a health threat), that disease was a serious problem (severity of the health threat) and they were sufficiently concerned about it to want to do something about it (health motivation). If they then thought that inter-dental cleaning was time consuming (barrier) but that this behaviour would lead to fresher breath and avoidance of the disease (benefits) they would be more likely to engage in interdental cleaning. Where patient beliefs were also supplemented by 'cues to action', e.g., internal cues such as an unpleasant taste in their mouth and/or external cues such as people telling them they had bad breath, the model predicts the person would be more likely to clean interdentally than someone who did not think they were susceptible, did not worry about periodontal and disease and saw barriers but no benefits in interdental cleaning.

It is evident how these variables predicted by the HBM can be used in clinic; in fact, many dental teams may well be using some of the model's components, particularly severity and susceptibility, as part of routine consultations. Warning patients of future risks of disease is probably part of most routine consultations! The theoretical evidence for the model's effectiveness however is rather patchy. Although the model arose out of early work that included studies on dental patients [21] there has not been extensive, convincing evidence about its ability to predict any wide-ranging health behaviours in oral health settings. In this early work, for example, it was shown that perceived susceptibility to the worst imaginable dental problems, coupled with the belief that regular visits to the dentist might prevent these problems, were useful predictors of patients' frequency of dental visits over the next 3 years. Later on [22] a US study examining women's success in engaging with preventive dental behaviour reported that such behaviour was strongly related to their health beliefs. The dental visit, as opposed to toothbrushing and dental flossing, had the highest level of predictability. These findings were taken as supportive of the role of health beliefs in predicting behaviour change and the Health Belief Model was put across as a viable predictor of the undertaking of healthy behaviours.

Protection Motivation Theory (PMT)

The understanding and accurate perception of risk is central to this theory that sees health behaviour change as being influenced primarily by emotion and in particular by a fairly basic one—that of *fear.* In this model, fear of ill health and in particular people's innate desire to protect themselves from harm is said to underpin people's efforts to engage in behaviours that are going to minimise risks and be protective to health.

Protection Motivation Theory [23] (PMT) proposes that when people receive health information, such as risk of disease, that they may find threatening, this process sets off two parallel thinking sequences: a risk appraisal process, i.e., where the person works out how much they feel threatened by the information they have received and a coping appraisal, where the person is said to attempt to evaluate the

extent to which they feel they have the resources and ability to deal with the threat. These appraisals are in turn said to result to either an adaptive, healthy and helpful or a maladaptive and unhelpful (and normally unhealthy) response. The model suggests that where people find a health threat serious and feel they are at high risk, but also feel they have the psychological resources to deal with it, they will form the intention to engage in a threat-minimising behaviour and then, accordingly, perform the behaviour. For example, where a patient is told that they are susceptible to developing oral cancer and that oral cancer is a serious disease this information is going to activate their threat appraisal system. For the person to engage in health behaviour to deal with their risk of developing oral cancer, the model proposes that their parallel coping appraisal system also needs to be activated through a conversation that will highlight the person's resources and ability to mitigate the risks and deal with the threat. In the absence of such a conversation, the model proposes that the patient is unlikely to engage in health behaviour change but rather, be left in a place where they feel threatened and unable to cope.

The difficulty with both of these models lies in the fact that they both seem to perceive people as objective, cognitive processors of information. Although fear features to a large extent as an emotion in PMT, the HBM in particular treats people as information processors devoid of any emotion. That is a major shortcoming of the HBM in that a lot of decision-making in humans is known to be '*primarily determined not by facts but by emotions*' [24]. For this reason, work looking to examine how risk information might impact behaviour change in dental settings has used PMT rather than HBM as a theoretical model to underpin its design. This work is presented next.

Risk Assessment to Change Behaviour in Periodontal Disease

A study by our team [25], based on the PMT model described above and using a reliable, validated oral health risk calculator [5, 26, 27] showed that data from a standardised risk calculator assessment and the use of a theory-based communication protocol can be effective in preparing periodontal disease patients engage in behaviour change with the view of reducing their future risk of the condition.

This study, carried out at a large UK dental school periodontal clinic, recruited adults (N = 102) with moderate/advanced chronic periodontitis who had been referred to the clinic for an assessment. Patients completed a self-report measure, based on the PMT model, to examine their thoughts about their susceptibility and severity to periodontal disease (that is their threat appraisals), as well as their beliefs about their self-efficacy and response efficacy to dealing with the threat of this condition (that is, their coping appraisals), before a periodontal assessment and again at the end of the visit. They also completed an assessment of their emotions surrounding periodontal disease, looking at both positive emotions (e.g., cheerful, happy) and negative emotions (e.g., scared, jittery). They were then randomly allocated to one of two groups: a 'treatment as usual' group and an intervention group. Both groups had the same routine clinical assessment, but the two groups were then treated differently post consultation.

Those in the intervention group spent 5–10 min going through an individualised calculation of their periodontal disease risk using the PreViser Risk Calculator (www.previser.co.uk). Using the objective analysis of a patient's oral disease risk and severity in the form of colour coded charts provided by the software, a standard script explaining the risk information provided by PreViser was developed and followed throughout. Specifically, patients were taken through the PreViser output where they had a conversation with a researcher about the idea of risk. Here, patients were offered one-to-one conversations about factors, such as smoking, for example, that might adversely impact one's risk score. The patient's specific risk profile and where their risk sat in a 1–5 risk score scale, was then brought to the conversation. The discussion focused on lifestyle and oral health factors that might impact those risk scores. An explanation of the patient's personalised PreViser disease scores was then offered, with a particular emphasis on what the number meant, in relation to the PreViser 1–100 disease score scale. The conversation ended with an exploration of patient reactions to their own risk and disease scores and ways the patient felt they might follow periodontal treatment advice in an attempt to reduce these risks and increase coping appraisals. Usual treatment group participants on the other hand, engaged in a question and answer session about general oral health.

At the end of this session, all participants completed the same assessments they were exposed to at the start of the consultation, measuring their thoughts and feelings about periodontal disease as well as their intentions to adhere to the dentist's periodontal disease management advice. The study found that those patients who had received the treatment as usual consultation in the absence of a conversation about individualised risks based on a PreViser assessment, felt that periodontal disease was serious and that they were quite susceptible to it. In other words, post consultation this group of patients reported feeling rather threatened by the prospect of the disease. In contrast, the intervention group reported feeling as threatened as the usual care group but, in addition, the intervention groups reported greater belief in their own ability to follow through with periodontal disease treatment, greater faith in the effectiveness of the periodontal disease treatment on offer and higher intentions to engage with such treatment. What this study was successful in showing was that a personalised discussion of risk scores with these patients had the additional benefit of preparing them to engage in behaviour change by activating not just their threat but also their coping appraisals. This is important as preparedness to engage in lifestyle change as seen in an intention to do so, is key to actually engaging in behaviour change.

In a more recent study [28] we endeavoured to add to the findings of this work by investigating whether the psychological preparedness we had witnessed in the periodontal patient study described above would in fact translate into clinical outcomes. In this second study we recruited and tested adults ($N = 97$) with a history of moderate oral hygiene from a general dental practice in London, UK. The patients were again, randomly allocated into one of three conditions; a treatment as usual group, a group where, as in the earlier study, personalised risk information using PreViser was offered, and a third group who in addition to the risk conversation they were also supported in goal-setting, planning and self-monitoring their

planned behaviour change. Patients were assessed before a routine clinical assessment, 4 weeks later and then again at 12 weeks on a range of self-reported psychological (PMT), behavioural (self-reported brushing and interdental cleaning) and clinical (Plaque and bleeding) outcomes. The strength of the study lay in assessing a combination of psychological behavioural and clinical outcomes and to our knowledge was the first study to show the impact of a simple behavioural intervention based on risk assessment on clinical treatment outcomes in periodontal disease settings. The study reported some really encouraging results. Both intervention groups appeared to show a plaque reduction (of around 10%) at 4 weeks which was then maintained at 12 weeks. The treatment as usual group's plaque came down by around 3% but that difference was not substantial enough to be considered statistically reliable. Bleeding reduced in all three groups but more so in the intervention groups—again, a difference that was observable 12 weeks after our initial intervention.

It would appear then that using a reliable, objective method to calculate risk and then standardised communication protocols to deliver this information to patients in a way that is, as predicted by theory, likely to increase patients' motivation to engage with behaviour change may well be a fruitful exercise in the dental clinic.

The limitation of this work is of course that both studies have only considered periodontal disease patients and in both cases the very long-term outcomes on behaviour and clinical indicators remain to be examined. These data however are encouraging in that they seem to suggest that objective risk assessment can be fruitfully used as a tool to support behaviour change in patients seen in dental settings and as such might be a low-cost effective way of arresting disease and improving health outcomes.

Summary and Next Steps

This chapter has highlighted some research findings from general psychology, medicine and dentistry that point to the shortcomings of people's understanding of risk-related information. It has also shown how the use of objective risk assessment calculators can augment clinical communication of such risk information so that such information is used not just to educate patients (i.e., in an information-giving exercise) but to motivate them and support them in considering engaging in health behaviour change. The limitations of the work carried on so far lie in the fact that the data we have are rather limited by context and clinical condition; for instance, little is known as to whether communicating risk information in ways that are underpinned by psychological theory may be effective in modifying behaviours that give rise to caries. The small samples used in the studies and the demographics of such samples may be areas to consider in designing future work in the field.

Nevertheless, the work described here is potentially a sound basis upon to suggest that the use of clinical risk calculators and the subsequent communication of these findings to patients using established behavioural methods may be a good means of helping patients engage in health behaviour change.

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Part III

Principles in Risk Assessment

Risk Driven Care Pathways in Publicly Funded Care

Eric Rooney

Contents

Introduction

Previous chapters have set out the approach to risk assessment for various diseases. This chapter focuses on the use of risk-driven care pathways in publicly funded care. The reasons why the public sector supports risk assessment are explored, and examples of policy approaches are described. The development and specific use of risk assessment in publicly funded dental systems are outlined, along with emerging evidence around their acceptance and effectiveness.

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The Public Sector Case for Risk Assessment

Why should governments and the public sector invest in risk assessment models? The reason lies not in risk assessment for its own sake, but in its key role in preventing disease and empowering the public to start taking responsibility for their own health. Throughout the world the importance of preventing disease where that is possible, rather than simply treating it when it occurs, has been recognised. Both the direct costs (health services provision) and the indirect costs (impact on education, workforce and the economy) of poor health and disease have a substantial impact on the public sector purse and the economic prosperity of nations. Globally, people are generally living longer, but in their later years of life, the population elders are the greatest users of health services and resources [1]. In the UK, the Treasury, concerned at the future costs of healthcare, commissioned a review: "Securing our Future Health: Taking a Long-Term View" which highlighted the importance of public engagement and prevention in its most optimistic forecast for future investment [2].

Over and above the economic aspects, a societal and moral case has been advanced that governments have a responsibility to promote a healthy population and reduce inequalities in health

> "…health inequalities that could be avoided by reasonable means are unfair. Putting them right is a matter of social justice." (Sir Michael Marmot) [3].

Inequalities in health are frequently associated with social circumstances, as illustrated by differences in life expectancy, and also in the difference in years of disability-free life expectancy (Fig. 1).

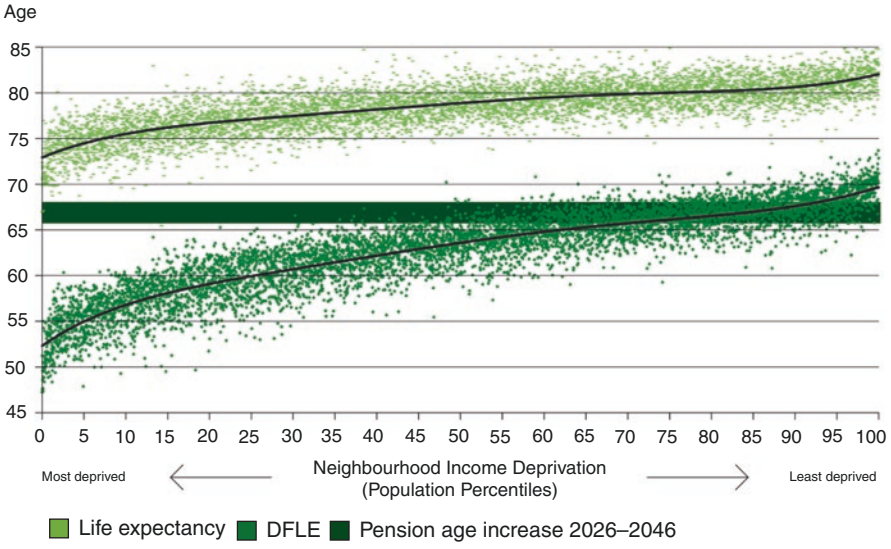
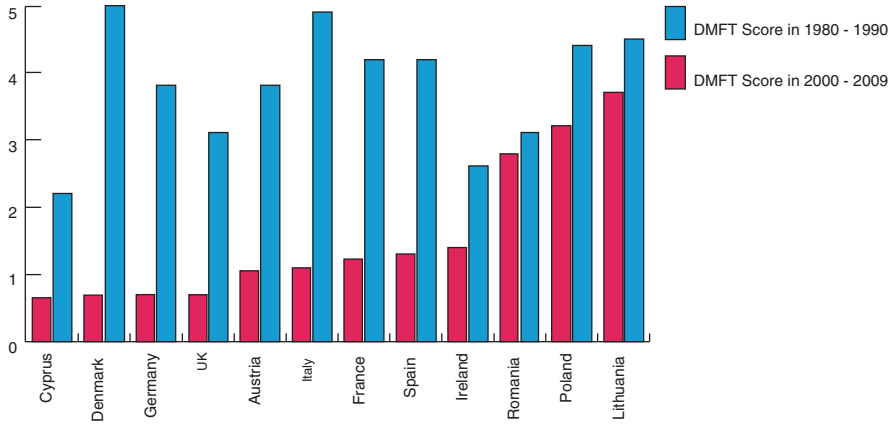


Fig. 1 Inequalities in life expectancy and disability-free life expectancy by neighbourhood income deprivation level. Source: [3]



Notes:
- Ireland: DMFT score for children receiving fluoridated water at home since birth
- DMFT score for Poland in 2003 was ascertained from examination of 180 children in Gdansk region
- DMFT score in UK in 2008-2009 is for England only

Fig. 2 Inequalities in improvements in oral health (caries) in Europe. Source: [7]

Inequalities in oral health have also been widely observed both between and within countries and also show clear association with socio economic circumstances and lifestyle behaviours [4–6]. Although there has been an overall trend towards improvement in oral health across Europe, there are considerable inequalities between countries in the rate of improvement (Fig. 2) [7] (Direct comparisons of DMFT scores between countries should not be made due to differences in methodology).

It is the focus on controlling health service costs, the recognition that poor health impacts on economic prosperity, and the moral case for health and reduction in inequality that drive the support and use of risk assessment and prevention in publicly funded healthcare.

Health Policy

Almost universally, governments have come to realise that simply treating disease is not a sustainable approach to improving health and wealth. People are living longer and as advances in medical science allow increasing opportunities for treatment, health costs are rising, particularly the spend towards the end of life. In recognition of this and the case for risk assessment and prevention set out above, Governments are broadening their objectives for their health systems. For example, the countries of the European Union set their health vision for 2020 as being to [8]:

- Reduce premature mortality in Europe by 2020
- Increase life expectancy in Europe
- Reduce inequities in health in Europe
- Enhance the well-being of the European population

- Provide universal coverage in Europe
- Establish national targets set by Member States

In England the Department of Health and Social Care sets out its objectives for the NHS in its mandate [9]. This includes:

- To commission services to improve local and national health outcomes, and reduce health inequalities
- To lead a step change in the NHS in preventing ill health and supporting people to live healthier lives

Prevention Strategies

In seeking to deliver their health and well-being policies, approaches to prevention adopted by policy makers may be described as Primary (preventing disease before it starts), Secondary (identifying disease early) or Tertiary (preventing complications or recurrence of disease), and can be focussed either on populations or individuals. Whether aimed at populations or individuals, understanding the risk of being affected by a disease or condition is an important factor in the design of the preventive approach.

Prevention at a population level

- At a population level, the main focus is on primary prevention. It is widely accepted that high salt intake is a risk factor for cardiovascular disease and the European Observatory on Health Systems and Policies estimates that cutting salt intake through regulation and food product reformulation led to a gain of 44,000 life-years in good health in England, with savings in health care expenditures largely offsetting implementation costs [10].
- Fluoridation of water supplies has been shown to reduce levels of dental caries in the population [11, 12] and recent fiscal policy, introducing a tax on high levels of sugar in soft drinks in England, has led to the reformulation of many soft drinks products with an expectation of having some impact on dental caries as well as obesity.

Prevention at an individual level

- At an individual level, all three approaches to prevention are evident in the policy approach, based around the stratification of patients into those who are presently free of disease, those who have it where it can be treated or reversed, and those who will need to live with their condition and manage it.
- For example, advice on smoking and alcohol, dietary advice and the use of fluoride toothpaste for caries, and toothbrushing and plaque control for periodontal disease [13] are all appropriate whether the patient has never had the disease, currently has active disease, or is recovering from, or living with the disease.

It is widely recognised that that most effective strategies for prevention and improved health involve a combination of population and individual approaches

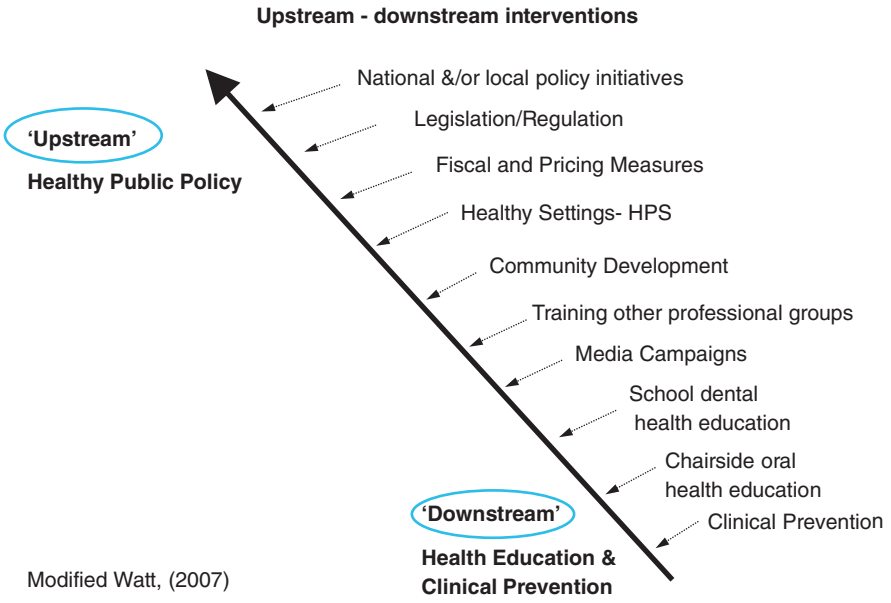


Fig. 3 The Upstream Downstream approach to promoting good oral health and preventing poor oral health. Source: [25]

aimed at pre-diseased states, post-treatment states and increasingly long-term condition management.

The use of a range of interventions, recognising the determinants of health, is the most effective way not only of improving health overall, but in tackling the inequalities in health which impact on people's lives.

Figure 3 illustrates the range of interventions from public health policy (upstream) to individual preventive advice and treatment in the dental surgery (downstream)

There has long been debate about whether it is best to focus prevention on whole populations, or to target at-risk groups or individuals. Current thinking recognises the need to provide everyone with the basic knowledge, information and opportunity to lead a healthy life, but if we want to reduce inequalities, then we should proportionately increase our support and interventions for those more at risk. This policy approach is described as "Proportionate Universalism" [3].

This approach has been employed in Scotland's Childsmile programme [14] which aims to reduce inequalities both in dental health and access to dental services. All children receive the core approach and those most at risk receive additional interventions and support (Table 1).

Risk Assessment and the Use of Care Pathways

In seeking to maximise the effectiveness and efficiency of both public and privately funded healthcare, health systems have increasing looked to the use of care pathways. The European Pathway Association defined care pathways as "a methodology

Table 1 Universal and targeted interventions in Scotland's Childsmile programme

Every child in the population has access to:
• Free dental packs to support toothbrushing at home
• A tailored programme of care within primary care dental services
• Free daily supervised toothbrushing in nursery
Additional support is targeted at children and families in greatest need through
• Additional home support and community interventions
• An enhanced programme of care within primary care dental services
• Clinical preventive programmes in priority nurseries and primary schools and facilitation into dental services as appropriate.
• Daily supervised toothbrushing in the first two years of primary school

Source: [14]

for the mutual decision-making and organization of care for a well-defined group of patients during a well-defined period" [15]. A more detailed definition has been suggested in the Seattle Dental Care for Elders Pathway:

> A care pathway describes at a fairly high and broad level the necessary evidence-based or evidence-informed steps or stages, which will take a patient towards an expected or planned outcome with a high degree of certainty. Each stage may deploy much more detailed clinical guidelines, protocols, policies or procedures either developed specifically for the pathway or use existing established guidelines or protocols. The process of pathway development should recognise the factors that affect implementation and this should influence pathway design [16].

There has been much discussion about the status of care pathways in a medico-legal context, but it must be remembered that they are there to guide the clinician. The degree to which clinicians deviate from care pathways is a matter of judgement and the clinician's understanding of the alternative evidence.

It is often the case that one of the stages of a care pathway will involve some degree of risk assessment, and that this risk assessment may be revisited over the course of care, particularly when dealing with long term, rather than acute conditions.

Examples of Risk-Driven Care Pathways in Publicly Funded Care

Across the world, cardiovascular disease, respiratory disease and cancer are the main cause of death [17], and in England this is supplemented by liver disease [18]. Risk factors associated with these and other diseases may be classified as modifiable or non-modifiable. Modifiable (extrinsic) factors include smoking habits (cardiovascular and respiratory), alcohol intake (liver disease) and non-modifiable (intrinsic) factors include age and genetic factors (family history), although gene therapy approaches may offer advances in this area in future. In recognition of the impact of cardiovascular disease and the potential for prevention through the management of modifiable risk factors, publicly funded health systems commonly invest in risk-based programmes, with the European Society of Cardiology

Table 2 Associations and common risk factors between oral and systemic diseases

	Cardiovascular disease	Obesity	Cancer	Diabetes
Caries		Sugar and diet		Sugar and diet
Periodontal disease	Inflammation, smoking, sugar and diet	Sugar and diet, inflammation	Smoking, inflammation	Inflammation
Tooth wear		High carbohydrate drinks		High carbohydrate drinks
Oral cancer	Smoking, inflammation	Alcohol	Smoking, alcohol	Inflammation

recognising eight different cardiovascular disease risk estimation systems. In England the QRisk system has been introduced into the national Health Checks programme offered by the NHS. In addition to using risk for the prevention of disease, primary care clinicians are increasingly using risk-based systems to identify factors in elderly patients which may lead to hospital admission. In this case the objective is not disease specific, but involves a range of factors with the aim of reducing the pressure on acute care and providing a better quality of life for the patient.

A number of the modifiable risk factors for our major killers are common to oral diseases and have been discussed in the relevant preceding chapters. These include smoking, alcohol and diet. Table 2 shows the relationships.

It therefore makes sense that we begin to think about the benefits of our risk-based pathway approach in dental care not just in terms of oral health but of general health also, and this approach has been tested within the NHS dental system in England.

In 2009 the Department of Health commissioned an independent review of NHS dentistry which set out the priorities for public investment in oral health and suggested the focus should be public health, prevention and the delivery of care through a pathway approach [19].

Following the review, a risk assessment and pathway was developed by a multidisciplinary group involving academics, policy makers, general dental practitioners, dental specialists and members of the public. This has been running in pilot practices in England since 2011 with minor modifications over time. The pathway includes a risk assessment stage and focuses on four oral diseases: caries, periodontal disease, tooth surface loss and soft tissue pathology. The pathway also includes two existing guidelines on evidence-based prevention and on the appropriate period for oral health review, both of which are risk based. Figure 4 shows the overall pathway in schematic form.

The risk assessment element of the approach takes patient factors and clinical factors into account and suggests a risk category of red, amber or green that the clinicians use to guide their conversations, preventive advice and treatment for patients.

Figure 5 shows the factors involved and the way risk is determined for caries and periodontal disease.

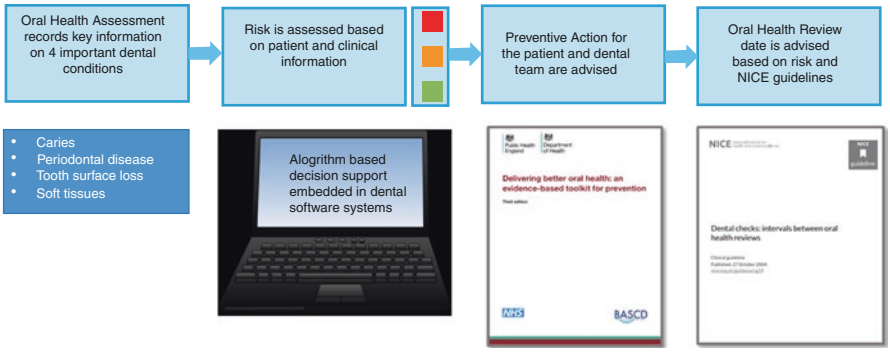


Fig. 4 Dental contract reform in England—pilots and prototype care pathway

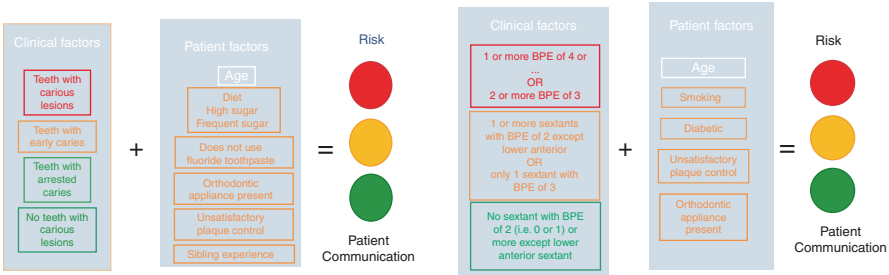


Fig. 5 Dental contract reform in England—pilots and prototype risk assignment

Outcome evaluation of the programme shows changes in individual risk for patients from red to amber and amber to green, and maintenance or improvement in both caries and periodontal disease in patients having more than one oral health review [20].

In terms of process, use of the pathway by clinicians is high, with around 90% of patients receiving the care pathway approach, and dentists feeling the pathway enables them to provide better care with the potential to improve oral health. Patients also find the risk driven pathway acceptable and beneficial, with 76% reporting they had changed the way they cared for their teeth/gums [21]. The pathway includes common risk factors for our big killers as discussed earlier, and although there were initial concerns regarding the focus within the pathway on general as well as oral health, most patients felt comfortable discussing diet, smoking and alcohol. There is also evidence that recall intervals for oral health review are being based on risk scores, although both patients and dental professionals remain reluctant to set 24-month intervals for those at low risk as recommended by the National Institute for Care Excellence (NICE).

This risk-based pathway and similar protocols which look to make patients "partners in care", taking responsibility for their own health, are not for

everybody and not necessarily at all times in their life. The preventive activities often require behaviour change for the patient and this is a complex area. It is generally recognised that patients may well have the requisite information and know what they should do but their balancing of the risk within the wider complexity of their life affects their readiness to change. Most behaviour change models focus on professional intervention when the patient is ready. Recognising this, the Very Brief Intervention (VBI) approach recommends asking whether the patient wants to change before delivering or referring for more in depth prevention advice. NICE offers guidance on individual behaviour change, including VBI [22].

Risk Driven Pathways in Health Systems

Although this chapter has focused on publicly funded care, the delivery of risk-driven pathways needs to take place within health delivery systems. Increasingly, countries are identifying that the traditional competitive model between hospitals, community services, primary care and social services is not sustainable and there is an increasing focus on collaborative working. The need to address care services and preventive services at both an individual and population level means that greater integration across traditional professional and organisational boundaries is required. There is evidence from the USA that groups of clinicians and care workers looking after populations of between 30 and 50,000 are able to work together to improve the quality and efficiency of care, and this has been developed further by the national association of primary care in the UK [23]. Their programme recognises the potential for dental practices and their teams to play a wider role in supporting these local populations [24]. For example, periodontal input being employed as part of the local diabetic pathway, or dental support being made available in local care homes. In the dental surgery, as well as oral health care and prevention, the opportunity for "making every contact count" (MECC) has been recognised. This means provision of advice on smoking cessation, alcohol consumption, and blood pressure monitoring as part of local risk driven pathways to improve general health is now on the dental agenda.

Figure 6 brings together these ideas and illustrates the components of an integrated health system.

Key to the delivery of integrated systems of care is the ability to use and share data across organisations and clinicians to support individual patient care, but also as "big data" to support risk profiling, targeting of interventions and evaluation of outcomes. At the individual level, many current systems in general medical practice can identify different risk factors within the patient's record and produce risk scores for patients that will support the development of care plans aimed at preventing unplanned hospital admissions. This benefits both the patient and the overall healthcare system.

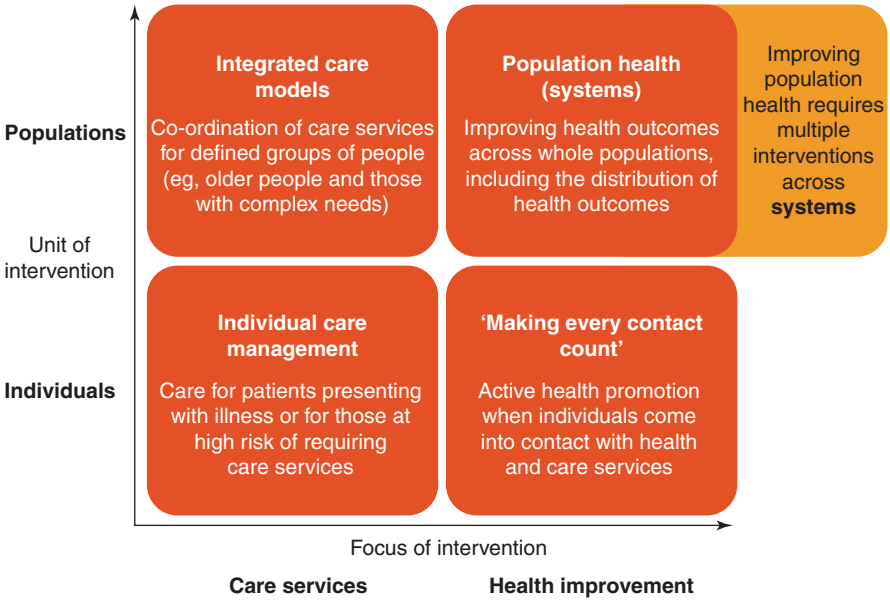


Fig. 6 Integrated population health systems. Source: [26]

Conclusions

In this chapter the challenges facing governments in publicly funded healthcare have been identified and an economic and moral case advanced for developing an increasing focus on prevention.

The importance of considering preventive approaches at both a population and an individual level have been highlighted, recognising that a clear understanding of the aetiology of disease and the risk factors contributing to development and progression are key to both effectiveness and efficiency.

Examples have been given showing how the intensity of preventive programmes may be related to risk through the proportionate universalism approach for populations, and how risk is incorporated into care pathways for individuals. Specific dental examples have been given and have proved popular with both the dental profession and the public.

The growing recognition of common risk factors across diseases (including dental diseases) has been explored along with the drive by heath systems to focus on risk-based pathways, leading to planning of much greater integration across health and social care delivery systems.

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Risk Driven Capitation Models

Roger Matthews

Contents

Introduction

It has been said that: 'There are no good ways to pay dentists; however, fee for service, salary and capitation are the three worst' [1]. Whilst this may be a tongue-in-cheek overview, there is little doubt that there are strong proponents for each of these, the three most common remuneration approaches in primary dental care, as well as for 'blends' of all three models.

In those areas of healthcare provision that follow a surgical model, fee-for-service has historically predominated. This is true of primary dental care, reflecting as it has done a past predominance of 'activity-driven' practice. However, dentistry differs significantly from other healthcare domains, especially that of primary medical care. Indeed, the advent of a preventive approach to the management of the most

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Table 1 Contrasting primary dental and primary medical care

Aspect	Primary dental care	Primary medical care
Principal disease management	Almost wholly preventable	Variable impact of preventability
Nature of diseases	Mainly chronic, widespread and specific	Acute and chronic, mainly sporadic and varied
Patient presentation	Mainly asymptomatic and regular	Mainly with symptoms or concerns, irregular
Impact of diseases treated	Mainly non-catastrophic	May be life-threatening
Professional career paths	c.80–90% generalist	c.50% generalist; 50% specialist
Historical focus of care	Invasive, activity-based	Advice, management and activity spectrum
Professional structure	Relatively individualist	Generally well-structured and organised

prevalent oral diseases, and the maintenance of health and wellbeing has further accentuated this distinction (see Table 1).

The adoption of a preventive approach to common oral diseases—which has come about through a greater understanding of their nature, underlying processes, causation, epidemiology and progression—has largely taken place within the last hundred years and accelerated in the past half century [2, 3]. The important relationships between oral and general health are also reflected elsewhere in this publication. Such improvements in scientific knowledge, whilst fundamental to a preventive healthcare approach, have additionally been mirrored by important societal changes over the same time periods.

The organisation and availability of health care, including in many cases oral and dental care has changed fundamentally in most developed societies. Bismarck is generally credited with the introduction of social healthcare provision in Germany in the nineteenth century [4], and other developed countries have followed suit to varying degrees, at different speeds, and—notably—in a broad and individualised spectrum of ways. However, one factor common to all approaches is the transformation of healthcare delivery from a binary (clinician and patient), to a three-party system, whereby a state, approved insurer, plan provider or social enterprise becomes a significant factor in the funding of care provision.

This change brings with it both advantages and tensions [5]. No longer is the healthcare relationship a bipartisan contract with relatively straightforward objectives and deliverables on each side. Issues of risk, efficiency and effectiveness, prioritisation, funding and affordability come into play. Indeed, to a great extent, the quotation at the beginning of this chapter clearly hints that what is acceptable or indeed a pre-requisite for one party, may not be to the other two.

It should also be noted that a further important societal change has, in parallel with scientific knowledge and healthcare organisation, radically impacted oral healthcare: the rising significance of aesthetics and appearance in the context of wellbeing [6]. As the mouth is a prominent human feature, dental professionals and their patients now place a great emphasis on the maintenance and preservation of healthy—moreover, socially desirable—oral tissues over an extending lifespan. All

of these factors have increased the importance of 'dental benefits' within healthcare systems in response to consumer demand.

Dental Payment Systems

Funding for primary dental care has evolved, in consequence of the 'revolutions' identified in the preceding section, to address the concepts of accessibility, affordability and quality of care. Whereas prior to the second half of the twentieth century the overwhelming majority of primary dental care was offered on a 'fee for service' basis, with the nature and quality of services dependent almost entirely on an individual patient's ability to pay, rising societal demand has given rise to a number of systems: state funded, privately funded or a blend of both. As with healthcare funding overall, a plethora of approaches has emerged. Four principal approaches now exist:

Direct Payment

As well as direct lump-sum payment, clinicians may offer terms such as payment by instalments (either self-managed or via a third party) as well as loyalty discounts, or pro bono care for dependants. There may be a tendency for patients to be treated on an episodic basis. Also referred to as 'out of pocket' and 'fee for service' (FFS). Historically predominant.

Societal Payment

Where an administration legislates that certain individuals (for example: children, the under-privileged, lower earning, those with disabilities, the armed forces) or certain treatments (e.g. pain relief, facial trauma, oncology) should be made available free of charge or discounted, then policies may be enacted to provide funding from general or hypothecated taxation (the 'Beveridgian' model). Alternatively, mandated social insurance by approved bodies may be chosen (the 'Bismarckian' model). Whilst such prioritisation is found in many developed societies, it is not universal. A Government may also provide subsidies from general taxation for routine dental treatment to the whole population as in Sweden, or the United Kingdom (UK). Such a system may by its nature be rationed explicitly or implicitly, or subject to means testing or co-payment by patients and tariff structures for providers.

Pooled Payment

Although often described as 'dental insurance', the high, indeed nearly universal, prevalence and nature of dental diseases necessarily implies that their occurrence, management and treatment are not truly insurable risks (i.e. the result of infrequent

or catastrophic events). The insurance principle of 'many pay in order that few benefit' is hence inapplicable. The majority of such approaches therefore are focused on either spreading the cost of care over a period of time (frequently implying a moratorium period before claims are covered), or limiting the cost of care (through negotiated-fee clinician networks, schedules of maximum benefits, deductibles, co-payments, moratoria or a combination of these). Some providers will however include limited cover for truly non-foreseeable or major risks (such as emergency dental care or orofacial trauma).

Capitation Payment

A per-capita payment implies regular payments (as with pooled payment premiums) of a fee designed to cover the cost over a period of time for the provision of dental care to an individual patient, family or group. Fees may be set at standard rates by a third party and/or may be adjusted for the particular individual and hence the likely anticipated costs incurred by the clinician. A closed network of practices (or a single identifiable clinician or clinic) is an integral element and catastrophic cover as above may be included. Co-payments for more complex treatments may be a feature, or indeed capitation may be blended with FFS for more costly or infrequent procedures such as dental implants. Laboratory or other external costs, such as prescription medicines, advanced diagnostic procedures or referrals for specialist care may be excluded or covered by patient co-payment.

Dental Capitation Systems

Capitation as a system for funding primary dental care exists in a number of countries, each with their own national characteristics. It is proposed to briefly consider and contrast the systems and risk assessment approaches in those countries where primary dental care under a capitation system has been relatively widely used.

Sweden and the United States (US) have both adopted forms of capitation in dentistry and the history and features of these will be briefly described. Within the United Kingdom (UK), a distinct format of capitation has been adopted in the private sector, whilst Government reforms are currently (2011–2020) piloting a societal derivative. Clinical risk assessment in the UK will receive more detailed review.

Sweden

Both public and private primary dental care provision is available in Sweden through the Public Dental Service (PDS) and in 1974 a national dental insurance programme commenced covering all adults over the age of 19 years [7]. Treatment for children and young adults was funded by County (Regional) Councils through general taxation. A national tariff was introduced for all adults with generous Council subsidies, with all treatment on an FFS basis.

Proposals for the piloted introduction of capitated primary dental care funding were presented to the Swedish Government in 1990. Based on the UK 'Denplan' approach (see the UK section below), this pilot was approved initially for trialling in a single PDS clinic in Göteborg (Gothenburg) and commenced in 1991.

Zickert et al. [8] reported on the first 6 years of this pilot, during which 3114 patients were treated under capitation. A random sample of 100 capitation patients and a matched control group of fee-for-service patients from the same clinic were reviewed for change in oral health status (DMFS increment; periodontal pocketing ≥6 mm; bleeding pockets). Health deterioration was significantly less in all categories for capitation patients.

Patients were initially clinically risk assessed and assigned to three risk groupings: low, medium and high. Risk assessment criteria were based on:

- Dental history
- General health and medication
- DMFT
- Salivary laboratory tests for cariogenic micro-organisms and flow-rate
- Periodontal bone loss
- Proportion of bleeding pockets on probing
- Oral hygiene status
- Existing restorations at risk of fracture
- Presence of third molar impaction

Since one aim of the pilot programme was to influence patient motivation towards preventive care, levels of clinical risk were assigned scores which facilitated alignment with fee bands. Lowered risk thus potentially equated, for the patient, into lower future fees. The three risk bands were subsequently each divided in two, to provide six increments, later in the pilot programme (the 'Denplan' system uses five risk bands).

Under the original 'risk band' tariff system, each patient paid 300/650/1000 Swedish Kroner per month (equivalent in 2020 terms to £25/£54/£83/month) with a matching sum contributed by the County Council. Whilst this reflected the comparatively high cost of healthcare in Sweden, it was also indicative of the generous social contribution and higher taxation rates in Scandinavian countries. However, 98% of the patients who chose it expressed preference for the capitation system.

Subsequently, all of the 20 County Councils in Sweden have adopted capitation as a means of payment for publicly funded dental healthcare [7]. There have been a number of central Government legislative changes in the intervening years and capitation is now offered in all Counties/Regions albeit with differing numbers of risk bandings and some regional variations in funding costs. Under this 'Dental Care for Health' (Frisktandvård) model, patients are risk assessed using a PDS model which includes the Cariogram© tool.

The online Cariogram tool uses four dimensions of risk for, and resistance to, dental caries: diet; bacterial colonisation and plaque level; fluoride availability, saliva flow and buffering; past caries experience and relevant medical history [9]. The output gives a percentage risk of future cavity formation. Evidence for

correlation of standardised caries risk assessment tools (CRA) remains weak: whilst Anup and Vishnani [10] reported overall support for Cariogram in 17 of 19 studies reviewed, Cagetti et al. [11] reported that seven of 32 papers reviewed showed sensitivity was low/medium low, although specificity was slightly higher. However, overall validity was limited.

A number of recent studies have considered the impact and effectiveness of capitation-funded primary dental care in Sweden. A longitudinal six-year study of 6299 dental patients showed that capitation patients demonstrated fewer manifest carious lesions than fee-for-service patients, after controlling for age, gender, education and pre-baseline caries incidence [12]. Capitation patients also received fewer restorative interventions. Notably, dentists working in the PDS are salaried [13].

Hakeberg and Boman [14] reported a telephone self-perception survey of 3500 patients across Sweden showing that satisfaction with care was higher amongst the capitation-funded cohort. Whilst overall satisfaction with Swedish dental care was generally high, 58% of capitation patients self-reported being 'very satisfied' compared with 25% of fee-for-service patients. Patients who selected the capitation option (19.7% of those receiving care under PDS) were younger and rated themselves as having better health, higher activity rates and higher household income than FFS patients.

On this last point, Petersson and Twetman [15] sound a note of caution. In their study of young adults choosing between FFS and capitation care, they note that those with better baseline oral health tended to choose capitation. Given the known and reported socio-economic gradient of caries incidence, there is a clear possibility that disadvantaged individuals with higher risks of oral disease are further excluded from care by lower risk capitation patients and by the fees charged for prospective entrants at high risk. Ironically, they also report that an 'inverse care law' may apply in capitation patients, whereby more preventive advice was given to the 'low' and 'medium low' risk patients than those in the 'high' and 'very high' risk groups. They identify the potential for capitation systems to worsen inequalities in population delivered dental health. Norderyd provides a detailed description of two classical Swedish risk-based prevention schemes in Chap. 'Impact of Risk Based Prevention in Public Oral Health' of this textbook.

United States

Capitation dentistry in the United States (US) may be considered to have originated in a limited form in 1954, when a longshoreman's union set up a mutual fund for delivery of dental care under a capitated system [16].

Growth of the dental insurance market accelerated in subsequent decades. In 1960, when it was estimated that 75% of the US citizens held medical insurance, only 0.5% had dental coverage [17]. However, by 1980, it was anticipated that up to 75 million US patients would hold pre-paid dental plans of some form [18].

Studies were undertaken in the 1970s to calculate the costs of providing primary dental care using predictability and analysis of clinical treatment time [19, 20]. These were based on 'whole population' models, with little emphasis given to the individual disease risks of patients. In general, caries and prosthodontics were given primary attention.

There was much professional concern and antipathy to the growth of many for-profit intermediaries and the many options offered. Capitation was mainly group-based and as Schoen—an early enthusiast for the introduction of capitation—noted, surveys revealed that administration costs were high, premiums were low and under-utilisation and under-treatment were endemic [21]. The option of capitation fell from favour. Atchison and Schoen were also critical of the quality and thoroughness of care and record-keeping in both FFS and capitation practices [22].

Rhodes [14], in looking back over a 13-year period, noted that whilst employer-based dental plans accounted for 50% of dental practice receipts, only 19% were capitation based. In an analysis of one of the largest capitation providers, he found that reimbursement increments fell behind dentists' practice costs in each year. Between 1996 and 2008, fees (including co-payments) fell by 19.6% whilst costs rose by 46%. In the same period, average wages increased by 34%.

Dental capitation in the US overall has not, it seems, met with the approval of many in the dental profession. The need to assess risk for disease, care and treatment on a population basis (since most plans are based on group enrolment) means that there has historically been little scope to address individual clinical risk and predisposition to oral disease. This has been combined with the need to maintain premiums at a low/affordable level whilst delivering profit to intermediaries or work within publicly funded budgetary constraints. However, recent advances in individual clinical risk assessments have been facilitated by the development of more effective and practical algorithms as further detailed in the chapter by Loeb and Mills (*qv*).

United Kingdom

National Health Service

In 1985, trial capitation projects were set up in the UK for paediatric primary dental care to test the possible efficacy of such an approach with the National Health Service (NHS) [23]. Although the outcome of these trials was equivocal [24, 25], and included concerns about the prevalence of 'supervised neglect' by clinicians, the UK Government, proposed a revised national NHS Dental Contract in 1990 adopting some of the principles of capitation in endeavouring to move away from the purely FFS schedule adopted at and since the creation of the NHS in 1948.

No clinical risk assessment as such was involved in the reformed NHS Dental Contract and child capitation fees were (initially) set by age range alone.

As well as providing capitation-based NHS care and treatment for children and young people up to the age of 18, a small 'continuing care' payment for adult patients also became payable to dentists for each person who registered with a specific NHS-contracted dentist (the vast majority of the profession at that time). The intention was to move towards a preventive and longer-duration pattern of care and away from episodic and relatively activity-heavy 'six-monthly' treatment plans. In the event, these reforms had unintended consequences, as more patients than anticipated registered for care, leading to significant fee reductions for dentists and subsequent amendments to the scheme [26].

In 2006, a further reform to the NHS dental contract resulted in a novel (and untested) model of primary dental care funding. This met with significant criticism from professionals and politicians [27], and in 2009, Steele published a government-commissioned report on further reforms [28]. Capitation was recommended as an option because 'A dentist who knows that they have a long-term commitment has a strong incentive to provide good preventive advice and support and to carry out the treatments that they believe will have a long-term benefit to the patient'. Piloting (and subsequently prototyping) of capitation and blended capitation/FFS models as suggested by Steele and based on an initial patient risk assessment, commenced in 2011. Although interim reports have been issued [29–31], no independent studies of the validity of the risk assessment model have so far been published. This is discussed in more detail in Rooney's chapter on Risk Driven Pathways in Publically Funded Care (Chap. 'Risk Assessment for Behaviour Change': *qv*)

Denplan

In the early 1980s, Stephen Noar, a general dental practitioner in southern England, devised and operated a system whereby his patients paid privately a monthly sum for the provision of all clinically necessary care. He risk-assessed patients, and set their corresponding monthly fees paid, according to their existing oral health status and the extent of past interventions, measured by restorations present, periodontal status, plaque control and any prostheses worn.

His stated intention was to better align the interests of patients (better dental health) with those of the clinician (effectiveness and clinical freedom), whilst not disadvantaging either party. He hoped that by providing oral health education and ongoing care, patients would be motivated over time to improve their health, lower their assessed risk of future disease and reduce their fee level, commensurate with a reduced level of intervention.

He refined and systematised this approach and in 1986 a commercial version of this capitation scheme was launched. Known as Denplan Care, it subsequently became the template for a number of similar offerings within the UK dentistry.

This approach differed significantly from both the NHS system and from the US systems of managed dental care in that the level of monthly fee charged to each patient was set by the individual practitioner. This was achieved firstly, by reflecting the specific costs and overheads of, and desired return from, their practice, and secondly, by carrying out a baseline clinical risk assessment for each patient. The resultant monthly fee was thus specific to both the clinician and the patient.

The Denplan system set out five levels of fee according to the assessed risk, and likely future care needs. Band A represented good oral health and low future risk; band E significant past disease and interventive experience and high ongoing risk. Restorations were categorised and scored according to their complexity, as were removable and fixed prostheses, whilst periodontal status and oral hygiene (plaque control) were also rated to give a total numeric 'risk score' facilitating patient assignment to a fee band.

By trial over time, Noar empirically evolved numeric 'cut-off points' signifying a transition from one fee band to another.

A further critical aspect of this approach was that patients should be 'orally healthy' before admission to the scheme: any existing disease should be treated

definitively and oral health education provided at the outset. There was, however, discretion for a practitioner to accept a patient into the Denplan scheme by 'excluding' (either permanently or for a set future period) outstanding treatment needs at the outset to enable the patient to register as a scheme member. Thus, a restoration of doubtful prognosis could be monitored for a time, or treatment provided later at additional cost.

Elective, aesthetic and complex treatments (including orthodontics, implants and specialist referral care) were excluded, as were laboratory fees, for which patients would pay separately. A 'supplementary insurance' was subsequently incorporated into fees to cover out-of-hours emergency care, oral trauma and mouth cancer.

The Denplan system initially was adopted by a relatively small proportion of the profession, but in the fractious circumstances which followed the inception of the reformed NHS contract in 1990, increasing numbers of the UK dentists saw in it an opportunity to 'convert' their practices increasingly towards the private sector at fee rates affordable for their patients, whilst retaining full control of their practice costs and income.

Patient fees could be adjusted as desired by the dentist on an annual basis allowing patients to have fully adequate notice of any change in budgetary requirements.

The basic Denplan risk assessment and scoring system remained in place for a considerable time and is still used by many participating dentists today (see Tables 2 and 3). In 2016, there were over 6000 participating dentists in all parts of the UK and over 1.5 million registered capitation patients.

Denplan's services were charged as a 'premium' per patient monthly payment (these charges were allowed for in fee calculations) on a sliding scale dependent on the number of patients enrolled. Services included all patient and practice literature, enrolment forms, registration and ongoing maintenance services (such as changes of bank account, address, additional family members etc.), telephone, e-mail and on-site advice and support and annual fee mailings to patients.

Variants of the original 'full capitation' programme were offered over time, the most significant (Denplan Essentials) being a blend of capitation (covering regular examinations, prophylaxis, radiography and preventive advice) with FFS payment for any necessary additional treatments (with any discounts at the discretion of the dentist).

In 1998, Denplan piloted a clinical quality assurance programme—'Denplan Excel'—in advance of which an internal survey was conducted amongst Denplan-registered patients and dentists with a view to enhancing patient satisfaction with their care and improving outcomes.

One finding was that, despite the intended aim of encouraging patients to improve their health and reduce their fee banding, patients had little perception of any change in their overall oral health status over time. Most reported that they knew either that 'no treatment' was required following routine recall examination, or that 'some treatment' was needed. Moreover, Denplan statistics show that only around 1% of patients have risk assessment band changes in a calendar year [32].

To address this deficiency, a search was conducted for a comprehensive but practicable measure of current oral health status. The adopted solution was a derivative of the Oral Health Index (OHX) originally developed by Burke and Wilson [33],

Table 2 Denplan (original) numeric risk scoring system 1986—date

Score item	Score detail	Numeric score
Restorations	Amalgam per tooth surface	1
	Composite per tooth surface	2
Crown or retainer	Per tooth	4
Crown post	Per tooth	4
Root filling	Upper incisor per tooth	4
	Lower incisor per tooth	8
	Canine or premolar per tooth	8
	Molar per tooth	12
Gingival condition	Good	0
	Mild gingivitis	2
	Moderate gingivitis	4
	Severe gingivitis	6
Periodontal status	No significant bone loss	0
	Some/physiological bone loss	6
	Significant bone loss	18
Prostheses worn	Per bridge pontic	6
	Per denture: 1–5 teeth	10
	Per denture: 6–10 teeth	16
	Per denture: 11+ teeth	24
Plaque control	Excellent	0
	Good	2
	Average	6
	Poor	12

Table 3 Denplan banding by numeric score

Band	Numeric score range
A	0–10
B	11–50
C	51–90
D	91–140
E	141+

modified by Burke and Busby [34] as the Oral Heath Score (OHS) which was trialled in 209 Denplan practices for 18 months to assess its usability for clinicians and comprehension by patients [35]. The OHS outputs an oral health status score with a maximum (optimal) score of 100.

Results were positive in both aspects and the Denplan Excel Quality Programme was launched in 2001.

In 2008, as part of a review of Denplan Excel, a study commenced to introduce standardised predictive disease risk assessment alongside the existing oral health status report for patients. This incorporated a version of the online tool (PreViser) developed and validated by Page et al. [36, 37].

In 2011, the first pilot of the Denplan Excel PreViser Patient Assessment (DEPPA) tool for combined clinical risk assessment and oral health status commenced [38]. DEPPA combines current knowledge of risk factors for dental caries, periodontal disease, tooth wear and mouth cancer with the existing Denplan OHS.

The patient report produced by DEPPA includes a revised fee band calculation which introduced weighting for future disease risk and assigned a higher weighting for periodontal disease severity. This allows the dentist to consider whether the DEPPA results suggest (but do not enforce) any change. A validation study of the DEPPA system has also confirmed overall consistency with the UK national dental health surveys conducted by Government [39] (Figs. 1 and 2).

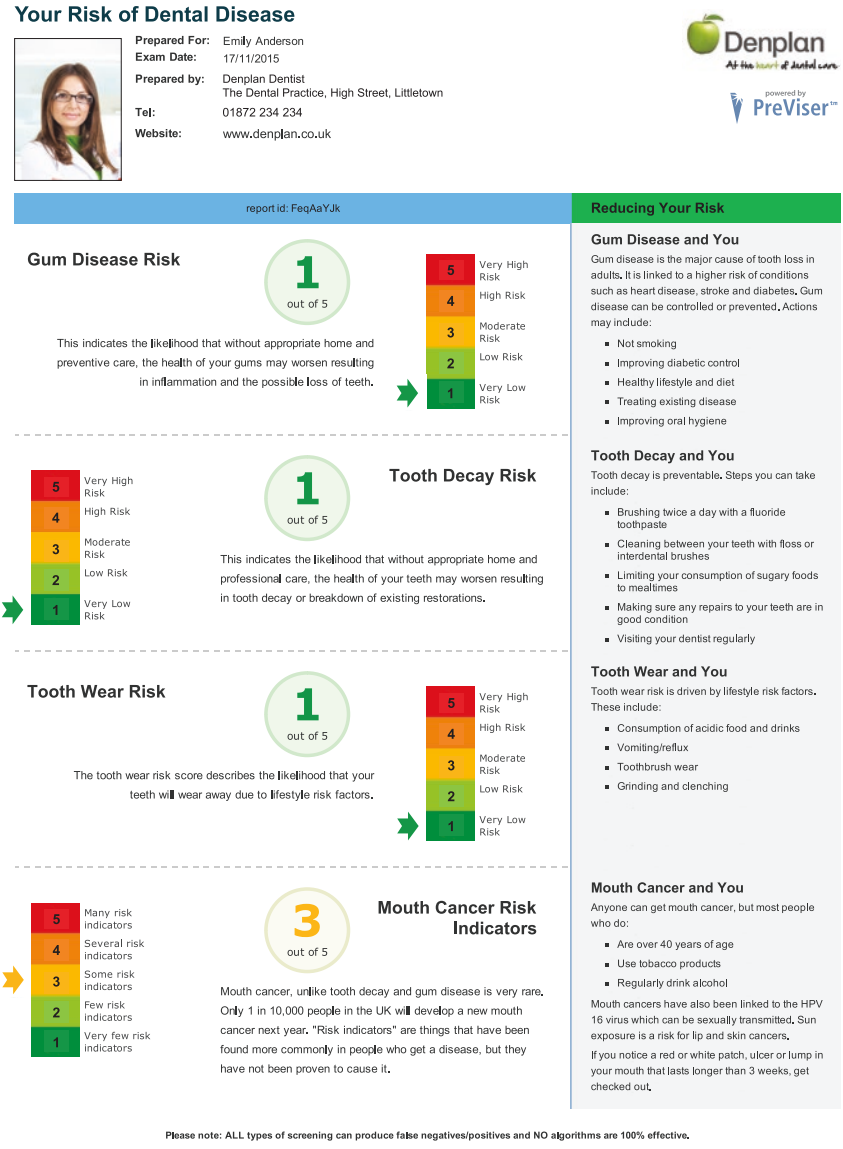
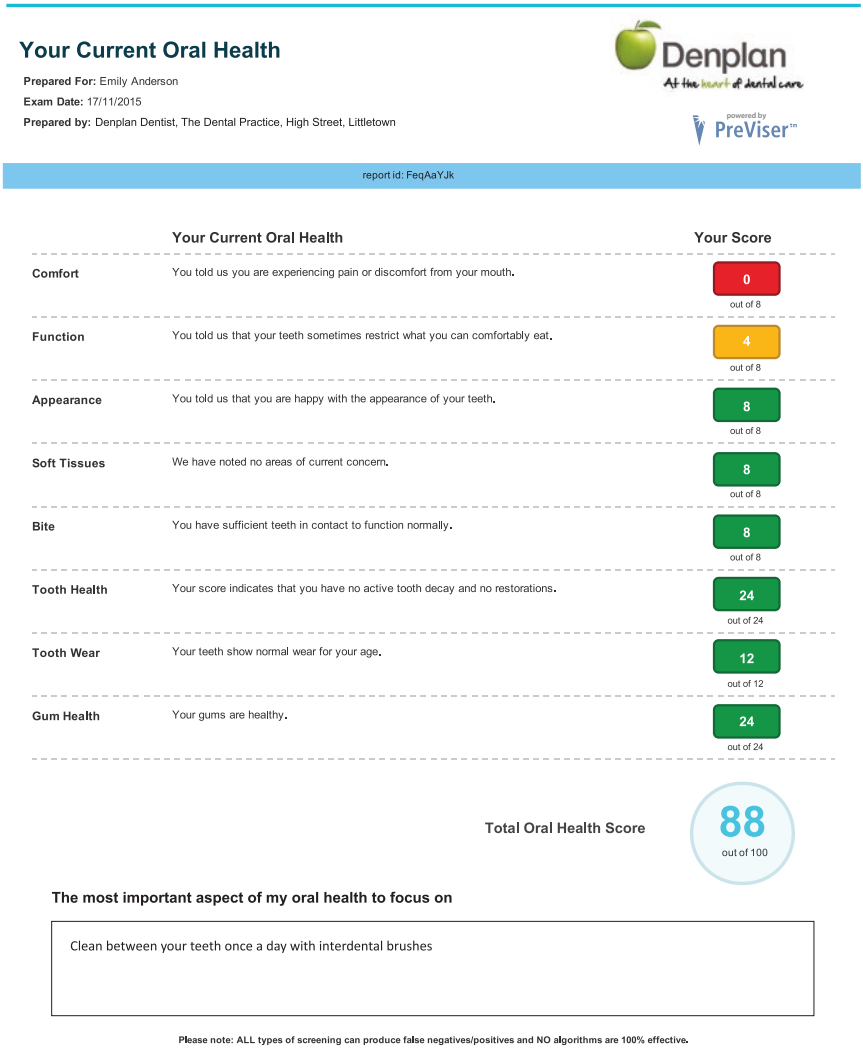


Fig. 1 Sample DEPPA report: page 1: risk for future disease (Reproduced with the permission of Simplyhealth Group Ltd)



Your Current Oral Health

Prepared For: Emily Anderson
Exam Date: 17/11/2015
Prepared by: Denplan Dentist, The Dental Practice, High Street, Littletown

Denplan
At the heart of dental care
powered by PreViser™

report id: FeqAaYJk

	Your Current Oral Health	Your Score
Comfort	You told us you are experiencing pain or discomfort from your mouth.	0 out of 8
Function	You told us that your teeth sometimes restrict what you can comfortably eat.	4 out of 8
Appearance	You told us that you are happy with the appearance of your teeth.	8 out of 8
Soft Tissues	We have noted no areas of current concern.	8 out of 8
Bite	You have sufficient teeth in contact to function normally.	8 out of 8
Tooth Health	Your score indicates that you have no active tooth decay and no restorations.	24 out of 24
Tooth Wear	Your teeth show normal wear for your age.	12 out of 12
Gum Health	Your gums are healthy.	24 out of 24

Total Oral Health Score 88 out of 100

The most important aspect of my oral health to focus on

Clean between your teeth once a day with interdental brushes

Please note: ALL types of screening can produce false negatives/positives and NO algorithms are 100% effective.

Fig. 2 Sample DEPPA report: page 2: current oral health score (OHS) (Reproduced with the permission of Simplyhealth Group Ltd)

Over 150,000 DEPPA reports have now been generated by over 800 Denplan Excel practices. Continuing review of this anonymised database has shown a relationship between general health and lifestyle factors and oral health [40], and between periodontal status (as measured by the tool) and patient-reported outcomes (PROs) [41].

Moreover, as risk and disease scores are shown to increase with age, the cost of providing oral health care tends to rise significantly with age and where capitation is employed as a method for funding, these costs either need to be passed onto those patients, or a conscious decision made to subsidise older age groups [42].

In 2017, a further online clinical risk assessment—YDEPPA—for young adults and children aged under 17 years was launched after piloting. Based on the principles of DEPPA, it is revised to be suitable for parents/carers or patients in the age bands 0–2 years; 3–6 years and 7+ years. Additional data collected include: orthodontic need (age 7+), dentoalveolar trauma, dental erosion and developmental abnormalities. The output again provides an OHS with a maximum score of 100. 'Preventive care need' is apportioned in three bands: Low need: 90+; moderate need 81–90; high need <80. Dedicated preventive care plans are provided to patients or carers with the report for each age range. Further research and validation of YDEPPA is ongoing [43].

Summary

Capitation as a means of both delivering and receiving primary dental care has benefits for both the patient and the responsible clinical team. For the patient, these benefits include improved certainty of budgetary planning and the knowledge that care will be provided on an ongoing basis, with an emphasis on prevention, oral health maintenance and optimisation.

The clinical team has, in an ideal capitation system, a similar certainty of the income that the practice will achieve, the satisfaction that comes from working 'with' the patient and not merely 'on' the patient, and the clinical freedom to provide the most appropriate treatment necessary to optimise or maintain oral health for patients.

Capitation imposes—as do all systems of dental remuneration—responsibilities on both parties: for the patient, to attend as required for care and treatment, to follow preventive and health-maintaining advice and counselling, and to pay the requisite fees on a regular basis. The clinical team must, firstly, be clear about their joint and several objectives, and be passionate about achieving them. They should be motivated to deliver good quality advice and treatment, in the knowledge that failure does not attract additional fee income.

In order for capitation to be effective, it is important that all parties recognise that it is not merely a form of 'dental insurance' or 'indemnity'. Indeed, indemnity implies a return to the *status quo ante*: once any restorative or surgical treatment is instituted, there *is* no going back.

Capitation should ideally approach as clearly as possible the ideal that each patient pays, over time, the average cost of their actual contact time with the dental team plus any incidental expenses (materials, equipment and tests) that may be incurred. To achieve such an ideal, clinical risk assessment is essential.

Numerous tools for clinical risk assessment have been developed. Whilst more recent systematic review of tools for caries risk (as noted above) [11] have concluded that overall, their validity and prognostic accuracy was only fair, a 2015 review of periodontal risk tools [44] considered that selected tools did predict disease progression and tooth loss. However, at present, reports of predictive tool application specifically to capitation funding systems are limited.

In the overwhelming majority of capitation systems found in the US, risk assessment has been broadly based on the population or group model which will inevitably introduce a 'gains and losses' approach. Particularly for the dental team that is attuned to fee-for-service, this may be, understandably, hard to acknowledge or work within. Arguably, the US risk assessed capitation models are designed primarily to attenuate fees for lower patient premiums.

Swedish models of capitation in the PDS undertake risk assessment on an individual basis, although the risk categories themselves may vary from region to region. Published data on caries risk assessment is available but not for comprehensive oral health, and detailed evidence for overall effectiveness and cost: benefit has not yet been published. Notably, capitation and fee-for-service care in the PDS in Sweden is provided under salaried dentist payment.

It has been argued that dentists providing care and treatment under capitation may be influenced to provide less care and treatment and fewer visits, whilst those utilising fee-for service may tend to maximise both the care and treatment value and appointments. Numerous studies have confirmed that the methodology of clinician remuneration may influence clinical activity [45]. However, whilst confounding factors such as sub-optimal payment constraints, third-party fee tariffs or even systems of national healthcare may play a part in influencing clinician behaviour, evidence overall of such bias remains inconclusive.

In this author's view, a well-validated, objective and individualised clinical risk assessment should form an essential basis for dental capitation payment systems. Further research on validation, cost: benefit and social equity is needed.

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Risk Assessment in Dental Health Economics: A View from a Third Party Provider

Carl F. Loeb and Shannon E. Mills

Contents

Background

Within the context of this article, economics can be defined as the study of transactions between a willing consumer and a willing provider of goods or services. In its simplest form an economic transaction consists of an agreement between two parties—the consumer and the seller, where the consumer has identified a desire or need for a product or service that another party offers. An economic transaction occurs when both parties agree to a transfer of value from the consumer to the seller and in return a transfer of goods or services from the seller to the consumer. That value can be monetary (I will pay $"x" for "y" goods or service), by barter for something of equal value (I will trade you 2 hours of my help on your project if you will give me 3 hours of your help on mine), or emotional/philanthropic (I will give $"x" or "x amount of time" to your charity because I believe in your mission).

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Within dentistry, fee for service models are commonplace and involve a patient being aware of or informed of a need and a clinical provider willing to satisfy that need at a transactional cost. Also commonplace in dentistry and healthcare in general are economic transactions involving third or fourth parties who each add their own contribution to the ultimate transaction. For example, an employer may decide to be the party that pays for the healthcare needs in whole or part for their employees, or a government may choose to manage a healthcare system as a function of government by assuming the role of a payer funded by the collection of taxes. If an employer wishes to provide healthcare to employees as a means of attracting or retaining healthy and productive workers, that employer will typically engage an insurance company as a fourth party to manage the healthcare benefit for employees on their behalf.

While economic transactions can and frequently do occur when a lack of comprehensive information about the goods or services purchased is available to buyers and sellers, the result is unlikely to be optimal. For example, a seller of an old violin may think they are just cleaning out the attic, but if the violin is a Stradivarius and the seller lacks this information the result will not be optimal for the seller. For a healthcare transaction to be economically optimal for all parties, highly specific information is required to be available to the different stakeholders that differs depending on their respective roles in the transaction. The consumer/patient must be aware of a need that establishes a basis for placing an economic value on the satisfaction of that need. That need may be reactive to a problematic condition, involving perhaps the need to repair a painful tooth, or preventive, to mitigate the possibility of future disease developing. For example, if a patient is unaware they have periodontal disease, the patient will have no reason to place value on the treatment of that disease or in the adoption of preventive actions that may prevent the disease state from worsening. The provider/dentist must be able to correctly identify a need a patient may be unaware of and adequately explain why it should be addressed if the patient is to place value on a remedy to that need. Or, if the patient is aware of that need, the dentist has to be able to adequately define a course of action or different options to manage their need as a necessary basis for an exchange of value. Ideally, the provider will also be able to communicate the probable endpoint of treatment so the patient has an idea of what the final value for the transaction will be. That final value may be a reduction of pain, improved appearance, improved oral or systemic health or quality of life. Where recommended treatment occurs over time via multiple interventions, understandable measures of progress towards the desired endpoint will help ensure patient involvement in the *process* of getting healthy, not just the reward of being healthier.

Similarly, if an employer or government entity is paying for the healthcare, that funder will require information that demonstrates the healthcare services for the patient are actually accomplishing something of value to the employer, to society, as well as the patient themselves. If a third party insurance company is involved, ideally that company will have the information necessary to correctly administer the dental benefit and appropriately approve any preventive or therapeutic interventions required, or question or deny insurance coverage for unneeded or inappropriate services.

Healthcare Presents Challenges to Efficient Cost/Benefit Analysis

Dentistry, and healthcare in general, presents some novel impediments to economically efficient transactions by distorting normal cost/benefit analysis. The involvement of third party payers of healthcare reduces or eliminates the burden of cost to the patient for treatment required. When a third party subsidizes or covers the cost of the transaction, cost naturally becomes far less important in the final decision to accept or reject treatment. Perversely, third party payments may also diminish the patient's ability to quantify the benefit of accepting treatment in many cases. It would be a rare dentist that has not had a patient conclude that a recommended therapeutic or preventive intervention is unneeded on the basis that a third party payer is not willing to cover the service, even though benefit contract limitations may bear scant correlation to a diagnosed clinical need.

A further confounding factor in patient cost/benefit analysis of improved oral hygiene is that humans, like all animals, experience olfactory habituation, meaning the continual presence of an odor will, over time, become unnoticeable. One theory as to why this adaptation exists is its utility in helping animals detect the trace odor of a predator. If that odor was to be overwhelmed by one's own personal stench, that danger signal may be missed. Since periodontitis is frequently not accompanied by any pain or other discomfort, a patient on their own may not be able to detect a likely olfactory marker of the disease, and unless convinced otherwise by an attending care provider, be unaware of the potential impact of that disease on their overall health.

The Role of Digital Information in Improving the Economic and Outcomes Efficiency of Healthcare Decisions

Prior to the advent of digital systems that assist clinicians in objectively identifying and determining how best to manage the specific needs of an individual patient, the information available to healthcare stakeholders was sometimes too imprecise to facilitate effective healthcare decisions at the individual patient level. For example, a patient can be told that brushing and flossing their teeth is important. However, if they lack convincing information that their own failure to brush and floss is demonstrably causing or is likely to cause them harm, they may not feel sufficiently motivated to act to improve their health. If a third party payer lacks specific and accurate information on which of their insured patients have elevated risk of oral disease, they may lack the motivation or ability to target spending towards those who will benefit the most from enhanced preventive care that is targeted to reduce the need for future costly therapeutic interventions.

The specific and relevant health information that is required for optimal healthcare decisions should include at a minimum three main elements to satisfy the needs of all stakeholders.

- First, the risk of oral disease needs to be known, so current health can be maintained and any possible deterioration in health can be measured, then further prevented or mitigated.
- Second, an accurate description of a patient's current health status must be known so that appropriate therapeutic interventions can be determined and the success of those interventions evaluated against a known health status baseline.
- Finally, a prognosis that describes the expected outcome of preventive and therapeutic interventions needs to be known so that patients or funders can evaluate whether the care offered will result in sufficient benefit to warrant the cost of care in money and time.

Risk, severity, and prognosis are different entities that are nevertheless closely coupled. For example, a patient may be at elevated risk of periodontitis but not currently have disease, or they may have periodontitis and be at relatively low risk of the disease progressing. A 28-year-old patient at a given level of risk and disease severity may have a positive prognosis for a proposed treatment plan, while a 28-year-old patient at the same risk and disease severity level who is deeply depressed may have a neutral to negative prognosis because they are less willing to participate fully in improving their health. If the risk and severity of disease are known, the prognosis associated with a treatment plan then becomes the basis for a patient or funder to pay for that treatment plan, since both the value and the efficacy of the treatment plan can be quantified to some degree. If the prognosis for a patient is the same with or without prevention or treatment, then the economically rational decision may be to do nothing, or direct care resources into other channels that may do the patient some good. The interplay between these three separate but coupled entities can be seen in data generated in a private study by over 18,000 low income US Medicaid patients in the state of Iowa whose oral health status was measured using a digital risk and disease severity toolset.

Table 1 shows how this population distributes across three broad categories of oral health—individuals with no active carious lesions or defective restorations, individuals with between 1 and 4 active lesions or defective restorations, and individuals with greater than 4 lesions or defects.

Each individual within this population has a specific risk for tooth decay, each individual has a specific severity of caries currently, and each individual has a prognosis for an outcome following any required treatment or preventive regime.

In this dataset, those within Group A—no current defects—likely have lower risk of disease and a good prognosis for continued health with good preventive care. Group B individuals by definition have elevated risk of caries since they currently have some level of disease, and will have differing prognoses based on personal

Table 1 Variable costs of care based on risk and severity

Total sample size: 18,648, all age >29	Group A	Group B	Group C
% of population	31%	36%	33%
Average # of active lesions or defective restorations	0	2.1	10.2
Cost of preventive + therapeutic intervention ($mm)	$1.25	$8.5	$26.5

health characteristics, the skill of the provider, and the availability of financial resources to obtain care. Finally, Group C has demonstrated high risk of disease, and because the average number of defects in this group was over ten per individual, Group C clearly have significantly increased disease experience. However, it is also likely that the individuals in this group would have a very poor prognosis for future optimal oral health since such a high level of current defects suggests other lifestyle issues that may interfere with attaining oral health.

Without knowledge of objective risk and severity being available to the patient, provider, and the funder, optimal decisions leading to the best prognosis are more difficult to make.

In a world of limited resources, an economic analysis of this adult Medicaid data suggests that the most cost-efficient use of financial resources would be providing optimal prevention services to Group A; appropriate therapeutic services and follow-on preventive services to Group B to attain and retain health; and a focus on helping those individuals in Group C positively addresses the lifestyle challenges they may have that are contributing to their very poor oral health status before spending a great deal of money attempting to fix the oral effects of the problem rather than addressing the problem itself. In the latter case, there is an additional dilemma in that pain relief must be provided and that may frequently result in the patient not returning for care or engaging in the prescribed preventive regime of care.

An example of a digital toolset that provides patients, dentists, dental insurers, and third party payers of dental care with the information required for rational healthcare decisions is one developed over the last 15 years by PreViser Corporation, a US-based health informatics company. The information generated by this web-based system includes a numeric risk score on a 1–5 scale for periodontitis, caries, and oral cancer, a periodontitis and caries severity score on a 1–100 scale, and a periodontal stability score, also on a 1–100 scale. These scores are returned instantly on submittal of a small subset of clinical observations collected during a routine oral examination, and entered into a web page. As shown in the example in Fig. 1 the assessment is returned in the form of a Red, Amber, Green (RAG) rating and informative patient report aimed at helping the patient understand their current needs so they can effectively place an economic value on addressing those needs.

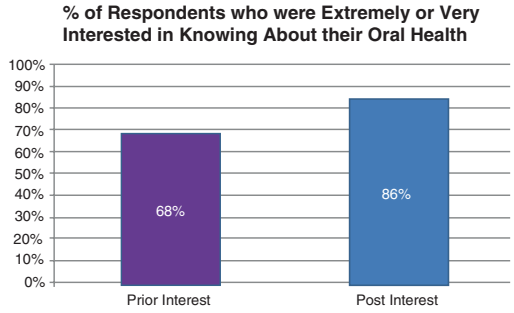
The impact of providing this information to patients and dentists is potentially far reaching. In 2011, an internal study conducted by a large US-based oral care and consumer products company examined patient attitudes towards the data on the PreViser patient report. 150 patients were asked before and after they received the PreViser report how interested they were in understanding their personal oral health status, and as seen in Fig. 2, the impact of receiving the PreViser report increased a desire for that knowledge by 26% from baseline.

After quantifying the interest the patients had in knowing more about their oral health, a follow-up question was asked to measure the degree to which the patients felt they were knowledgeable about their health. Prior to receiving the PreViser report 48% of the participants felt they were “Extremely or Very Knowledgeable” and as illustrated in Fig. 3, once a patient received the PreViser report the

Fig. 1 Comprehensive oral assessment, courtesy PreViser Corporation

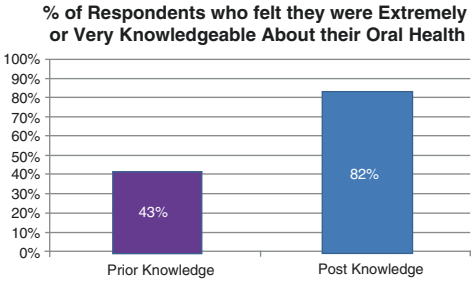
percentage of patients who thought they were now "Extremely or Very Knowledgeable" increased from 48% to 82%. In other words, the patient's interest in knowing more about their oral health was satisfied and an equivalent percentage of patients who felt they had that knowledge now better matched those interested in having that knowledge.

From an economic perspective, this increase in knowledge about the value of a product (the patient's personal oral health) may be expected to result in the patient



Prior to your most recent dental visit when you received the PreViser Oral Health Report, how interested were you in knowing about your overall oral health? / After you received the PreViser Oral Health Report, how interested are you in knowing about your overall oral health? N=150

Fig. 2 Patient interest in oral health increases with more informative health information



Prior to your most recent dental visit when you received the PreViser Oral Health Report, how knowledgeable would you say you were about your overall oral health? / After you received the PreViser Oral Health Report, how knowledgeable do you consider yourself to be about your overall oral health? N=150

Fig. 3 Impact of risk and disease information on patient oral health understanding

making more informed decisions on whether or not to adhere to dentist recommendations, or to pay for services not covered under a dental benefit plan based on more complete information on the personal impact of such decisions.

When the primary information a consumer has about a product is limited to its cost, the consumer will make a purchase decision heavily influenced by that price, without reference to other quality measures. They may choose a more expensive product, believing it must be better if it costs more. Or, they may choose the cheapest product since they have no information to justify paying more. In either case, the actual quality of the product is unknown and therefore does not influence the ultimate decision. In oral care, if the consumer has no knowledge of the value of oral health other than its cost, simply not spending the money is a rational choice. When this occurs, the dentist has failed to provide the consumer with intelligible information on a health care need, so naturally the patient places low value on what the dentist is offering. However, when the patient is provided with highly specific and individualized health information, the calculus changes.

Trends in the Use of Digital Information to Improve the Quality of Healthcare Decisions

The use of digital oral risk and disease severity information to manage dental benefit plans is currently being adopted within the US dental industry, primarily by the Delta Dental family of insurers who manage such plans for over 70 million Americans. Northeast Delta Dental, a Delta Dental company who operate in the states of Maine, Vermont, and New Hampshire, is a pioneer in the use of PreViser technology to rationalize the underwriting of enhanced and evidence-based benefit design. At the time of writing this, approximately 95% of all general dentists in these three states are using the PreViser tool described above to assess Northeast Delta Dental insured patients in order to determine whether those patients may qualify for enhanced preventive benefits under the company's Health Through Oral Wellness™ plan (www.healththroughoralwellness.com). Under this benefit plan, a patient receives additive benefits on top of a base plan design if their PreViser generated risk and disease scores indicate they would benefit from enhanced care. A breakdown of those current enhanced benefits can be seen in Fig. 4.

An interesting feature of Northeast Delta Dental's implementation of the Health Through Oral Wellness™ benefit plan is that if a patient initially presents with elevated risk or severity of disease scores that qualify them for enhanced benefits, those benefits are never reduced even if the patient's PreViser scores measuring their health have improved. Northeast Delta Dental decided to implement this feature to ensure access to the benefits that helped make the patient healthy in the first place. The impact of highly specific oral health information on patient decision-making can be seen from initial pilot data on one of Northeast Delta Dental's larger accounts.

In 2011, this group was insured under the Health Through Oral Wellness™ program and employees and the dentists caring for them began receiving PreViser assessment reports as part of their routine dental visits. As can be seen from Fig. 5, an immediate and permanent change in employee attitudes towards dental care can be seen.

Prior to the initiation of the Health Through Oral Wellness/PreViser pilot program, the average employee received around 1.5 prophylaxes/cleanings per year—CDT 1110, adult prophy, CDT 1120, juvenile prophy, or CDT 4910, periodontal

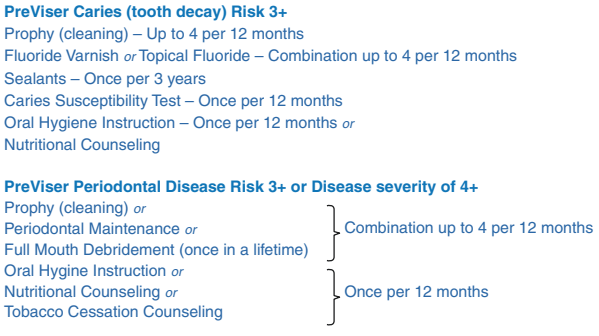


Fig. 4 Quantifiable oral health data enables patient centric care

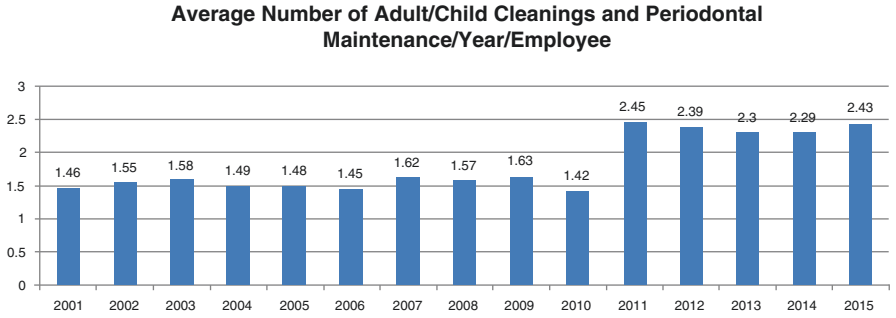


Fig. 5 Risk and disease data increases preventive care delivery

maintenance, and this pattern had persisted for a decade. After the initiation of the pilot program, this average increased by 60% and did not decline over the subsequent 5 years. Of note in this data is that the employees of this group already had the benefit of up to four prophylaxes per year without the need for a PreViser score to justify it, but did not avail themselves of this benefit. It is reasonable to assume that the significant increase in dental visits may be attributed to an increased awareness on the part of the patient of the value of the services the dentists offered. In economic terms, by receiving personalized risk and disease severity scores, the consumer/patient had the information required to make them more willing to invest time and money visiting the dentist in exchange for the services rendered, whose value was now more clearly understood.

Clinical Impacts of Digital Risk and Health Data

While making individualized risk and disease severity information available to patients appears to impact the patients' willingness to invest in better oral care, it may also have an impact on the behavior of clinicians. Within the Delta Dental system, between 3% and 7% of adults over the age of 29 received a procedure for treatment of, or maintenance following treatment for periodontitis. However, the prevalence of periodontitis in the general US population exceeds 45% [1]. This significant gap between prevalence and reported treatment is an indication that the appropriate clinical diagnosis and treatment of inflammatory periodontitis can be improved. There are likely many different reasons for the under diagnosis and treatment of periodontitis that relate to how dentists are trained and compensated. However, if the average consumer/patient is unaware of a need for treatment, and the average provider/dentist is overlooking or simply not focusing on that aspect of patient health, then there is no basis for a rational economic transaction to occur between consumer and provider that addresses this unknown need. Providing objective periodontal disease risk and severity measures to both patient and clinician should spotlight the need for treatment, with an expecting closing of the gap between delivery of, and need for therapeutic interventions.

The economic consequences of risk and disease severity data that can be used to develop treatment plans with optimal prognoses extend to the economic health of the dental practice as well. While it is likely that many dentists feel that expensive procedures like crowns or implants may be the most profitable part of the practice, that may not necessarily be the case. In a case study of a private practice dental office in a rural location in Minnesota who for 14 years had used digitally generated oral health scoring on every patient each time a patient was seen, some surprising results were found. This dentist practices in a town with a population of under 2500 with four competitor dental offices. The active patient list exceeds 1500 patients and the dentist reports unusually high profitability that he attributes to the following factors:

- Almost all patients are as healthy as they can be due to significantly increased compliance with treatment plans justified by the digital oral health scores.
- The creation of a common language describing oral healthcare needs built around digital scores that is understood by patients and clinical staff and which facilitates patients placing appropriate value on the dental services offered.
- The reduced time it takes the dentist to perform required procedures on healthy patients vs. diseased patients. Since the dentist is compensated the same amount however long it takes to perform a procedure, reducing the time the procedure takes effectively increases the dentist's compensation since more procedures can be performed in a given work day.
- Improved word of mouth referrals to new patients from existing patients who have quantifiable information that the dentist is making or keeping them healthy.

Summary

Healthcare has been dominated by a paternalistic model of the relationship between patient and provider that is characterized by the assumption that only the healthcare expert is qualified to make healthcare decisions, with the patient a passive recipient of that presumed expertise. However, that model is changing to one where the patient is an active participant in all care decisions not requiring specific technical expertise, like lab testing or radiographic analysis [2]. This change is expected to not only improve patient outcomes, but the healthcare system itself [3]. While patient engagement and involvement in care planning and delivery is now recognized as an essential element to improving care, it is dependent on information the patient, provider, and funder can readily consume, understand, and act upon. Encouraging the transition from a paternalistic approach to care delivery to one that is patient-centered and data driven is now a cornerstone of what healthcare institutions, government regulators, and benefit funders define as quality of care, and continued improvement in the tools needed to engage patients more actively in their care will continue to be emphasized [4]. Friendly staff, attractive office surroundings, and convenient hours of operation may all be important components of a satisfactory patient experience, but are not substitutes for individualized information

that helps a patient or funder make an economically rational decision on the need for dental care, and provide the basis for determining whether quality of care has been delivered. Individualized and objective risk and disease information that stakeholders can easily understand will continue to increase in importance as the healthcare system, including dentistry, comes under increasing demand for accountability of outcomes.

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Part IV

Risk Assessment in Dental Practice

Leading the Oral Healthcare Team in Risk Assessment

Mike Busby

Contents

What Is Leadership?

> Our practice will strive, within a patient centred care framework, not only to achieve optimal oral health outcomes with our patients, but also to support optimal general health outcomes for them. We will pursue these objectives by making holistic and evidence based disease risk assessment the corner-stone of our care. Individual oral healthcare plans will be tailored within a risk based minimally invasive preventive philosophy. When risk assessment highlights significant systemic health risk we will refer patients to their General Medical Practitioner. Our practice will be funded by risk based capitation payments in order to facilitate our philosophy of care. We will strive to support a happy working environment which permits the whole team to feel fulfilled in their work.

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This would be my mission statement were I to be setting up my own practice in 2020, based upon 45 years of experience and reflecting on the dramatic changes in dentistry over the past decade. Even at this advanced stage in my career I felt a passion for the objectives described above as I wrote the paragraph. The paragraph describes what I really believe in. It may be hypothetical, but it is my future vision for a twenty-first-century dental practice.

Having a passionate vision of what you want your dental practice or organisation to be like is the foundation stone of good leadership. For the purpose of illustration I will use this personal vision throughout this chapter, keeping in mind that only your vision can work effectively for you! Further, 'your vision' needs to be shared with your team members fully. The ease with which a vision can be truly shared is likely to be in inverse proportion to the size of your dental organisation or individual practice.

Morison and McMullan [1] recognised the need for both greater teaching and research into leadership skills in dentistry. They conducted a qualitative study based on semi-structured interviews with nine individuals who held professional leadership positions within the UK dental services. The following are two selected quotes on what makes a good leader from these interviews:

> Somebody who has a vision and can communicate this vision and can help other people catch this vision. And has the ability to take this vision forward despite whatever challenges come on-board

> It's about listening and being able to be a strong advocate of your discipline and seeing the big picture at the same time

Even though there is a paucity of literature on the specifics of dental leadership, these quotes illustrate that the principles of leadership in dentistry can be held to be much the same as in any other sphere of human endeavour.

Let us start then with three of the hundreds of definitions of leadership offered in the general business literature [2].

> *Leadership is the capacity to translate vision into reality*
> Warren Bennis

> *Leadership defines what the future should look like, aligns people with that vision, and inspires them to make it happen, despite the obstacles.*
> John Kotter

> *Effective leadership is not about making speeches or being liked; leadership is defined by results*
> Peter Drucker

The consensus therefore is that leadership is about having a relevant vision which inspires you and your team to achieve results. On that basis Busby [3] proposed the following definition of dental practice leadership:

> The ability to continuously define a future practice vision which inspires you and your team towards success.

This definition is also drawn from Radcliffe's 'Leadership Plain and Simple' model [4].

Radcliffe's Model

This is the model for leadership that has underpinned the structure of this chapter because it illustrates so well, in the author's opinion, that leadership skills can be developed by anyone. Leadership is not the province of a chosen few who are blessed with special charismatic qualities. The principles of good leadership are quite simple. We are all capable of leading as long as we are 'up for something'. As Radcliffe [4] explains further:

> You need to have an ambition or dream or goal for your team, organisation, colleagues or yourself and it's got to matter to you

If you want to successfully lead a dental team in oral health risk assessment you will need a passion for this approach to healthcare which is detailed throughout this book. The chances are that if you have got this far through the publication you already have that passion!

In this chapter, each of Radcliffe's three pillars will be discussed in turn, including leadership skills in general and exploring some practical ways in which you can lead your team in oral health risk assessment. However, first it is important to decide what style of leader you want to be if you have not already done so.

Leadership Styles

A straightforward classification of leadership styles which has stood the test of time is that of Lewin et al. [5] who described three styles of leader:

Autocrats will decide on the vision and the policies for delivering it. Autocrats simply brief their team on what is expected of them. This is a decisive and fast style which can be effective with a leader who is followed without question by their team. However, it ignores the potential of team members to contribute ideas to the vision and consequent policies. This style can lead to team frustration and even revolution!

Democratic leaders will have the final say on the vision and policies, but they will consult with team members about them. This can enrich the decision-making process while still characterising a decisive style. However, it can also lead to frustration when opinions appear to be ignored.

Delegating leaders support their team to share in both creating the vision and the consequent policies. With the emotionally intelligent and well-qualified teams frequently found in small to medium-sized dental practices, this style can work to facilitate a highly motivated and effective team. However, it can lead to total chaos if the team fails to align and engage.

Each of Lewin's styles therefore has advantages and disadvantages. In practice most leaders will tend to adjust their style according to the issue being dealt with. The author's personal view is that the delegating style makes true team engagement more likely and that, in most dental practices, it is a much easier style to realise than in larger organisations. Therefore, this chapter will therefore principally discuss techniques mostly appropriate to a delegating style as Radcliffe's model is examined in more detail step by step. However, it will still be obvious how Autocrats and Democrats may amend techniques to fit the model.

Future–Engage–Deliver

In respect of this first pillar 'Future' of leadership, Radcliffe [4] states:

> Leadership is not about your competencies, skills and personality. It's first and foremost about being in touch with what you care about and then going for it.

Kemp [6] in a publication specifically aimed at dental practice suggested that, in order to 'get in touch' with what you care about most, you ask yourself the following question:

> How would I like my dental practice to be remembered?

This idea aligns with Covey's [7] suggestion in the general literature that we should write our own obituaries and work backwards.

Radcliffe [4] simply suggests the question:

> What do I care about?

Or as previously suggested [2]:

> What would my ideal practice be like?

When considering these questions it is vital to have an open and relaxed approach. At this stage in proceedings it is important not to reject ideas by allowing practicalities such as 'I can't see how' and limiting beliefs such as 'I'm too old' or 'I'm too young', for example, to intervene. From the realms of corporate jargon, this is 'blue sky thinking time'. The author would suggest simply listing your key points at this stage. The author's list before writing the paragraph at the beginning of this chapter was simply:

- Patient-centred
- Optimal oral health
- Supporting good general health

- Risk-based minimally invasive preventive philosophy
- Risk-based capitation funding
- Happy team

Your list will, and indeed should, be different. It might be more 'blue sky' than the above somewhat functional list. It must be a list which makes you feel very positive about the future. For the purpose of leading the team in risk assessment it might mention this aspect of practice specifically. However, that is not essential because it might be dealt with in the 'Deliver' domain (see below) as long as your vision points towards this in some way.

It is now time to consider your list in the context of what is happening in the real world in order to turn the 'blue sky thinking' into the 'possible' by regarding the 'big picture' and the reality 'on the ground'. Employing a P.E.S.T analysis may help with this phase.

The P.E.S.T analysis sets a structure for the consideration of the real world in which you practice from a Political, Economic, Social and Technological perspective. To illustrate how it may be used, Table 1 sets out a P.E.S.T analysis of the

Table 1 An example of a P.E.S.T analysis

Political	NHS England is working towards capitation funding and risk-based patient assessment. Regulatory bodies (GDC and CQC) strive to mandate holistic patient-centred preventive care. In excess of one million patients already subscribe to risk-based capitation plans in the UK private sector. The major provider of such plans offers a validated online patient risk assessment tool. However, medical practice and dental practice are usually entirely separate entities in the UK *Conclusion*: Political conditions are becoming increasingly favourable for the passions on my list although dental and medical practices are a little too remote from each other ideally
Economic	Brexit may harm the economy in the UK which has been in modest growth in recent times. This could impact on dental funding unfavourably. Although this is probably the consensus it depends on which economist you consult *Conclusion*: Especially with the relatively modest sums needed to fund minimally invasive preventive care and the total failure of economists to agree on future prospects I will not allow economic considerations to affect the pursuit of my passions
Social	In general social attitudes in my area are favourable towards accepting personal healthcare responsibility in areas such as nutrition, exercise, smoking, etc. There has been little research on public attitudes towards minimally invasive preventive oral healthcare specifically, but logic suggests popularity for this approach over traditional restorative approaches. Significant proportions of the workforce are seeking fulfilling careers in which they can feel involvement and worthy purpose. We are in an IT-driven society which may soon come to expect personalised IT-based biofeedback on their health *Conclusion*: My passions are in harmony with social attitudes in my area
Technological	Validated risk assessment tools are available for aspects oral health and general health. Materials and equipment for minimally invasive preventive approaches are in good supply. Ethical attitudes to such approaches are highly favourable and further education including master's degrees is available *Conclusion*: The technological climate for the pursuit of my passions is highly favourable

author's list from the point of view of practising in Southern England in the United Kingdom in 2020. All four aspects of the analysis will be significantly influenced by the location of your practice or organisation.

The analysis resulted in a 'green light' for the author to proceed with a vision of the future around the things he cared most about in dental practice. The most futuristic aspect of this vision is the fully holistic approach to risk assessment. Integrated tools still need to be developed to include general health risk in a dental setting. It will also be important to investigate social attitudes to the collection of more general health metrics in a dental practice. Further, communication would need to be improved between medical and dental practices. Including futuristic aspects in your vision may stimulate you to get involved in developments personally.

Following the PEST analysis it should be possible to articulate your vision more fully and the author's is presented, as you have already seen, in the first paragraph of this chapter. So, you are now equipped with a summary of what will guide your practice over the next few years about which only you feel very motivated and positive at this stage.

Future–*Engage*–Deliver

The vision you have compiled in the 'Future' aspect must energise and motivate you. If it does not, then keep working on it until it does! You cannot expect to engage and motivate your team unless you are inspired by a future you can visualise.

Senge [8] described a continuum of possible team attitudes and responses to your vision which could be summarised as follows:

- *Commitment*—The team are fully engaged and are fully energised in the pursuit of your vision. They will even devise their own policies in full alignment with what you are striving for. You are in harmony with your team because you have also engaged with their passions. You want the same things from your practice. Team members display personal leadership towards your shared goals.
- *Enrolment*—The team are happy to work towards your vision through the policies you set.
- *Compliance*—The team are prepared to work towards your vision through the policies you set.
- *Apathy*—Team members want a job but they do not really care about your vision; they will comply with policies to keep their job.
- *Resistance*—Team members are actually against your ideas and policies and will seek to follow other ideas at the earliest convenience.

If you are striving for a primarily Delegating leadership style 'commitment' from team members is your objective. 'Resistance' and 'apathy' towards the risk management approaches to patient care are clearly states to avoid whatever your style. The big question for this section is therefore how can you achieve

'commitment' or at least 'enrolment' from your team to your future vision for the practice? Radcliffe [4] asserts that the key to success in this pillar lies in having:

> Relationships big enough to get the job done

From a practical point of view relationships with your team members are built through the various meetings which you hold with them. In a small to medium-sized dental practice the most common 'meeting' is likely to be the day to day encounters 'on the job'. So, a leader must not forget to be constantly mindful of the 'big picture' themselves but also to take everyday opportunities to remind team members of how risk assessment fits into the overall practice purpose.

The more formal meetings with team members are also vital in developing 'enrolment' and even 'commitment'. Five different types of more formal meetings and the documents which might relate to these encounters will therefore be discussed.

Recruitment Meetings

The job interview is often the first opportunity to communicate your vision to applicants who want to join your team. Indeed, the job advertisement placed in order to attract staff presents a chance to at least precis your vision before potential team members even apply. The job description document should also convey the team culture and vision. The terminology used at this stage will be determined by how well your vision might be understood by new applicants. Nevertheless, the recruitment meeting is a golden opportunity to explain your vision to applicants, and to assess their suitability to engage with it, and play a part in its delivery.

On a very practical note Richer [9] talking about the retail sector described how his company placed job adverts in their own sales catalogue. He states:

> Some of our best staff were customers. That way we attract people who already love the product and know what goes on in the shops.

In other words perhaps recruiting team members from your patient base could have considerable merit because they may already have a feel for your vision based on their experience of care. Some of the author's most successful team members during his practising life were patients before they were recruited. That included two of the dentists who eventually became partners and two long serving hygienists.

Training Meetings

Appointing a mentor, who the new team member 'shadows' for the early period of their employment, is another good idea recommended by many authorities including Richer [9]. This mentor will be an employee fully committed to your vision, so

that they can enthuse the recruit. The mentor will ensure that the new recruit reads and understands your practice manual. This document will contain information on many important but routine aspects of working in a dental practice (infection control and data protection for example). However, text describing your vision should have a prominent place.

Specific training courses including initial and further professional qualifications will all form part of training for the whole team with you leading by example. Specific training on the use of risk assessment in a risk-based preventive philosophy is obviously relevant to our particular leadership purpose in this chapter.

Day to day learning on the job is probably the most important development for all members of the team including the leader. Good leaders, in the author's opinion, foster a reflective and open learning culture. There are many everyday opportunities in practice to emphasise the big picture which is your vision and how risk assessment is such an important part of the whole.

Appraisal Meetings

The performance review of all team members should be ongoing. Nevertheless, most organisations formalise this periodically with a specific meeting using a document designed to produce a personal development plan (PDP) for each team member. This appraisal document gives another opportunity to put your vision centre stage. All resulting PDPs should be constructed keeping in mind the clear objectives which your vision prescribes. The appraisal documentation can draw the attention of all team members to this focus.

Routine Team Meetings

The essential purpose of regular team meetings is to construct, confirm and review policies, protocols and procedures for the delivery (see next pillar) of your vision. These policies will include the many essential or even mandatory routine aspects of running a modern dental practice. An agenda will usually be employed to guide these meetings and minutes will summarise the outcomes. Both of these documents provide an opportunity to precis the vision in the text (at the start of each document) to remind everyone of the big picture. It will be evident below that team meetings at which the surgery's oral health policy is formulated and reviewed will be the most important group meetings in respect of risk assessment.

Planning/Strategy Meetings

As a leader you will need to periodically review your visio**n** and the strategies for delivering it. Depending on the size of your dental organisation, these meetings might include partners, practice managers and possibly outside advisers. For the

small to medium-sized dental practice, it is perfectly possible to include the whole team on these occasions. This would give leaders wanting to adopt the delegating style a chance to ask the whole team the 'future' questions suggested in Sect. 3. The opportunity exists therefore to explore the collective future vision for your ideal practice and so to clarify what all team members really care about. If your relationships with each team member have been positively built around the other encounters described above, it would be highly surprising if you found drastic variation from your passions. If significant differences did exist it would certainly be better to be aware of these! This exercise, if well conducted, can make team members feel fully involved in something with a worthy purpose in which they believe. In the Senge [9] continuum 'commitment' is fostered. The works of both Maslow [10] and Kovac [11] support the idea that feelings of inclusion and belonging at work are key factors in team motivation.

Future–Engage–*Deliver*

> *Vision without action is merely a dream. Action without vision is merely passing time. Vision with action can change the world*
> Nelson Mandela

If you are doing a good job on 'Future' and 'Engage', then the groundwork for 'Delivery' is in place. Nevertheless, success is far from guaranteed. The author would suggest that there are three key elements to 'Delivery'.

1. Having clear policies and plans to describe exactly what each aspect of your vision means and how you will strive towards success in each area.
2. Having the energy and delegation protocols to constantly manage the implementation and review of these plans and policies.
3. Having clear metrics which monitor your progress.

Leaders of dental organisations will be acutely aware of the mandatory burden of practice policies. To help in motivating statutory compliance within the team it is usually possible to fit this important workload within the parameters of your vision. For example infection control and data protection policies might be seen as being part of supporting the patient-centred aspect of the author's vision because they are so important to patients.

We have seen in 'Future' that the author's hypothetical vision in fact comprises six aspects which are listed in the section. All six aspects will need to be delivered. However, for the purposes of this chapter we are most concerned with 'Delivery' of risk assessments. So, finally we will examine how we might specifically lead the team to deliver risk assessments in practice using the three 'Delivery' elements described above.

You will remember in the 'Future' section it was suggested that it was not essential for your vision to specifically include risk assessment in its text.

The author's hypothetical vision, in fact, does specifically refer to the important cornerstone that risk assessment would take in our approach. However, if your vision was less specific but still referred in some way to the pursuit of good oral health that would be fine. This is because in the 'delivery' of good oral health, the key to success in any dental organisation is to adopt an oral health policy. As you may have believed even before you started reading this book, including risk assessment in your overall patient assessments is such a logical, effective, efficient and ethical step towards achieving optimal oral health outcomes with your patients, it naturally follows that it will have an important place in your oral health policy.

Practice Oral Health Policy

Because there are so many mandatory policies to implement in modern dental practice (this is particularly the case in the heavily regulated United Kingdom Profession), it is understandable that many dental leaders and managers might be resistant to the use of non-mandatory policies. Nevertheless, how can good oral health, which must surely be the central purpose of any dental practice, be delivered effectively and efficiently without a policy that guides the team?

Policies are the basic science of management, but they are useless if they are simply documents on a computer or in a filing cabinet. They must be genuinely shared by the whole team and reviewed on a regular basis in the light of scientific evidence and the practice monitoring of outcomes.

Figure 1 sets out the author's suggestion for the format of an oral health policy noting that consistency within your dental organisation will improve patient perceptions and facilitate teamwork in the approach to patient care. As you will see from Fig. 1 risk assessment has a prominent and essential place in the 'starting line' of this protocol in order to consistently categorise patients for the subsequent risk-based prevention phase of care.

It will be clear from other chapters in this book that you currently have some choice in patient assessment tools which might include risk assessment. The UK government is developing such tools which could eventually become mandatory in

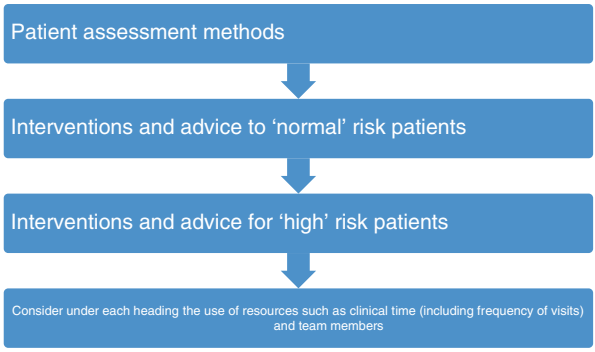

Fig. 1 Suggested format for practice oral health policy

state services. Your selected tool might be paper-based, or you may well opt for IT-based systems such as those available internationally from the PreViser Corporation of Washington, USA.

The author has had the privilege to work with PreViser as part of the team developing the Denplan PreViser Patient Assessment (DEPPA) [12] for the UK market. DEPPA uses the red, amber, green (RAG) protocol to highlight both disease risk and current health status to both clinicians and patients in its reports. Further, DEPPA offers numerical scores for both disease risk and status and an encrypted patient anonymous and centralised database, so that clinicians can audit their outcomes against those from hundreds of other practices. DEPPA also offers risk-based fee code guidance for capitation payments and therefore is an ideal fit for the author's preferred practice funding (see opening paragraph).

Your team will need to decide collectively on your broad clinical approaches to patients according to risk. There is now a considerable evidence base to support you in these deliberations. Public Health England and the Department of Health for example are now on their third edition [13] of a publication designed to summarise this evidence in a 'toolkit for prevention'.

Energy and Delegation

Radcliffe's leadership model in this chapter has been sectioned such that 'Future–Engage–Deliver' appear to be separate phases of leadership, whereas in reality they in fact merge. The leader will need the spirit and energy to be constantly re-engaging the team with the vision and to get things done through the guidance of the policies.

That energy will originate in the leader's passion for the vision. However, it soon dissipates if leaders fail to look after their own health and well-being. A healthy lifestyle including a sensible diet, exercise and relaxation away from the practice are all therefore integral to good leadership. Emotional as well as physical energy is paramount. Good leaders develop high levels of emotional intelligence as described by Goleman [14] so that they can manage their own emotions and, as Ratcliffe [4] has stressed, their relationships with other team members strongly. These are, once more, learnable skills. Managing relationships can be very easy when all is well. The challenge often comes when developmental feedback needs to be exchanged. Good leaders will manage to leave team members still feeling positive even after they have received criticism.

As we have already discussed in 'Engage' the various types of meetings are the vehicle for relationship building. It is very important to avoid using group meetings for either individual criticism or even praise; these are best left for one-to-one meetings. In respect of criticism the reasons are obvious. In the case of praise, just imagine being the 'quiet' team member who unobtrusively gets on with delivering the team vision without drawing attention to themselves. Think how their motivation might be affected by more 'forward' team members constantly being publically

praised because they are deliberately drawing attention to their successes. Individual performance rewards can also be divisive [15]; rewarding the whole team for collective performance seems a more open and effective approach.

The key meetings in respect of leading risk management will be the team meetings at which the oral health policy is reviewed periodically. In a small to medium-sized dental practice the whole team should discuss the approach to risk assessment. It may be the case that a member of the reception team, with a sufficient level of privacy, might be the ideal team member to interview patients on lifestyle and health history. Even if this is not the case the reception team will need to answer patient questions on risk assessment. Similarly the dental nurse will need to be fully engaged to facilitate your data collection. The hygienist may take on a significant proportion of the risk assessment workload and she/he or the oral health educator might be best placed to communicate your findings to the patients. Therefore, being involved in policy review has significant advantages. This brings us to delegation.

Richard Branson is unlikely to be flying your Virgin Atlantic plane or even serving the food and drink. Leaders of large organisations usually only face their customers directly on very big issues and even then it is usually through mass media outlets, not face-to-face. Almost all of Virgin Atlantic's 'delivery' is therefore delegated. Most dental leaders conversely are treating patients in their practices on a regular basis and are therefore right in the forefront of 'delivery' personally. Nevertheless, the dental leader does not have to do everything personally. Considering risk assessments specifically there are significant opportunities to delegate aspects to team members depending upon preference and the regulations under which you practise.

Delegation takes place at either one-to-one or group meetings. The leader needs to ensure that team members have adequate training in order to carry out delegated duties. Once more the periodic practice oral health policy reviews present the best opportunity for discussing delegation protocols in respect of risk assessments. Significant efficiencies can be achieved by delegation.

Metrics to Monitor Progress

You have the policies to deliver and you have agreed on how these will be implemented, but are they successful? Do they result in the outcomes prescribed by your vision? Perhaps the first step in respect of patient risk assessment is to audit whether they are actually happening in your practice, and if so, are they happening to the extent described in your oral health policy. For example you may have decided to conduct risk assessments for all patients annually. How close are you to this goal? This can be monitored by a record audit when using paper-based systems or automatically reported through computerised risk assessment.

Another outcome that you are likely to expect is that patient perceptions of their personal biofeedback on disease risk are positive. You might measure this qualitatively and/or quantitatively. It is a fairly simple matter to include a question or questions in patient surveys.

One of the objectives of delivering a risk-based preventive approach is to lower future disease risk by working with patients on their individual modifiable risk factors. Periodic audits of your RAG grades of risk can demonstrate whether you are reducing the numbers of regular patients with increased risk over time.

Finally, the ultimate goal for all dental practices is to improve and maintain the oral health of its patients. There are numerous ways that this ultimate outcome could be monitored. For example, you could audit whether the proportion of your long-standing patients diagnosed with severe periodontitis falls over time. Likewise are you measuring a reduction in caries incidence amongst your patient base over time? These data are readily available to users of DEPPA who can also compare their outcomes with those of colleagues in hundreds of other practices. DEPPA, uniquely, reports the Oral Health Score (OHS) for each patient, which is a validated and composite measurement of current oral health status [16]. Reports for each clinician are available giving average OHS compared to a national reference sample result from other users. Practices can easily monitor whether the oral health of their patients is improving, on average, over time.

Outcome measurements, whichever you choose to use, need to be discussed periodically at oral health policy review team meetings. This means that your outcomes together with the changing evidence base can continue to inform your policy.

Summary Bullets

- Develop a practice vision statement which inspires you and your team to conduct patient disease risk assessments as an integral part of your service.
- Continuously engage with the team in such a manner that they maintain a commitment to the importance of patient disease risk assessment for your collective success.
- Continuously develop a practice oral health policy to guide the team in the implementation of patient disease risk assessments in practice and how these assessments will inform your risk-based preventive approach.
- Continuously measure your results to monitor your success. Use this monitoring, along with the developing evidence base, to inform oral health policy revisions.

Declaration of Interest Until 31/12/18 I was a dental adviser to Simply Health Professionals. Mike Busby.

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Implementation of Patient-Based Risk Assessment in Practice

Leonardo Trombelli and Roberto Farina

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Rationale for Patient-Based Periodontal Risk Assessment

Periodontal care encompasses a series of personalized preventive and treatment measures that primarily aim to avoid the future deterioration of the periodontal condition of a specific individual to a severity and extent that leads to tooth loss and consequently negatively impacts upon function, aesthetics, and quality of life. When considering the management of periodontal diseases, even when manifest in their mildest forms (plaque-induced gingivitis or as Stage I/II, Grade A periodontitis cases), it is evident that periodontal care cannot be simply based on eliminating/controlling disease-associated symptoms, but also on the control of modifiable risk factors affecting disease progression. Moreover, following the active phase of periodontal treatment, patients should be enrolled in a stringent secondary prevention

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program (based on self-performed and professional control of the supra- and sub-gingival dental biofilm) whose modalities and frequency should be tailored to patient susceptibility for disease recurrence [1]. These considerations assume greater relevance when considering that periodontal treatment is costly, generally uncomfortable, and frequently associated with undesired sequelae such as dentine hypersensitivity. As a direct consequence, the standardized application of the same protocols (for intensity, modality, and monitoring) for primary and secondary prevention as well as for the active treatment phase itself will rarely meet the individual's needs, resulting in under-provision of care to some and over-provision to others, with unwanted side effects and suboptimal allocation of resources [2]. Therefore, to deliver personalized and precision periodontal care, periodontal risk assessment should be incorporated in the periodontal evaluation as a third dimension, to comprehensively assess and interpret factors that may have an impact on future disease onset or progression and upon a patient's anticipated response to periodontal treatment [3].

Periodontal risk assessment is not just a working philosophy or a treatment approach, but rather a pragmatic and effective method to categorize patients, teeth, and sites according to their probability to exhibit future disease incidence or progression. This option is currently available to clinicians at two of the most important phases of therapy: at first visit/initial presentation and at reevaluation after active treatment (at either the beginning of supportive periodontal therapy, SPT, or during the course of SPT). At the first visit, the practitioner needs to identify individuals at high risk of disease incidence (if still healthy) or progression (if already diseased). This information is essential to inform and motivate patients to undergo preventive/treatment strategies that have been tailored on their individual risk profile. At the completion of active periodontal therapy, the practitioner needs to determine whether the resulting periodontal condition will be compatible with long-term periodontal stability [4], assuming that the patient will be supervised and regularly maintained through a personalized SPT program.

It has been recognized that periodontal risk assessment is a necessary component of all comprehensive dental and periodontal evaluations as well as part of all periodic dental and periodontal examinations. In a position paper, the American Academy of Periodontology stated that risk assessment may help dental professionals predict the potential for developing periodontal diseases and allow them to focus on early identification and to provide proactive, targeted treatment for patients at risk of progressive/aggressive diseases [5]. Similarly, in a recent consensus report, the European Federation of Periodontology supported the usefulness of risk assessment tools, recognizing their validity to capture the complexity of the patient profile, inform clinical decision-making, and communicate potential preventive targets to the patient [2]. The development of a new periodontal classification system that embeds prognostic determination (World Workshop for the Classification of Periodontal Diseases and Conditions, [6]) reinforces the importance of risk assessment in the comprehensive patient evaluation. In this respect, the new case definition of periodontitis incorporates a framework for implementation of biological grade (risk or actual evidence of progression) of the disease. In the grading system,

the patient is assigned a grade A, B, or C (corresponding to slow, moderate, and rapid rate of progression, respectively) depending on direct and indirect evidence of periodontitis progression as well as exposure to true risk factors (smoking and diabetes) [6].

Need for Risk Assessment Tools to Provide an Objective and Standardized Way to Define Patient Periodontal Prognosis and Treatment Need

During a recent interview of oral care providers, one hygienist said: "*It's host resistance. It's smoking. It's health. It's medical condition. It's psychological condition. They're all risk factors that you have to either ask or assess with your intuition and kind of put that all together and how individually all those things affect this one person.*" Similarly, a dentist declared: "*Frankly we don't use tools…all we do is you know, like intuitive finding. You look at the patient, and you set some bells off in your head… I know my knowledge base, I see some signs and symptoms, and then I kind of move onto another thing*" [7]. These responses clearly delineate a scenario where dental care providers still tend to interpret risk-based dental care as a process based on a subjective assessment, the latter consisting of identifying risk factors elucidated during the examination and history-taking process and then making an arbitrary judgment as to the magnitude and role these factors may be playing in disease status/progression. This clinical behavior appears to still dominate in clinical practice despite the availability of risk assessment tools [7].

With this context in mind, a group of researchers conducted an experiment where European periodontists, expert US periodontists, and general dentists were asked to assign risk scores for periodontal deterioration to each subject of a cohort consisting of 107 patients, assuming no treatment would have been performed. Subjective evaluations of periodontal risk were performed by assembling information from the medical and dental history, full-mouth periodontal charting, and periapical radiographs, and the level of inter-examiner agreement was assessed [8, 9]. Interestingly, a high level of disagreement was observed between risk scores assigned by different periodontal professionals, irrespective of the level of experience/competence of the tested clinician. In a separate study, 74 general dental practitioners and 46 dental hygienists were asked to express their judgments related to the periodontal prognosis of three simulated cases [10]. The three cases were chosen with a different disease condition as defined by the severity of bone loss, number of pockets, and level of periodontal inflammation (bleeding on probing). The three standardized cases reflected a well-maintained periodontitis patient, an untreated periodontitis patient, and a gingivitis case, respectively. Surprisingly, 64.4% of professionals participating in the study assigned the same prognosis to the three simulated cases, and only 3.4% of interviewed professionals recognized that the untreated periodontitis case had a worse prognosis compared to the other two [10].

Heterogeneity in risk scores assigned through subjective risk assessment as observed among clinicians may affect the appropriateness of treatment planning. In

this respect, different operators evaluating the same patient not only generated varying risk scores but also established different treatment needs [11].

The arbitrary nature of risk assessment may arise due to the difficulty of including all relevant parameters in an integrated decision. For example, the study by Persson et al. [8] showed that individual risk assessment was based almost exclusively on parameters related to disease severity (e.g., radiographic bone levels, periodontal pockets), whereas relevant risk factors (such as diabetes and smoking) were not accounted for in the risk evaluation [8, 9].

Tools for Patient-Based Periodontal Risk Assessment

These observations on the poor accuracy and reproducibility of subjective risk assessment called for the development of patient-based risk assessment tools, i.e., instruments that include a standardized, composite measure of risk expressing the probability for disease incidence/progression in a specific individual. Such tools aim at obtaining more uniform and accurate information, in order to optimize clinical decision-making, improve oral health for patients, and reduce health care costs [12].

Available risk assessment tools include the following:

- *The Health Improvement in Dental Practice Model (HIDEP) system model* [13]

  Health Improvement in Dental Practice Model (HIDEP) is a computer-based tool. Its model combines preexisting examination methods, risk estimation systems, and treatment suggestions into a new entity. According to HIDEP, the patient is classified as healthy, with a risk ranging between 0S (lowest risk) to 4S (highest risk), or sick, with a disease status ranging between 1 (mild symptoms) and 4 (severe symptoms). In HIDEP, the process links the risk score to prevention/treatment schemes.
- *The Periodontal Risk Calculator (PRC) (in either its original version or as incorporated in broader oral health assessment tools)* [14–17]

  Periodontal Risk Calculator (PRC) is a web-based tool that was later incorporated into broader oral health risk assessment tools (PreViser; DenPlan Excel/Previser Patient Assessment—DEPPA) for calculation of risk related to periodontal disease and other diseases and conditions (e.g., caries, non-carious dental lesions, and oral cancer). According to the PRC, the patient is assigned a risk score ranging from 1 (lowest risk) to 5 (highest risk). Based on the patient risk score and the contribution of each parameter, the system also provides general suggestions on which active interventions may be the most relevant to reduce disease risk.
- *Periodontal Risk Assessment (PRA)* [18] *and its modifications* [19, 20]

  The Periodontal Risk Assessment (PRA) is a spider web-shaped diagram composed of six vectors, each corresponding to a risk factor/indicator. The contribution of each risk factor/indicator to the patient risk is graphically reported on the respective vector (the greater the contribution of the risk factor/indicator, the greater the distance from the center of the diagram). Three concentric areas are

identified on the polygon, with different distance from the polygon center. Irrespective of the version of PRA (original or modified), the patient is assigned a low, moderate, or high risk score based on the largest area that has been reached by a predetermined number of vectors. For example, in the original version of the PRA, the patient is assigned a low risk if all parameters are in the low risk area or, at the most, one parameter is in the moderate risk area; a moderate risk if at least two parameters are in the moderate risk area and not more than one parameter is in the high risk area; and a high risk if at least two parameters are in the high risk area.

- *PerioRisk* [21]

  PerioRisk is based upon five parameters derived from a patient's medical history and clinical recordings. Each parameter is allocated a parameter score (ranging from 0 to 4 for 4 parameters and from 0 to 8 for one parameter) according to predefined tables (Figs. 1a–e). The algebraic sum of the parameter scores is then calculated and relates to a patient risk score between 1 (lowest risk) and 5 (highest risk) (Fig. 1f). Recently, a simplified version of the *PerioRisk* (which was named as *SmartRisk*) was also proposed and evaluated. Risk profiles of the *SmartRisk* system were generated by adding the number of cigarettes per day and the number of sites with PD $\geq$ 5 mm [22].
- *Dentition risk system (DRS)* [23]

  The dentition risk system (DRS) is a computer-based, online tool that calculates chronic periodontitis risk for the dentition (Level I) and, if an elevated risk is found, prognosticates disease progression tooth by tooth (Level II). For patient-based risk calculation, numeric or dichotomous values are assigned to eight systemic predictors and nine local predictors, and then entered into the algorithm after adjustment with relative weights for each factor (unpublished). A risk score related to the dentition ($DRS_{dentition}$) is then generated as a continuous value.
- *Risk assessment-based individualized treatment (RABIT)* [24]

  Risk assessment-based individualized treatment (RABIT) calculates the risk of periodontitis progression as well as other aspects of oral health according to a series of unpublished parameters and calculation algorithms. In RABIT, the patient is assigned a low, moderate, or high risk, and prevention/treatment schemes are associated with the individual risk score.

The principles of application of any patient-based risk assessment tools are reported in Table 1, and the output (in terms of visualization of final risk score/category) is shown through two clinical paradigmatic cases (Figs. 2 and 3).

Overall, available scientific evidence indicates that risk assessment tools may discriminate subjects with different probability of disease progression and tooth loss [12]. The level of scientific support from longitudinal cohort studies, however, differs substantially among tools (Table 1). Differences reside in the number of longitudinal cohort studies investigating each method, in the level of evidence supporting the use of each method at the first visit or under SPT, and in the level of agreement between the study findings for each method. Among the periodontal risk assessment tools described above, the following four were currently validated on

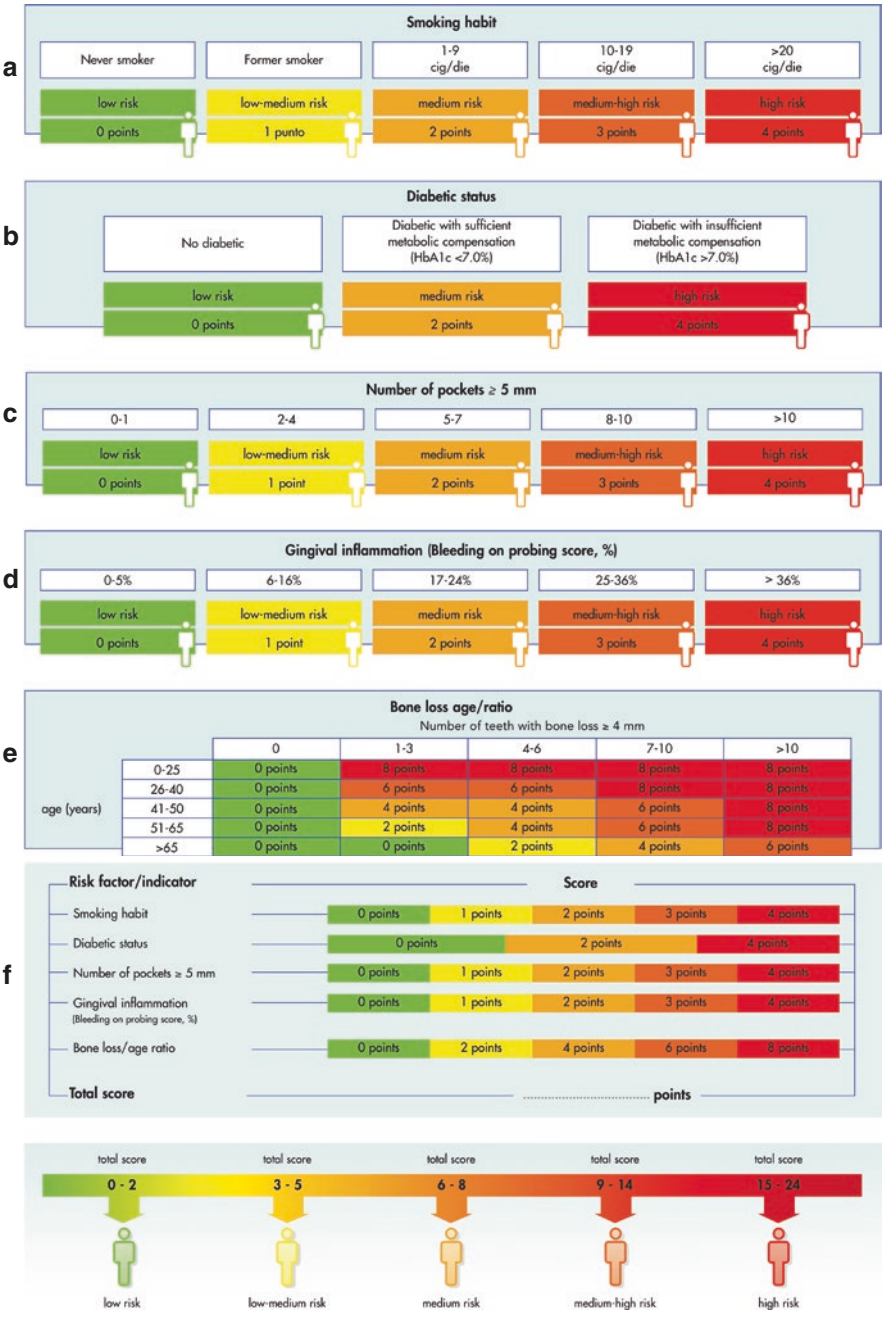


Fig. 1 PerioRisk method [21]. Generation of the score related to (**a**) smoking status, (**b**) diabetic status, (**c**) the number of pockets with probing depth ≥5 mm, (**d**) the Bleeding on Probing Score, and (**e**) the extent of bone loss/age, and (**f**) determination of the patient risk score (the parameter scores obtained from **a**–**e** are added and the sum is referred to a risk score ranging from 1 to 5)

Table 1 Risk assessment tools. Re-adapted from: Lang et al. [12]

Risk assessment tool (Author and year; COUNTRY)	Risk calculation based on	Algorithm for risk calculation (according to the original version of the risk assessment tool)	Output	Supporting evidence from longitudinal cohort studies
Health Improvement in Dental Practice Model (HIDEP) [13]	Fourteen parameters (partly shared with the caries risk assessment methods within the same tool): Total number of teeth, total number of intact teeth (teeth without restorations, caries, or crowns), number of caries lesions (initial lesions included), caries experience, fluoride exposure, saliva diagnostics (including secretion, buffering capacity, lactobacilli criteria, and streptococcus mutans), sugar intake frequency, oral hygiene screening, professional risk estimation for caries and periodontitis, gingival bleeding, probing of periodontal pockets, radiographic examination, registration of tartar, and/or overhang	Each parameter is assigned a default weight. If the sum of all parameters = 0, the subject is assigned to 0S risk category. If not, the risk group (ranging from 1S to 4) is determined on the basis of a combined evaluation of the bone levels and the Gingival Index	The patient is classified as: – Healthy, with a risk ranging between 0S (lowest risk) and 4S (highest risk). The S indicates need of support – Sick, with a disease status ranging between 1 (mild symptoms) and 4 (severe symptoms)	–
Periodontal Risk Calculator (PRC) (as either original version or incorporated in broader oral health assessment tools) [8, 9, 14–16]	Nine parameters: Age, smoking history, diabetes, history of periodontal surgery, pocket depth, furcation involvements, restorations or calculus below the gingival margin, radiographic bone height, and vertical bone lesions	Unknown (not published)	The patient is assigned a risk score ranging from 1 (lowest risk) to 5 (highest risk)	*Risk scores were associated with tooth loss and bone loss in absence of periodontal treatment* [16, 17] *Risk scores were associated with mean tooth loss rate during SPT* [15] *Risk scores were not associated with tooth loss during SPT* [25]

(continued)

Table 1 (continued)

Risk assessment tool (Author and year; COUNTRY)	Risk calculation based on	Algorithm for risk calculation (according to the original version of the risk assessment tool)	Output	Supporting evidence from longitudinal cohort studies
Periodontal Risk Assessment Model (PRA) and its modifications [18–20, 41]	Six parameters: Bone loss/age, number of pockets ≥5 mm, number of missing teeth, BoP score, cigarette smoking, and systemic/genetic factors (such as diabetes and Il-1 gene polymorphism) In the modified version by Renvert and Persson [41]: – Bone loss/age is replaced with extent of alveolar bone loss at the most compromised site – Systemic/genetic factor was omitted In the modified version by Chandra [19]: – Environmental factors, systemic/genetic factors are specifically defined as diabetes status and interplay of dental—systemic factors that accounts for dental factors; – Bone loss/age is replaced with attachment level/age – Other background factors are included to include estimated socioeconomic or stress factors – The scores on each trajectory ranged between 1 and 5 based on a coding system rather than using actual factor thresholds such as bleeding on probing percent, or numbers of pockets ≥ 5 mm. In the modified version by Lu et al. [20] – BoP score is replaced with bleeding index ≥2; – Sites with PD ≥6 mm, and not sites with PD ≥5 mm, are counted – Bone loss is measured as a full-mouth average, and not at the most compromised site	PRA is a spider web-shaped diagram composed of six vectors, each corresponding to a risk factor/indicator. The contribution of each risk factor/indicator to the patient risk is reported on the respective vector (the greater the contribution of the risk factor/indicator, the greater the distance from the center of the diagram) Three concentric areas are identified on the polygon, with different distance from the polygon center. The patient is assigned a low, moderate, or high risk score based on the largest area that has been reached by a predetermined number of vectors: Low risk if all parameters are in the low risk area or, at the most, one parameter is in the moderate risk area; moderate risk if at least two parameters are in the moderate risk area and not more than one parameter is in the high risk area; high risk if at least two parameters are in the high risk area	The patient is classified as low, moderate, or high risk	*Risk level (as assessed before treatment) did not significantly predict SPT outcomes in terms of tooth loss* [25, 26] *Risk level (as assessed at the completion of active periodontal treatment) significantly predicted SPT outcomes in terms of tooth loss and/or periodontitis progression* [15, 20, 27–31]

UniFe/PerioRisk and its modifications [21, 22]	Five parameters: Smoking status, diabetic status, number of sites with PD ≥5 mm, BoP score, bone loss/age In the modified version (*SmartRisk*) by Trombelli et al. [22], risk score is calculated as the sum of the number of cigarettes per day and the number of sites with PD ≥ 5 mm	Each parameter is allocated a parameter score (ranging from 0 to 4 for four parameters and from 0 to 8 for one parameter) according to predefined tables. The algebraic sum of the parameter scores is calculated and relates to a risk score between 1 (lowest risk) and 5 (highest risk)	The patient is assigned a risk score ranging from 1 (lowest risk) to 5 (highest risk)	*Risk scores were associated with tooth loss in patients under SPT* [22]
Dentition risk system (DRS) [23]	Systemic predictors: Age, family history of periodontitis, systemic disease, skin test result (assesses patient's inflammatory reactivity), patient compliance, and disease awareness, socioeconomic status, smoking habits, and therapist's experience with periodontal care Local predictors: Plaque, endodontic pathology, furcation involvement, angular bony destruction, radiographic marginal bone loss, pocket depth, bleeding on probing, marginal dental restorations, and tooth mobility	Numeric or dichotomous values are assigned to each variable and entered into the algorithm after adjustment with relative weight factor (unpublished)	A risk score related to the dentition ($DRS_{dentition}$) is generated as a continuous value	*Risk scores were associated with bone loss in patients under supportive care at 4-month intervals* [23]
Risk assessment-based individualized treatment (RABIT) [24]	Unpublished parameters	Unknown (not published)	The patient is classified as low, moderate, or high risk	–

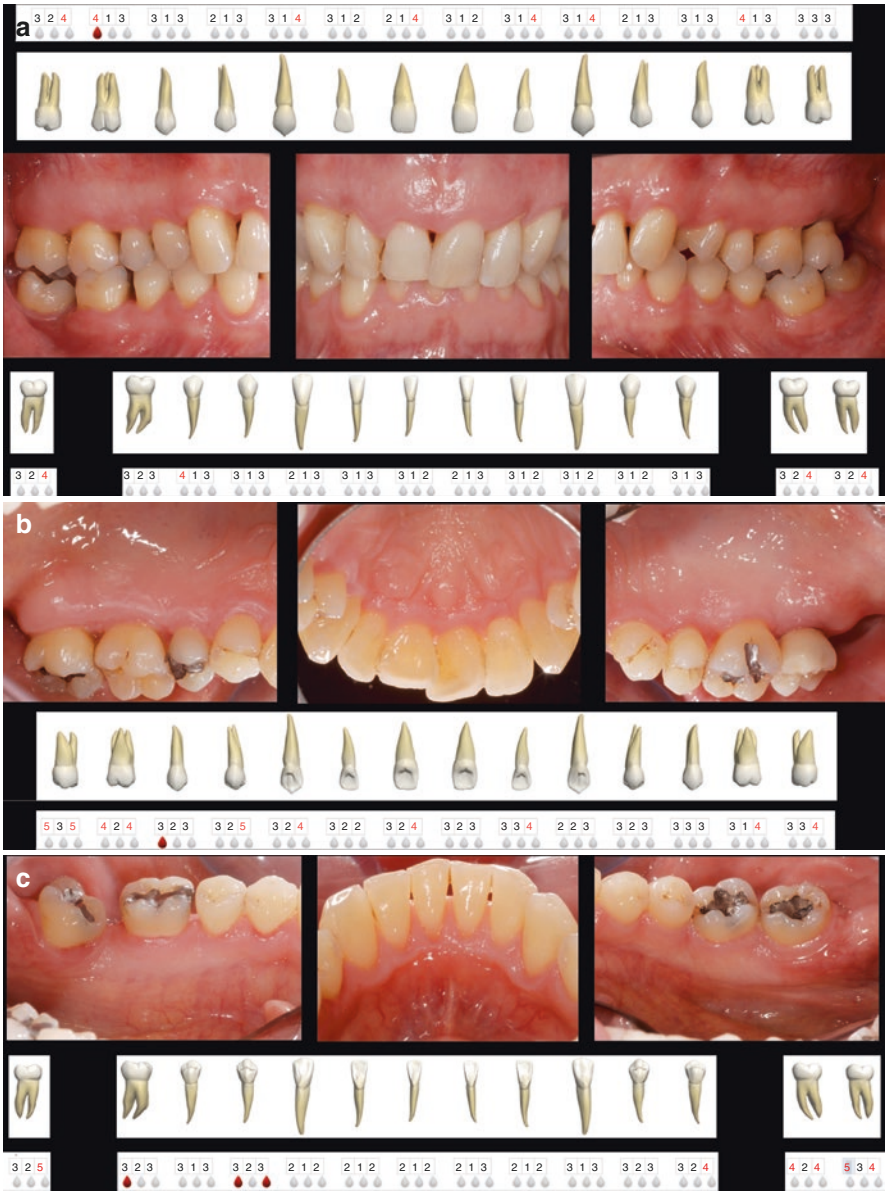
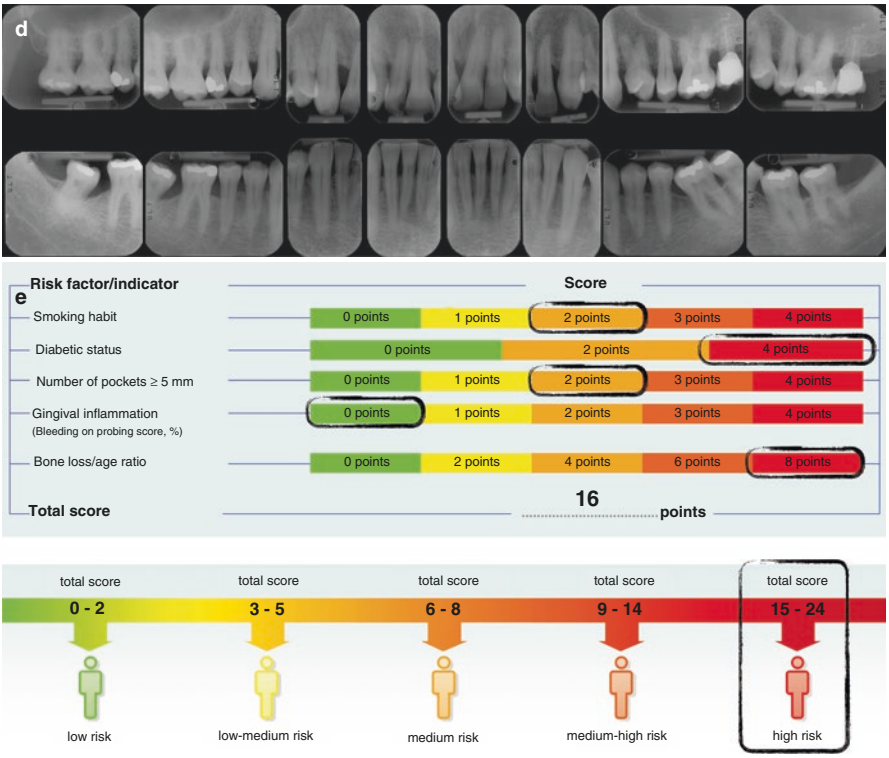


Fig. 2 Stage III periodontitis case with a high risk for periodontitis progression as assessed at the completion of active periodontal therapy. Probing depths (in mm) and bleeding on probing (present/absent) as assessed at the (**a**) buccal aspects, (**b**) palatal aspects of maxillary teeth, and (**c**) lingual aspects of mandibular teeth. Severity of bone loss is shown in periapical radiographs in figure (**d**). High risk for periodontitis progression was determined using (**e**) the PerioRisk method [21], (**f**) the Periodontal Risk Assessment (PRA) [18], where the patient is assigned a high risk since two parameters are in the high risk area, and (**g**) the Periodontal Risk Calculator (PRC) [16]



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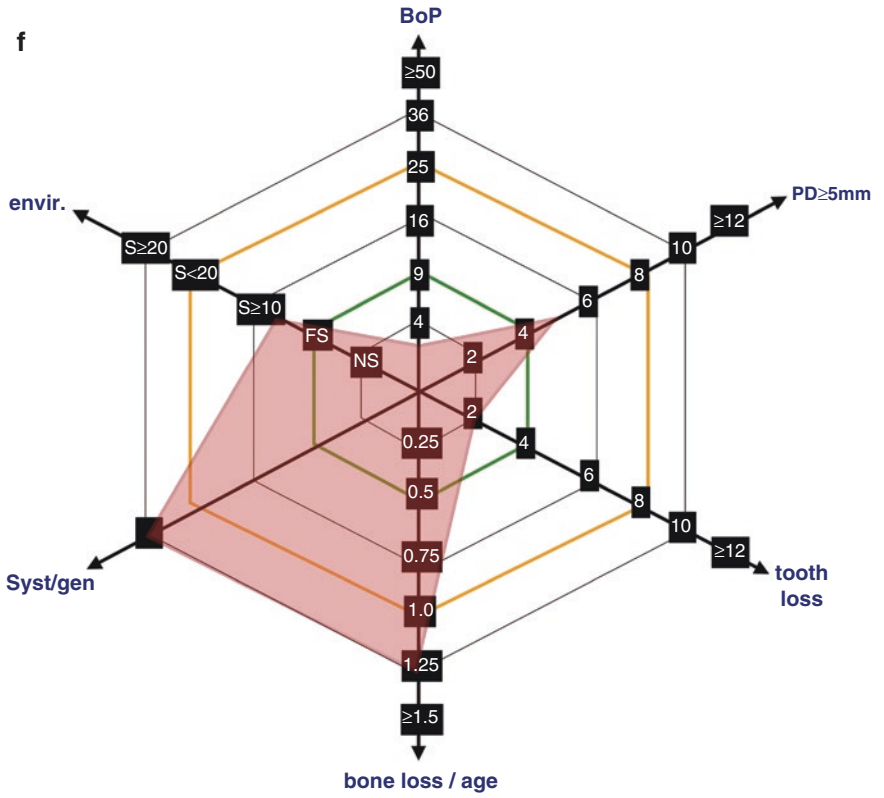

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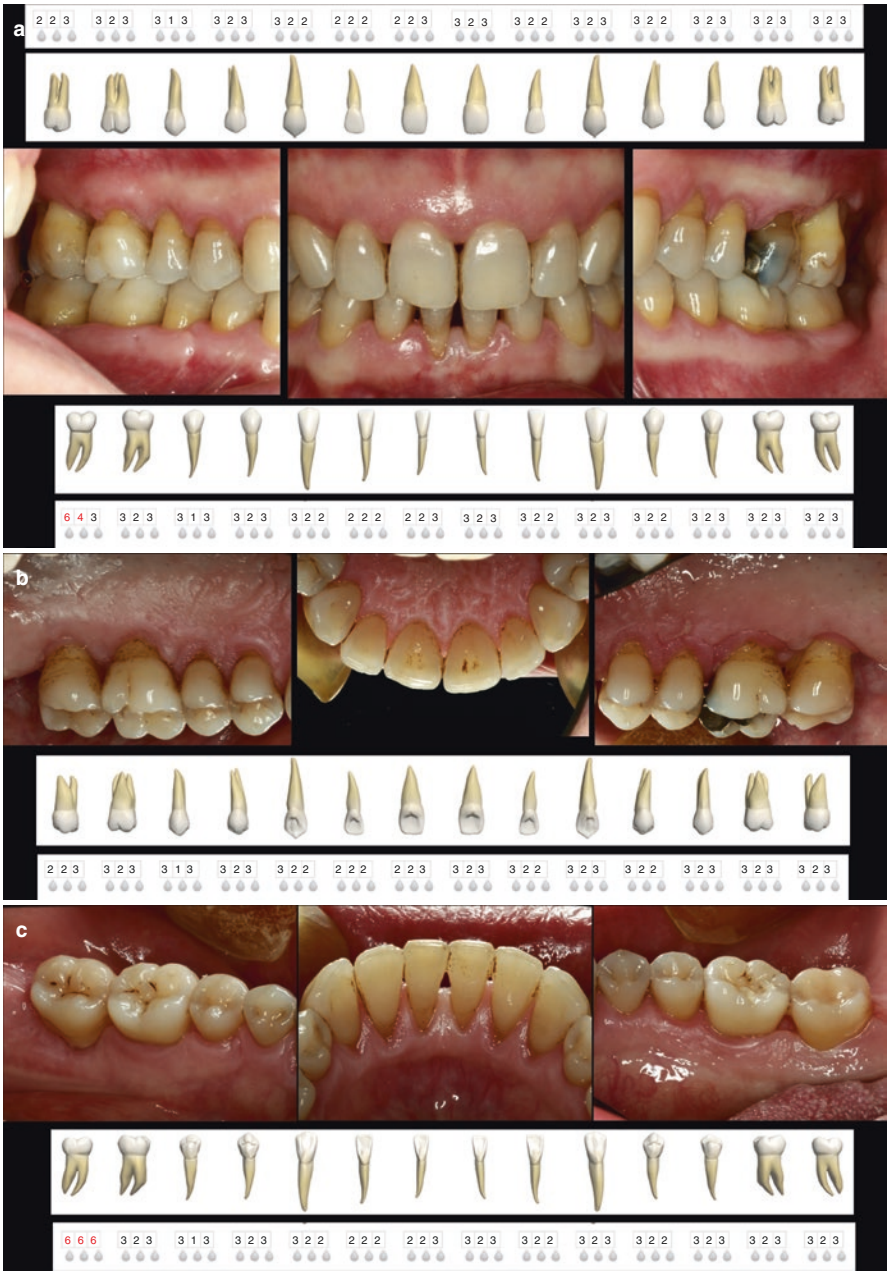
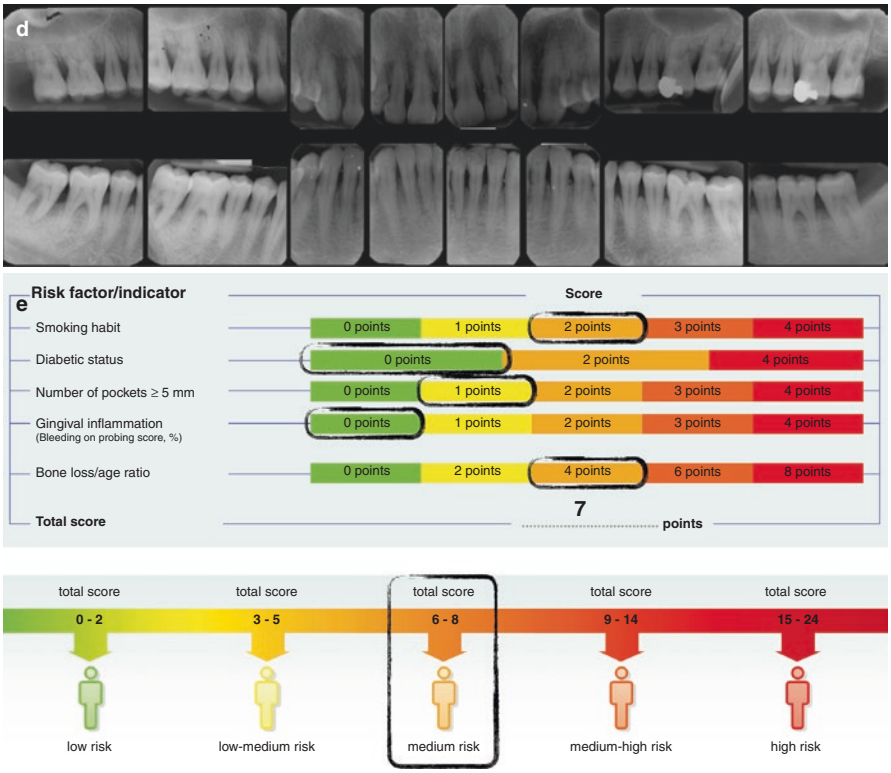


Fig. 3 Stage III periodontitis case with a moderate risk for periodontitis progression as assessed at the completion of active periodontal therapy. Probing depths (in mm) and bleeding on probing (present/absent) as assessed at the (**a**) buccal aspects, (**b**) palatal aspects of maxillary teeth, and (**c**) lingual aspects of mandibular teeth. Severity of bone loss is shown in periapical radiographs in figure (**d**). Moderate risk for periodontitis progression was determined using (**e**) the PerioRisk method [21], (**f**) the Periodontal Risk Assessment (PRA) [18], where the patient is assigned a moderate risk since two parameters are in the moderate risk area and no parameters are in the high risk area, and (**g**) the Periodontal Risk Calculator (PRC) [16]



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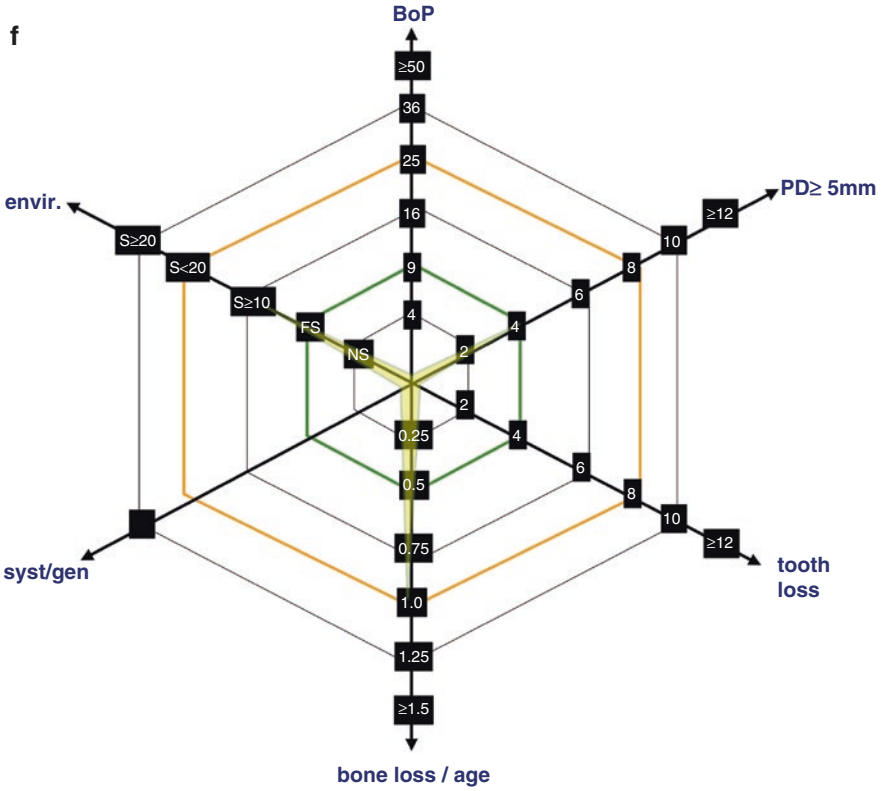

Fig. 3 (continued)

longitudinal data: PRC (in either its original version or as incorporated in broader oral health assessment tools) [8, 9, 15–17, 25], PRA and its modifications [20, 25–31], PerioRisk [22], and DRS [23].

Among tools with longitudinal validation, only the PRC is evidenced by a study where risk scores (as retrospectively calculated on data collected at first visit) were associated with disease progression in the almost complete absence of periodontal treatment [16, 17], while the other tools were applied in patients undergoing SPT. Indeed, the PRA tool was designed for assessing risk following initial periodontal therapy, whereas the PRC was designed primarily for baseline (pretreatment) risk assessment, and secondarily for post-therapy comparison of derived scores. Except for two studies that failed to find a significant association between tooth loss and risk scores generated by either PRA [25, 26] or PRC [25], available evidence indicates that risk assessment tools may effectively predict tooth loss and/or periodontitis progression [15, 20, 22, 23, 27–31].

To date, various studies have compared the risk scores generated with two different tools when applied on the same population [14, 19, 21, 22, 25, 32–34].

A group of studies compared the prognostic assessments performed according to PRA and those performed according to its version modified by Chandra [19, 32–34] or other risk assessment tools such as PRC [25]. When patient distribution according to risk scores of each method was evaluated [19, 32–34], substantial agreement between PRA and modified PRA was found, particularly in high risk categories [19, 34]. These analyses, which were either limited to descriptive considerations [32, 33] or substantiated by inferential statistics [19, 34], all share the common limitation of being based on the comparative evaluation of patient distribution within a cohort according to risk level rather than the assessment of the level of within-subject agreement between methods. Overall, therefore, current evidence suggests that both original and modified versions of PRA may be used to evaluate patient prognosis, but future studies will be needed to clarify whether one of the two methods must be preferred.

Two studies reported the results of comparative evaluations including the *PerioRisk* system. In the first study, a substantial level of agreement was observed between *PerioRisk* and PRC in a cohort of 109 randomly selected patients [21]. Interestingly, a simplified version of *PerioRisk* (which was named *Smart Risk*) recently showed a significantly greater prognostic value compared to the *PerioRisk* [22].

May Risk Assessment Tools Help in Tailoring Preventive and Treatment Strategies?

Robust evidence on the manner in which secondary preventive strategies should be tailored, based upon risk level, is still lacking [1, 35]. However, some information may be derived from studies where longitudinal evaluation of patient groups with a different prognosis and maintenance protocols has been carried out [22, 30].

Recently, a retrospective study was conducted on a cohort of 109 subjects with a diagnosis of gingivitis or periodontitis and undergoing SPT for a mean period of

5.6 years [22]. Groups with different risk scores [21] following active therapy were included in SPT protocols (with a similar frequency of recalls). In patients with a baseline risk score of 3 (moderate), 4 (moderate-high), and 5 (high), a significant difference in the mean number of teeth lost during SPT was observed (0.3, 0.9, and 1.8, respectively). The tooth loss rate per year of SPT was 0.07, 0.14, and 0.32, respectively, with a borderline significant difference between risk groups ($p = 0.053$) [22].

In a retrospectively selected cohort of 160 subjects [30], patients with a low, moderate, and high risk of periodontitis according to the PRA [18] were proposed for three different frequencies of SPT, ranging from at least one session per year (low risk group) to 3–4 sessions per year (high risk group). Based on tooth loss in patients fully complying with the suggested SPT program, the authors found that the low, moderate, and high risk groups lost on average 1.18, 0.80, and 1.71 teeth, respectively, during the follow-up [30].

Overall, these results reinforce the need for tailored secondary prevention strategies in patients with different risk profiles during SPT. The higher tooth loss in patients with a high risk profile despite the more frequent SPT visits compared to the other groups, observed in the Matuliene study [30] as well as in other cohorts [17, 36], could be argued to reinforce our current inability to effectively match the adopted secondary preventive protocol with the individual risk level.

Value for Risk Assessment Methods as Educational/ Communication Tools

Asimakopoulou et al. [37] have investigated the adjunctive effect of periodontal risk assessment when used as an educational/communication tool in 102 adults with moderate/advanced periodontitis. While all patients underwent routine periodontal assessment, patients in the test group received additional information on individual risk level. More specifically, information consisted in (1) calculation of periodontal risk with the PRC [17] and disease severity score, including an explanation of the concept of risk; (2) explanation of the current periodontal disease scores; (3) explanation of the patient's risk profile; (4) explanation of the contribution of lifestyle and oral health factors to risk score; and (5) exploration of patient reactions to these scores. Patients in test group showed a significantly higher consciousness of disease seriousness and a significantly higher intention to adhere to treatment instructions after consultation [37]. These findings support the use of this system in clinical practice and obtain greater patient adherence to the suggested preventive and treatment protocols.

Perceptions of the Oral Health Profession on Risk Assessment Tool

Overall, the perception of oral health professionals toward risk assessment tools (either entirely dedicated to periodontal risk assessment or included in broader oral health risk evaluation tools) is very positive. Oral health providers report a favorable

attitude toward using such tools [38] and feel that their use improves patient care outcome and practice productivity [38, 39]. Moreover, high utilization rates (98%) and levels of satisfaction were found in providers following comprehensive training [38], and clinicians reported a high level of agreement with the periodontal judgments generated by a risk assessment tool [40].

In a recent study, practitioners' perceptions of risk-based dental care, methods to perform risk assessment, as well as the benefits from and barriers to performing risk assessment were collected in a group of 27 general dentists and 25 hygienists working in solo and small group dental practices [7].

Perceived benefits of risk assessment in dental care can be summarized into the following points. Risk assessment:

- Helps dentists practice preventive dentistry, by categorizing patients based on their risk level, identifying the most relevant risk factors, and producing documentation that may inform the patient and serve as a reminder to dentists and patients during future visits.
- Helps dental providers play a bigger role in patients' health, by identifying risk levels and factors that may have an impact on the patient's overall health.
- Helps educate patients, by improving communication especially with the assistance of well-designed and validated risk assessment tools.
- Helps boost the business of dental practices, by improving patients' satisfaction and acceptance of treatment and increasing revenue over time.

On the other hand, the main perceived barriers that hamper the application of risk assessment in clinical practice were:

- Lack of solid scientific validation, since providers raised some doubts on the accuracy and validity of the risk assessment as derived from the current evidence.
- Time-consuming and under-reimbursed, since providers estimated that relevant extra time (to be quantified and reimbursed by the patient) was needed to gather and document all information regarding risk. The creation and validation of more user-friendly risk assessment tools was encouraged.
- Financial cost, due to the need for specific equipment and a service fee.

Clinical Recommendations for the Practitioner

Current evidence on periodontal risk assessment supports the following clinical recommendations for oral care providers:

- The use of periodontal risk assessment tools should be considered as a standard of care and should be applied at the first visit (not strictly periodontal, but oral) as well as at the beginning and during SPT. Unfortunately, at present none of the existing tools has been consistently validated for application at both phases. In

particular, PRA, PerioRisk, and DRS have been validated only for use after active periodontal therapy; PRC has been validated for use at first visit; however, contrasting results have been shown when used during SPT (Table 1).
– Risk scores generated by periodontal risk assessment tools should be used to (i) identify factors with the most relevant impact on individual patient prognosis and plan a preventive/treatment regimen targeted on elimination/control of such factors and (ii) quantify and monitor the impact of preventive and treatment interventions on risk level.
– Risk scores generated by periodontal risk assessment tools should be used to inform the patient regarding his/her disease condition and prognosis, and to motivate the patient for adherence to suggested preventive/treatment plan.

Conflict of Interests Prof. Trombelli and Prof. Farina have developed one of the risk assessment tools described in the present chapter [21], but declare not to have financial interests related to either this tool or its description in the chapter.

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Medicolegal View and Implications

Kevin Lewis

Contents

Introduction

Many people assume that medicolegal risks are in some way different from the range of clinical risks discussed elsewhere in this book. Quite often—although not always—they are a tangible manifestation of the same risks, or a failure to recognise them and/or manage them appropriately.

Medicolegal challenges can take many different forms, and perhaps surprisingly this is heavily dependent upon where in the world you happen to practise. In some parts of the world there are many separate self-governing jurisdictions with or without an over-arching Supreme Court or its equivalent to provide the checks, balances and general stabilising influence to maintain consistency of approach. In this chapter, we will explore examples of this from Europe, the USA and other countries. In some countries or individual jurisdictions, the levels of (clinical negligence/medical

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malpractice) litigation against dental practitioners are many times greater than in others. The same is true of the level of scrutiny by professional registration bodies and regulators (Dental Councils and Boards). But a likely precursor of most forms of challenge is an element of concern or dissatisfaction. Such concerns or criticisms can be voiced by professional colleagues, and lead directly to a medicolegal challenge or investigation of some kind. However, they may also plant the seeds of dissatisfaction in the mind of a patient which is then expressed as what we might call a 'complaint'—made either direct to the practice or to a third party (perhaps one offering practical assistance with complaint resolution through conciliation, mediation, etc.). Complaints are made at a local (practice) level far more frequently than either litigation or regulatory investigation, and if they are managed well at this stage, they do not escalate beyond the practice and/or involve other parties [1].

The degree to which dental patients think and act like empowered consumers is another variable and reflects attitudes in wider society as well as more practical considerations such as whether or not the patient has paid for the treatment personally and, if so, how much. Around the world there is a wide spectrum of mechanisms for funding the provision of dental care, and each country has its own infrastructure of legal and regulatory systems, and complaints pathways that are appropriate to local society and its culture. However, even within the same country and systems there is no shortage of evidence to support the proposition that individual variation between patients is the single most important determinant of where things finish up, and this in turn reflects their personality, past experience, expectations and not least what happened, how they feel about it and what kind of remedy or reparation they are seeking [2–6]. The same clinical scenario can unfold for several patients and what happens next will be different for each of them. Hidden from view in this analysis is another key factor—the relationship and interactions between the patient and the clinician [7, 8].

In some countries (such as the UK) where the level of both litigation and regulatory scrutiny is high, effective risk management becomes particularly important for clinicians as a defensive, self-protection strategy.

This involves

- Awareness: recognising and understanding the risks
- Minimising and controlling those risks wherever possible
- Taking steps to contain the risks and their potential consequences
- Risk sharing and transfer (including professional indemnity/malpractice insurance)

However, risk management is, for other reasons, equally important and valuable in countries where dental professionals are much less likely to be sued or investigated by their professional regulator or another such body. Separating the issue of compensation from any consideration of fault, blame, penalty or sanction brings wider benefits for healthcare systems as well as changing the dynamic between healthcare providers and those who have suffered harm. In New Zealand, for example, a no-fault compensation scheme exists so that individuals who have been accidentally

injured or suffered harm cannot sue anyway so they do not need to identify who was responsible or attribute blame. Instead they can apply for compensation for the personal injury they have suffered, including adverse outcomes of medical and dental treatment. This is paid through a state agency (the Accident Compensation Corporation—ACC). Accountability and quality improvement are achieved in other ways, as they are in Scandinavia, Iceland and elsewhere where various versions of a 'no-fault' approach also apply: this will be further explored in Section 'Risk, Fault, Blame, Harm and the Law', later in this chapter.

In short, the same clinical risks will not carry the same medicolegal risks for every treating clinician. Moreover, the clinical risks that might preoccupy researchers, academics and clinicians are in many cases not those at the top of the leader board in terms of their propensity to result in a complaint, clinical negligence claim or regulatory investigation. Adverse (or sub-optimal) clinical outcomes do not necessarily result in medicolegal challenge—indeed, the large majority of them do not.

Risk Assessment Models

The traditional models of risk assessment provide a logical starting point for this discussion, but they do not tell us the whole story. In bridging the gap in our understanding between clinical risk factors and medicolegal risk factors, we must first appreciate the difference in perspective between oral health professionals on the one hand and patients on the other. Patients will often be perfectly happy with an outcome that a lot of clinicians would be deeply troubled by. And yet a recurring feature of so many complaints and negligence (malpractice) claims is a patient who is dissatisfied with an outcome that the clinician believes to be entirely satisfactory or even much better than many other clinicians could or would have achieved in the same circumstances.

One of the traditional tools for assessing risk is to consider firstly the *likelihood* of an event occurring, and secondly, the potential *severity* of the consequences if that were to happen. Combining these two determinants, the resulting risk matrix (Fig. 1) creates a clear differentiation between the most and least important risks leaving a somewhat less well-defined grey area in between, the legal implications of which will be explored in Section 'Evidence-Based Dentistry: A Note of Caution' below. But health care is one of those fields where it is also necessary to consider *frequency*, i.e. how often one is exposed to that same combination of risks. One or more mitigating factors may come into play in one situation, but one cannot rely on this happening every time—it may be a simple case of the wrong thing happening on the wrong day, for the wrong patient. This turns our two-dimensional risk matrix into something resembling a three-dimensional Rubik's cube because of the interplay between the three dimensions. In a single instance, the highest risks are still those with the highest combination of likelihood and potential impact. But viewed over a period of time, or a career, exposing yourself to these risks again and again makes it more likely that the adverse outcome will not be avoided by some kind of mitigating factor that might come to the rescue on a single occasion (see Fig. 2).

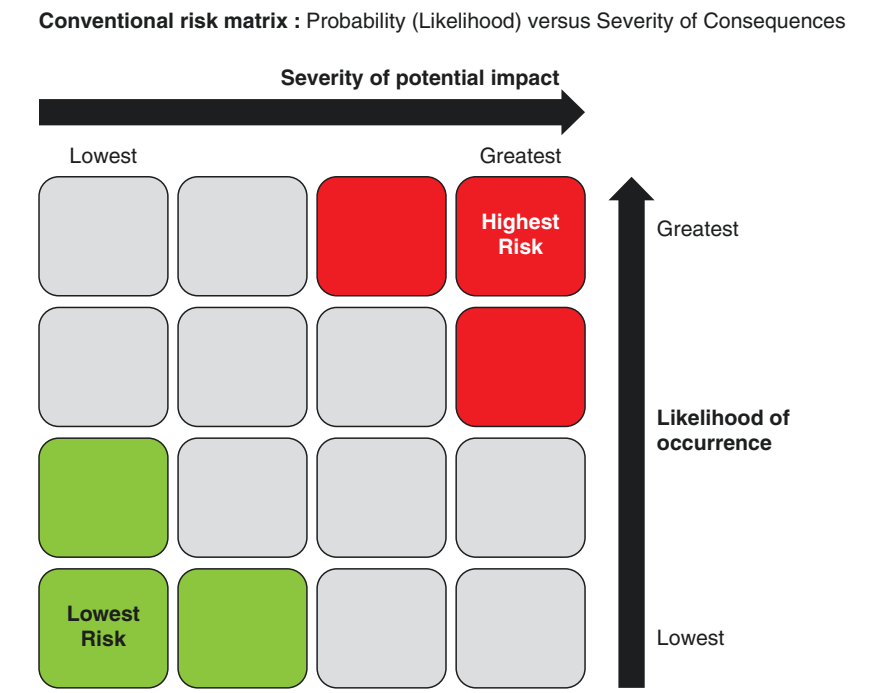

Fig. 1 Conventional risk matrix: Probability (Likelihood) versus Severity of Consequences

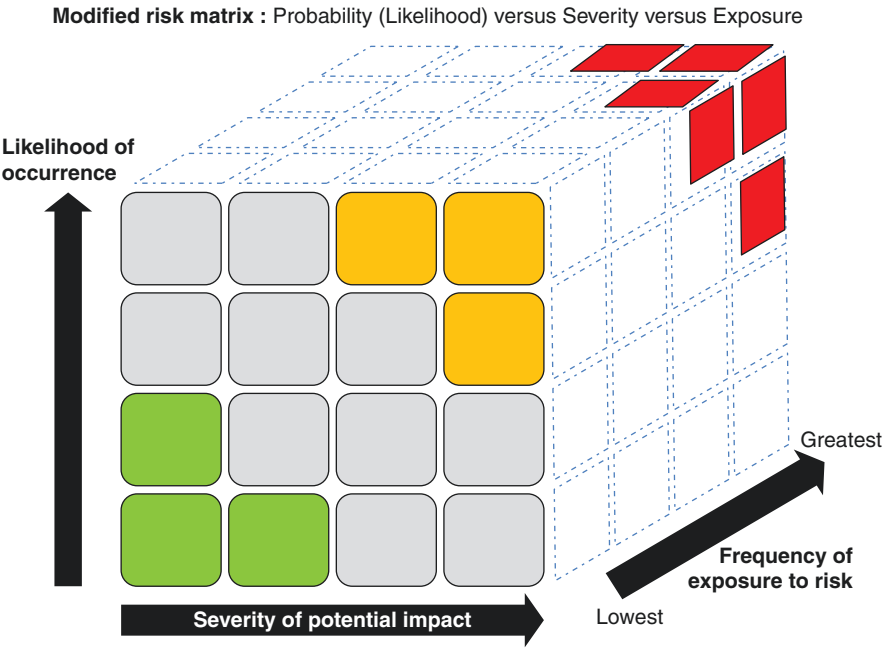

Fig. 2 Modified risk matrix: Probability (Likelihood) versus Severity versus Exposure

To illustrate what this means in practical terms, a specialist endodontist will be exposed to the particular risks associated with endodontic procedures all day, every day. A general dental practitioner might carry out endodontics much less often, so at first sight might appear to be less exposed to these risks. But—as with most hypothetical models—reality is more complicated than that. The accumulated knowledge, experience and skill of the specialist may well reduce the likelihood of many of the risks and complications occurring, and/or allow any adverse consequences to be mitigated or avoided. A separated (fractured and retained) endodontic file could be one such instance; the experienced specialist will probably encounter this complication less often, and also be more likely to be able to recover the separated instrument and successfully complete the cleaning and obturation of the root canal system. The distribution of risks will therefore be different for specialists and generalists, even after taking into account the fact that the generalists are transferring risk each time they refer their most complex cases to the specialist, and the specialists are accepting risk by spending more of their time on more difficult and challenging cases.

Standard of Care

In most countries around the world there is an underpinning assumption that clinicians owe a duty of care to the patients they treat, with the expectation that they will exercise a proper degree of skill and care in all aspects of the professional services they provide. It is not left up to individual clinicians to decide what represents an appropriate standard, and although the practical details and terminology vary from one part of the world to another, the applicable benchmark is generally that of a standard accepted as being appropriate by a responsible body of professional peers—that is, other clinicians working in the same field and professing to have the same skills.

Risk impacts upon this in a number of ways, including the following:

(a) Clinicians are expected to keep themselves sufficiently up to date with scientific knowledge and literature relevant to their field, to enable them to discharge their duty of care.
(b) Clinicians are expected to understand risk factors in the aetiology and progression of disease, and their likely impact upon the prognosis for any treatment that is under consideration. This is the patient's risk, but a failure to make that clear and to assist patients in managing their risk appropriately creates risk for the clinician also. Familiar examples of this include the 'duty of care' obligation for a clinician to provide relevant health information and to give appropriate advice and recommendations regarding oral hygiene, diet, smoking cessation and in relation to other risk factors. The patient needs to be made aware of the potential or likely consequences of failing to follow the advice given—with appropriate emphasis applied for the disengaged, reluctant or non-compliant patient. In medicolegal terms this has come to be viewed as an extension of the well-established concept of 'informed refusal' [d] in that the patient is taking a considered and informed decision to disregard or not to act upon the advice they have been given by a health professional in their best interests.

Even then, the clinician faces the further risk of having to demonstrate—through their clinical records, perhaps—that the risks had been made sufficiently clear and that they took steps to ensure that the patient also understood the information and advice given to them. In a world that looks for fault and blame, one often encounters a reluctance to accept any personal responsibility for one's own actions and failings, and a desire to find somebody else to blame.

(c) In the context of risk in a dynamic field such as dentistry (and health care generally) this is particularly important when considering new approaches to treatment, using new materials and techniques. An individual clinician's attitude to risk will affect the extent to which they are likely to be 'early adopters' of new techniques, and how circumspect they might be. The flip side of this is how receptive a clinician might be to a fundamental change in approach—minimum intervention being a perfect example—and how willing to abandon outdated approaches even if (and especially if) they have become highly proficient in them. We will return to this question in Section 'Attitudes to Risk and Its Management' below.

(d) Clinicians are expected to plan and carry out treatment in a way that is mindful of any risks involved, the aim being to achieve an outcome which optimally manages risks and maximises any potential benefits to the patient.

(e) Clinicians are required to share information, and help patients to a position of understanding, so that they can properly weigh up their options and come to a well-informed, reasoned and considered conclusion about what treatment to undergo, when and from whom. This information should always include the purpose, nature, likely effects and risks of the treatment (including the likelihood of its success) and any alternatives. In many countries this forms the basis of the principle of 'informed consent' (sic) although increasingly there is an emerging acceptance that the term 'valid consent' is preferable not only because the mere sharing of information is less important than the achievement of *understanding* on the part of the individual patient, but also because the law itself requires more than information sharing per se, in terms of the age and competence (mental capacity) of the patient at the time the consent is being sought.

Material Risks

At this point it is necessary to explain something of a medicolegal conundrum in terms of which risks the law expects a clinician to be aware of, and which of them they should be making patients aware of (and in what terms). And here again the answer—to both questions—depends upon where in the world you happen to practise.

Until the early 1970s clinicians would not (in general) be vulnerable medicolegally if, as part of the consent process, they had warned patients about risks to an extent that would be considered reasonable, by a responsible body of fellow clinicians practising in the same field. This started to change first in the USA [a][b] and then Canada [c], and it was only in the 1990s that a more patient-centric approach gathered

momentum elsewhere in the world. It was the 1972 case of Canterbury v Spence [a] in the USA that first shifted the issue (of which risks to disclose) away from following the prevailing consensus amongst clinicians to considering the perspective of a reasonable person in the patient's position and what information they needed. It started the ball rolling away from the era of medical paternalism, and paved the way for the patient-centred doctrine of 'informed consent' to gain traction [d,e].

A common theme of the early legal decisions was a requirement to warn patients, in meaningful terms that they could understand and relate to, about important risks. In courts around the world these were variously described as 'significant risks', 'particular risks', 'material risks', 'major risks', 'rare risks', 'recognised risks', 'known risks', 'common risks', 'serious risks', 'special risks' and so on—usually with little or no guidance for the busy clinician as to what these terms were intended to mean. There was, however, a clear implication that a healthcare professional was (or should be) much better placed than a patient to appreciate the relative risks of different treatments in different situations, and the patient had a right to share the benefit of those insights before being asked to consent to a procedure.

In Australia, the landmark High Court decision in 1992 in the case of Rogers v Whitaker [f] was unequivocal in stating that patients must be warned of all material risks, defining such risks in the following terms:

> A material risk is one that in the circumstances of a particular case,
>
> – A reasonable person in the patient's position, if warned of the risk, would be likely to attach significance to it (the "objective test"), or
> – If the practitioner is or should reasonably be aware that the particular patient, if warned of the risk, would be likely to attach significance to it (the "subjective test").

The following year (1993), the Supreme Court of Canada mirrored this change of direction (at least to some extent) in the case of Ciarlariello [g],

> The appropriate approach is… to focus on what the patient would like to know… The critical question will always be whether the patient would want to have the information.

and by 1994 the courts in South Africa [h] had elected to follow the newly established Australian direction, adopting the definition of 'material risk' from Rogers v Whitaker in its entirety and, indeed, adding an interesting specific requirement that patients '*must have appreciated and understood the nature and extent of the harm or risk*' that might result from the procedure.

During this same decade, however, the courts in Ireland seemed unable to agree whether the disclosure of risks should be a patient-centric decision or remain one for responsible clinicians to make. But a High Court judgement in 2000 in the watershed (dental) case of Geoghegan [j] included the following statement from the presiding Judge:

> A clinician has a professional duty to warn a patient of <u>all</u> risks, including those risks of which he may be unaware.

This statement feels at first reading to have all the qualities of an oxymoron. But it needs to be placed in its proper context. Firstly, this was elective treatment and it was argued that in the absence of any medical necessity for treatment, the standard of risk disclosure by the clinician must be higher (reflecting the likely imbalance of knowledge and understanding between the parties). Secondly, the outcome that eventuated from this procedure was accepted as not having been published widely in the literature, but on the balance of probabilities was 'known'—albeit indirectly—and 'remote' (much <1%). However, it was also 'gravely serious' in its consequences as the patient was left with severe, intractable pain. In these circumstances the Judge was effectively saying that the fact of being unaware of the existence of a risk in a specific situation like this does not relieve the clinician of the obligation to have warned a patient about the possibility, however remote.

One by one, many other countries have since shifted to a more patient-centric approach to risk disclosure, mostly ending up with a requirement to disclose and explain those risks to which a reasonable person in the patient's position, if warned of the risk, would be likely to attach significance [k]. In short, the 'objective test' which forms the first limb of the Rogers v Whitaker Australian judgement. We have already seen that some countries moved faster and further than others; Canada and South Africa having swiftly added the 'subjective test' of the specific, individual patient rather than being content with the less demanding 'reasonable patient' (objective) perspective that still applies in many parts of the world even today.

In the UK, a 2004 majority ruling in the House of Lords in the case of Chester v Afshar [l] underlined the lengths to which courts can be prepared to go, to support patient autonomy and self-determination. But left behind in one of the dissenting opinions in this case (from Lord Hoffman) was a refreshing glimpse of common sense that often gets overlooked:

> The purpose of a duty to warn someone against the risk involved in what he proposes to do, or allow to be done to him, is to give him the opportunity to avoid or reduce that risk. If he would have been unable or unwilling to take that opportunity and the risk eventuates, the failure to warn has not caused the damage. It would have happened anyway

The UK has since gone further, following a 2015 Supreme Court decision in the case of Montgomery [m]. Its approach to material risk closely mirrors the 1992 Australian decision in the Rogers case and, indeed, has also adopted the whole of its definition of material risk.

This raises the bar significantly for clinicians—especially when treating patients that they do not know well. It takes time to understand a patient well enough to form a view on what risks they might consider 'material'. And until you advise them of the risk, they cannot assist you by telling you whether or not they attach significance to it. One patient may be highly risk averse and cautious, while another has a much greater risk 'appetite'; the same clinical risk might be material for one but not the other. Another patient might be blinded by the perceived benefits of a particular treatment ('cosmetic dentistry' being a common example), and not fully engage

themselves with any downside risks involved—the clinician may be telling them in clear terms but they may not be listening, or they may not act upon the information and advice being offered because their mind is set on having the treatment.

It is also worth mentioning in passing that in a world where patients are increasingly regarded as consumers, a requirement to disclose and discuss all the risks to which the individual patient might attach significance can quickly expand to include not just 'risks' as we as healthcare professionals might define them, but *any* kind of information that might affect a patient's decision or that a patient might wish to be told [9]—for example, about the personal health and well-being of the clinician, or their training and experience (both generally and in relation to a proposed procedure) [i], their success and failure rates, or any commercial motivation that might influence their advice and recommendations [b]. This pushing out of the envelope has already been seen in many parts of North America [10], Europe and elsewhere—with varying responses on the part of the courts. It obviously creates a moral hazard as well as a legal one because anyone can argue after the event that they would never have agreed to undergo the procedure at all had they been made aware of some piece of information, however obscure, to which they claim they would have attached significance. The fact that the courts, not patients and their lawyers, will be the final arbiter of this is unlikely to provide much reassurance for the clinician facing the stress of litigation.

Courts in the USA have also considered the responsibility of clinicians to ensure that patients understand not only the risks associated with a given procedure, but where applicable, the risks of electing *not* to undergo the procedure (so-called informed refusal) or postponing the treatment [d].

Evidence-Based Dentistry: A Note of Caution

One of the most valuable tools in the modern clinician's armamentarium is the existence of a meaningful evidence base and its ease of access. In theory this should make life and treatment outcomes more predictable, unwelcome surprises less frequent and our ability to share meaningful information with patients as part of the consent process, much easier.

Some would even argue that patients must be provided with every last detail of the evidence base, to enable them to assess the information objectively for themselves and to compare alternative treatment options. Not only is this another onerous prospect for the clinician, it also fails to recognise two important aspects of the consent process.

Firstly, it is not sufficient for the clinician to present the patient with information in terms that would be meaningful to another clinician. The evidence base is invaluable for the purpose of informing a clinician, but this is usually very different from what an individual patient needs to know, and how this information needs to be presented to each specific patient in the context of their personal situation and capacity to understand.

Secondly, while both the evidence base and clinical guidelines relying upon it provide information regarding risks and risk factors, and what treatment is most likely to succeed, or fail, it often takes no account of the particular situation, predisposition and circumstances of an individual patient [11]. Take, for example, a clinician who gives a standard explanation to every patient who is considering the provision of dental implants that (for example) the 'success rate' of implants is known to be *x%*.

Patient (A) is a young, healthy patient with no relevant risk factors, considering the provision of a single implant-supported crown placed in the lower molar region. Patient (B), on the other hand, is an elderly patient with a long history of periodontal disease, having eight units of implant-supported fixed restorations placed in the anterior maxilla. The patient has a heavy and longstanding dependence upon tobacco and alcohol, patchily controlled diabetes, extensive loss of alveolar bone in the area in question and a high lip line. The clinician's standard commentary on the 'success rate' of implants is clearly irrelevant and inappropriate to both of these patients.

This further illustrates the danger of giving the same information in the same way to every patient, and the importance of personalising any information provided, for each individual patient—just as so many of the courts around the world are increasingly urging us to do.

Similarly one might be describing the risk of temporary or permanent lingual nerve injury resulting from the surgical removal of a lower third molar. Quoting the risk in generic percentage terms is meaningless without also helping the patient to understand what the practical consequences might be for them personally—this will obviously reflect many procedure-specific factors but it might also depend upon the patient's occupation. Quoting the risk of an adverse outcome in terms of a generic percentage of likelihood across all cases is not even meeting the test of the '*reasonable person in the patient's position*'. Patients are not interested in what might happen to other patients, but what is likely to happen to them personally, in the circumstances of their own individual case.

Another way of looking at this issue is to consider, for example, five possible ways to deal with a given clinical situation (Fig. 3). Option A is the one most strongly supported by the evidence base, striking an optimal balance between the risks and benefits. But B is also supported by a reasonably good base of evidence. At the other extreme, option E has no evidence to support it at all. In between, options C and D are possible but less likely to be successful than A or B.

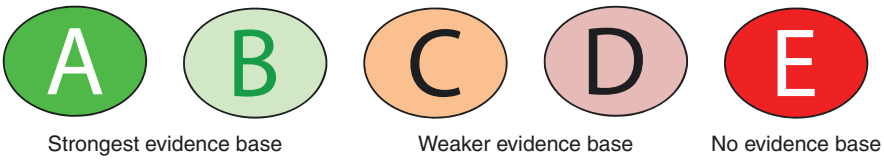

Fig. 3 Treatment options, the consent process and the evidence base

A specialist (or anyone else) who is very familiar with the evidence base may well feel that they would only be prepared to offer A and B (or even A alone) for the patient's consideration. In one sense this is a perfectly reasonable position to adopt, but should not be confused with a situation where the patient is never even made aware of the other options. Patients have a right to make irrational decisions or to proceed in ways that are contrary to their best interests and therefore to choose even E if that is their choice.

The clinician has every right to decline the provision of E, giving their reasons, but somewhere between B and E a line is crossed which is different for each clinician. No clinician should be persuaded to carry out treatment which is likely to leave the patient worse off, but patients do have a right to choose C or D if it appears to be a cheaper, quicker, simpler or more attractive alternative to A or B in some respect that matters to them. Even when they are told that C, D and E carry greater risks and a poorer long-term prognosis, some patients will still favour these options. The patient has a right to know that they may be available elsewhere as well as the fact that the clinician considers them less likely to succeed (or more likely to fail or cause other problems) and is therefore not prepared to provide them.

Perversely, clinicians who are the most familiar with the evidence base tend to be vulnerable to claims which allege that they denied the patient the option of even considering C, D or E, particularly if they chose not to mention them at all. On the other hand, clinicians who are less experienced or less familiar with the evidence base may not themselves provide options A and B and consequently might advocate C, D or E in ways which fail to present the risks and limitations in a balanced way (or at all). If the treatment then fails, risks transpire or complications arise, the allegation that they expose themselves to is that they could and should have acted in the patient's best interests by offering the patient a second opinion or perhaps a referral to a colleague who could provide A or B.

'Clinical pathways' are a natural extension of the evidence base and have the potential to compound the above problem if (for example) a third-party funder will only pay for treatment provided in line with prescribed/recommended pathways. This does not relieve a clinician of the responsibility to take into account the patient's own value systems and preferences—they have a right to at least know about, and the option to choose, a treatment approach which may differ from a clinical pathway. The clinician may be contractually prevented from providing treatment outwith the pathway, and has no obligation to provide treatment which they are not comfortable to provide and/or which they do not believe to be in the best interests of the patient: but this does not give the clinician a right (or an excuse) to run roughshod over the patient's autonomy.

Attitudes to Risk and Its Management

In 2014, Professor John Adams received the Lifetime Achievement Award from the Institute of Risk Management. In *Risky Business* [12], he had suggested that one can predict risk behaviours by looking at an individual's attitude to risk and

the personal 'risk thermostat' that each of us owns. He describes the concept of 'virtual risks', where in the absence of known, solid scientific evidence about given risks, individuals are at the mercy of their own judgements and attitudes, and their predispositions to view whatever evidence is available, in particular ways.

Adams (Fig. 4) describes a 'fourfold typology' of these predispositions:

- INDIVIDUALIST—a cheerful optimist who believes that if you cannot prove it is dangerous you can assume that it is safe. He believes that science provides solutions and is confident in his ability to fix things after the event.
- EGALITARIAN—a worried pessimist who believes that unless you can prove that it is safe, then you should assume that it is dangerous. He believes that science creates new risks and there will always be unseen risks that you do not know about.
- HIERARCHIST—believes that all risks can and should be quantified and managed. He does not like uncertainty, so he looks for evidence and demands information, systems and processes and measurement in order to gain as much control as possible over risk and uncertainty.
- FATALIST—a pragmatist who feels powerless in the face of forces that he feels unable to control. What will be, will be—and the best you can do is keep your eyes open, duck if you see something about to hit you and accept that bad stuff happens sometimes, whatever you do.

It is interesting to note in passing that the two quadrants to the left of the midline are essentially problem-solving dispositions, while the two to the right rely on problem finding (i.e. the ability to anticipate risks and problems).

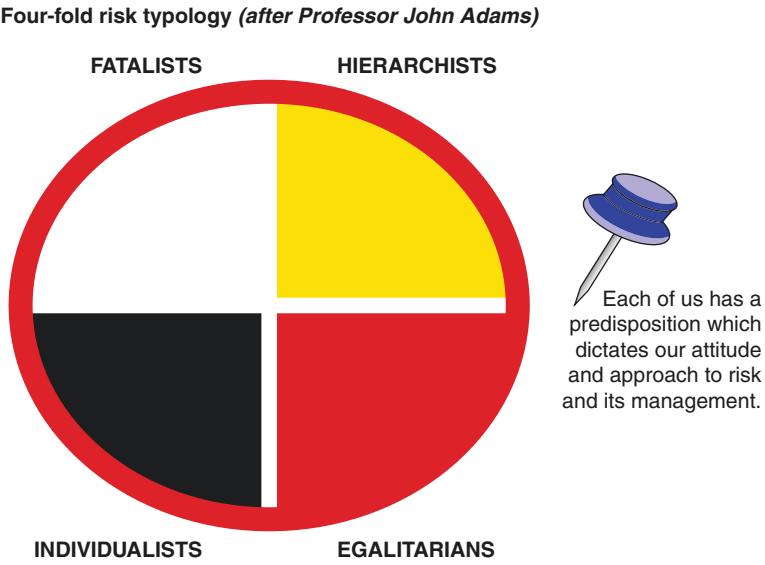

Fig. 4 Four-fold risk typology (after Professor John Adams)

Adams makes a compelling case that you should not ignore risk and simply hope for the best, but nor should you become excessively risk averse and negative. Instead he advocates an approach whereby you try to understand risks, effectively manage those that you are able to influence and accept those that you cannot control. Your approach should be positive and proactive, but you should not become so consumed by risk that you stop looking for solutions, perhaps because you are so busy looking for problems.

Having worked internationally in the risk field for almost 50 years, Adams fears that modern society is in danger of becoming paralysed by risk and the fear of criticism and litigation. He has argued that a generation of children have been so overprotected from risk that they have learned no mechanisms for managing risks proportionately as adults. It is a challenging and thought-provoking perspective.

Within the world of dentistry we encounter individuals with all of the above characteristics. One dentist will think it sufficient to learn a new procedure at a half day course, and will schedule the first patient to have this procedure carried out the following day. Another more cautious dentist will attend many courses, talk to colleagues and seek independent validation of their competence before taking the plunge. Meanwhile, many of the dentists who experience multiple claims (malpractice suits) and complaints seem to have no 'risk radar' whatsoever, or perhaps suffer from an unshakeable overconfidence—or occasionally, arrogance. This 'blind spot' can influence how they present and describe any risks to patients, and through the so-called framing effect [13] perhaps lead patients to share the clinician's skewed and unrealistic assessment of the risks, or simply to find themselves excessively reassured into a misplaced complacency about the risks. This is precisely what was found to have occurred in the UK case of Chester v Afshar [l].

We can all have the occasional bad day, but most of us will reflect on why it happened, learn valuable lessons and take steps to prevent a recurrence. If nothing else, an adverse experience makes us more circumspect. But if you go through life shrugging your shoulders and adopting a 'stuff happens' viewpoint you are unlikely to benefit from any reflective learning. You may even start from the premise that nothing is ever your fault.

Understanding our own 'default' predisposition to risk is an important part of developing a healthy, constructive approach to risk and its effective management. Spend a few moments reviewing Fig. 4 and think about your own approach to risk (and the attitudes of others with whom you live and work).

Risk, Fault, Blame, Harm and the Law

Fault-based systems of legal redress for personal injury in health care generally require three preconditions to be satisfied before a finding of negligence (or malpractice) can be made. The details and terminology vary between jurisdictions but in broad terms:

- The clinician must have owed a duty of care to the injured party.
- The standard of care provided has not met a generally accepted standard, and therefore the duty of care has not been satisfied.
- Harm must have resulted from the breach of duty.

These requirements for the patient to have been harmed, for fault to be shown and for blame to be attributed, are felt by many to be a direct obstacle to quality improvement. They are a legal construct designed to determine responsibility, and resolve disputes over personal injury. The focus is on looking back forensically at what happened in the past, rather than forwards at how things might be improved in the future.

Any balanced assessment of risk also needs to distinguish those adverse outcomes that might just as easily happen in the absence of fault (i.e. misfortune/ mishap/misadventure) from those that result from some kind of human error, mistake or active failure whether by act or omission. Interestingly enough, the law in most countries requires clinicians to warn about risks in the former category, but not the latter. But as public expectations of health care continue their relentless upward spiral, there is less acceptance and tolerance of sub-optimal outcomes. When bad things happen—in health care and elsewhere in life—the search begins for someone to blame, and hold accountable. In fault-based legal systems, the law does health care a disservice because it encourages an excessively defensive reaction on the part of healthcare providers and their representatives and advisers. Ultimately it does patients a disservice too, because it is by its nature adversarial and sometimes upsetting for all parties. It prolongs the time to achieve resolution and closure. Instead of being open and transparent about mistakes, reflecting upon them and sharing those experiences and lessons, fear of litigation might drive clinicians to be more tempted to cover up or deny mistakes. Instead of admitting and learning from errors, lapses, failures and adverse outcomes, and pooling that information to benefit others [14], the likely mindset of the defendant clinician when challenged is to refute allegations and justify their own actions.

Another argument in favour of no-fault compensation mechanisms is that they remove one of the main drivers for defensive medicine. This in turn has economic and other benefits [15], such as

- Reducing the likelihood of unnecessary tests and procedures being undertaken to 'protect' the clinician medico-legally, rather than to benefit the patient
- Increasing the willingness of clinicians to consider and undertake 'riskier' procedures that have the potential to provide significant benefits to patients, instead of protecting themselves by choosing not to offer those procedures to patients at all [16]

The 'golden thread' must surely be the repositioning of the compensation process as primarily a patient safety strategy rather than a risk management or rebalancing/restoration strategy. If patients and clinicians can both, with their different perspectives, operate in a high-trust, low-fear environment, then risk management,

patient safety and optimising clinical outcomes become a higher priority for all the right reasons. It fundamentally changes the risk environment.

For health professionals, investigations by professional regulators can be even more threatening than litigation, because of the possibility that one's registration/licence and continued ability to practise may be placed at risk. Unlike the courts, there is no requirement here for the patient to have suffered avoidable harm, but the standard of care is likely to come under close scrutiny nevertheless. A regulator will also be much more influenced by evidence that the clinician has reflected upon an adverse event, has learned any relevant lessons and is better equipped to manage risks more effectively in the future (this is not a consideration for the court in a negligence claim/malpractice suit). Being able to demonstrate insight not just into why something happened, but what needs to happen to make it less likely to happen again, is a precious asset when under medicolegal investigation by a regulator or perhaps an employer. Developing reflective learning skills and a proportionate 360° perspective on risk in all of its forms is the key to safe and successful practice.

In some countries (such as Australia) there is a legal requirement for all claim settlements above a specified financial threshold, to be reported to the professional regulator, in the interests of visibility. Whether or not the regulator feels the need to act further upon this information will depend upon the details, but it does make them aware of any clinicians who are experiencing multiple claims.

In contrast, in a well-established 'no-fault compensation' environment such as has existed in New Zealand since 1974 through its Accident and Compensation Corporation (ACC), one might be concerned at an apparent lack of accountability and a reduced sense of responsibility. In fact one finds the reverse because patients are empowered as consumers, and feel more like equal partners in their own health care. Furthermore, dental patients in New Zealand have access to free and independent second opinions from a Peer Review system provided by the profession itself, in addition to a range of healthcare complaint pathways—all being cost-free and easily accessible. And the regulatory oversight of the registration body is also focused squarely on maintaining professional competence rather than simply punishing underperformance when it comes to light. This tapestry of different mechanisms all routinely share information, so arguably there is more transparency than in many fault-based systems. It is designed so that patients speak up, health professionals listen and in most cases lessons are learned and improvements are made. The level of compensation on offer through ACC is not high, but ultimately it is funded by taxpayers and employers so a sustainable balance needs to be struck for society as a whole.

On the other hand, some commentators [17] have concluded that there will always be a need for accountability of some kind or another in health care: the public expects it, while also wanting all the benefits of a more open, no blame and learning culture. For this reason the mechanism of compensation is only one small part of the story, because the regulatory risk and the fear of sanction or de-registration drives behaviours as much (or more) than the risk of being sued per se. In most countries, insurers and indemnity providers cushion the financial aspect of that risk.

All of the Scandinavian countries, and also Iceland, have adopted variations on 'no-fault' compensation systems, reflecting a socio-political philosophy of greater state responsibility for and intervention in health care. Around 60% of dentists in Sweden, for example, and 45–50% of those in Finland work in the public health service—but this falls to 20% in Denmark and there is no equivalent service in Iceland. But across the Nordic countries dentistry for young children, adolescents and the elderly is mostly provided in the public health sector and uptake is very high. The close involvement of the state also manifests itself in direct reimbursement of private (especially specialist) fees in many situations. The compensation schemes are a natural extension of that state paternalism and intervention, compensating patients whose injuries which could have been avoided. It is akin to a recognition by the state that risks exist, and in an imperfect world those risks need to be managed collectively, in the interests of all parties. Unlike New Zealand, however, patients do still have access to the courts as a fall-back protection which works as an appeal process. The Nordic systems have long been held up as a constructive alternative to a fault-based system and supporters of this approach cite many benefits [18], but—especially in Denmark—the 'fault' element is not removed as completely as in New Zealand.

In Scotland, an expert group was convened in 2009 to consider the possibility of establishing a no-fault compensation system. This group's recommendation in 2011 was for a scheme to be introduced based upon the Swedish model and there followed a public consultation in 2012 and a response from the Scottish Government in 2014. For now the issue still remains under consideration: the recurring dilemma for healthcare systems and governments is the extent to which no-fault compensation schemes can or should be centrally funded from taxation or through direct levies at the point of use.

Both France (since 2002) and some states of the USA have adopted no-fault compensation for specific, limited situations (such as catastrophic injuries or 'serious and unpredictable injuries') alongside, in both cases, a tort-based system which still remains available. Unlike the Nordic countries and New Zealand, the motivation in the USA was not socio-political but economic: historically the state's direct involvement in US health care has been patchy at best, and private malpractice insurance was becoming unaffordable. Anything that threatened the continued availability of a sustainable obstetric specialty was a risk to society, not just to the medical profession.

Opponents of no-fault compensation systems maintain that their very existence necessitates costly administrative processes, wasting resources that could and should have been used in other ways to improve patient safety. These critics, unsurprisingly, often include the legal profession and claimant (plaintiff) law firms whose commercial interests are best served by fault-based systems and a continued reliance upon litigation. It is true, however, that no-fault systems are better equipped to deal with adverse outcomes of actual clinical procedures, than with injuries of a 'failure to diagnose' or 'failure to treat' or 'failure to warn' nature. It is equally true that attitudes to risk, remedy and quality assurance are partly cultural in nature and it is perfectly possible to learn from mistakes and improve performance in a fault-based environment if the will is there.

Communication

A useful perspective comes from the work of Bunting [19], who found that many complaints are triggered not just by the actual event(s) that tipped the patient over the edge into complaining (the 'precipitating factors')—like an adverse clinical outcome of some kind—but also because other things had already happened (the 'predisposing factors') to create doubts and concerns. These predisposing factors included poor communication, a perceived lack of interest, rudeness or a lack of respect—in short, interpersonal issues. In isolation, neither predisposing factors nor precipitating factors are generally sufficient to make a patient complain—it is the combination of the two that motivates the patient to take things further.

Young and inexperienced dentists may not (yet) have developed the full range of clinical skills that will help them to minimise technical deficiencies and the operative risks that result. This creates an even greater imperative for them to recognise the importance of developing their interpersonal skills, while also adopting a cautious and responsible approach to the treatment they provide, which recognises risk and the need to manage it effectively.

Good communication—including but not limited to verbal skills, listening skills and non-verbal communication—creates a better and stronger relationship between patient and clinician and this in turn reduces the medicolegal risk, irrespective of the clinical risk and any adverse outcomes that flow from it. So the corollary lesson to draw from Bunting's work is that clinicians who are not great communicators need to take risk management very seriously in their clinical decisions and technical approach, because they are more vulnerable and exposed than their colleagues if things do not go to plan.

A dentist who—for whatever reason—has a somewhat cavalier approach to risk is likely to encounter more medicolegal problems in the course of their career than a more prudent and risk-alert colleague. If you are not aware of the existence and importance of risks, you cannot manage them effectively nor train and develop other team members to do so. Nor are you well placed to explain these risks to patients and to help them to make informed decisions in the light of advice they are given, or regarding which risks they are willing to accept.

DiMatteo and co-workers [8] looked at whether or not it was possible to predict patient satisfaction and tolerance of adverse outcomes. In a study of 500 patients, split between those who had sued and those who had not, the conclusions were that

- Patients noticed and responded to the non-verbal communication of the physician.
- Patients formed views about the physician and his/her skills and level of care, based on their interpretation of that 'body language'.
- Physicians with the best non-verbal communication skills tended to engender significantly higher levels of patient satisfaction.
- When deciding whether or not to litigate, a dissatisfied patient would be strongly influenced by how they felt about the physician, as well as the actual treatment outcome.

Summary

We have seen how risk factors affect the likely outcome of patient care, and what might happen medico-legally if things do not go to plan. Some of these factors relate to the patient, some to the clinician, some to the treatment itself and some to the environment in which it is provided. All four have the capacity either to mitigate risk or to increase and compound it.

The medicolegal view of risk outlined in this chapter is intended to challenge many traditional perceptions and provide food for thought. The clinical evidence base is an invaluable tool for assisting clinicians in the assessment of clinical risk, but in the absence of any consideration of the individual patient, the individual dentist and the relationship between them, it is far less helpful where medicolegal risk is concerned—and can be actively misleading. This medicolegal view complements other risk perspectives, and relies on many of the same principles. But in a professional environment where risk is ever-present and the stakes are high, we discover a surprisingly positive message for clinicians and those who design the systems in which they work. We are not helpless onlookers in a world of all-pervasive risk after all, but involved and empowered participants in the safe and successful delivery of oral care and treatment.

Citations for Legal Cases Referred to in Text (Listed in Chronological Order)

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[b] **Gates v Jenson** [1979] 595 P2d 919 *(Washington, USA)*
[c] **Reibl v Hughes** [1980] 2 S.C.R. 880 - *(Canada)*
[d] **Truman v Thomas,** [1980] 27 Cal 3d, 611 P2d 902 - *(California, USA)*
[e] **Nixdorf v Hicken,** [1980] 612 P2d 348 - *(Utah, USA)*
[f] **Rogers v Whitaker** [1992] HCA 58, (1992) 175 CLR 479 - *(Australia)*
[g] **Ciarlariello v**. **Schacter**, [1993] 2 S.C.R. 119 - *(Canada)*
[h] **Castell v De Greef** [1994] 4 All SA 63 (C) - *(South Africa)*
[i] **Johnson v Kokemoor** [1996], 545 NW2d 495 – *(Wisconsin, USA)*
[j] **Geoghegan v Harris** [2000] 3 IR 536 - *(Ireland)*
[k] **Ravid Moshe v. Clifford** [2003]. HCJ 779/98 - *(Israel)*
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