H

Habilitation

► Rehabilitation

Habit Strength

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Synonyms

Habitual automaticity

Definition

A habit can be defined as a learned behavioral response to a situational cue. The repeated performance of a behavior in a specific context leads to the development of a behavioral habit that is triggered by features of the environment that have covaried frequently with past performance of the behavior. Such features of the environment might include performance locations, preceding actions in a sequence, the presence of particular people, or an internal thought or feeling. As a consequence of repetition in the same cue-contexts, a habit becomes capable of being triggered directly by perception of the cue. This is referred to as cue contingent automaticity. A person might experience his or her habit as something "I cannot help doing."

Habit strength is a function of the frequency with which an action has been repeated in a stable context and has acquired a high degree of habitual automaticity. Verplanken and Orbell (2003) developed and validated a metacognitive 12-item instrument to measure habit strength, the Self-Report Habit Index (SRHI). This is a generic instrument that asks respondents whether their performance of a target behavior occurs frequently; requires conscious awareness, thought, and effort; and is difficult to control.

From an empirical perspective, stronger habits are associated with heightened attention to cues associated with the performance of a habit and an increased likelihood of making an action slip when the cue is detected (Orbell & Verplanken, 2010). Removal of the cue (e.g., by changing one's environment) disrupts the performance of a previous habit (Wood, Tam, & Guerrero Witt, 2005). Strong habits also disrupt the ability to enact a counterhabitual intention. Strong habits may be useful in health contexts where, for example, good adherence is required and may be

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promoted by interventions that promote repetition in stable contexts (Orbell & Verplanken, 2010).

Cross-References

▶ Intention Strength

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Habitual Automaticity

▶ Habit Strength

Habitual Performance

▶ Physical Fitness

HADS

▶ Hospital Anxiety Depression Scale

Hamilton Anxiety Rating Scale

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Definition

The Hamilton Anxiety Rating Scale (HAM-A) is a widely used 14-item clinician-administered rating tool in the public domain used to measure the severity of anxiety symptoms among individuals previously diagnosed with anxiety disorders (McDowell, 2006). The HAM-A was originally developed by Max Hamilton in 1959 as an assessment tool to evaluate anxiety symptoms among people diagnosed with "anxiety neurosis." Since that time, anxiety neurosis has been reconceptualized and the HAM-A is used among individuals with a variety of anxiety disorders (panic, phobia, and generalized) (McDowell, 2006). The 14 items reflect 13 categories of anxiety-related symptoms including anxious mood, tension, fear, insomnia, intellectual/cognitive symptoms, depressed mood, general somatic (muscular and memory symptoms), cardiovascular, respiratory, genitourinary, and gastrointestinal symptoms, with one item capturing the rater's assessment of behavioral symptoms. The HAM-A takes approximately 15–30 min to administer and score and contains two subscales – psychiatric anxiety (psychological distress) and somatic anxiety (physical symptoms of distress) (Hamilton, 1959).

The HAM-A is not designed to be used as a diagnostic tool and has poor discriminant validity between anxiety disorders and depression. Instead, it is a standard primary outcome measure used to asses the efficacy of clinical interventions for DSM-IV anxiety disorders – most commonly

generalized anxiety disorder – within psychopharmacologic randomized controlled trials and psychotherapeutic clinical trials (Roemer, 2001). It also used for monitoring anxiety symptoms during treatment. The severity of the item is determined on a five point scale (0 = not present, 4 = severe). A computerized version as well as a pen-and-paper format is available (Kobak, Reynolds, & Greist, 1993), and a structured interview guide has also been developed to standardize its administration (SIGH-A), as its administration is not predefined in the initial instrument (Williams, 1988). A six-item abbreviated scale capturing psychic anxiety, tension, restlessness, inability to relax, startle response, worry, and apprehension called the Clinical Anxiety Scale was also proposed by Snaith, Baugh, Clayden, Husain and Sipple (1982). The HAM-A has also been translated into several languages, including Spanish, German, and Polish (Roemer, 2001).

Cross-References

- ▶ Anxiety
- ▶ Anxiety and its Measurement
- ▶ Anxiety Disorder
- ▶ Stress

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Hamilton Rating Scale for Depression (HAM-D)

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Definition

The Hamilton Rating Scale for Depression or Hamilton Depression Rating scale (HAM-D, HRSD, or HDRS) is a 21-item clinician-administered multiple-choice measure of depression symptom severity. The first 17 of the 21 items contribute to the total score (Hamilton, 1960) and items 18–21 give additional information not part of the scale, such as paranoia and diurnal variation (Hedlund & Vieweg, 1979). Other versions have been developed, ranging from 7 to 29 items (e.g., Hamilton, 1964; Williams, 1988). In all versions, symptoms are defined by anchor point descriptions (ranging from 3 to 5 possible responses), which increase in severity. Clinicians consider intensity and frequency of symptoms based on patient response and observations. A score of ≤ 7 is widely thought to indicate remission on the HAM-D_{17} (Frank et al., 1991).

The HAM-D was first published in 1960 and reviewed subsequently (Hamilton, 1964, 1980). Due to its comprehensive coverage of depressive

symptoms, strong psychometric properties (Hedlund & Vieweg, 1979), and the total score demonstrating high concurrent and differential validity as well as strong reliability (Carroll, Fielding, & Blashki, 1973), the HAM-D is considered by many to be the "gold standard" of assessing depressive symptomatology. However, most individual items demonstrate fair to poor agreement (Cicchetti & Prusoff, 1983). Use of the Structured Interview Guide, published in 1988, increased the reliability of the items (Williams, 1988). Self-report and computerized versions have been developed to improve the psychometric properties of individual items (Williams, 2001).

The HAM-D is primarily applied for research purposes to determine severity of depressive symptoms throughout treatment and in response to psychotherapy or antidepressants (O'Sullivan, Fava, Agustin, Baer, & Rosenbaum, 1997; Williams, 2001). More specifically, in the area of behavioral medicine, the HAM-D is used to measure the severity of depression in people with comorbid chronic illness. As assessment of depression can be particularly complicated in this population due to the co-occurrence of somatic features of depression and physical illness, the HAM-D has been criticized for its sensitivity to somatic symptoms (Maier & Philipp, 1985; Sutton, Baum, & Johnston, 2004). Consequently, researchers have evaluated the utility of the HAM-D for assessing depression in chronic illness. An early study assessing somatic comorbidity in a sample of elderly patients found that eight of the HAM-D scale items rated as positive scores for depression by psychiatrists were rated by internists as being related to somatic conditions (Linden, Borchelt, Barnow, & Geiselmann, 1995). Additionally, researchers compared depression rating scales in chronic fatigue syndrome and found that the HAM-D overestimated the number of depressed patients (Henderson & Tannock, 2005). Nevertheless, a more recent study evaluated the scale in depressed participants with multiple sclerosis and found that the majority of items (12 out of 17) captured depressive symptoms and adequately differentiated from somatic symptoms (Moran & Mohr, 2005). Results suggest that this co-occurrence must be considered when using the HAM-D in behavioral medicine settings.

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Handgrip Strength

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Definition

This term refers to a common measure used often in rehabilitation medicine to determine the maximum forearm muscular isometric strength. Given that muscle strength has general characteristics, the handgrip strength test may often indicate general muscular strength. The test includes a dynamometer, with a scale in kilogram, where people are asked to perform their maximal press with their hand. Different protocols exist concerning the angle of the arm and hand in relation to the body, the number of pressing trials, and the duration of pressure, normally lasting 3–5 s. This test can be used to indicate various health factors in different populations.

A review of the value of the handgrip strength test in dialysis patients found this test to correlate with general muscle mass, nutritional status (of importance in dialysis), and future complications (Leal, Mafra, Fouque, & Anjos, 2011). A review of 114 studies in the general population and 71 studies with arthritic patients found a strong age-related decline in handgrip strength, and much lower scores among arthritic patients, suggesting a relationship between inflammation and performance on this test (Beenakker et al., 2010). In some pain patients, this test is also helpful in assessment of their condition. For example, handgrip strength is lower in patients with fibromyalgia, and is inversely related to their levels of pain, fatigue, and stiffness (Aparicio et al., 2010). This test was used in several cohort studies to predict risk of death. For example, in elderly French women, a low handgrip test score significantly predicted risk of mortality, independent of confounders (Rolland et al., 2006). In patients with congestive heart failure, low handgrip strength also predicted risk of death, independent of confounders (Izawa et al., 2009). Furthermore, handgrip scores also prospectively predict decline in activity of daily living and in cognitive performance in the elderly (Taekema, Gussekloo, Maier, Westendorp, & Craen, 2010). Thus, the handgrip test is a simple, rapid, and objective test which provides information on important physical factors and has predictive validity in relation to functional, cognitive, and vital status measures.

Cross-References

▶ Functional Capacity, Disability, and Status

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Happiness

▶ Well-Being: Physical, Psychological, Social

Happiness and Health

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Synonyms

Physical well-being; Positive affect; Positive emotion; Subjective well-being

Definition

Positive emotions (including happiness) arise as the result of pleasurable engagement with the environment and may present themselves in a variety of forms (e.g., enthusiasm, calm, contentment). Traditionally, physical health is defined as the objective absence of disease or illness, but can also include perceptions of wellness.

Description

While the concept that happiness is tied to better health is not novel and is widely accepted by the public, the research in this area remains in its infancy. Due to the surge of interest in positive psychology over the last decade, researchers are beginning to unveil the predictive and protective effects of positive emotions on health. There are however many remaining critical research questions. This section will focus on the most robust and striking findings in the literature on positive emotions and physical health, in addition to a brief discussion on some of the important methodological concerns for the field.

What Is Positive Affect?

While there is some debate in the literature as to what adjectives and precise feelings make up positive affect (PA), it is typically considered to be the general positive emotions or feelings (e.g., happiness, enthusiasm, calm, or contentment) resulting from pleasurable interactions with the environment. These feelings may persist for long periods of time and define an individual's general disposition (often called trait PA) or they may be transient moments of emotion that last for minutes or days, typically referred to as state PA, positive mood, or emotion (emotion being the shorter lasting of the two). Research on PA and health primarily focuses on trait PA given its more likely *long-lasting* effects on physical well-being; however, on occasion, studies will assess the effects of state PA (commonly assessed via a one-time mood assessment asking questions like "How happy are you this week?"). While shorter time assessments of positive feelings are less likely to influence long-term health outcomes, they are known to have transient effects on physiology and are highly correlated with dispositional measures of PA.

Measuring Positive Affect

PA is most frequently assessed via self-report scales asking about the frequency, duration, or intensity of positive feelings. There are a host of different scales to do this with wide discrepancies between them. In the health field, the most frequently used multi-item tool is the 20-item Positive and Negative Affect Schedule (PANAS), which assesses affect by having individuals rate the degree to which each emotion word (e.g., enthusiastic or irritable) describes their typical mood with flexibility in the assessed time period covered. This scale focuses on aroused emotions and is therefore not useful for individuals interested in assessing the health impact of low-energy states (e.g., calm). There are however many other mood adjective checklists that include low-arousal emotions such as those using Circumplex Models of Emotion (Russell, 1980), which includes measures of both arousal and valence, or the extended 60-item version of the PANAS (the PANAS-X)

(Watson & Clark 1999). Studies have also utilized single-item questionnaires (e.g., "Are you happy?"), confederate report, positive items drawn from other scales (e.g., depression measures), or even autobiographical writing samples. Given the known high levels of social desirability and response bias to emotion scales, future research would benefit from greater use of unobtrusive and non-self-report methodologies to determine an individual's level of PA. One other critical measurement concern relates to the role of negative affect (NA) in the PA health association. At the trait level, PA and NA are often weakly correlated; however, they are sometimes considered to be opposite ends of the same spectrum by many researchers. It may be the case that benefits of PA on health are simply attributable to the absence of NA. The majority of studies do not test for the independence of these affect variables in relation to their health impact; however, those that do frequently report that PA is beneficial to health irrespective of NA. Also critical is to better understand what types of PA are beneficial to health. Given the divergent physiological impacts of high- versus low-arousal emotions (e.g., ecstasy versus relaxation), it is not unrealistic to anticipate differential health results. Nevertheless, this is rarely considered and frequently unmeasured due to the choice of affect items within scales.

Associations Between Positive Affect and Health

In their major review, Pressman and Cohen (2005) evaluated the results of over 150 studies on PA and health and physiological outcomes. They consistently found that greater PA was associated with increased longevity in individuals older than 55, in studies with years to decades of longitudinal follow-up. For example, in one creative study by Danner, Snowdon, and Friesen (2001), autobiographical writing samples from 180 young nuns entering the convent were coded for positive and negative emotion word usage. At a 50-year follow-up time point, researchers found that nuns who used higher levels of positive words lived almost 11 years longer than their counterparts who used the fewest positive emotion words. This finding was not attributable to negative word usage. Similar results have been demonstrated in multiple studies of healthy, community-dwelling older individuals revealing that those individuals who report greater amounts of PA at baseline live *years* longer than their less positive counterparts.

There is also consistent evidence that positive emotions are protective against a multitude of morbidity outcomes including decreased falls and injuries, reduced heart attack and stroke incidences, fewer hospitalizations for coronary complications, and improved pregnancy outcomes. An exemplary example of this is the viral challenge work of Cohen, Doyle, Turner, Alper, and Skoner (2003). In this study, PA (determined via interviews averaged over several weeks) was found to prospectively predict the decreased likelihood of developing an objective cold (and cold symptoms) after being experimentally exposed to a novel virus. These results were independent of the influence of trait NA, which was only tied to the *perception* of having a cold as opposed to actual incidence. This finding was replicated, in that positive emotion styles predicted fewer objective flu cases and fewer flu symptoms reported when the flu virus was experimentally administered.

Additional findings from the literature generally show that cross-sectionally, individuals with higher PA report fewer symptoms and generally feel better. What remains unknown is whether this is a true physiological process (e.g., altered opioid levels) or whether PA simply leads to altered attention to or perception of symptoms. Similarly, it may also be true that feeling of health lead to greater positive emotion. Most studies to date do not address these mechanistic and directional questions.

Finally, survival studies of diseased patients have provided *some* indication that PA may lead to improved health outcomes, but not in every circumstance. Research on those with early-stage life-threatening diseases (e.g., HIV, stage I–II breast cancer) indicates that PA may lengthen life. This may be due to physiological changes (outlined below) or due to greater adherence to treatments and/or positive behavioral changes,

but these mediators have not been thoroughly evaluated. To date, there is little and mixed evidence regarding the effects of PA in late stage disease (e.g., stage IV breast cancer, end-stage renal disease). There are several possible reasons for this: high PA during the end stages of life may indicate unusual or inappropriate coping and possible underreporting of important symptoms. It is also likely that the small physiological changes tied to PA may be too weak to alter the course of disease in its late stages.

How Could Positive Affect Improve Health?

Pressman and Cohen (2005) proposed two pathways by which PA might benefit health and prevent disease. The first is the main effect hypothesis, which contends that PA influences health via its positive influences on health practices, physiological functioning (e.g., immune, cardiovascular, endocrine), and social relationships (also known to have health benefits). The second theory indicates that PA may be tied to better health via its beneficial impact on the stress response. Specifically, it may ameliorate the negative impact of stress by altering perceptions of severity, reducing detrimental physiological responses, and by helping individuals build resources (e.g., physical health, social support) to aid in coping and stress recovery. It is likely that both pathways play some role, although to date no one has directly contrasted the pathways in a single study. There is, however, growing evidence for both types of connections.

Critiques and Future Directions

Future research needs to distinguish what *types* of positive factors are most important to health outcomes and when. To date, most studies focus on "happiness"; however, there is equally good evidence for multi-adjective scales in addition to related positive constructs (e.g., optimism, life satisfaction). It is also important for researchers to better understand at what intensity and frequency positive emotions must be felt to show real physiological benefits and to what extent changes are independent from negative feelings. There is also a need to better understand the mediators of the PA health association and to have studies that prospectively test both health and physiological pathways together. Finally, it is an exciting notion to consider the possibility that PA-inducing interventions might improve health in a meaningful fashion, but it is too soon to determine whether or not these types of studies have robust effects.

General Conclusions

Happiness and other positive emotions have been linked to a greater lifespan, reduced disease susceptibility, improved health perceptions, and better outcomes for those with early-stage diseases. Meanwhile, researchers continue to explore the extent to which positive emotion can be beneficial, when it is most important in disease prevention and treatment, and finally the mechanisms by which it is most helpful.

Cross-References

- ▶ Affect
- ▶ Affect Arousal
- ▶ Emotions: Positive and Negative
- ▶ Health Psychology
- ▶ Mood
- ▶ Optimism, Pessimism, and Health
- ▶ Positive Affect Negative Affect Scale (PANAS)
- ▶ Positive Affectivity
- ▶ Positive Psychology
- ▶ Well-Being

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Hardiness

▶ Locus of Control
▶ Resilience
▶ Resilience: Measurement
▶ Salutogenesis
▶ Williams LifeSkills Program

Hardiness and Health

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Synonyms

Personality hardiness

Definition

Hardiness is a personality construct composed of three traits – control, commitment, and challenge – that are theorized to make one resilient in the face of stress. Individuals high in hardiness tend to believe and act as if life experiences are controllable (control), to engage meaningfully in life activities and to appraise these activities as purposeful and worthy of investment even in the face of adversity (commitment), and to view change in life as a challenge toward growth and development rather than as a threat to security (challenge). Based on existential personality theory, the combination of these characteristics is believed to provide individuals with the courage and motivation to cope adaptively with life stress, thereby buffering its adverse effects on health.

Description

Hardiness has historical significance because it played a significant role in the re-emergence of research examining the relationship between personality and health, and it foreshadowed the current positive psychology movement that focuses on transformation, growth, and resilience in the face of adversity (e.g., optimism, benefit finding, posttraumatic growth). Hardiness was developed by Maddi and Kobasa (Kobasa, 1979; Kobasa, Maddi, & Kahn, 1982) out of a longitudinal study of executives at Illinois Bell Telephone who were facing work upheaval due to deregulation. Executives were studied before, during, and after deregulation to identify characteristics of those who remained healthy and thrived in this time of heightened life stress versus those who showed signs of strain. Individuals who displayed little strain differed from their high strain counterparts on the characteristics of control, commitment, and challenge.

Associations Between Hardiness and Health

Evidence has accumulated across the decades to suggest that hardiness is associated with lower levels of physical and psychological strain following exposure to stress. Higher hardiness has been associated with lower reports of physical symptoms and psychological distress in both cross-sectional and longitudinal analyses. Such associations have been found across samples experiencing diverse stressors including

school-related stress in undergraduates, work-related stress among business executives, bus drivers and lawyers, and military personnel undergoing stressful military procedures. The characteristics of hardiness have also been consistently associated with better performance under stress as revealed in higher GPAs, athletic performance, and leadership skills.

Research has also examined the biobehavioral mechanisms by which hardiness may attenuate adverse responses to stress. There is compelling evidence that characteristics of hardiness facilitate adaptive cognitive appraisals in the face of stress. For example, high hardy individuals make more positive appraisals when experiencing laboratory-induced threat, and appraise the same life stressors as less threatening and more controllable than do low hardy individuals. Hardiness is also associated with more adaptive coping characterized by higher problem-focused and support-seeking coping, better health behaviors, and lower avoidance coping. Consistent with hardiness theory, these more positive perceptions of stress and active versus passive coping strategies have been found to mediate associations between hardiness and health.

Controversies Regarding Hardiness and Health Associations

Despite such encouraging findings, numerous criticisms of this literature have led some researchers to question the evidence supporting an association between hardiness and health. Concerns have centered on: (a) problems with the measurement of hardiness, (b) problems with the measurement of health outcomes, and (c) inconsistent evidence that hardiness "buffers" the adverse effects of stress.

Measurement of hardiness. Progress in the field has been hampered by a number of problems with the measurement of hardiness. First, the measure of hardiness has gone through multiple iterations and no standard measure of hardiness exists. The use of multiple measures makes it difficult to evaluate findings across studies. Second, the existing measures have not consistently supported the three-factor structure theorized to underlie the hardiness construct, raising questions about whether hardiness should be examined as a single composite variable. Research that has examined the three constructs individually suggests that control and commitment are more consistently associated with lower strain than is challenge. No study has provided compelling evidence that all three components are necessary to promote adaptive responses to stress. Third, the items on the initial hardiness scales were negatively keyed, raising questions about whether the scale was measuring the absence of maladaptive traits (e.g., neuroticism) rather than the presence of adaptive traits. Construct validity studies have demonstrated that hardiness scores are strongly correlated with neuroticism, and that some associations between hardiness and lower strain are reduced or eliminated when shared variance with neuroticism is statistically controlled. The most recent version of the hardiness scale – Personal Views Survey III-Revised (PVS III-R) – appears to have partially addressed these issues. However, the psychometric properties of this scale have not been published in a peer-reviewed journal.

Measurement of physical health outcomes. Another concern with the hardiness and health literature is that health outcomes are commonly measured with self-reported somatic complaints or other subjective signs of strain, rather than with objective signs of illness. Such outcomes are imperfect measures of health; they are heavily influenced by illness cognition and illness behavior processes that occur with heightened distress. The use of such health measures combined with the overlap between measures of hardiness and neuroticism have raised concerns that hardiness-health associations reflect shared variance with neuroticism. Few published studies have examined associations between hardiness and more objective signs of physical health (e.g., psychophysiological reactivity to stress; blood pressure; immune function; mortality), and those that exist have yielded inconsistent findings.

Evidence of stress buffering. Although developed in the context of work-related stress, the question of whether hardiness "buffers" the adverse health effects of stress is not fully answered. If hardiness works by buffering stress,

its associations with health outcomes should be stronger under high versus low stress conditions, as evidenced by a statistical interaction between hardiness and stress. Many studies have not been designed to test this stress-buffering hypothesis (e.g., hardiness is often tested among samples exposed only to high levels of stress), and those that have provide inconsistent support for stress buffering. Nevertheless, the consistent finding of adaptive perceptions of stress noted above suggests that hardiness may reduce one's level of psychological stress even in the face of objectively similar stressful life events.

Conclusion

The construct of hardiness continues to be studied in a variety of settings around the world, and interventions to increase levels of hardiness have been developed. This recent wave of hardiness research has focused on psychological strain and performance-based outcomes more than on physical health outcomes, but may provide answers to some of these ongoing controversies.

Cross-References

- ▶ Benefit Finding
- ▶ Biobehavioral Mechanisms
- ▶ Construct Validity
- ▶ Coping
- ▶ Coping Styles
- ▶ Health Behaviors
- ▶ Individual Differences
- ▶ Life Events
- ▶ Mediators
- ▶ Neuroticism
- ▶ Optimism, Pessimism, and Health
- ▶ Passive Coping Strategies
- ▶ Perceived Control
- ▶ Perceptions of Stress
- ▶ Personality
- ▶ Positive Psychology
- ▶ Posttraumatic Growth
- ▶ Problem-Focused Coping
- ▶ Psychological Stress
- ▶ Psychometric Properties
- ▶ Resilience
- ▶ Self-Report
- ▶ Social Support
- ▶ Somatic Symptoms
- ▶ Stress
- ▶ Stress: Appraisal and Coping
- ▶ Stressor
- ▶ Symptoms
- ▶ Work-Related Stress

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Harm Minimization

- ▶ Harm Reduction

Harm Reduction

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Synonyms

Harm minimization; Risk reduction

Definition

Harm reduction is a public health framework that refers to policies, programs, and practices that focus on reducing potentially adverse health, social, and economic consequences related to engagement in high-risk behaviors. Harm reduction has been controversial as it focuses on preventing or reducing harm and not necessarily on preventing or eliminating risky behavior. Although it has been used in many different settings, harm reduction is most often associated with issues related to substance use and became a more prominent framework in the mid-1980s as a public health response to the HIV epidemic among injection drug users.

In the field of substance abuse, harm reduction provides an alternative to abstinence. The harm reduction framework recognizes that there are many people who are unable or unwilling to stop using illicit drugs. Subsequently, it focuses on reducing the societal and individual harms that may occur as a result of drug use. Needle exchange programs are an example of a harm reduction approach to HIV among injection drug users. These programs focus on providing clean syringes so that individuals who continue to inject drugs do not get infected with HIV. Through a nonjudgmental approach, education, and offering clean equipment, the programs strive to prevent adverse health outcomes (e.g., HIV infection) among injection drug users. These programs do not encourage or promote drug use but instead offer realistic options to individuals who are unable to quit their drug use. This framework has been proven to be effective in reversing and preventing the HIV epidemic among injection drug users (Des Jarlais, 2010).

The harm reduction framework encompasses multiple levels as policy, environments, and individual behaviors can all be targeted and modified to reduce harm. The framework acknowledges that risky behaviors occur along a continuum ranging from minimal risk to excessive risk. The goal is to identify feasible and realistic options along this continuum that can be adopted to reduce risk. Instead of seeking to criminalize or moralize behavior, harm reduction seeks to meet individuals in their current situation and identify ways to reduce the harmful outcomes to society and the individual that may be a result of engaging in risky behavior. According to a recent editorial in the International Journal of Drug Policy, harm reduction started as a public health strategy informed by social justice and over time has increasingly drawn attention to structural issues and the need to reform policy so that disenfranchised populations can avoid harm (Stimson & O'Hare, 2010).

Cross-References

▶ HIV Infection

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Harmful Drinking

▶ Binge Drinking

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Biographical Information

Laura L. Hayman earned her BSN, MSN, and PhD at the University of Pennsylvania. Her program of research and scholarship focuses on primary prevention of obesity and cardiovascular disease (CVD) in children, adolescents, and families. Her research, in collaboration with colleagues from several disciplines, has included clinical, school, and population-based studies of biobehavioral risk factors for CVD. Her recent work combines both individual/clinical and community-based approaches to identifying children at risk for obesity and cardiometabolic conditions, and theory-based interventions designed to increase physical activity and promote healthy lifestyle behaviors.

Hayman has served on numerous national and international interdisciplinary advisory and expert panels relevant to primary prevention of obesity and CVD in childhood and adolescence. She has also served in leadership roles in the American Heart Association, the Society of Behavioral Medicine, and the Preventive Cardiovascular Nurses Association. She holds fellowships in the American Heart Association, the American Academy of Nursing, the Society of Behavioral Medicine, and the Academy of Behavioral Medicine Research.

Major Accomplishments

Christian R. & Mary F. Lindback Award for Distinguished Teaching, University of Pennsylvania, 1983

Katharine A. Lembright Award for Achievement in Cardiovascular Nursing Research, American Heart Association, 1997

Fellow, American Heart Association, Council on Cardiovascular Disease in the Young, and Council on Nutrition, Physical Activity and Metabolism, 2003

Member, Academy of Behavioral Medicine Research, 2006

C. Tracy Orleans Distinguished Service Award, Society of Behavioral Medicine, 2007

Distinguished Achievement Award, Council on Cardiovascular Nursing, American Heart Association, 2009

Spirit of Nursing Award, College of Nursing and Health Sciences, University of Massachusetts, Boston, 2010

National Meritorious Achievement Award, American Heart Association, 2010

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HbA1c

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Synonyms

A1C; Glycated hemoglobin; Glycosylated hemoglobin; Hemoglobin A1c

Definition

HbA1c, or glycosylated hemoglobin, is a measure of how much glucose is irreversibly bound (glycated) to hemoglobin, and can be used to assess the degree of exposure to glycemia in the preceding 2–3 months (corresponding to the life span of the red blood cell where hemoglobin is contained). In a person with normal blood glucose levels, the amount of glycated hemoglobin is around 4–6%, representing an average blood glucose level between 70 and 120 mg/dl. In individuals with diabetes, HbA1c can be measured

every 3 months with a goal of keeping the value as close to normal as possible, or at least under 7% in most patients. The higher the HbA1c, the greater the risk over time (usually measured in years) of developing microvascular complications, such as diabetic retinopathy, nephropathy, and neuropathy. HbA1c remains the best predictor for future diabetes-related chronic complications that is available in the clinical setting.

Cross-References

- ▶ Diabetes
- ▶ Hyperglycemia

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Head Injury

▶ Traumatic Brain Injury

Headache with Aura

▶ Migraine Headache

Headaches, Types of: Cluster, Migraine, and Tension

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Definition

Cluster headache, migraine, and tension-type headache are three major types of primary headaches. Primary headaches are headaches with no apparent underlying organic disease process.

Description

The International Classification of Headache Disorders, 2nd edition (Headache Classification Subcommittee of the International Headache Society, 2004), is a widely used classification of headaches, and it contains diagnostic criteria for headaches. Cluster headache, migraine, and tension-type headache are classified as primary headaches, and their diagnosis is based on the pain characteristics and associated symptoms. Generally, neuroimaging is not necessary for the diagnosis; however, it is considered to exclude underlying abnormalities of the brain in some cases. Assessment of psychosocial aspects is also important especially in migraine and tension-type headache because psychosocial factors can be precipitating and aggravating factors of headache and headache may affect psychosocial condition.

In these headaches, treatment consists of acute therapy and prophylactic therapy. Prophylactic therapy is important because frequent use of analgesics places patients at risk for medication overuse headache.

Cluster Headache

Cluster headache is a headache which is severe, strictly unilateral, and orbital, supraorbital, or temporal in location. Attacks usually occur in series for a period of weeks or months which is called a cluster period. Cluster periods are separated by remission periods which are usually months to years. The attack lasts 15–180 min, and its frequency ranges from once per 2 days to eight times a day. The attack accompanies ipsilateral autonomic symptoms such as ptosis, miosis, lacrimation, conjunctival injection, rhinorrhoea, nasal congestion, and forehead and facial sweating. Pain is severe enough to disturb daily activities, and most patients are restless or agitated during attack.

The prevalence of cluster headache is less than 1%, and prevalence is three to four times higher in men than in women (May, 2005).

Pathophysiological involvement of hypothalamus is suggested by time pattern of attacks. Neurovascular factors are also important.

Acute therapy for cluster headache includes inhalation of pure oxygen and triptan. Subcutaneous injection and nasal spray are preferable to oral administration. As preventive therapy for cluster headache, verapamil is established. Lithium, methysergide, and corticosteroids are also used. Non-pharmacological treatment is generally ineffective.

Migraine

Migraine is further classified into two major subtypes: migraine without aura and migraine with aura.

Migraine without aura is recurrent headache disorder whose pain is generally unilateral, pulsating (throbbing), moderate to severe in intensity, aggravated by daily physical activities, and accompanied by nausea and/or photophobia and phonophobia. The attack lasts 4–72 h, and its median frequency is 1.5 per month. Migraine with aura is characterized by a complex of reversible focal neurological signs (visual, sensory, motor, or speech signs) which gradually progresses in 5–20 min and last for less than 60 min generally before headache. Typically, headache of the same quality as migraine without aura follows aura, but sometimes, the quality of headache is different and even headache can be absent. Symptoms such as fatigue, difficulty in concentrating, neck stiffness, sensitivity to light or sound, nausea, blurred vision, yawning, pallor, or emotional lability sometimes occur several hours to 2 days prior to migraine (either with or without aura), and they are called premonitory symptoms. Migraine may be aggravated (i.e., increased in the severity or frequency in a relatively long term) by psychosocial stress, frequent intake of alcohol, and other environmental factors. An attack may be triggered by menstruation, chocolate, etc.

Prevalence has been reported to be between 5% and 25% in women and 2% and 10% in men.

Trigeminovascular theory is now a broadly accepted pathophysiological mechanism of migraine (Silberstein, 2004). Perivascular trigeminal terminals are stimulated by certain causes and vasoactive substances such as calcitonin gene-related peptide (CGRP) are released. Vessels dilate and neurogenic inflammation occurs, which leads to pain and accompanying symptom such as nausea. Central pain modulation is also thought to be involved. Cortical spreading depression is associated with aura.

Acute therapy for migraine consists of specific (triptans and ergots) and nonspecific (analgesics) (Silberstein, 2004). Triptans are $5HT_{1B/1D}$ receptor agonists and have effects of vasoconstriction and inhibition of vasoactive substances release and of neurogenic inflammation. Prophylactic therapy includes calcium channel blocker, beta-blocker, ergots, antidepressants, and anticonvulsants (Silberstein, 2004). Refraining from drinking alcohol and eating certain foods (chocolate, cheese, etc.) may also be effective for prevention of migraine attacks. Relaxation therapy, thermal and electromyography biofeedback, and cognitive behavior therapy are also used as prophylactic therapy.

Tension-Type Headache

Tension-type headache typically causes pain which is bilateral, pressing or tightening, and mild to moderate in intensity, and is not aggravated by daily physical activities. Although anorexia may accompany, neither nausea nor vomiting does. Photophobia and phonophobia can coexist.

Tension-type headache is the most common type of primary headache, and its life prevalence estimates range from 30% to 78%, and tension-type headache is slightly more in women than in men (Headache Classification Subcommittee of the International Headache Society, 2004). Its prevalence is most at 40s (Loder & Rizzoli, 2008).

Increased muscle tension was formerly thought to be a major cause of tension-type headache; however, now it is thought that peripheral factor (hypersensitivity to pain in the head and neck tissue) plays a major role in less frequent headache (i.e., infrequent and frequent episodic tension-type headache), while central factor

(alteration of pain sensitivity in the central nervous system) plays a major role in more frequent headache (chronic tension-type headache).

Acute therapy for tension-type headache is analgesics (nonsteroidal anti-inflammatory drugs (NSAIDs) and acetaminophen). Over-the-counter analgesics are commonly used. As pharmacological prophylactic therapy, amitriptyline is the most widely researched (Millea & Brodie, 2002). With less evidence, other antidepressants such as selective serotonin reuptake inhibitors (SSRIs) and tizanidine are considered as prophylactic therapy. Relaxation therapy, electromyography biofeedback, and cognitive behavior therapy are also used for prophylaxis.

Cross-References

▶ Headaches: Psychological Management

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Headaches: Psychological Management

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Definition

Psychological management of headache includes assessment of psychosocial aspects of headache, screening and treating psychiatric comorbidity, and application of psychological treatments to manage pain.

Description

Psychological management of headache has been researched mostly in tension-type headache and migraine.

When diagnosing headache, assessment of psychosocial aspects is also important because psychosocial factors can be precipitating and aggravating factors of headache and headache may affect psychosocial condition. In both migraine and tension-type headache, 50–80% of patients report that psychological stress is a precipitating or aggravating factor of headache according to some reports. Identifying individual precipitating or aggravating factors is fundamental for the prophylaxis of headache. In both migraine and tension-type headache, anxiety and depressive mood are reported to be higher than healthy controls and social activities are also affected. Overall assessments of those psychosocial conditions are necessary to understand the burden of headaches.

Comorbidity with mood disorder and anxiety disorder is also reported to be high in both migraine and tension-type headache (Holroyd, 2002). In addition, psychiatric comorbidity is reported to be a possible risk factor for chronification of headache. Therefore, screening and treating psychiatric comorbidity is also necessary for headache management.

Representative psychological treatments of headache are relaxation therapy, biofeedback therapy, and cognitive behavioral therapy (Holroyd, 2002).

In tension-type headache, relaxation therapy in the form of progressive muscle relaxation and autogenic training, electromyographic biofeedback therapy (reducing muscle activity in forehead or neck and shoulder muscles), and cognitive behavioral therapy are used. Although relaxation therapy alone is suggested to be effective, it is reported that the percentage of patients

whose headache was improved was increased when biofeedback therapy was added. Cognitive behavioral therapy is also conducted in combination with other therapies as well as alone. Cognitive behavior therapy increases the effectiveness of relaxation therapy when it is added to relaxation therapy.

In migraine, relaxation therapy in the form of progressive muscle relaxation and autogenic training, thermal biofeedback therapy (warming hand), and cognitive behavioral therapy are generally thought to be treatment options for prevention of migraine as psychological treatment. However, it is reported that cognitive behavioral therapy did not appear to enhance the effectiveness of relaxation therapy or thermal biofeedback.

In cognitive behavioral therapy, it is assumed that irrational cognition and maladaptive behavior underlie pain, psychological stress, and mood disturbances and those cognition and behavior are the targets of intervention. Usually, treatment program comprising several components is conducted and relaxation therapy is often included. Treatment aims at achieving adoptive coping behavior to pain as well as approaching psychosocial factors. Psychological treatments have a feature that they all aim at self-control in common. There is not any recommendation about which of these psychological treatment to choose for specific patients.

Psychological treatment is usually used either with or without medication. In chronic tension-type headache, the combination of cognitive behavioral therapy and tricyclic antidepressants are reported to possibly improve outcome relative to monotherapy.

The mechanism of how these psychological treatments improve headache is still unclear. Previous studies that showed the effect of psychological treatment on headache was not limited to patients with psychiatric comorbidity, and it is not likely that the improvement of comorbid psychiatric disorders fully mediates the improvement of headache.

Cross-References

▶ Headaches, Types of: Cluster, Migraine, and Tension

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Health

▶ Well-Being: Physical, Psychological, Social

Health Anxiety

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Synonyms

Health phobia; Hypochondriasis

Definition

Health anxiety refers to an excessive concern or preoccupation about being ill based on the misinterpretation of somatic symptoms despite medical reassurance indicating otherwise.

Description

Health anxiety refers to an excessive concern or preoccupation about the meaning and potential

consequences of somatic symptoms. Individuals high in health anxiety are more likely to believe that their physical symptoms are signs of a serious disease than their low anxious counterparts despite medical reassurance indicating otherwise.

Several environmental, biological, and psychological (e.g., behavioral and cognitive) factors have been implicated as causes of health anxiety (Abramowitz & Braddock, 2008; Kirmayer & Looper, 2006). Evidence from research examining the environmental antecedents of health anxiety indicate that individuals, especially females, who have experienced serious illness as children, suffered the death of a loved one from a devastating medical condition, or were victims of physical, sexual, and/or emotional abuse in childhood are at higher risk of developing health anxiety when compared to individuals without a history of traumatic experiences (Stein et al., 2004). Another environmental risk factor is parental modeling of illness behaviors via observational learning (Mineka & Ben Hamida, 1998). For instance, children can learn from overprotective parents that any somatic symptom is a sign of a serious disease which can result in the regular use of emergency and/or primary care clinics to care for nonthreatening symptoms. Finally, informational transmission (e.g., media and Internet) can serve as a trigger for health anxiety and health anxious-related behaviors (Abramowitz & Braddock, 2008; Rachman, 1991). Examples of this include mass psychogenic illness, mass anxiety hysteria, "Koro" in Southeast Asia, and medical student's syndrome.

Family and twin studies suggest that heritability may play a role in the development of health anxiety and hypochondriasis; however, evidence about the specific genes involved is inconclusive (Smoller, Gardner-Schuster, & Covino, 2008). Research on the neurobiological basis of health anxiety suggests that hyperactivity in the amygdala and limbic regions and an inability of higher cortical executive areas to inhibit limbic responses may be involved in initiating and maintaining anxiety episodes (Martin, Ressler, Binder, & Nemeroff, 2010). Additionally, neuropharmacological studies have shown that either decreased inhibitory control utilizing GABA, or an increase of the excitatory neurotransmitter Glutamate, or a synergistic combination of both systems play key roles in the development of health anxiety. It must be noted, however, that these neurobiological antecedents are not unique to health anxiety but rather shared by various anxiety disorders.

The identification and understanding of psychological antecedents of health anxiety have received wide attention from researchers (Marcus, Gurley, Marchi, & Bauer, 2007). Individuals high in health anxiety have been found to: (1) have catastrophic beliefs about their somatic symptoms, (2) be more sensitive to and aware of their somatic symptoms (i.e., somatosensory amplification), and (3) be more likely to interpret their somatic symptoms as signs of a serious disease. Health-anxiety beliefs can be elicited by various triggers such as benign physical symptoms, nonthreatening disease, and hearing or reading about illnesses from different sources (e.g., friends, media). Once triggered, these beliefs are responsible for maintaining a feedback loop that results in automatic hypochondriacal thoughts which, in turn, increase attention to somatic sensations that may confirm illness (i.e., confirmatory bias). Increased somatic vigilance compounded with a confirmatory bias help perpetuate health anxiety.

Health anxiety is best understood as a continuum ranging from mild to severe. Although many people have benign health anxiety, it can become extreme and result in hypochondriasis, a somatoform disorder (Faravelli et al., 1997; Looper & Kirmayer, 2001). The prevalence of hypochondriasis in the general population (0.2%) is low, while nonclinical levels of health anxiety (6%) are more common (Looper & Kirmayer, 2001). To diagnose an individual with hypochondriasis, he or she must believe that his/her somatic symptoms result from a serious disease despite medical reassurance. These symptoms must last a minimum of 6 months and cause significant

distress or interfere with social, occupational, or other forms of functioning (American Psychiatric Association [APA], 2000). Hypochondriasis is typically treated with a combination of cognitive behavioral therapy and psychotropic medication (e.g., SSRI, Pimozide, and Clomipramine).

Health anxiety can be assessed with different tools including self-reports and structured clinical interviews. The most widely used self-reports include the Short Health Anxiety Inventory (Salkovskis, Rimes, Warwick, & Clark, 2002), the Illness Behavior Questionnaire (Pilowsky & Spence, 1994), and the Health Anxiety Questionnaire (Lucock & Morley, 1996). Structured clinical interviews include the Composite International Diagnostic Interview (World Health Organization [WHO], 1990) and Structured Clinical Interview for DSM disorders (First, Spitzer, Gibbon, & Williams, 1995). Clinical interviews should be used when the goal of assessment is the diagnosis of clinical levels of health anxiety.

Cross-References

- ▶ Anxiety
- ▶ Anxiety and Heart Disease
- ▶ Hypochondriasis
- ▶ Neuroticism
- ▶ Pain Anxiety

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Health Assessment

► Physical Examination

Health Assessment Questionnaire

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Synonyms

Activities of daily life assessment; Disability assessment; Self-reported patient outcome measure

Definition

The Health Assessment Questionnaire (HAQ) was developed three decades ago by James F. Fries, MD, and colleagues at Stanford University (Fries, Spitz, Kraines, & Holman, 1980) as a model of patient-reported outcome (PRO) assessment for assessing physical function. Three reviews examined the HAQ's history, its reliability, validity, and applicability (Bruce and Fries, 2003; Ramey, Fries, & Singh, 1995; Ramey, Raynauld, & Fries, 1992).

The HAQ has been administered globally and validated in patients with a wide variety of rheumatic diseases, HIV/AIDS, and in studies of normal aging, in diverse disciplines and different cultures, and in dozens of languages without impacting reliability or validity with properly designed adaptations. The HAQ is usually self-administered. However, it can be administered face-to-face or over telephone by a trained interviewer. Further, the HAQ has been validated for Internet administration (Bruce, Fries, & Lingala, 2011). The HAQ is available online (The Arthritis, Rheumatism, and Aging Medical Information System, 2011).

The original HAQ was developed using classical test theory methodology, is sensitive to change, and a good predictor of future disability and costs. However, it did not benefit from use of modern psychometric approaches. Modern methods, such as Item Response Theory (IRT) (Embertson & Reise, 2000), which quantitatively assess item properties, enable development of more precise instruments (Rose, Bjorner, Becker, & Fries, 2008).

Recently, items in the HAQ, along with the SF-36's PF-10, have undergone extensive revamping using both classical and IRT methods as part of the Patient-Reported Outcomes Measurement Information System (PROMIS) (Reeve et al., 2007). PROMIS is part of the National Institutes of Health (NIH) Roadmap Initiative aimed at re-engineering the clinical research enterprise. Work in PROMIS resulted in a 20-item revised HAQ and the IRT-derived PROMIS PF-20, both of which more precisely measure physical function and are available for use on the PROMIS website (http://www.nihroadmap.nih.gov) (US Department of Health and Human Services, 2011). Investigation of the psychometric functions showed that instruments utilizing these items are more patient-centered, more validly translatable, and have better clarity in diversely educated groups. In addition, they also show responsiveness and precision that is

better than the parent instruments, the original HAQ and PF-10 (Fries, Krishnan, Rose, Lingala, & Bruce 2011).

Cross-References

- ▶ Health Economics
- ▶ SF-36

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Health Behavior Change

- ▶ Behavior Change
- ▶ Behavior Change Techniques
- ▶ Behavioral Medicine
- ▶ Health Behaviors
- ▶ Health Communication
- ▶ Health Education
- ▶ Health Promotion and Disease Prevention
- ▶ Lifestyle, Modification
- ▶ Population Health

Health Behavior Predictors

- ▶ Psychosocial Predictors

Health Behavior Variables

- ▶ Psychosocial Variables

Health Behaviors

- ▶ Aerobic Exercise
- ▶ Alcohol Consumption
- ▶ Alcohol Abuse and Dependence
- ▶ Healthy Eating
- ▶ Illness Behavior
- ▶ Lifestyle
- ▶ Lifestyle, Healthy
- ▶ Meditation
- ▶ Medication Compliance
- ▶ Relaxation: Techniques/Therapy
- ▶ Tobacco Cessation
- ▶ Tobacco Use

Health Beliefs

▶ Beliefs
▶ Illness Cognitions and Perceptions

Health Beliefs/Health Belief Model

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Definition

Rosenstock's Health Belief Model (HBM) is a theoretical model concerned with health decision-making. The model attempts to explain the conditions under which a person will engage in individual health behaviors such as preventative screenings or seeking treatment for a health condition (Rosenstock, 1966).

Description

Under the HBM, a person's likelihood for health behavior is assumed to be related to four main variables. First, action is more likely if the person perceives himself to be *susceptible* to or at risk for the condition. For example, if Lucy has a history of breast cancer in her family, she may see herself as more susceptible to developing breast cancer, and thus, be more likely to get a mammogram each year. Second, the likelihood for action depends on the *perceived seriousness* of the condition. Seriousness may be judged based on the amount of emotional arousal produced by thinking about the condition as well as the anticipated physical, social, and psychological consequences of developing the condition. For example, Lucy's mother passed away from breast cancer so she deems it to be a serious condition requiring preventative action. Third, the *perceived benefits* of performing the action are considered. Lucy considers the effectiveness of a mammogram in detecting breast cancer when determining whether to get the screening. Finally, the *perceived barriers* of performing the action are weighed. Lucy knows that a mammogram can be uncomfortable and scheduling an appointment is inconvenient. However, for Lucy, the benefits outweigh the barriers. Additional modifying variables like age and sex have been introduced with the assumption that they influence the above beliefs.

The variables of HBM are intended to measure a person's psychological readiness or intentions to act (Kirscht, 1988), and on the whole, research has found the HBM to be predictive of people's individual health behaviors (Janz & Becker, 1984). Self-reported susceptibility, benefits, barriers, and severity were shown to be correlated with health behavior outcomes such as attending preventative screening, seeking medical care, and utilizing health clinics. However, the HBM has been unable to consistently predict adherence to a medical treatment regimen or terminating an unhealthy behavior such as smoking (Kirscht, 1988). Additionally, while the variables of HBM may measure a person's individual level of readiness, the optimal level of readiness for health behavior change is still unknown.

Further critiques state that HBM ignores self-efficacy (a person's belief that he has control over a particular behavior) which has been shown to play a large role in behavior change (Kirscht, 1988). Thus, while the predictive validity of HBM with regard to health behaviors seems firm, the usefulness of focusing only on HBM factors in interventions has been contested (Davidhizar, 1983; Kirscht, 1988).

Cross-References

▶ Adherence
▶ Health Behaviors
▶ Self-efficacy

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Health Care

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Definition

Health care is a general term comprising services provided to improve health in the general population as well as to cure diseases and relieve symptoms in diseased patients. Health care may denote the organization of services (e.g., private vs public health care), a facility (e.g., hospital or health care center), as well as the actual delivery of care (e.g., to provide health care or to obtain health care). The term may comprise preventive services, such as vaccination, and mother and child care as well as curative services.

Health Care Access

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Definition

Health care access is the extent to which patients and groups have access to health care. It depends on factors related to general living conditions in society, and to organization of health services as well as factors in individuals. General conditions comprise availability overall of economic resources and how they are distributed, manpower for health care, and availability of health care facilities and transport systems to reach them. Organization of health services comprises how they are organized in primary and secondary care, adequate staff at various levels, geographical distribution, and insurance system to cover costs. Factors in individuals and groups are related to their knowledge about health care services, capacity to pay for fees and transport, and knowledge of the language and other cultural codes needed to access health care.

Health Care Costs

▶ Health Economics

Health Care System

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Definition

The concept “health system” has been developed and defined by the World Health Organization (WHO), and is now widely used internationally by scientists and policy makers. The WHO defines health system as “all organizations, people, and actions whose primary intent is to promote, restore, or maintain health.” The concept thus includes not only public and private health care facilities and staff, but also health insurance organization, water control, occupational health, and safety legislation. The WHO has described a health system framework consisting of six building blocks: Service delivery; Health workforce; Information; Medical workforce, vaccines, and technologies, Financing, and Leadership/Governance.

Cross-References

▶ Health Insurance: Comparisons

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Health Care Utilization

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Definition

Health Care Utilization is the quantification or description of the use of services by persons for the purpose of preventing and curing health problems, promoting maintenance of health and well-being, or obtaining information about one's health status and prognosis.

Description

Health Care Utilization refers to the use of health care services. People use health care for many reasons including preventing and curing health problems, promoting maintenance of health and well-being, or obtaining information about their health status and prognosis.

Utilization is often reported in a variety of different methods:

1. The number of services used over a period of time divided by a population denominator (e.g., in 2008, there were 320.1 ambulatory Care Visits to Physicians' Offices per 100 persons living in the USA).
2. The percentage of persons who use a certain service over individuals eligible for that service in a period of time (in the USA in 2008, 75% of all women aged 18 years and over reported having a Pap smear in the last 3 years).
3. An aggregate number without a denominator (in 2008, there were 39.9 million discharges from US hospitals).

Health care utilization can vary by many factors. Sociodemographically persons at extremes of age (very old or very young) have higher health care utilization. Women also have higher utilization than men, partially explained by need for obstetric and gynecologic care. By race and ethnicity, members of minority groups have lower utilization of certain health care services. For example, despite a higher burden of cardiovascular disease, after adjusting for factors that predict utilization, African-Americans are less likely to receive invasive cardiac procedures. Latinos are less likely to have colorectal cancer screening tests. By health status, persons with poorer health have higher utilization.

However, of all the factors that drive utilization, perceived need for health care by the patient is probably the single most important independent factor. In addition, since many people may not fully know how medical conditions are prevented, diagnosed, or treated, perceived need also includes the perceptions on what health care is needed for a particular person by their providers and others who make health care recommendations to patients. In addition to need, there are many other factors that also impact utilization. Often these are conceptualized as predisposing, enabling, and need related factors. Examples of predisposing factors include a person's propensity to seek care as well as cultural norms on health care seeking behaviors. Ability to pay or health care coverage is the most important enabling factor. However, other important enablers include accessibility and location of services, language and cultural barriers, and availability of resources to appropriately provide such services.

Data on utilization can be gathered and compiled from various sources. One is administrative or claims data collected from those delivering

health care services or serving as payers of those health care services (such as insurers). An example is data on the number of cardiac catheterizations performed among Medicare beneficiaries which can be examined from the Medicare Provider Analysis and Review (MEDPAR) files. Data can also be collected from providers using surveys. An example of this type of utilization data is the CDC's National Ambulatory Medical Care Survey in which a representative sample of office-based providers are queried to provide data on health care services delivered over a 1-week period. Another example is the Nationwide Inpatient Sample containing discharge abstracts from a 20% stratified sample of US community hospitals (part of AHRQ's Healthcare Cost and Utilization Project). A limitation of these methods is that collecting and compiling accurate such data through these approaches can be resource intensive, particularly in countries with multi-payer and delivery systems. Also, it will not capture services delivered outside the health care sector being sampled. An example is data on alternative medicine.

Another approach used in many countries to collect information on healthcare utilization is through population-level surveys using self-reported data from patients themselves. In the USA, examples are the CDC's National Health Interview Survey and AHRQ's Medical Expenditure Panel Survey. Since these are based on patient self-reports, accuracy is always a concern. Thus, careful attention needs to be paid in design, collection, and analysis of data from such surveys so that the data presented is valid and accurate.

Utilization data is used for a variety of purposes. Cross-sectional data can be used to compare services received across different settings, to relate provider characteristics to patient utilization, to compare utilization rates among subpopulations, and to assess how the health care delivery system is being used and by whom. It can provide interested parties with information to help determine if utilization is appropriate or inappropriate, high or low quality, and expensive or inexpensive, and highlight areas that may warrant in-depth examination. For example, data on a higher than expected rate on cesarean sections or less use of cancer screening tests by certain population subgroups may highlight areas in need of attention. Longitudinally, health care utilization data is also used to monitor changes in the use of health care resources and to forecast future health care expenditures, or as the basis for projecting future healthcare needs such as facilities, personnel, or supplies.

Cross-References

- ▶ Health Care Costs
- ▶ Health Care System
- ▶ Health Economics
- ▶ Medical Utilization

References and Readings

A comprehensive listing of sources of health utilization data for the United States can be found at the Partners in Information Access for the Public Health Workforce at http://phpartners.org/health_stats.html

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One comprehensive source of comparable statistics on health care utilization among industrialized countries is found in the OECD interactive database at www.oecd.org/health/healthdata

Health Communication

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Synonyms

Health education; Health promotion; Social marketing

Definition

In the context of behavioral medicine, health communication is best defined as transmission or exchange of information designed to modify behaviors related to health. This can be envisioned broadly to encompass communication in medical settings (e.g., patient counseling to increase adherence to regimens), in communities and populations (e.g., media and community outreach campaigns to increase condom use), and in the political sphere (e.g., internet messaging platforms designed to spur advocacy to influence policies that affect health).

Description

Modalities include (1) face to face, telephone, and telecommunicated interpersonal communication between individuals and groups; (2) text and graphic messaging in print and electronic form, e.g., newspaper stories, posters, leaflets, websites; (3) audiovisual messaging in mass communication, e.g., television and documentary film; and (4) new media integrating multiple modalities through interactive web-based and mobile applications.

Skills training and guidelines for health communication are widely available online (e.g., U.S. Centers for Disease Control, 2011). Key tasks for effective health communication include assessment of audiences' health literacy, composition of appropriate messages through cultural competency, selection of channels of communication to effectively reach defined audience segments, and creation of message content to influence specified intermediate cognitive or emotional factors to achieve measurable changes in behavior. Audience research is central to effective health communication, as observations, interviews, focus groups, and surveys can be used to prioritize the segmentation of audiences within populations and identify most suitable channels and forms of communication for those audiences, and the knowledge, attitudes, perceptions, feelings, competencies and other factors that will provide the most effective message content for achieving behavior change.

Health communication is an important part of health education and promotion (Glanz, Rimer, & Viswanath, 2008), which also is concerned with changing behavior through both communication and the modification of environmental circumstances to facilitate or incentivize healthy behaviors. However, changing environmental circumstances requires effective media advocacy and related political communication skills. Social marketing (Andreason, 2006), in which techniques and strategies from advertising and sale of consumer products are adopted for noncommercial purposes, has become the dominant professional model for much health communication and been associated with numerous successful behavior change campaigns in both the developed and developing world (Wakefield, Loken, & Hornik, 2010). Alternatives and complements to the marketing model include behavioral journalism (McAlister, 2000) and the use of narrative storytelling (Kreuter et al., 2007), illustrated by reality-based television programs that follow individuals through the process of smoking cessation or weight loss, and edutainment in the form of radio dramas to promote family planning.

The basic theoretical foundation for health communication was articulated by William McGuire (2001) in his classic communication matrix model which considers how channels, sources, and message content influence exposure, attention, comprehension, yielding to persuasion, skill acquisition, trial of new behaviors, and long-term behavior change. This kind of sequential analysis of communication effects on individual behavior change has been elaborated in the transtheoretical model (Prochaska & DiClemente, 2005), which highlights specific processes in particular steps such as emotional arousal in the initial contemplation of change and feelings of self-efficacy and competence in the acquisition and maintenance stages of behavior change. Health communication effects on sequential processes in behavior change on the societal level are described in Rogers' (2003) diffusion of innovation model, which distinguishes between early adopters (who acquire innovations after being exposed to them via

media communication) and later adopters (who are more influenced by peer modeling, interpersonal communication, and conformity pressures).

The integrative model of behavioral prediction (Fishbein & Cappella, 2006) classifies mediating psychological factors that are influenced when communication yields behavior change: (1) modification of belief and expectations regarding behavior outcomes and values, (2) increases in perceived "normative" social pressure and anticipations of social sanctions, and (3) rise in "self-efficacy" expectations regarding personal or collective ability and competence. The latter factor is central to Bandura's (2001) social cognitive theory, which emphasizes a dual link comprised of peer modeling via mass media and social reinforcement in learning to perform healthy behaviors. Social cognitive theory also provides a formulation for self-management training methods widely used in patient education and behavioral counseling. Motivational interviewing (Rollnick, Miller, & Butler, 2007) is another notable theory-based technique for interpersonal communication to change behavior.

Innovation in health communication, while largely based on theoretical foundations noted above, has followed emerging technologies. Computer and web-based interactivity has opened new capacities for communicators to precisely segment differentiated audiences and tailor messages to their observed preferences. Internet and mobile applications for gaming have become notable modalities for health communication (Read & Shortell, 2011). New platforms for interactive social media and mobile messaging are providing opportunities for more compelling communication with patients in behavioral medicine, communities in health promotion, and advocates for policies to strengthen public health (Korda & Itani, 2011).

Cross-References

- ▶ Cultural Competence
- ▶ Health Literacy
- ▶ Motivational Interviewing
- ▶ Social Marketing

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Health Consequences of Smoking

► Smoking and Health

Health Departments

► Health Care
► Health Care Access

Health Disparities

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Synonyms

Health inequalities; Health inequities

Definition

A health disparity is defined as an observed difference in health outcomes (e.g., diabetes) or health status between the most advantaged group in a given category (e.g., the wealthiest) and all other groups in that category. Observed differences in the health outcomes are not only limited to differences between better- and-worse-off groups, given they reflect varying levels of social advantage and disadvantage (Braveman, 2006).

Description

The term "health disparity" is often times used interchangeably with the terms "health inequality" or "health inequity." The use of "health disparity" is most common in the United States, whereas the other terms are most often used outside of the United States (Carter-Pokras & Baquet, 2002). The underlying distinction between these terms is that the latter ones distinguish between health differences that are unfair, unjust, and unavoidable. To illustrate, differences in health among men and women that are due to sex-specific problems (e.g., ovarian cancer) would be attributed to biological variation and therefore unavoidable, whereas health differences due to social or environmental factors (e.g., socioeconomic status, unequal access to resources), for example, would be considered unjust and avoidable (Whitehead, 1992). However, the term "health inequality" requires both an ethical and moral consideration regarding what constitutes a difference as "unavoidable" and "unjust/unfair," and therefore leaves its definition open to interpretation (Braveman, 2006; Carter-Pokras & Baquet, 2002).

Health disparities are typically thought of as referring to racial/ethnic disparities in health status. This is partly due to the long legacy of racism and racial inequality in the United States, for example. In fact, it has been consistently argued that health disparities must not be stripped from the social, cultural, political, and historical contexts in which they occur. However, differences in health can be present along other social dimensions, other than those based on race and ethnicity. These may include differences in health indicators with respect to gender, socioeconomic status (e.g., education, occupation, income), disability, age, and sexual orientation, among other characteristics. Several social determinants of health disparities have been identified, including but not limited to socioeconomic status (e.g., education, income, poverty), residential segregation, differential access to resources, lack of health insurance, and differential exposure to different types of stressors. These factors can both initiate as well as sustain health disparities. Further, systematic differences in health outcomes, such as those noted for African Americans across a number of health indicators (e.g., heart disease, certain types of cancer, diabetes, HIV, infant mortality), can further compromise the health status of already disadvantaged social groups (Myers, 2009).

There is no clear consensus on how to best measure health disparities, given the differences that exist in the use of the terms "health disparities," health inequalities," and "health inequities." Likewise, different approaches to measuring health disparities exist given the research question one is trying to answer. Nonetheless, a direct way of measuring a health difference is by comparing the health of one group (the reference group) with the health of another group(s). In general, one could compare the non-minority to the majority population (e.g., non-Hispanic whites compared to Asian Americans); compare a group against the general population; or compare differences among segments of the population. Other considerations include subgroup comparisons; for example, comparing differences within the Latino category (e.g., Mexican Americans against Cuban Americans). However, for any of these approaches, lack of clarity exists in who the reference group should be, although the most widely used approach has been to identify specific social groups a priori and examine differences in health status between them (Carter-Pokras & Baquet, 2002). Additionally, the U.S. National Center for Health Statistics has advocated for using the group with the most favorable rate in a given health indicator as the reference category, since it can potentially avoid dealing with concerns regarding who is considered to be the "most" socially advantaged group (Braveman, 2006; Keppel, Pearcy, & Klein, 2004). Other approaches to measuring health disparities have included obtaining relative indicators of health (e.g., black–white ratios), as well as measuring the distribution across individuals in a population on health status (similar to the measurement of income distribution in a population) (Asada, 2005). Depending on the field and question of interest, any of these approaches may be used, though they all have advantages and disadvantages to them.

Importantly, health disparities do not equate to *health care disparities*. In contrast to a health disparity, the Institute of Medicine, in its report "Unequal treatment: Confronting Racial and Ethnic Disparities in Health Care," defines a health care disparity as a difference in access to and quality of health care treatment between population groups that are not justified by access-related factors, treatment preferences, or the underlying health condition(s) of the population groups. Important to note is that limited access to treatment and good quality health care services play a significant role in observed differences in health status, particularly in producing racial/ethnic health disparities (Institute of Medicine, 2002).

Significantly, although specifying that both a difference in health exists and that they are socially patterned are important first steps to addressing the problem, they alone are not enough to reduce or eradicate health disparities. Indeed, addressing health disparities will require multi- and interdisciplinary research approaches to better understand the causes of specific types of health disparities, as well as multilevel interventions that target social disparities known to contribute to differences in health status (Braveman, 2006; Myers, 2009).

Cross-References

- ▶ Ethnic Differences
- ▶ Gender Differences
- ▶ Minority Health
- ▶ Racial Inequality in Economic and Social Well-being
- ▶ Social Class
- ▶ Social Epidemiology

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Health Economics

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Definition

Health economics applies the principles of economics to address problems of health and health care. It identifies the factors that contribute to the health of individuals and populations and identifies the most productive ways of using whatever resources are available for improving health.

Description

Health economics is an area of economics that applies the principles of the discipline of economics to address problems of health and health care. Both health and health care are commodities with characteristics that make them different from standard goods and services that are bought and sold in private markets. This means they require particular attention from economists in order to consider the use of resources devoted to producing health care and changing health.

Health as a Commodity

Health cannot be purchased directly. Instead it is "produced" by the levels and combinations of health "determinants," that is, factors that influence health and illness. Some of these factors can be purchased directly (e.g., exercise equipment, healthy foods, health care), while others may be in the form of public goods (or bads) such as air pollution. The individual may have little control over exposure to some of these determinants.

Although *health care* is an important determinant of health, other factors might also influence an individual's health, for example, an individual's genes, his lifestyle (Does he smoke?), his places of home and work, his diet and activity levels as well as limitations placed on choices about many of these factors by income and wealth. The relationship between health determinants and health outcomes is often complex and conditional on other health determinants. For example, the improvement in health produced from a heart bypass procedure may depend on the environment in which an individual lives and works (Are there factories close by that pollute the air that he breathes? Is he exposed to unhealthy work conditions?), the lifestyle he follows (Does he smoke?), the skill levels of the doctors treating him, etc. Economics provides a means of analyzing the production of health both at the level of an individual but also in terms of the production of health in populations.

The estimation of health production functions (the relationship between health determinants and health outcomes) enables us to consider the following:

1. The returns to investment in health determinants across a range of *different levels* of investment. For example, is the relationship between the quantity of health care and the health outcome produced constant for all levels of health care or does the change in health produced from health care change with the level of investment in health care? This is similar to the dose–response relationship in clinical research.
2. Whether the returns to investment differ among a range of *different health determinants*.

For example, does investing resources in public health programs to reduce smoking produce more health outcomes than investing the same amount of resources in additional cardiac care services?

3. Whether the return to investment in a particular health determinant is conditional on the levels of other health determinants. For example, is the health outcome associated with a public health program to reduce smoking conditional on the socioeconomic circumstances of the population targeted by the program.

Health Care as a Commodity

Health care represents a range of services aimed at improving health or reducing the risks of health loss. It is often labor intensive requiring the inputs of a mix of skilled professionals (physicians, nurses, dentists, etc.) together with nonlabor inputs such as capital equipment (hospitals, beds, diagnostic and surgical equipment) into a health care production function. The production function represents the particular technology (or production process) used to combine inputs to produce health outcomes. For example, primary care physicians may work independently, or in groups or as part of multidisciplinary health care teams.

Often opportunities arise for substitution between inputs. For example, nurse practitioners are trained to be able to perform services provided by family physicians. The production of primary care services could be changed by deploying more nurse practitioners and fewer family physicians. Decisions about the choice of production function need to be informed by evidence of the difference in outcomes and costs of the different ways of producing primary care services. Substitution can also occur between human and physical capital often as a result of new technologies. Cataract replacement surgery used to involve an inpatient stay requiring considerable inputs of physician time. The introduction of new laser technology has reduced the amount of physician time required, with the procedure now taking only a few minutes delivered in an outpatient clinic.

In addition, health care often involves episodes of care that are made up of a complex series of complementary services (e.g., prevention, treatment, and rehabilitation). The health outcomes of each item of care within an episode may not be simply additive. Failure to provide one element of the package of services may undermine the outcomes of the other elements.

Both the demand and supply of health care are complex issues that cannot be analyzed in the same way as many other commodities. Because of the complex nature of the association between health care use and health outcomes, individuals are unable to determine what services they need to address their health problems. Instead they rely on the advice of their health care provider. In an unregulated market, any individual could set themselves up as an "expert" in diagnosing the cause of an individual's health problem, recommending a treatment and delivering that care. However, changing provider as a result of poor advice would not avoid the potentially profound consequences of poor health care decisions (serious injury, illness, disability, or death). Supply is, therefore, organized through a system of strict licensure that involves restrictions on entry to the market to individuals with defined qualifications as well as professional codes of practice in order to protect the public interest.

Health care is not demanded for its own intrinsic value. On the contrary, individuals would generally prefer to not consume health care since it is often unpleasant, uncomfortable, or painful. Instead, the demand for health care is derived from the demand for the health outcomes it is expected to produce. Providers, in addition to being a major input in the supply of health care, also influence (or induce) the demand for health care through their role as advisor, or agent, of the patient. Supplier-induced demand is not a problem per se because the whole purpose of a licensure system is to have "experts" advising individuals what services they need to improve their health. However, it can become a problem where the earnings of providers respond to the

level and type of health care delivered. As a result changes in levels of services used over time need not reflect (only) changes in need for those services among patients but also responses of providers to income opportunities. This means that the traditional market of supply and demand does not exist for health care and hence market mechanisms fail to achieve the socially optimum allocation of health care resources.

Health care economics is that part of health economics concerned with the supply of health care and the evaluation of health care services and patient uptake of and compliance with treatment. In the context of scarce health care resources, it considers the impact on the health and well-being of individuals and populations of using the available resources in alternative ways by comparing both the effects (outcomes) and costs of different health care interventions (Economic evaluation). Such evaluations are, in isolation, simply descriptive information on the expected rate of return on additional investment (what extra outcome can be produced by investing more resources in this particular treatment?). In addition, consideration needs to be given to the opportunity cost of the additional investment (what has to be forgone in order to provide the additional investment required) and how to ensure the services supported by the additional investment will be produced by providers and consumed by patients in the way intended. Hence, health care economics extends beyond the area of economic evaluation of health care interventions to also incorporate the study of the behavior of providers and consumers. So, for example, there may be interest in introducing a new screening service. Health care economics would involve inter alia the following:

1. Estimating the additional costs and effects of the new service compared to existing practice
2. Calculating the expected rate of return of the additional costs
3. Considering the alternative ways of supporting the additional investment within the existing resource constraint and the forgone effects associated with taking the resources required from these other uses
4. Analyzing the behavior of providers and patients when presented with the opportunity to deliver/use the new service

This final set of challenges involves studying the funding, planning, management, and delivery of health care. Health problems can be caused by problems associated with low income and wealth, and health problems can lead to reductions in income and wealth as they can restrict normal activities. As a result an individual's need for health care is greatest when his ability to pay for health care is lowest. Health economics is, therefore, concerned with addressing this "conundrum" by analyzing alternative approaches for funding provision, allocating resources, and managing performance.

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Health Education

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Synonyms

Patient education

Definition

The World Health Organization defines health education as "any combination of learning experiences designed to help individuals and communities improve their health, by increasing their knowledge or influencing their attitudes." Because knowledge alone may not be powerful enough to motivate change, health education works to enhance knowledge, attitudes, and skills to positively influence health behaviors of individuals and communities.

Adult learning theory is an important construct to consider for effective health education. Malcolm Knowles has identified five crucial assumptions about the characteristics of adult learners. These characteristics are (1) self concept, as a person matures, they move from a dependent personality to a self-directed one; (2) experience, an accumulation of experiences are a resource for learning; (3) readiness to learn, an adult's readiness to learn is oriented to the tasks of their social roles; (4) orientation to learning, adult learning shifts from subject-centered to problem-centered; and (5) motivation, an adult learner's motivation to learn is internal.

Health education is provided in a variety of settings and can be targeted at individuals, groups, or larger populations. Although health education is generally considered primary prevention as a health promotion strategy, it can also occur at the secondary and tertiary levels. Health education at the primary prevention level is aimed at educating to promote healthy behaviors and to prevent the occurrence of illness or injury; at the secondary and tertiary levels, health education focuses on teaching strategies to detect problems early by identifying risk factors, and rehabilitation to optimize function and prevent complications of disease.

Health education is a dynamic process that requires planning and evaluation of interventions. Important steps include assessing the need for education of a target population, setting learner-centered goals and objectives, implementing the educational intervention, and evaluating and revising education to meet the targeted goals. Other considerations needed for education to be effective include one's readiness to learn, and personal, cultural, political, and environmental factors that may impact learning.

Cross-References

- ▶ Behavior Change
- ▶ Behavioral Intervention
- ▶ Community-Based Health Programs
- ▶ Education, Patient
- ▶ Empowerment
- ▶ Health Behavior Change
- ▶ Health Communication
- ▶ Health Promotion and Disease Prevention
- ▶ Intervention Theories
- ▶ Lifestyle Changes
- ▶ Risk Factors and Their Management
- ▶ Theory of Planned Behavior

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Health Inequalities

► Health Disparities

Health Inequities

► Health Disparities

Health Information Record

► Electronic Health Record

Health Insurance

► Health Insurance: Comparisons

Health Insurance Portability and Accountability Act (HIPAA)

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Synonyms

Patient protection

Definition

Health Insurance Portability and Accountability Act of 1996 (HIPAA)

HIPAA is a federal law that addresses a variety of health care subjects in various titles. These address health insurance coverage, enrollment and preexisting conditions, fraud and abuse, administrative simplification, electronic billing and coding for health care services, and the protection of certain individually identifiable health information that is obtained by "covered entities." These titles affect how health care claims are documented and billed and amended laws governing health insurers. Tax laws were amended to establish medical savings accounts and address the deductibility of health insurance premiums by self-employed individual, long-term care insurance, and provide other benefits. With respect to fraud and abuse, HIPAA also, for example, provide for advisory opinions, increased and expanded fraud and abuse investigation and enforcement penalties and tools for regulatory agencies and outline when inducements by health care providers to Medicare and certain other health care beneficiaries are prohibited. The use, disclosure, and retention of protected health information is addressed under both a privacy rule and a security rule. Covered without limitation entities include health care providers such as physicians, nurse practitioners, physician assistants, psychologists, health care facilities such as hospitals, nursing homes and pharmacies, health insurance companies and health plans, and entities that process nonstandard health information they receive from another entity into a standard format (a health information clearing house). There are also regulations governing the sharing and use and accounting of information by business associates of covered entities. Any discussion of HIPAA should also include reference to the statutes and regulations enacted under the Health Information Technology for Economic and Clinical Health (HITECH) Act, enacted as part of the American Recovery and Reinvestment Act of 2009. Subtitle D of the HITECH Act addresses the privacy and security concerns associated with the electronic transmission of health information, in part, through several provisions that strengthen the civil and criminal enforcement of the HIPAA rules.

Cross-References

► Patient protection

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Health Insurance: Comparisons

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Synonyms

Health care system; Health insurance; Risk pooling

Definition

The term *health insurance* is generally used to describe a form of insurance that pays for medical expenses of the insured. The main aim of a health insurance is to spread the financial risk arising from ill-health. Health insurance may apply to a limited or comprehensive range of medical services and may provide for full or partial payment of the costs of specific services. Moreover, the insurance could be provided either through a governmental national insurance program, or from private for-profit or non-for-profit insurance companies. Some health care systems rely more on private health insurance than others, for example, the health care system in the United States.

Description

A *health care system* includes all activities and structures whose primary purpose is to influence health in its broadest sense. The goals for health systems are good health, responsiveness to the expectations of the population, and fair financial contribution (World Health Organization (WHO), 2000). *Health care systems* are organized differently, that is, the way health care is provided and how it is financed differs between countries. Most health care systems are characterized by both providers (primary care centers, hospitals), who supply health care services, and purchasers (insurers, health authorities) who buy health care for a certain population. Health care systems can be funded via social health insurance, general taxation to the state, county or municipality, direct or out-of-pocket payments, voluntary or private health insurance, and donations or community health insurance. Most countries' systems constitute a combination of these. One common feature is that all health care systems implicitly *pool* the risks associated with individual health care needs (WHO, 2000). A risk pool allows a large group of people to share the risk that they may become ill and need expensive care. Funds dedicated for health care are collected through pre-payment (e.g., via insurance) and are managed in such a way as to ensure that the risk of having to pay for health care is borne by all the members of a pool and not by each contributor individually.

Since improving access to health care services has been a fundamental objective of health systems in the past 30 years, most countries today have a *national health insurance*, which means that it insures a national population. By having a national health insurance governments ensure that people have access to affordable health services without risking, ending up in financial difficulties. Very often national health insurance systems are established by national legislation. The clear benefit of national insurance is that the pool of pools is very large representing the national population. The contribution from the individual is regulated by the government and paid into the pool over a lifetime. This is different from *private health insurance*, where the price is set in a competitive insurance market and health risks are matched with the price of the insurance.

National health insurance can be administered by the public sector, the private sector, or a combination of both. These insurance programs differ both in terms of how the money is collected, and how the services are provided. Even if several countries raise part of the revenue for health in the same way, they may operate differently in how they pool funds and how they purchase and provide services. This is why the

traditional way of categorizing health financing systems into tax-based or social health insurance is not longer useful (WHO, 2010). In some countries, payment is made by the government (or local governments) directly from tax revenue, for example, Canada and Sweden. In other countries, for example, the UK, an additional contribution is collected for all workers, paid by employees and employers based on their earnings. In both of these cases the collection is administered by the government. The collection of compulsory contributions can also be administered by non-profit organizations like in the case of France. This is sometimes related to as a single-payer health care system. The health care providers may be either publicly or privately owned. The Netherlands, on the other hand, has adopted a completely different funding approach, where competing health insurance funds receive the compulsory contributions. These insurance funds can be either public bodies, private for-profit companies or non-profit companies. They are all obliged to provide a minimum standard of coverage and are not allowed to discriminate between patients by charging different rates according to age, occupation, or previous health status. Other countries' national health insurance plans, for example, Germany and Belgium, are largely funded by contributions by employers and employees to sickness funds. With these programs, funds usually come from three sources (private, employer-employee contributions, and national/subnational taxes). These funds are usually not for profit; institutions run entirely for the benefit of their members.

Some national insurance plans also provide compensation for loss of work due to ill-health, or covering things such as pensions, unemployment, and occupational retraining.

Countries' national health insurance systems also differ in terms of the amount of out of pocket payments that are required. Out-of-pocket expenses are direct outlays of cash made by the patient when seeking care, which may or may not be later reimbursed. Many countries still rely greatly on out-of-pocket payments from individuals to health service providers to fund their health systems. Some countries have abolished out-of-pocket payments completely, for example, the United Kingdom, and the patient is not paying anything when seeking health care, while in other countries patients are expected to pay part of their medical expenses and to pay more for higher level of services, for example, as provided in Singapore. In other countries like France, patients pay medical bills and are later reimbursed by sickness insurance funds. These outlays made by patients, usually just represent a symbolic part of the real cost of services in developed countries, as in the majority of these the government subsidizes basic healthcare. However, in many developing countries these financial outlays made by patients lead to severe financial difficulties as a consequence. A high proportion of the world's poor have no access to health services merely because they cannot afford to pay at the time they need them (Preker et al., 2004).

Separate to national health insurance there is in many countries also the possibility to have private health insurance. Private health insurance schemes are financed through private health premiums, that is, payments that a policyholder agrees to make for coverage under a given insurance policy. A contract is issued by an insurer to the covered person. Commonly private health insurance is voluntary; however, it can be compulsory for employees as part of their working conditions. Premiums paid by the covered person are non-income-related, although the actual purchase of private health insurance can in some cases be subsidized by the government. An important distinction between private and national health insurance is that the pool of financing is not channeled nor administered through the government. Private health insurance can be a controversial form of insurance because of the conflict between the need for the insurance company to make profit versus the need of its customers to remain healthy, which many view as a basic human right.

Some countries' health systems depend greatly on private health insurance, for example, the United States (US) which has a complex health care system. In the USA, public programs (e.g., Medicare, Medicaid, and Veterans Health Administration) provide coverage for health care of those

citizens that meet their eligibility requirements. The US insurance system has made discussions, and in 2010, a new law came into force making it mandatory to have health insurance.

The health sector is extremely complex, and health care expenditure represents a major use of a nation's resources and has been growing during the last three decades. Factors such as an aging population, the increased personal use of health care, and medical advances that have opened the way for more treatment options and diagnostics have contributed to a rise in the demand for health care and increased the costs for health care. At the same time enormous pressure is being put on the health system in terms of their capacity to improve outcomes and ensure consumer satisfaction, in an equitable, efficient, and financially sustainable manner. Health financing is, therefore, an issue of growing importance.

Cross-References

▶ Health Care System

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Health Literacy

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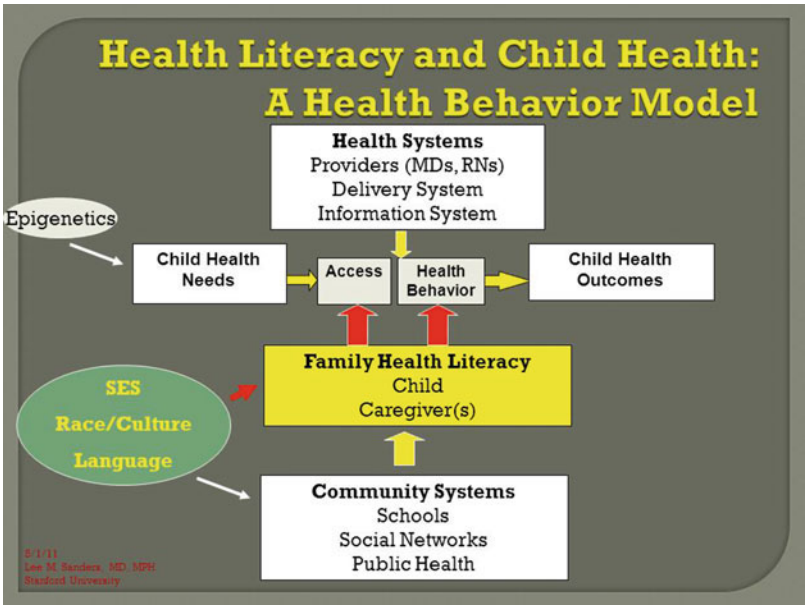
Synonyms

Literacy

Definition

Health literacy refers to an individual's ability to understand and use written and spoken health information. An applied version of the broader construct of "literacy" (i.e., the ability to read and understand all written information), health literacy encompasses the tasks necessary for a patient to navigate the modern medical system. This includes tasks such as dosing medication according to written instructions, interpreting a food label, following an immunization schedule, completing personal health information at the doctor's office, understanding a patient-centered brochure, completing health-insurance forms, or finding health information on the Internet. At least one in three US adults has limited health literacy (Kutner, Greenberg, Jin, & Paulsen, 2006; Nielsen-Bohlman, Panzer, & Kindig, 2004; Yin et al., 2009). According to the 2003 National Assessment of Adult Literacy (NAAL), 78 million US adults (36% of the population) are unable to perform "basic" health literacy tasks, such as using an immunization schedule, following recommendations from a preventive-health brochure, and interpreting a growth chart (Doak, Doak, & Root, 1996; Rich, 2004). Unfortunately, most health information in the USA is written with a complexity of form and language too difficult for most US adults to understand (Davis et al., 1994; Kutner et al., 2006; Rothman, Housam, & Weiss, 2006; Sanders, Federico, Klass, Dreyer, & Abrams, 2009; Yin et al., 2009)

Health literacy is a critical and potentially modifiable factor that influences health behaviors and may help reduce health disparities. Health literacy may contribute to the health-behavior model of health outcomes by attenuating the relationship between social factors and health behaviors. Many of the leading sources of morbidity and health disparities (e.g., preterm birth, obesity, chronic lung disease, cardiovascular disease, type 2 diabetes, mental health disorders, and cancer) are the result of literacy-sensitive health behaviors acquired across the life course (e.g., physical activity, nutrition, smoking,



Health Literacy, Fig. 1 This conceptual model proposes collective health literacy ("Family Health Literacy") and institutional health literacy (the "Health Systems") as modifiable determinants of child health outcomes. Note the contribution of other social determinants (e.g., SES, culture, language) as moderating factors and of health behaviors as mediators

risky sexual behaviors). Recent studies among adults have established an independent association between lower health literacy and decreased access to preventive-care services, increased use of urgent care services, increased risk for depression, and worse chronic-illness outcomes. Controlling for income, gender, and age, several studies have demonstrated that adults with limited literacy skills are significantly less likely than those with stronger skills to receive basic preventive care, including vaccines, weight management, and screening for breast, cervical, and prostate cancer (Bennett et al., 1998; Scott, Gazmararian, Williams, & Baker, 2002; Schillinger et al., 2002). In similarly adjusted analyses, children living with low-literacy caregivers have decreased access to primary preventive care, are more likely to be uninsured, less likely to access needed social services, less likely to be breastfed, and more likely to be exposed to second-hand tobacco smoke (Sanders et al., 2009). Adolescents who read below grade level are at an increased risk for violent and aggressive behavior, substance use, and sexually transmissible illnesses (Abrams & Dreyer, 2009)

As a result of these research findings, leading government agencies and national medical organizations – including the National Institutes of Health, the Institute of Medicine, and the Agency for Research in Healthcare Quality – have developed guidelines that call for more strategic attention to individuals' health literacy as a way of addressing major health disparities and public health challenges in the USA (Kutner et al., 2006; Nielsen-Bohlman et al., 2004; Yin et al., 2009). Experimental, clinical, community-based, and policy approaches to attenuating literacy-related health disparities have been proposed and tested. Evidence suggests that the most effective solutions apply to simplifying systems of care, particularly in the domains of medication delivery, chronic-illness management, and informed consent (Doak et al., 1996; Edwards, Elwyn, & Mulley, 2002; Rich, 2004; Sanders, Thompson, & Wilkinson, 2007; Weiss, Francis, Senf, Heist, & Hargraves, 2006). The most innovative and effective strategies apply interdisciplinary solutions that integrate cognitive behavioral theory, visual images, cultural sensitivity, and new interactive technologies.

Cross-References

- ▶ Community-Based Participatory Research
- ▶ Informed Consent

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Health Navigators

- ▶ Promotoras

Health Outcomes Research

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Definition

Health outcomes research refers to the entire evaluation of all health professionals' efforts in ameliorating patients' health conditions, in relation to various dimensions of health. Furthermore, it refers to the work not only of health professionals but of health-related organizations and their policies as a whole as well. Thus, in contrast to research on the effectiveness of specific medical or health interventions on specific health indices (e.g., blood pressure, survival), health outcomes research is a broader concept, both on the side of the intervention agents as well as on the side of the outcomes. Health outcomes research thus can guide health policy makers, health economists, as well as specific types of health professionals and clinicians.

The setting or context of health outcomes research includes clinics, hospitals, patients' homes, or even entire regions. The typical measures used in health outcomes research are also broader than those commonly used in medical intervention trials. While in the latter, outcomes mainly include physiological parameters (e.g., HbA1C, tumor markers, pulmonary functioning), health outcomes research additionally focuses on patients' satisfaction with health care, daily functioning, and, more broadly speaking, on their well-being. In addition, health outcomes research uses the method of meta-analysis to summarize and infer from multiple, yet comparable, clinical studies of a given treatment. Measures of functional status include physical (e.g., carrying), role (e.g., being a parent or worker), and social functioning (e.g., taking part in social events) in general or as influenced by a disease. The Brief Pain Inventory assesses the level of interference of pain in these factors. Measures of well-being include dimensions such as mental health, health perceptions, pain, and general life satisfaction. A common measure assessing most of these dimensions is the SF-36 (Aaronson et al., 1992). This measure is also used in health economics studies to assess quality-adjusted life years, a major outcome in health outcomes research, as it encompasses survival time with its quality.

Cross-References

► Randomized Clinical Trial

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Health Phobia

► Health Anxiety

Health Plan

► Health Policy/Health-Care Policy

Health Planning

► Health Policy/Health-Care Policy

Health Policy/Health-Care Policy

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Synonyms

Health plan; Health planning; Health program; Health strategy

Definition

A nation's, state's, city's, or other community's decisions regarding matters affecting health.

Description

Health policy refers to the systematic and/or organized approach to decision making regarding matters affecting health by a national or regional governmental entity (such as state or city) or other organized group (company, agency, etc.) (Patrick & Erickson, 1993). Most often, the term is used synonymously with health-care policy. However, the latter more narrowly applies to decisions affecting the formal medical system. Ideally the goal of such decision making would

be toward improving the well-being of members of the community (Bodenheimer & Grumbach, 2009; Weiner, Famadas, Waters, & Gikic, 2008). Further, such a decision-making process should be largely informed and driven by factual knowledge and evidence from the natural and social sciences. However, in reality, health policy is heavily influenced by many factors outside the scientific realm such as economic and political forces. In addition, such policies also heavily reflect a region's and society's ethics and values (Bodenheimer & Grumbach, 2009; Weiner et al., 2008). Thus, health policies vary widely around the world.

An example is the ways countries choose to finance their formal health-care sector, for which there are five major approaches: direct taxation; social health insurance, with mandatory premiums; voluntary or private health insurance; out-of-pocket payments; and charitable care (World Health Organization, 2005). In some countries such as Norway, the vast majority of health care is financed through direct government taxation. In Taiwan, social health insurance, financed by a payroll tax, covers nearly all health care. In the USA, a mixed market exists, with the government paying for slightly under half of all costs, and most of the rest covered by private insurance and/or out-of-pocket payments. In extremely poor countries, such as Mali, much of the care is provided by charitable organizations (World Health Organization, 2005).

Health-care policy also includes decisions around how health care is organized and delivered and the amount of money that should be devoted to health care. While it is generally agreed that countries spending less than $60 per person annually on health care have difficulty providing minimal essential services (World Health Organization, 2005), absolute funding levels do not necessarily correlate with health or health-care outcomes. For example, the USA spends nearly twice as much as most other developed countries on health care, yet is often ranked lower than many other countries with respect to measures of health outcomes and access to care. How health-care funding is allocated is also a major focus of health policy deliberations. In many countries, more population-based medical interventions, such as immunization programs, are prioritized as it is felt that these may result in a more efficient allocation of limited health-care resources. In poorer countries, such population level interventions may also be administered by transnational organizations such as the World Health Organization or nongovernmental organizations operating at the national, state, or local levels (World Health Organization, 2005).

Another example of differences in health-care policies across countries is evident with respect to counseling and therapies meant to promote behavior change, such as dietary counseling or counseling to promote tobacco cessation. Given extensive evidence that such programs can reduce complications related to obesity and long-term tobacco use, many countries provide coverage for such services. In contrast, until recently many insurance plans in the USA provided limited or no coverage for counseling directed at shaping health behaviors.

Health policy also includes programs and legislation which may influence health-related behaviors but are not typically considered part of the formal health-care sector. An example is outdoor smoking bans, which not only protect nonsmokers from secondhand smoke but are also associated with decreases in tobacco use among smokers. Laws allowing police to issue tickets to drivers of cars with unbelted passengers are associated with increased seat belt usage and a corresponding decrease in motor vehicle accident-related deaths. Another example is land-use policies that create pedestrian-friendly built environments that promote healthy behaviors, such as walking, and result in lower obesity rates (U.S. Centers for Disease Control). Often times, the health effects of such policies may not even be apparent at the time they are implemented. For example, the intention of a federally mandated decrease in highway speed limits in the 1970s was to improve conservation of fuel, but resulted in fewer automobile accident-related deaths. Thus, while health-care policy often dominates health policy deliberations, interventions

outside of the formal health-care delivery system may also have a major influence on health (Connolly, 2008).

Cross-References

- ▶ Centers for Disease Control and Prevention
- ▶ Community-based Health Programs
- ▶ Health Departments
- ▶ Institute of Medicine
- ▶ National Cancer Institute
- ▶ National Heart, Lung, and Blood Institute
- ▶ National Institute of Diabetes and Digestive and Kidney Diseases
- ▶ National Institute of Mental Health
- ▶ National Institute of Nursing Research
- ▶ National Institute on Aging
- ▶ National Institute on Alcohol Abuse and Alcoholism
- ▶ National Institutes of Health
- ▶ Robert Wood Johnson Foundation
- ▶ Smoking Prevention Policies and Programs
- ▶ Tobacco Control
- ▶ World Health Organization (WHO)

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Health Program

- ▶ Health Policy/Health-Care Policy

Health Promotion

- ▶ Health Communication

Health Promotion and Disease Prevention

- ▶ Centers for Disease Control and Prevention
- ▶ Health Communication
- ▶ Health Education
- ▶ Health Literacy
- ▶ Health Policy/Health-Care Policy
- ▶ Healthy Cities
- ▶ Healthy Eating
- ▶ HIV Prevention
- ▶ Prevention: Primary, Secondary, Tertiary
- ▶ Preventive Care
- ▶ Preventive Medicine Research Institute (Ornish)
- ▶ Worksite Health Promotion

Health Psychology

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Synonyms

Behavioral medicine; Medical psychology; Psychosomatic medicine

Definition

Health psychology is a relatively new subfield of psychology that studies psychological factors related to how people stay healthy, why they become ill, and how they respond when they do become ill. The American Psychological

Association's official definition of health psychology comes from Matarazzo (1982): "Health Psychology is the aggregate of the specific educational, scientific, and professional contributions of the discipline of psychology to the promotion and maintenance of health, the prevention and treatment of illness, the identification of etiologic and diagnostic correlates of health, illness, and related dysfunction and to the analysis and improvement of the health care system and health policy formation."

Health psychology emphasizes the biopsychosocial model where physical well-being and disease reflect a complex set of interrelated processes including biological factors (e.g., genetics, hormonal fluctuations), psychological factors (e.g., mood, personality, health behaviors), and social factors (e.g., cultural norms, health policy, social support). Health psychologists may focus their professional activities on consultation, intervention, public health policy and administration, and/or research. They commonly collaborate with other health care professionals in multidisciplinary settings in order to provide optimal care for patients and to improve health care systems, policy, and public health. From its inception, health psychology has had a dual focus on research and practice, reflecting the philosophy of the broader discipline of psychology.

The field of health psychology was formally recognized in the USA in 1978 with the establishment of the Division of Health Psychology (Division 38) within the American Psychological Association. A confluence of factors contributed to the development of the field of health psychology at this time including (a) research demonstrating compelling mind-body associations (e.g., Neal Miller's work on the conditioning of physiological processes), (b) recognition that the leading causes of mortality (e.g., coronary heart disease) could be prevented, delayed, or treated through health behavior change, and (c) the possibility to curb health care costs through prevention and low-cost behavioral initiatives. The mission of Division 38 was – and still is – to advance the contributions of psychology as a discipline to understanding health and illness through basic and clinical research, to promote education and services in the psychology of health and illness, and to inform the psychological and biomedical community of these research and service activities. Parallel movements and related fields have developed over the years but remain distinct from that of health psychology. Behavioral medicine is an interdisciplinary organization devoted to integrating biomedical and psychosocial factors in health and illness, in contrast to the intradisciplinary focus of health psychology; health psychologists engage in behavioral medicine when they collaborate with colleagues outside of psychology (e.g., medicine, nursing, public health, etc.). Medical psychology, or clinical health psychology, is a term most commonly used to describe the work conducted by clinical psychologists who practice in medical settings. Another interdisciplinary field, psychosomatic medicine, which developed somewhat earlier than health psychology and behavioral medicine, focuses similarly on understanding biobehavioral links between psychology, psychiatry, internal medicine, physiology, and other disciplines.

Division 38 (Health Psychology) of the American Psychological Association, the Society of Behavioral Medicine, and the American Psychosomatic Society are organizations that promote research and practice of health psychology and related fields. Many scholarly journals are dedicated to disseminating research generated by health psychologists. The official journal of Division 38 is Health Psychology, but there are also international journals that publish peer-reviewed research in health psychology (e.g., Journal of Health Psychology; Psychology and Health; Health Psychology Review). Health psychology research is also routinely published in journals linked to the interdisciplinary organizations of behavioral medicine (Annals of Behavioral Medicine) and psychosomatic medicine (Psychosomatic Medicine). Finally, consistent with the goal of informing the biomedical community about the research and service activities of health psychologists, health psychologists increasingly publish their research in relevant medical journals.

Cross-References

- ▶ Behavioral Medicine
- ▶ Medical Psychology
- ▶ Psychosomatic

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Health Risk

- ▶ Cancer Risk Perceptions

Health Risk (Behavior)

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Definition

Health risk is a comprehensive term covering all phenomena that carry a hazard to health of individuals or communities. Health risks can be divided into social, environmental, and behavioral risks. Another, partly overlapping, division is into health risks at population level, community level, or individual level. However, it is important to understand that health risks operate on various levels, so even if one talks about health risks on individual level, such as an individual's smoking, alcohol consumption, or lack of physical activity, these are often related to social and environmental conditions under which people live. Behavioral health risks, such as those related to eating habits, physical activity, stress, etc., have often been referred to lifestyle risk factors, although more specific terms are usually preferred.

Health Science

- ▶ Occupational Therapy

Health Strategy

- ▶ Health Policy/Health-Care Policy

Health Survey Questionnaire

- ▶ SF-36

Health Systems

- ▶ Health Insurance: Comparisons

Health-Related Quality of Life

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Synonyms

Quality of life

Definition

In 1948, the World Health Organization defined health as "the state of complete physical, mental and social well-being and not merely the absence of disease or infirmity" (World Health Organization [WHO], 1948). Since then, QoL has become increasingly important in health-care practice and research. The term "health-related quality of life" (HRQoL) narrows QoL to aspects relevant to health. However, HRQoL is a comprehensive and complex concept for which no universally accepted definition is available (Fayers & Machin, 2000). Two aspects of HRQoL are central in most definitions. First, it is a multidimensional concept that can be viewed as a latent construct which describes the physical, role functioning, social, and psychological aspects of well-being and functioning (Bullinger, 1991; Calman, 1987; Spilker, 1990). Second, in contrast to QoL, HRQoL can include both objective and subjective perspectives in each domain (Testa & Simonson, 1996). The objective assessment focuses on what the individual can do, and it is important in defining the degree of health. The subjective assessment of QoL includes the meaning to the individual; essentially it involves the translation or appraisal of the more objective measurement of health status into the experience of QoL. Differences in appraisal account for the fact that individuals with the same objective health status can report very different subjective QoL.

HRQoL as an Outcome

It has become clear in the last decade that HRQoL is an important outcome variable on its own independent of medical outcomes. HRQoL outcomes can guide decisions on alternative treatments or effectiveness of interventions at a *patient group level* (Koot, 2001). In clinical research trials in children, HRQoL has long been neglected as an outcome, but this changed rapidly over the last 10 years (Clarke & Eiser, 2004). An important step towards a more structured and frequent use of patient-reported outcomes (PROs) in drug development is represented by the US Food and Drug Administration (FDA) guidance, issued in 2006. This describes how the FDA evaluates PROs, including HRQoL, to be used as effectiveness end points in clinical trials (U.S. Department of Health and Human Services, 2006). This guidance emphasizes the importance of considering HRQoL separate from medical effectiveness. From the *individual patient perspective*, HRQoL can guide the choice of best treatment, made by the patient himself/herself and the health-care professionals (Koot, 2001). Evaluating the impact of diabetes on the adolescents' HRQoL and vice versa can help both the patient and physician decide on the optimal individual treatment (de Wit et al., 2008).

HRQoL in Children

Attention to the QoL of children has evolved rapidly from the 1980s. Advances in medical care have changed the emphasis in pediatric medicine from the diagnosis and management of infectious disease to prevention and control of chronic conditions. This means that health-care professionals should have insight into the child's views and experiences. Early attempts to rate children's QoL were based on data provided by mothers as children are often regarded as unreliable respondents. However, children and parents do not necessarily share similar views about the impact of illness. As children grow older and develop their own life, the HRQoL reports of parents become of less relevance. It has been shown that parents and children agree more on objective domains of HRQoL (i.e., physical functioning) than on subjective domains, like emotional and social functioning (Eiser & Morse, 2001; Janse, Sinnema, Uiterwaal, Kimpen, & Gemke, 2008). Therefore, the child's HRQoL is included more and more in decisions about their care and treatment.

Cross-References

▶ Quality of Life
▶ Quality of Life: Measurement

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Healthy Cities

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Definition

The term healthy cities (HC) refers to a policy and activity at the village, town, or city levels to promote health. This follows the World Health Organization's (WHO) conceptualization of health as one's physical, psychological, and social well-being and current health professionals' growing emphasis on people's self-care. Furthermore, HC reflect the emerging need to allocate resources to disease prevention and to the maintenance of health and well-being, beyond treatment of existing illnesses alone. When this is done at the level of a town or city, "peer pressure" becomes positive and can influence people toward more healthy lifestyles including balanced diets, physical activity, smoking cessation, moderate alcohol consumption, and provision of communal social support. Furthermore, recognizing that environmental factors influence health (e.g., crowding, pollution), HC also provide an excellent opportunity to change one's environment in order to foster health and well-being. Such initiatives are supported by the WHO via fostering programs and networks, inside and between countries (Goldstein, 2000). Additional core values in the HC project are equity, community participation, and community empowerment (Tsouros, 2009), particularly fostered by the European Healthy Cities Network (Heritage & Dooris, 2009).

An example of a HC project, which was tested, includes the Minnesota Heart Health Program (MHHP), where three intervention towns/cities were compared to three control towns/cities. The MHHP focused on health education with the aim to reduce cardiovascular morbidity and mortality. It succeeded to mobilize many community leaders, large segments of the adult population, and repeatedly exposed health-education information to residents via multiple channels of communication (Mittelmark et al., 1986). van Oers and Reelick (1992) developed quantitative indicators for evaluating HCs and also showed that such evaluation can feedback into local policy making, thus influencing health-related decisions at the city levels. According to initial findings from the European Healthy Cities Network, 80% of such cities used various forms of community participation, and more than two thirds of cities tried to empower their citizens (Heritage & Dooris, 2009). Empowerment is of

course central to health since it fosters self-efficacy, a major predictor of health outcomes (e.g., Ironson et al., 2005). This term reflects an important area of intervention for behavior medicine, where its theoretical models, methodological rigor, and clinical practice could contribute to societies' health at the "macro" level.

Cross-References

- ▶ Health Behavior Change
- ▶ Prevention: Primary, Secondary, Tertiary
- ▶ Self-Care

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Healthy Eating

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Synonyms

Food pyramid

Definition

A healthy diet is one that maintains a state of well-being and reduces the risk of chronic diseases such as obesity, cancer, heart disease, and diabetes. A healthy food intake will have an adequate amount and balance of macronutrients (protein, carbohydrates, and fats), micronutrients (vitamins and minerals), and fluids. This goal can be accomplished with different dietary patterns.

Description

A healthy diet produces an appropriate body weight. Maintaining a healthy weight, with a body mass index between 18.5 and 24.9, is achieved by balancing total calorie intake with calorie requirements. The 2010 report of the Dietary Guidelines for Americans states that too many calories from foods high in solid fats and added sugars are contributing to obesity. These same foods offer few nutrients other than calories. Eating with mindfulness and an awareness of what, when, and how much is eaten is a useful technique for weight loss and maintenance. Limiting solid fats such as butter and lard and using limited amounts of mono- and polyunsaturated fats from plants and seeds support healthy weight and normal blood lipids.

The base of a heart-healthy intake is nutrient-rich plant foods. In addition to essential vitamins and minerals, plant foods provide phytonutrients. Phytonutrients are chemical compounds that occur naturally in plants. Phytonutrients have a beneficial effect on health but are not yet established as essential nutrients. Examples of phytonutrients are lutein and zeaxanthin in dark greens that reduce the risk of cataracts and sulforaphane in broccoli that reduces the risk of cancer. It is recommended that half of the food consumed at a meal be plant based.

The recommendation for dietary fiber is between 25 and 35 g a day. Dietary fiber assists with weight management, control of blood glucose levels, and healthy blood cholesterol levels. Dry beans, whole grains, fruits, and vegetables with skin are sources of fiber. Many different

foods, including breakfast cereals and yogurts, are fortified with extra fiber. The Nutrient Facts Label on all packaged foods lists fiber content.

Dietary protein provides essential amino acids to build body proteins and is also a calorie source. Most Americans are eating the required 0.8 g protein/kg body weight/day. Major sources of protein are lean meats, chicken, fish, dry beans, and soy products. Proteins from dry beans and soy products have the added benefit of fiber.

For a healthy diet, sodium intake should be less than 2,300 mg for healthy adults and less than 1,500 mg for individuals with hypertension, African Americans, and middle-aged and older adults. Seventy-five percent of sodium intake comes from processed and fast food. Increasing plant-based foods, cooking and eating at home, and using low-sodium canned products assist in reducing sodium intake.

Potassium helps to reduce the impact of sodium on blood pressure and is deficient in the average American diet. Most fruits and vegetables are good sources of potassium. Excellent sources are bananas, melon, oranges, spinach, fat-free milk, tomatoes, and vegetable juice.

Most fluid requirements are met through water and beverages, and a lesser amount through food. Adequate fluid is necessary for maintaining body temperature, lubricating joints, protecting spinal cord and other sensitive tissues, and ridding the body of waste. Greater fluid intake is necessary in hot climates, among physically active people, during illness such as a fever, diarrhea, or vomiting. Primary source of fluid should be water and calorie-free drinks.

A healthy meal is one half vegetables and fruits, ¼ whole grains, and ¼ lean meats or high-protein plant foods. The Food Guide Pyramid is a resource for further information about portion sizes, meal plans, and food tracking.

Cross-References

- ▶ Cholesterol
- ▶ Eating Behavior
- ▶ Fat, Dietary Intake
- ▶ Nutrition
- ▶ Nutrition Data System for Research (NDSR)

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Healthy Eating Guide

- ▶ MyPlate

Healthy Lifestyle

- ▶ Lifestyle, Active

Healthy-Years Equivalents (HYEs)

- ▶ Benefit Evaluation in Health Economic Studies

Hearing Disturbances

- ▶ Tinnitus and Cognitive Behavior Therapy

Hearing Impairment

► Hearing Loss

Hearing Impairment (Noise Pollution Related)

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Synonyms

Noise-related hearing loss

Definition

Hearing impairment is the alteration in the perception and interpretation of sound secondary to changes in the auditory system as a result of exposure to noise.

Description

Hearing is a complex phenomenon that depends on the capture, transmission, and interpretation of sound waves from the environment. Ultimately, hearing occurs in the brain where incoming information is synthesized and interpreted. However, appropriate and accurate transmission of sound has to occur for correct interpretation to be possible. Exposure to a single loud noise or chronic exposure to noise above a sound pressure level of 85 dB (decibels) can damage the inner elements of the ear responsible for the translation of sound into signals that can be transmitted by the auditory nerve to the central nervous system. Although other components may be affected, of special concern is the impact that loud noise can have on the hair cells within the cochlea that transform sound into electrical signals that can be transmitted via the auditory nerve to the auditory cortex and related structures The level of noise exposure needed to cause permanent hearing loss may vary with the individual because of genetic or other environmental conditions. The hair cells that are responsive to high-frequency sounds, located near the base of the cochlea, are especially vulnerable. Thus, hearing loss as a result of noise exposure tends to present with a distinct audiogram pattern which usually includes a "notch" at about 4,000 Hz (Hertz; 4 kHz). A "notch" is a drop in hearing acuity at a given frequency, indicating that the sensory receptors responsive to that frequency have been damaged. Current data suggest that the damage from noise may be caused by oxidative stress. Individuals vary in their susceptibility to noise exposure, and other concurrent exposures, such as to certain medications or environmental toxins, may act synergistically with noise to further damage the hair cells. Strategies are being tested to try and protect the sensitive inner ear components before noise exposure and/or to minimize the traumatic response to the noise exposure. However, most current efforts are aimed at preventing exposure to excessive noise or minimizing the level of exposure. For example, the Occupational Safety and Health Administration has established specific requirements for hearing protection in settings where noise exceeds a given level as well as a permissible noise exposure.

Cross-References

► Hearing Impairment

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Hearing Loss

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Synonyms

Hearing impairment

Definition

A decrease, alteration, or distortion in the perception of sound.

Description

Hearing involves the perception and interpretation of sounds transmitted from the environment and occurs within the central nervous system. Sound waves enter the ear canal and stimulate the ear drum or tympanic membrane. The movement of the tympanic membrane changes the acoustic energy into mechanical energy that can then be transmitted by the three tiny inner ear bones or ossicles to the oval window of the inner ear or cochlea. The mechanical energy transmitted to the oval window initiates movement of the fluid within the inner ear. The sound energy now carried by the inner ear fluid stimulates sensitive hair cells and is transformed into electrochemical energy that can be transmitted by the auditory nerve to the auditory centers of the central nervous system. It is at the central level that sound is perceived and interpreted. Disturbances anywhere in the transmission process can lead to hearing loss. Thus, hearing loss can be conductive (disturbance in transmission from the external environment to the inner ear), sensorineural (involving alterations in the inner ear and/or auditory nerve), mixed conductive/sensorineural, or central. The cause of hearing loss can be multifactorial. Conductive loss can be caused by cerumen or wax in the ear canal which blocks sound transmission. The accumulation of wax is more common in older adults because of changes in its consistency with age. Conductive loss can also occur as a result of changes in the middle ear related to the ossicles or other diseases. Sensorineural losses can be caused by noise, ototoxic agents (e.g., environmental toxins or ototoxic medications), chronic conditions, or age. Chronic conditions such as diabetes mellitus are increasingly recognized as associated with increased incidence of hearing loss, possibly through their impact on the cardiovascular system. Age-related hearing loss is usually called "presbycusis" and is characterized initially by loss in the perception of high-frequency tones. Because consonants (such as s, p, t) are high frequency while vowels (a, e, i, o, u) are low frequency, individuals often feel they can "hear but not understand" or that individuals mumble because they are only hearing the vowels in any given word. This leads to distortion and misinterpretations. For example, "time" and "dime" may be confused. Hearing loss can occur at any time in the life cycle but becomes increasingly common with age. While hearing loss is usually not considered a life-threatening condition, it is associated with multiple negative outcomes including isolation, depression, and altered interpersonal relationships. There is also increasing interest in the relationship between hearing loss and cognition. Approaches to the treatment of hearing loss usually focus on enhancing amplification in the frequencies that are most affected. Newer hearing aids have a greater capacity to be individualized but remain aids. Individuals need to understand that they will need to relearn how to hear and to get use to hearing sounds that they may not have heard in some time. The underlying damage to the ear is

not repaired. In addition to hearing aids, other assistive listening devices are available to facilitate communication or participation in activities or events (510).

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Heart

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Definition

The heart is the principle organ of the circulatory system and is responsible for pumping blood through the pulmonary and systemic circulations.

Description

In humans, the heart has four structural chambers: two upper ones, the left and right atria, serving as reservoirs for blood return from the pulmonary and systemic venous vasculature, respectively; and two lower chambers, the left and right ventricles, that eject blood forward into the aorta and the pulmonary arteries. In a typical cycle, peripheral venous blood returns to the right atrium via the superior and inferior venae cavae, and then empties into the right ventricle through the tricuspid valve, both passively and with contribution from right atrial contraction. Contraction of the right ventricle then opens the pulmonic valve and propels the deoxygenated blood through the main pulmonary artery on the way to the lungs. After passing through lung alveoli where gas exchange takes place, oxygenated blood from the pulmonary circulation eventually drains into the left atrium through the four pulmonary veins, and from there fills the left ventricle via passage across the mitral valve. Contraction of the left ventricle then opens the aortic valve, and oxygenated blood is pumped into the aorta to perfuse the other organs of the body. The blood supply to the heart itself is provided by the coronary arteries: the left main artery, arising from the left coronary cusp at the root of the aorta, and giving rise to the left anterior descending and the left circumflex arteries; and the right coronary artery, arising from the right coronary cusp. Venous return from the heart drains into the right atrium via the coronary sinus (Kusumoto, 2005).

Anatomically, the heart lies within the mediastinum and is encapsulated by the pericardial sac, which normally contains small amounts of serous fluid to minimize friction as the heart moves during each contraction. At the cellular level, the heart is composed of myocytes, which contain sarcomere units that form the basic contractile elements. The myocytes also possess gap junctions that allow propagation of electrical impulses from cell to cell. In addition, there are specialized myocytes with unique electrical properties that form the conduction system of the heart. Electrical impulses are typically

initiated in the sinoatrial node (SA node), located within the right atrium, then travel to the atrioventricular node (AV node), which lies along the inferior-posterior aspect of the interatrial septum near the opening of the coronary sinus. The AV node allows conduction from the atria to the ventricles, and its refractory properties help to prevent rapid ventricular response in case of atrial arrhythmias. From the AV node, the electrical impulses travel rapidly down the bundle of His and then the right, left anterior and left posterior bundle branches, eventually arriving at the Purkinje fibers that reach the rest of the heart. It is this carefully timed activation sequence that makes possible the ordered and efficient contraction that occur with each heartbeat (Kusumoto, 2005).

Numerous disease processes are known to affect the heart. Early in life, abnormalities of cardiac development can lead to various forms of congenital heart disease. Disorders of the conduction system can manifest as tachyarrhythmias and bradyarrhythmias, and can present at any age, either in isolation or in conjunction with other cardiac diseases. Valvular heart disease may involve stenotic or regurgitant lesions of any of the four cardiac valves or combinations thereof. The clinical syndrome of heart failure, defined as the inability of the heart to generate enough forward flow except at elevated filling pressures, can occur through diverse mechanisms including ischemia due to coronary artery disease to genetic disorders such as hypertrophic cardiomyopathy. Ischemic heart disease caused by coronary atherosclerosis is currently the most frequently diagnosed cardiac disorder, in part due to the increased life expectancy and changes in lifestyle and diet that occurred in the past century. Taken as a whole, cardiovascular diseases represent one of the most significant global public health burdens of the modern age, accounting for 30% of all deaths worldwide and up to 40% of all deaths in the industrialized world (American Heart Association Statistics Committee and Stroke Statistics Subcommittee, 2010; Gaziano, 2008).

Cross-References

▶ Heart Disease

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Heart and Estrogen/Progestin Replacement Study (HERS)

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Definition

The heart and estrogen/progestin replacement study (HERS) was a randomized, double-blinded placebo-controlled trial of the effect of 0.625 mg of conjugated estrogens plus 2.5 mg of medroxyprogesterone acetate daily on coronary heart disease (CHD) event risk among more than 2,700 postmenopausal with women with known CHD.

Description

The primary outcome of this trial was CHD events (nonfatal myocardial infarction (MI) plus CHD-related death). Secondary cardiovascular outcomes included coronary revascularization, unstable angina, congestive heart failure,

resuscitated cardiac arrest, stroke or transient ischemic attack, and peripheral arterial disease.

Overall, during more than 4 years of follow-up, there were no significant differences between the hormone and placebo groups in the primary outcome of CHD events (nonfatal MI plus CHD-related death) or in any secondary outcomes.

However, post-hoc analyses showed a statistically significant time effect, wherein participants taking hormone therapy experienced more CHD events in the first year of treatment, and fewer in years 3–5. This led the investigators to speculate that the early risk due to estrogen/progestin treatment may be due to a prothrombotic, proischemic, or proarrhythmic effect which was gradually outweighed by the beneficial effects of hormone therapy, potentially mediated by observed changes in low- and high-density lipoprotein cholesterol.

This apparent pattern of an early increase in CHD events followed by a later decrease led to the recommendation that women with CHD should not start hormonal therapy for the purposes of preventing CHD events, but that those who were taking hormones could continue to do so. As reported in HERS II, women in HERS tended to follow this advice and many of those randomized to hormones during the trial continued with open-label treatment. This provided an opportunity to continue outcome surveillance for several years (HERS II).

The HERS II study contained both cardiovascular and non-cardiovascular components. Participants from the initial HERS study were followed for an additional 4 years. The HERS cardiovascular study demonstrated the absence of late benefit of hormone therapy, effectively answering the important question raised by the original HERS study of whether the lower CHD event rate observed during the final years of the trial suggested the presence of a long-term benefit of hormone replacement therapy. The non-cardiovascular component of the HERS study demonstrated an increase in venous thromboembolism and biliary tract surgery over the near 7 years of follow-up and demonstrated unfavorable trends in the incidence of some cancers and incidence of fractures.

The follow-up study (HERS II) has been critiqued because randomized assignment was no longer blinded in HERS II, and event ascertainment may have been influenced by the knowledge of randomization assignment. Regardless, the HERS I/II provided useful information regarding the short- and long-term use of hormone replacement therapy, and supported the larger, more definitive trials on hormone replacement and cardiovascular and non-cardiovascular events, such as the Women's Health Initiative.

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Heart Attack

▶ Acute Myocardial Infarction

Heart Bypass Surgery

▶ Bypass Surgery

Heart Disease

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Definition

Heart disease encompasses a range of conditions, including coronary artery disease, heart failure or inadequate cardiac output, and cardiac arrhythmia. See ▶ Coronary Heart Disease (CHD) and ▶ Congestive Heart Failure for further explanations.

Cross-References

- ▶ Atrial Fibrillation
- ▶ Congestive Heart Failure
- ▶ Coronary Heart Disease

Heart Disease and Cardiovascular Reactivity

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Synonyms

Cardiac risk factor

Definition

There has been a long-standing notion that exaggerated responses to mental stress are linked to the development of future heart disease (Hamer & Malan, 2010). Although not clinically meaningful in themselves, if heightened responses to stress are elicited on a regular basis, they might become clinically relevant over time. Existing work has largely focused on cardiovascular reactivity to stress as a tool to predict future risk. Blood pressure and heart rate responses to mental stress are largely augmented by the sympathetic nervous system and release of catecholamines. The issue of whether stress reactivity contributes to the progression of underlying disease or only to the incidence of clinical cardiac events has led to research involving indicators of subclinical disease. Several studies have indicated that heightened blood pressure and heart rate responses to laboratory-induced stressors predict future progression of subclinical atherosclerosis and hypertension in initially healthy participants, independently from conventional risk factors such as blood cholesterol, resting blood pressure, and smoking (Chida & Steptoe, 2010). Few studies have examined the association between cardiovascular reactivity and the incidence of clinical cardiac events, and the available evidence is equivocal. Since individuals cannot be randomized to being high or low cardiovascular reactors to stress, it is impossible to prove or disprove beyond doubt that exaggerated reactivity is a causal risk factor in cardiovascular disease. On balance, there is substantive evidence to support the reactivity hypothesis although effect sizes are small and the clinical utility is questionable. Reasons for inconsistencies in the data include variability in the type of mental stressors employed and individual differences in responses that might be influenced by factors such as personality, race and ethnicity, genetics, chronic background stress, and lifestyle habits.

From a mechanistic standpoint, stress-induced blood pressure surges that contribute to increased shear stress in the arteries could promote endothelial damage and inflammatory responses that are thought to play a role in atherogenesis. Endothelial dysfunction plays a key role in the initiation of atherosclerosis because nitric oxide production from healthy endothelial cells has an anti-atherogenic effect by inhibiting cellular adhesion, migration, and proliferation responses. Individual differences in blood pressure reactivity have also been linked with activation patterns in corticolimbic brain areas (e.g., divisions of the

cingulate cortex, insula, and amygdale) that are jointly involved in processing stressors and regulating the cardiovascular system (Gianaros & Sheu, 2009). Thus, this data might provide the vital piece in the missing jigsaw puzzle explaining the interaction between brain and body.

Cross-References

- ▶ Blood Pressure Reactivity or Responses
- ▶ Cold Pressor Test
- ▶ Physiological Reactivity
- ▶ Stroop Color-Word Test

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Heart Disease and Emotions: Anger, Anxiety, Depression

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Definition

The negative emotions of anger, anxiety, and depression exist in several different forms – stable individual differences in negative emotionality (i.e., personality traits), brief emotional episodes, symptoms of emotional distress, and emotional disorders. Each of these emotion constructs has been examined as an influence on the development and course of coronary heart disease, a common cause of death resulting from the progression of atherosclerosis in the arteries that otherwise supply blood to the myocardium.

Description

Negative emotions have been suggested as an influence on the development and course of coronary heart disease (CHD) for centuries since the earliest descriptions of this common and costly medical condition. CHD begins as early as childhood or adolescence, with the development of fatty streaks within the walls of the coronary arteries. Over decades, these progress to more substantial coronary artery lesions, with the buildup of lipids and inflammatory processes at these sites. After decades of asymptomatic or silent progression of coronary artery disease (CAD), the symptoms of CHD appear in mid to later adulthood in the form of angina pectoris (chest pain due to myocardial ischemia), myocardial infarction, or sudden coronary death. Recent research indicates that negative emotion may play a role in CHD at each stage of this decades-long process. However, the various specific negative emotions and forms of these emotion characteristics are closely related, and it is not clear if they have independent or overlapping effects on the development and course of CHD (for reviews, see Smith, 2010; Suls & Bunde, 2005).

Virtually all major conceptual descriptions of basic emotions distinguish among anxiety and related emotions (e.g., fear), depression and related emotions (e.g., sadness), and anger. In contrast to a state of calm, anxiety is characterized by nervousness, tension, and apprehension. In the extreme, it is characterized by dread. Depression is characterized by sadness, sorrow, and unhappiness, and in the extreme by despair. Anger varies in intensity from irritation or annoyance to the extreme of rage. In terms of accompanying cognitive content, anxiety is associated with perceptions of threat and vulnerability to potential harm. In contrast, depression is associated with

a sense of loss, separation from important others, failure, and hopelessness. Anger is associated with thoughts of interpersonal transgression, frustrated goals, and unfair victimization or mistreatment.

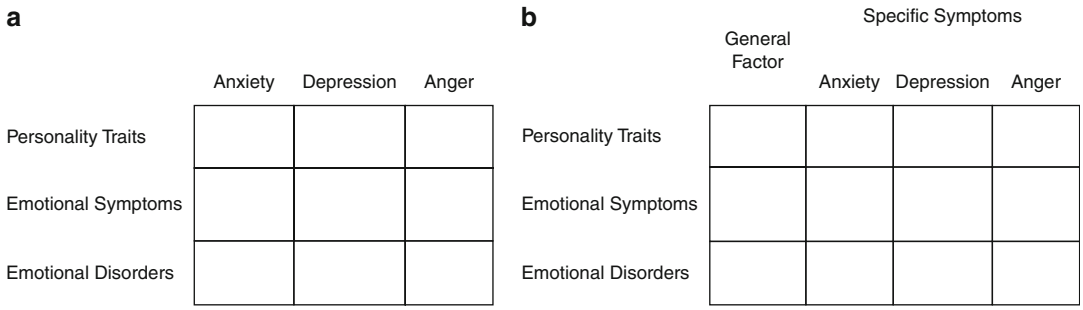
These affective phenomena take several forms, ranging from brief episodes well within the range of normal experience to much more enduring and maladaptive conditions. Episodes of negative emotion (e.g., anger during a pointed disagreement) have been examined as precipitants or "triggers" of acute coronary events (e.g., myocardial infarction, sudden coronary death), and the related findings suggest that these episodes can indeed evoke such life-threatening events, although this is most likely limited to individuals who have advanced CAD (Bhattacharyya & Steptoe, 2007). Episodes of negative emotion can be distinguished from related moods, in that emotions are more intense, briefer, and much more strongly related to physiological changes and the activation of associated behavioral response tendencies.

There are stable individual differences in the tendency to experience negative emotions and moods, such as the Five-Factor Model trait of neuroticism versus emotional stability. This trait contrasts individuals who are prone to anxiety, depression, anger, and related characteristics (e.g., self-consciousness, feelings of vulnerability or inferiority) with individuals who are generally calm. A closely related construct – negative affectivity – differs from neuroticism by including only the affective elements of this broad trait. In these trait models, individual differences in the tendency to experience anxiety, depression, and anger are seen as lower level, more specific aspects or "facets" of the broader trait.

These emotions are also seen in various forms of psychopathology. Anxiety disorders (e.g., generalized anxiety disorder, social phobia) and mood disorders (e.g., major depressive disorder) are common, and intense and destructive anger is the major feature of intermittent explosive disorder. Individuals can report elevated symptoms of anxiety, depression, or anger without reaching the severity, duration, or level of related impairment necessary to qualify for a diagnosed emotional disorder. Such elevations in symptoms are presumed to be less enduring than the negative emotionality seen in related personality characteristics, and diagnosable emotional disorders are seen as qualitatively distinct from personality traits in their severity, related features, and levels of general impairment or dysfunction. Hence, anxiety, depression, and anger are typically conceptualized and studied as distinct emotions, as are their various forms in emotional episodes, symptoms, disorders, and personality traits.

If these distinctions were in fact firm, the common approach of studying one emotional risk factor for CHD at a time would not pose a problem. Unfortunately, these distinctions are quite difficult to support empirically (Smith, 2010; Suls & Bunde, 2005). There is a high degree of correlation among anxiety, depression, and anger, and this is true in each of the various forms. Further, the distinctions between the various forms of negative emotions are problematic. Symptoms of emotional distress are closely correlated with related personality traits, and scores on such symptom inventories are more stable than should be the case if they were unrelated to personality (Suls & Bunde; Watson, 2009). Neuroticism and negative affectivity are closely associated with anxiety and mood disorders (Weinstock & Whisman, 2006), and symptoms of emotional disorders often appear to take the form of continuous severity distributions rather than the discrete structure implied by categorical models of emotional disorder (Haslam, 2007). Emotional disorders co-occur so frequently that alternative diagnostic frameworks involving "distress disorders" have been proposed (Watson, 2009). Hence, when one specific negative affect risk factor is related to the development or course of CHD, this association could easily involve another negative affect and/or another form of negative affect. This lack of specificity complicates the design and implementation of risk-reducing interventions, in that it is not clear which specific negative affect or which form of negative affect should be targeted.

Reviews of the many studies of negative emotional factors and the development and course of CHD suggest reliable associations for depression, anxiety, and anger when these characteristics are



Heart Disease and Emotions: Anger, Anxiety, Depression, Fig. 1 (**a**) Conceptual framework for overlapping negative affective risk factors (**b**) Alternative framework including general and specific aspects of negative affect (Reprinted with permission from Smith (2010) (Fig. 12.1, p. 161))

considered separately (Chida & Steptoe, 2009; Nicholson, Kuper, & Hemingway, 2006; Suls & Bunde, 2005). However, the far fewer studies attempting to parse the potentially overlapping effects of these negative emotional phenomena have produced mixed results. Studies of preclinical, asymptomatic atherosclerosis that have measured multiple negative affective characteristics have sometimes found that depression but not anxiety or anger predicts disease severity (Stewart et al., 2007), whereas others have found that anxiety and anger but not depression are related to CAD (Smith, Uchino et al., 2008). Studies of multiple indicators of negative affect as predictors of the initial occurrence of CHD have produced evidence of the unique or independent effects of anxiety (Phillips et al., 2009; Kubzansky et al., 2006; Shen et al., 2008), depression (Grossardt et al., 2009; Phillips et al., 2009), and general emotional distress (Boyle et al. 2006; Kubzansky et al., 2006). In studies of patients with CHD, the evidence is again mixed. Some studies suggest that anxiety and depression are independent predictors of recurrent cardiac events (Frasure-Smth & Lespérance 2008), whereas others suggest that anxiety but not depression has an independent effect (Tully et al., 2008).

Additional research is clearly needed to sort out the independent versus overlapping effects of the various negative emotions and their various forms, across the multiple phases of the development and course of CHD. The design of future studies must attend to the fact that anxiety, depression, and anger are correlated, and the overlap across various forms of these emotions is considerable. Figure 1 presents two conceptual models that can guide these efforts. In the first (panel A), anxiety, depression, and anger are each considered and assessed separately, as are the various forms of these emotional constructs. In the second, the general factor (i.e., general emotional distress) is considered and assessed directly, as are the unique aspects of anxiety, depression, and anger. For example, depression differs from anxiety and anger in its associated low levels of positive affect and physiological activation (Watson, 2009), and anger involves approach motivation (e.g., overcoming obstacles in frustrated goals, redress social transgressions or mistreatment), whereas anxiety is mostly associated with avoidance motives (i.e., minimize harm). Additional studies that measure these multiple aspects and forms of negative emotion could help define targets for risk-reducing interventions more precisely.

Interventions for CHD patients focused on a specific negative affect (e.g., depression) generally have not been found to be effective in reducing recurrent coronary events (ENRICHD Investigators, 2003). In contrast, more general interventions focusing on stress and negative emotion broadly considered have been found to be effective in improving the prognosis of CHD patients (Linden, Phillips, & Leclerc, 2007). If supported in future research, it is possible that this pattern of intervention results indicates that it is the general or overlapping aspects of negative emotion – rather than the specific aspects – that

are important risk factors and important intervention opportunities. However, much additional research is needed.

Cross-References

- ▶ Anger Management
- ▶ Anger, Measurement
- ▶ Anxiety and Heart Disease
- ▶ Anxiety Disorder
- ▶ Depression: Measurement
- ▶ Depression: Symptoms
- ▶ Depression: Treatment
- ▶ Type A Behavior
- ▶ Type D Personality

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Heart Disease and Smoking

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Synonyms

Cardiovascular disease (CVD); Coronary heart disease (CHD); Nicotine

Definition

Heart disease is a relatively broad term which refers to many diseases associated with the heart. Coronary heart disease (CHD) refers to a narrowing or even complete occlusion of the blood vessels which supply the heart with blood and oxygen. Cardiovascular disease (CVD) is often used as a synonym for CHD. Smoking is defined as inhalation of smoke from burning tobacco via a cigarette, cigar, pipe, or other smoke delivery device (e.g., hookah).

Description

Approximately 20% of adults smoke in the United States, and this percentage has remained fairly consistent across the last 10 years (Centers for Disease Control and Prevention, 2010). It is currently estimated that over 140,000 individuals in the US will die annually due to smoking-attributable CHD (Centers for Disease Control and Prevention, 2008). There is currently compelling evidence that smoking is causally related to cardiovascular diseases. The most extensive current compilation of evidence supporting a causal link between smoking and CHD is found in the 2010 Surgeon General's Report: *How Tobacco Smoke Causes Disease: The Biology and Behavioral Basis for Smoking-Attributable Disease* (U.S. Department of Health and Human Services, 1994). What follows is a brief overview of the evidence for a causal link between smoking and CHD including a discussion of the biological processes impacted by smoking that are implicated in CHD.

There are numerous studies which demonstrate a clear dose–response relationship between number of cigarettes smoked per day and risk for developing CHD. The recent Surgeon General's Report on Smoking (Centers for Disease Control and Prevention, 2010) indicated that risk for CHD is increased across all levels of smoking and that smoking even a few cigarettes per day (less than five) resulted in increased risk of developing CHD. This report also indicated that duration of smoking is associated with increased risk of CHD-related mortality.

Exposure to secondhand smoke also increases the risk of CHD. One study determined that the relative risk of CHD for smokers is 1.78 as compared with 1.31 in nonsmokers exposed to secondhand smoke. This study also revealed that exposure to secondhand smoking resulted in similar negative circulatory changes to those which occur in smokers. There is also evidence of decreased hospital admissions due to myocardial infarction rates in locations which have banned public smoking (Barnoya & Glantz, 2005)

There are multiple physiological changes which occur in response to smoking which are thought to be associated with CHD. Cigarette smoking results in decreased blood flow to the heart and increased blood pressure and heart rate (U.S. Department of Health and Human Services, 1994). Smoking damages and impairs regeneration of the inner lining of blood vessels (endothelium). Damaged blood vessels increase the risk of developing arterial plaques (atherosclerosis) which consequentially increases the risk of developing blood clots which can lead to myocardial infarct (U.S. Department of Health and Human Services). Cigarette smoking causes inflammation in the circulatory system and also impairs vasodilation of blood vessels, both of which increase the risk for negative cardiac events (U.S. Department of Health and Human Services). Smoking can also cause platelets in the blood to become sticky, and this is thought to increase the likelihood of clot formation (U.S. Department of Health and Human Services). Importantly, similar

physiological changes have been demonstrated in persons exposed to secondhand smoke (Barnoya & Glantz 2005).

Quitting smoking will reduce the risks of developing CHD (U.S. Department of Health and Human Services). The risk for CHD and CHD-related mortality drops in half after approximately 1 year of smoking cessation. After 10 years of smoking cessation, the risk for developing CHD-related issues is essentially equivalent to that of a nonsmoker (U.S. Department of Health and Human Services). Smoking cessation at any age is also clearly associated with increased longevity (Taylor et al. 2002).

Cross-References

- ▶ American Heart Association
- ▶ Atherosclerosis
- ▶ Cardiac Rehabilitation
- ▶ Cigarette Smoking Behavior
- ▶ Coronary Heart Disease
- ▶ Ex-Smokers
- ▶ Heart Disease
- ▶ Heart Failure
- ▶ Inflammation
- ▶ Secondhand Smoke
- ▶ Smoking and Health
- ▶ Smoking Behavior
- ▶ Smoking Cessation
- ▶ Tobacco Cessation
- ▶ Tobacco Use

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Heart Disease and Stress

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Definition

Stress is a hard concept to define precisely, but like obscenity (to paraphrase Supreme Court Justice Potter Stewart (*Jacobellis vs. Ohio. 378 U.S. 184, 197, 1964*)), you (sometimes, but perhaps not always) know it when you feel it. For any given system, stress implies a load that challenges the homeostasis and integrity of the system. Stress may be intrinsic to the system, for example, the weight of a suspension bridge's roadway platform, or extrinsic to the system, such as the effect of hurricanes, earthquakes, or traffic jams loading the bridge. Both intrinsic and extrinsic stressors pose the risk of bridge collapse. Whether a stressor is intrinsic or extrinsic, the system must have a capacity to withstand it in order to maintain structural and/or functional integrity. Thus for a suspension bridge, to continue the example, this capacity is built into the tensile strength of its suspension cables and the mass and strength under compression of the towers and buttresses supporting the cables and transmitting force to the surrounding ground. In behavioral cardiology, concern about stress and heart disease has focused on external psychological stressors, and related behaviors and affective and psychophysiological states, associated with

the onset and progression of heart disease, especially coronary artery disease and sudden cardiac death.

Description

For lack of a precise, gold-standard, quantifiable measure of stress as it relates to heart disease, stress is usually defined by subjective measures of perceived stress, by characterization and counts of common life experiences generally regarded as stressful, unwanted, or at least as disturbing homeostasis (e.g., daily hassles, hostile interactions, job strain, job loss, loss of a loved one, divorce, interpersonal conflict, moving to a new city, marriage, promotion to new work responsibilities), by experimental manipulations or ecological measurement of short-term challenges, and by exposures to epidemiologically significant events such as natural disasters, missile attacks during wartime, and terrorism. It is evident that stress is often associated with negatively valenced affective states such as anxiety, depression, and anger, but can also be associated with more positively experienced acute excitement and life experiences.

Evidence for the association of stress with heart disease is overwhelming. Numerous studies demonstrate that earthquakes are associated with a subsequent increase over background rates in myocardial infarction and sudden death. Iraqi missile attacks on Israel during the Gulf War in the 1990s, the destruction of the World Trade Center in New York in 2001, and sports events such as World Cup Soccer matches have been consistently observed to be associated with increased rates of acute coronary syndromes and lethal ventricular arrhythmias, even in individuals not physically endangered by proximity to the events. Acute episodes of anger appear to be an especially potent trigger of acute coronary events in vulnerable patients. In INTERHEART, a very large case–control study of first myocardial infarction involving over 24,000 subjects in 52 countries, chronic exposure to stress, measured by self-reports of problems with family relationships, work, or financial strain over a 1-year period, was associated with increased rate of MI, with population-attributable risk estimated at 12–33%. A dose–response relationship was observed between the number of stressful life events reported and risk of myocardial infarction, with an odds ratio for MI risk of 1.5 for individuals reporting two or more stressful life events compared to those reporting no stressful life events. Thus, epidemiological evidence supports the role of emotional stress both over the long term, in processes contributing to the development of coronary atherosclerosis, and in the short term, as a trigger of acute coronary events in vulnerable patients. Perceived stress is heightened in people with low socioeconomic status, which may also be associated with numerous other behavioral and physiological cardiac risk factors. Conversely, stress effects on cardiovascular risk are moderated by the presence of good social support. Studies comparing heart disease risk in men and women have found that for women, the relationship of stress at work to coronary heart disease risk is less clear-cut than in men, but stress at home and in interpersonal relationships is associated with increased risk of coronary disease-related events.

Stressful experiences result in derangements of autonomic nervous system regulation of the cardiovascular system, with reduced vagal tone and relatively increased sympathetic tone. Heart rate and blood pressure increase in response to acute mental stress; the extent of this increase depends in part on the nature of the stressor and also in part on individual characteristics such as trait anxiety and hostility. Interruption of sympathetic inflow to the left side of the heart by left stellate ganglionectomy reduces stress-induced ischemia and ventricular arrhythmias. Stressful experience is associated with hemoconcentration and increased blood viscosity, which may increase the risk of thrombosis, especially as clotting factors and platelet activation also increase during acute stress. Stressful experiences alter hypothalamic-pituitary axis function, with lasting consequences, such that childhood trauma is associated with elevated circulating levels of corticotrophin-releasing factor in adulthood, and stress in adulthood leads to hypercortisolemia.

Endothelial dysfunction and paradoxical vasoconstriction during acute mental stress are observed in atherosclerotic coronary artery segments. During mental stress, increased heart rate and blood pressure along with coronary vasoconstriction may result in myocardial ischemia, which may be manifest as ST segment changes in the electrocardiogram, reduced left ventricular ejection fraction and regional wall motion abnormalities; stress-induced impairment of left ventricular function is associated with an increase in the risk of recurrent coronary events and survival. Yet, although mental stress–induced ischemia occurs primarily in individuals with exercise-induced ischemia, it is notable that mental stress–induced myocardial ischemia is frequently asymptomatic, and occurs at a level of increased heart rate and blood pressure less than that required to achieve ischemia during exercise. Acute mental stress also induces an inflammatory response, with elevated levels of inflammatory cytokines IL-6 and TNF-alpha, which can destabilize atherosclerotic plaque and promote plaque rupture or superimposed thrombosis. The extent to which each of these mechanisms mediates the effect of stress on heart disease risk is unknown.

Sudden emotional stress can also lead to acute cardiac events in patients who do not have coronary artery disease. Takotsubo cardiomyopathy is a rare syndrome characterized by transient left ventricular dysfunction with apical ballooning and acute heart failure in response to emotional shocks, such as surprise parties or receiving unexpected bad news. The syndrome has been attributed to rapidly increasing sympathetic tone and catecholamine levels in response to the psychological stressor.

Studies of the effects of interventions to reduce stressful experience or improve resilience in managing stress on heart disease outcomes have had decidedly mixed results. Behavioral or cognitive behavioral stress management treatment groups to reduce angry responding and time urgency reduce the incidence of recurrent MI. Inclusion of "stress management" in cardiac rehabilitation programs for post-MI patients is associated with reduced recurrent cardiac events and reduced mortality, but individualized life stress monitoring and problem-solving interventions for post-MI patients resulted in no benefit in men and increased recurrent cardiac events in women in the Montreal Heart Attack Readjustment Trial (M-HART). Cognitive behavior therapy aimed at bolstering social support also had a negative effect on recurrent cardiac events in women in the ENRICHD trial. Overall, however, exercise and stress management programs appear to reduce recurrent cardiac events, improve subjective well-being, and may reduce hemodynamic and other physiological responses to stress.

Cross-References

- ▶ Coronary Artery Disease
- ▶ Social Support
- ▶ Stress

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Heart Disease and Type A Behavior

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Synonyms

Coronary heart disease (CHD); Type A behavior pattern (TABP)

Definition

A comprehensive meta-analysis of prospective studies between 1966 and 1998 failed to show an association between Type A behavior pattern (TABP) and coronary heart disease (CHD) (Myrtek, 2001), and since then there has been no evidence showing such an association. However, the recent meta-analytic investigation on prospective studies showed that anger and hostility, one of the key dimensions of TABP, are significantly associated with not only increased CHD events in initially healthy populations but also poor prognosis in the patients with existing CHD (Chida & Steptoe, 2009).

Description

Type A behavior pattern (TABP) was first noted by two American cardiologists, Meyer Fredman and Ray Rosenman, in 1959 (Friedman & Rosenman, 1959). They demonstrated that a group of 83 men, mainly executives, had seven times the risk of coronary heart disease (CHD) when compared with that in the other two groups of 83 men from unions and accounting firms and 46 unemployed blind men. The behavior pattern of group A is characterized by intense ambition, competitive "drive," constant preoccupation with "deadlines," and a sense of time urgency. Over time, together with input from psychologists, they refined the notion of TABP with an increasing focus on the elements of aggressiveness and easily aroused annoyance or hostility (Rosenman, 1978). Persons not showing these characteristics are labeled Type B behavior pattern. Data published in 1974 and 1976 from the Western Collaborative Group Study containing approximately 3,000 middle-aged men, exhibited a relative risk of 1.8 for nonfatal myocardial infarction (MI) or angina over 4 years in those were TABP, and a relative risk of 1.9 for fatal CHD, nonfatal MI, or angina over 8.5 years in those who were TABP (Jenkins, Rosenman, & Zyzanski, 1974; Rosenman, Brand, Sholtz, & Friedman, 1976). Nevertheless, as time passed, these original findings were not supported by an increasing number of subsequent studies (Brotman, Golden, & Wittstein, 2007; Everson-Rose & Lewis, 2005). More comprehensively, a meta-analysis of prospective studies between 1966 and 1998 failed to show an association between TABP and CHD (Myrtek, 2001), and since then there has been no evidence showing such an association.

Some researchers, therefore, changed their focus to investigate whether anger, hostility, and related constructs, one of the key dimensions of TABP, would be more closely linked to the development of CHD. Hostility is typically described as a negative attitude or cognitive trait directed toward others, anger as an emotional state that consists of feelings that vary in intensity from mild irritation or annoyance to intense fury or rage, and aggressiveness as a verbal or physical behavioral pattern manifest in yelling, intimidation, or physical assaults. These constructs, these terms often are used interchangeably and their interrelationship remains poorly delineated (Martin, Watson, & Wan, 2000; Schulman & Stromberg, 2007). Over the past 25 years, the body of research investigating associations between anger and hostility and CHD development and progression has grown. The recent meta-analytic investigation on prospective studies showed that anger and hostility are significantly associated with not only increased CHD events in initially healthy populations but also poor prognosis in the patients with existing CHD (Chida & Steptoe, 2009). The harmful effects of anger and hostility were slightly greater in the CHD patients than the healthy population

studies, making it possible that frequent anger episodes related to trait anger and hostility trait might accelerate recurrence of CHD.

If anger and hostility do influence CHD risk, effects might be primarily mediated via behavioral pathways, with anger and hostility promoting high-risk behaviors such as poor diet, less physical activity, smoking, poor sleep, or lower treatment adherence (Scherwitz et al., 1992; Shin et al., 2005; Siegler, Peterson, Barefoot, & Williams, 1992). Indeed, the apparently harmful effects of anger and hostility on CHD were no longer significant in either the healthy or disease populations after fully controlling for behavioral covariates such as smoking, physical activity or body mass index, and socioeconomic status (Chida & Steptoe, 2009). However, other unmeasured factors cannot rule out that could potentially have confounded the associations, and direct physiological pathways might also contribute. Anger and hostility may alter susceptibility to CHD via autonomic nervous dysregulation (Chida & Hamer, 2008; Thomas, Nelesen, & Dimsdale, 2004; Vella & Friedman, 2007), increases in inflammatory and coagulation factors such as interleukin-6, C-reactive protein, and fibrinogen (Markovitz, 1998; Stewart, Janicki-Deverts, Muldoon, & Kamarch, 2008), and higher cortisol levels (Steptoe, Cropley, Griffith, & Kirschbaum, 2000). Furthermore, a number of studies have demonstrated that anger and cynical hostility predict the progression of subclinical atherosclerosis (Matthews et al., 1998; Raikkonen, Matthews, Sutton-Tyrrell, & Kuller, 2004), suggesting that the associations between anger/hostility and CHD may be due to the impact of anger and hostility on the development of coronary atherosclerosis, although acute trigger effects may also contribute (Dimsdale, 2008; Mittleman et al., 1995; Möller et al., 1999).

Taken together, given a recent meta-analysis on randomized controlled trials endorsing the efficacy of psychological interventions in cardiac patients (Linden, Phillips, & Leclerc, 2007), clinical trials focusing on anger and hostility in CHD interventions are necessary for the more effective prevention and treatment of CHD.

Cross-References

- ▶ Aerobic Exercise
- ▶ Anger, Measurement
- ▶ Coronary Event
- ▶ Coronary Heart Disease
- ▶ Hostility, Measurement of

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Heart Doctor

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Heart Failure

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Synonyms

Congestive heart failure

Definition

Heart failure is a general term used to describe a clinical syndrome characterized by shortness of breath, exertional dyspnea, paroxysmal nocturnal dyspnea, orthopnea, peripheral and/or pulmonary edema, and exercise intolerance. Since individual experiences of these clinical indicators may vary, it is important to understand how heart failure is classified. The underlying etiology and associated pathophysiology are keys to appropriate diagnosis and management.

Several categories are used to describe, organize, and classify heart failure. Acute versus chronic heart failure are terms used to describe both the onset and intensity of symptoms. Acute heart failure refers to the sudden appearance of symptoms (e.g., usually over hours or days), which have progressed to a point at which immediate or emergency medical intervention is necessary. Chronic heart failure refers to the development of symptoms over a period (e.g., months to years). Chronic symptoms represent the baseline condition or symptoms that an individual lives with on a daily basis. If the cause of the acute onset of heart failure is not reversible (e.g., left ventricular damage from a myocardial

infarction), then the heart failure may become chronic. Other classification categories include left versus right-sided heart failure.

Right-sided heart failure refers to failure of the right ventricle to pump adequately, and is most commonly caused by left-sided heart failure. It can also be caused by pulmonary disease and primary pulmonary hypertension. Left-sided heart failure refers to failure of the left ventricle to fill or empty properly, which leads to increased pressures inside the ventricle and congestion in the pulmonary vascular system. Left-sided heart failure may be further classified into systolic and diastolic dysfunction.

Systolic dysfunction is usually estimated by ejection fraction, or the percentage of the left ventricular end-diastolic volume (LVEDV) that is ejected from the ventricle in one cycle. For example, if the LVEDV is 100 mL and the stroke volume is 70 mL, the ejection fraction is 70%. Normal ejection fraction is 50–70%. Systolic dysfunction is defined as an ejection fraction of less than 40% and is caused by a decrease in heart contractility. Diastolic dysfunction, which is more common among older adults, is less well defined and more difficult to measure (i.e., left ventricular function is preserved). Although heart contractility is normal or even increased (e.g., normal or even high ejection fractions), diastolic dysfunction is caused by impaired relaxation and/or filling (e.g., fast heart rate, stiff or poorly compliant ventricle, rhythm that is poorly organized).

Diagnosis and management of heart failure begins with a clinical history and physical examination, and confirmatory evidence of cardiac dysfunction on an echocardiogram. Once a diagnosis has been made, it is important to characterize the degree of impairment using the standardized New York Heart Association (NYHA) Functional Classification. The American College of Cardiology (ACC) and the American Heart Association Guidelines (AHA) outline four stages of heart failure that are useful for organizing the prevention, diagnosis, management, and prognosis for patients with heart failure; only stages C and D are applicable to the NYHA functional classification system.

Cross-References

- ▶ Dyspnea
- ▶ Heart
- ▶ Systolic Blood Pressure (SBP)

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Heart Patients

- ▶ Williams LifeSkills Program

Heart Rate

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Synonyms

Pulse rate

Definition

Heart rate is the number of beats of the heart per unit of time. Typically it is measured in beats per minute (bpm). Heart rate is based on the time interval between one R wave and the next. The R wave is the onset of ventricular depolarization. For further details, see Andreassi (2006).

Cross-References

▶ Psychophysiology: Theory and Methods

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Heart Rate Variability

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Synonyms

Autonomic; Parasympathetic; Sympathetic

Definition

Like many organs in the body, the heart is dually innervated. Although a wide range of physiologic factors determine cardiac functions such as heart rate (HR), the autonomic nervous system (ANS) is the most prominent. Resting cardiac autonomic balance favors energy conservation by way of parasympathetic dominance over sympathetic influences. In addition, the HR time series is characterized by beat-to-beat variability over a wide range, which also implicates vagal dominance as the sympathetic influence on the heart is too slow to produce beat-to-beat changes. There is an increasing interest in the study of this heart rate variability (HRV) among researchers from diverse fields. Low HRV is associated with increased risk of all-cause mortality and has been proposed as a marker for disease. In the following, I will briefly describe the nature and assessment of HRV.

Heart Rate Variability

The basic data for the calculation of all the measures of HRV is the sequence of time intervals between heart beats. This interbeat interval time series is used to calculate the variability in the timing of the heart beat. Relative sympathetic increases cause the time between heart beats (the interbeat interval) to become shorter and relative parasympathetic increases cause the interbeat interval to become longer. The parasympathetic influences are pervasive over the frequency range of the heart rate power spectrum whereas the sympathetic influences "roll-off" at about 0.15 Hz. Therefore high-frequency HRV represents primarily parasympathetic influences with lower frequencies (below about 0.15 Hz) having a mixture of sympathetic and parasympathetic autonomic influences. The differential effects of the ANS on the sinoatrial node, and thus the timing of the heart beats, are due to the differential effects of the neurotransmitters for the sympathetic (noradrenaline) and parasympathetic (acetylcholine) nervous systems. The sympathetic effects are slow, on the timescale of seconds, whereas the parasympathetic effects are fast, on the timescale of milliseconds. Therefore the parasympathetic influences are the only ones capable of producing rapid changes in the beat-to-beat timing of the heart.

Measures of HRV

A variety of measures have been used to operationalize HRV. Long-term measures like the standard deviation of all interbeat intervals in 24 h, short-term measures like the standard deviation of 5 min intervals and beat-to-beat measures like the root mean square of successive RR differences (RMSSD) have all been used. Power spectral analysis of interbeat interval time series is frequently used to quantify HRV. The power spectrum of short-term time series contains two major components, a high-frequency

(0.15–0.40 Hz) and low-frequency (0.01–0.15 Hz) component reflecting cardiac vagal influences and a mixture of vagal and sympathetic influences, respectively. RMSSD and the high-frequency component of the power spectrum are closely related and reflect vagal cardiac influence. More recently, measures derived from nonlinear dynamics have been used to describe aspects of HRV. One such measure is approximate entropy (ApEn). It quantifies the complexity or irregularity of time series data.

Cross-References

▶ Autonomic Balance

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Heart Rate Variability (HRV)

▶ Respiratory Sinus Arrhythmia

Heavy Episodic Drinking

▶ Binge Drinking

Height

▶ Body Mass Index

Helplessness

▶ External Locus of Control
▶ Passive Coping Strategies

Hematopoietic Stem Cell Transplantation

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Description

In general, hematological malignancies show good response to antineoplastic agents. However, less than half of patients with hematological malignancies can be cured with chemotherapy alone. Since some antineoplastic agents show linear dose–response relationship, increasing the dose of chemotherapy may increase the antineoplastic effects, but it is precluded by the adverse effects of antineoplastic agents, so-called dose-limiting toxicities. The dose-limiting toxicity of many antineoplastic agents is myelosuppression. Therefore, hematopoietic stem cell transplantation has been investigated to allow high-dose chemotherapy over the maximum tolerated dose by supporting the hematopoietic system with the infusion of hematopoietic stem cells from a donor (allogeneic transplantation) or the patients themselves (autologous transplantation). In addition to the effect of the high-dose chemotherapy, an immunological antineoplastic effect of donor cells, so-called graft-versus-leukemia/lymphoma effect, can be harnessed after allogeneic transplantation. Currently, the application of hematopoietic stem cell transplantation is extended to solid tumors and nonmalignant hematological disorders such as aplastic anemia.

Previously, bone marrow was the sole source of hematopoietic stem cells. However, in the 1990s, the peripheral blood stem cells, which were mobilized by the use of granulocyte-colony stimulating factor alone or in combination with chemotherapy in autologous transplantation, became widely used as a source of hematopoietic stem cells, since the hematopoietic recovery is faster after peripheral blood stem cell transplantation than that after bone marrow transplantation. In addition, cord blood cells are currently the third source of hematopoietic stem cells. A major drawback of cord blood stem cell transplantation is the longer duration between transplantation and hematopoietic recovery. However, cord blood units are readily accessible, since they are already frozen in cord blood banks and there is no need for time-consuming door coordination.

The procedure of hematopoietic stem cell transplantation starts with a conditioning regimen using high-dose chemotherapy with or without total body irradiation. The major object of the conditioning regimen is to eradicate malignant cells from the recipient. However, in allogeneic transplantation, conditioning regimens are also required to suppress host immune system to prevent rejection of donor hematopoietic cells. The latter is the sole object of the conditioning regimen in allogeneic transplantation for aplastic anemia. After the conditioning regimen, hematopoietic stem cells are infused to the recipients intravenously. In general, hematopoietic recovery is observed within 2–4 weeks. However, supportive managements to prevent or treat transplant-related complications are required at least several months (or much longer in allogeneic transplantation) after transplantation.

The major complications after allogeneic hematopoietic stem cell transplantation include toxicities directory due to the conditioning regimen, infection, and graft-versus-host disease (GVHD). Increasing the dose of chemotherapy or total body irradiation over the maximum tolerated dose may induce toxicities that are not observed after standard-dose chemotherapy. For example, a fatal cardiac toxicity may develop after high-dose cyclophosphamide as a conditioning regimen. To extend the application of allogeneic transplantation to the elderly or clinically infirm patients who cannot tolerate these conditioning regimens, reduced-intensity or non-myeloablative stem cell transplantation has been investigated. Clinical studies of reduced-intensity or non-myeloablative transplantation have shown that the incidence of transplant-related morality was decreased, but the increase in relapse rate was the problem.

Hematopoietic stem cell transplantation recipients experience various infections. In the early phase after transplantation (day 0 to day 30), neutropenia is the primary immunologic defect. In addition, mucosal damage caused by the conditioning regimen adds the risk of infection. Flora of the skin, gastrointestinal tract, and mouth are the primary causes of infection during this phase. The second phase (day 30 to day 100), infectious complications due to *Cytomegalovirus*, *Aspergillus*, and *Pneumocystis jiroveci* are common due to the impaired cellular immunity. The development of acute or chronic GVHD is associated with severely impaired cellular immunity. In the later phase (after day 100), impaired cellular and humoral immunity is the major risk factor for infectious complications, especially in allogeneic transplant recipients with chronic GVHD. The causative organisms of infectious complications in this phase include Cytomegalovirus, Varicella-zoster virus, Epstein-Barr virus, *Aspergillus*, and *Pneumocystis jiroveci*.

GVHD is an immunological reaction of the donor immunological cells targeting host antigens. Clinical manifestations of acute GVHD include erythroderma, diarrhea, and jaundice. On the other hand, chronic GVHD resembles autoimmune disorders such as scleroderma, Sjögren's syndrome, primary biliary cirrhosis, bronchiolitis obliterans, and immune cytopenias. GVHD can be observed even after human leukocyte antigen (HLA) matched transplantation, but both its incidence and severity are increased by the presence of HLA-mismatch between the donor and the recipient. A combination of calcineurin inhibitors (cyclosporine or tacrolimus) and methotrexate is the mainstay of the pharmacological prevention of GVHD, whereas steroid is the first choice of the treatment of acute GVHD. The use of these immunosuppressive agents increases the risk of infectious complications.

In general, the incidence of transplant-related mortality is less than 5% after autologous transplantation, but it exceeds 10–20% after allogeneic transplantation, even from an HLA-matched donor. In addition, transplant-related complications, especially the development of chronic GVHD, affect the quality of life of survivors after allogeneic transplantation. Therefore, the indication of hematopoietic stem cell transplantation should be considered by balancing the risk and benefit obtained after transplantation. Many clinical trials have been performed to evaluate the indication of hematopoietic stem cell transplantation in a variety of hematological disorders. However, information is not yet sufficient to identify patients who will clearly benefit from undergoing hematopoietic stem cell transplantation.

Cross-References

▶ Stem Cells

Hemodynamic

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Definition

Hemodynamics is a general term referring to the movement or flow of blood. More specifically, this term refers to the measurement of and general principles governing the flow of blood in the human body. Movement or flow of blood throughout the body is dependent upon many factors. Two of the most important of these factors are the pressure differences and resistances between different elements of the circulatory system, such as the cardiac chambers, large arteries, arterioles, veins, and venules. Along with the heart rate (HR), pressure and resistance are major determinants of the amount of circulatory blood flow. Measurement of certain hemodynamic parameters, such blood pressure (BP) and heart rate (HR), can be determined noninvasively. Resistance is a calculated parameter determined by flow and pressure. For example, systemic vascular resistance is equal to the cardiac output divided by mean arterial

pressure. As will be described, cardiac output is determined by heart rate and stroke volume. These measures are important in understanding the pathophysiology of cardiovascular disease diagnosis and treatment. However, more detailed measures of cardiac structure, function, and intracardiac pressures are frequently necessary, and advanced measurement modalities are required.

Description

In the field of cardiology and cardiovascular medicine, hemodynamics are most commonly measured by two different modalities in the clinical setting: two-dimensional/Doppler echocardiography and cardiac catheterization. Each modality has limitations, and these two techniques are frequently used in concert. Importantly, absolute pressures can only be directly measured by cardiac catheterization. In contrast, absolute pressure cannot be measured by echocardiography; only pressure differences can be measured.

Echocardiography measures: Cardiac ultrasound, or echocardiography, uses ultrasonic beams through tissue. M-mode and two-dimensional (2D) echocardiography are used for structural imaging, while Doppler echocardiography uses ultrasound to record the movement of blood, enabling the assessment of hemodynamics and cardiac physiology. Each tissue has different properties that influence the amount of ultrasound beam that is reflected back to the ultrasound machine, generating a very small electrical current that can then be integrated into a composite image of the structure. Doppler ultrasound measures the difference between the scatter of ultrasound by different tissues and the frequency of the transmitted ultrasound beam. Doppler echocardiography is especially useful for the hemodynamic measurement of blood flow across stenotic valves of the heart and for measuring the pressure differences between different chambers. Assessment of blood flow is approximated by measurement of cardiac stroke volume. Stroke volume, in turn, is measured by taking the product of the velocity-time-integral (VTI, measured by Doppler echocardiography) and the cross-sectional area (CSA, measured with 2D echocardiography) between certain chambers. Cardiac output is then determined by the product of stroke volume and heart rate. The change in pressure between two cardiac chambers, a useful principle in cardiac hemodynamics, is frequently assessed using a modified form of the Bernoulli equation, which is itself a derivation of basic principles of conservation of energy in closed systems.

Catheterization measures: During catheterization, a special type of catheter is introduced into either the internal jugular vein in the neck or a femoral vein in the leg. The catheter is advanced into different chambers of the right (venous) side of the heart, the side responsible for collecting and pumping deoxygenated blood from the systemic circulation into the lungs. Typically, these catheters have an inflatable balloon built into the catheter. Inflation of the balloon in a pulmonary artery allows for the measurement of the pulmonary capillary "wedge" pressure (PCWP), which is, in most circumstances, a clinically useful approximation of intravascular fluid balance. To measure pressures in the left (arterial) side of the heart, a similar type of catheter and wire system is introduced into the right femoral artery and advanced retrograde up to and across the aortic valve into the left ventricle. Routine hemodynamic pressure measurements obtained from the right side include right atrial pressure, right ventricular pressure, pulmonary artery pressure, and PCWP. Left-sided pressure measurements include measurement of aortic pressure and left ventricular pressure. Cardiac output can be measured directly with cardiac catheterization using the thermodilution or Fick method. In the thermodilution method, cold saline is injected into the proximal port of the pulmonary artery catheter and mixes with body temperature blood in the right ventricle. As blood flows past the distal port, a small sensor records the change in temperature over time. The area under this curve is proportional to the flow in the pulmonary artery which, assuming there is no intracardiac shunt, is proportional to cardiac output. Stroke volume is determined by dividing this measured cardiac output by the heart rate.

The Fick method of cardiac output uses measures of oxygen consumption measured by either exhaled breath analysis or determined by an age, sex, height, and weight specific nomogram and is dependent on hemoglobin and levels of arterial oxygen saturation.

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Hemodynamic Response/Reactivity

► Blood Pressure Reactivity or Responses

Hemodynamic Stress Responses

► Blood Pressure Reactivity or Responses

Hemoglobin A1c

► HbA1c
► Glycosylated Hemoglobin

Hemoglobin, Glycosylated

► Glycosylated Hemoglobin

Hemostasis

► Coagulation of Blood

Hepatitis Types A, B, C

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Definition

Hepatitis involves inflammation of the liver and is most commonly caused by a number of viruses. Hepatitis can be an acute or a chronic condition. Hepatitis A is an acute viral infection of the liver due to the hepatitis A virus (HAV), and is the least serious of the hepatitis viruses. Hepatitis B and C are viral infections of the liver due to the hepatitis B virus (HBV) and the hepatitis C virus (HCV). Both hepatitis B and hepatitis C can become chronic infections and result in chronic liver disease.

Description

HAV is the main cause of acute viral hepatitis and is endemic worldwide. The virus is spread by the fecal-oral route, and very rarely by blood transfusion, and is often contracted from a contaminated water or food source. Major epidemics of hepatitis A are uncommon in the United States of America (USA), and most common in regions of the world with poor sanitary hygiene and overcrowding. The severity of the illness tends to increase with age. More than 80% of childhood cases of hepatitis A are asymptomatic, which encourages the spread of the virus. Adults with hepatitis A are more likely to have clinical symptoms such as jaundice (a yellowing of the skin and whites of the eyes). Symptoms tend to appear 2–6 weeks after exposure to HAV, and mild symptoms may last several months, particularly in adults. The virus does not remain in the body when the infection is gone. Vaccination for HAV is available, but there are no specific treatments for unvaccinated individuals who are exposed to the virus or develop the infection.

HBV is also found worldwide. It is spread parenterally and by sexual contact. Hepatitis B infection is most prevalent in sub-Saharan Africa, China, and Southeast Asia. Less than 1% of people in modern Europe, the USA, and Canada carry the virus, and in these areas, horizontal transmission occurs predominantly in younger individuals (ages 15–39) by sexual contact or intravenous drug use. Vertical transmission of the virus from mother to child and horizontal transmission from household members to unimmunized children occurs more frequently in high prevalence countries and helps maintain the virus in these areas. Most cases of hepatitis B are acute, but a small percentage of individuals will not clear the virus from their bodies and develop chronic infection. Hepatitis B infections are typically asymptomatic, especially in infancy through young adulthood, but early symptoms of acute hepatitis may occur such as jaundice, appetite loss, low-grade fever, and nausea and vomiting. Symptoms may not appear for up to 6 months after infection with HBV. Chronic infection with HBV can result in cirrhosis and hepatocellular carcinoma (HCC), also known as primary liver cancer. Up to 40% of individuals with chronic infection die from liver-related causes. A vaccination for HBV is available, and unvaccinated individuals exposed to the virus may be treated with Hepatitis B Immune Globulin. Those who develop chronic liver disease may need liver transplantation.

Like HAV and HBV, HCV is a worldwide endemic. It is primarily spread parenterally, while vertical and sexual transmissions of HCV are much less common than in HBV. Intravenous drug use is the most common cause of the virus. More than 80% of acute HCV cases lead to chronic infection, with long-term infection often leading to cirrhosis and HCC. As with hepatitis A and B, hepatitis C infection is usually asymptomatic, with the possible appearance of symptoms such as jaundice, dark urine, abdominal pain, and others. There is currently no vaccination for HCV. However, there are treatments such as antiviral medications that have a high likelihood of removing the virus from the blood or preventing the development of cirrhosis or liver cancer. Chronic hepatitis C with comorbid alcohol liver disease is the most common reason for liver transplantation in the USA.

Relevant to the field of Behavioral Medicine, the behavioral nature of numerous risk factors for exposure to viral hepatitis has led to research examining relevant psychosocial and behavioral predictors of viral hepatitis and its symptoms, as well as behavioral interventions for the prevention of viral hepatitis. Behavioral interventions researched include those targeting risk factors for viral hepatitis such as intravenous drug use and unsafe sex practices that could result in exposure to viral hepatitis.

Cross-References

- ▶ Infectious Diseases
- ▶ Inflammation
- ▶ Needle Exchange Programs
- ▶ Sexual Risk Behavior

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Hepatocyte Stimulating Factor

- ▶ Interleukins, -1 (IL-1), -6 (IL-6), -18 (IL-18)

Herbal Medicines

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Definition

Herbal medicine is the use of plants to treat human illnesses and debility. Parts used can be roots, bark, flowers, seed, leaves, or sometimes total aerial parts.

Description

Until early twentieth century, materials required for herbal medicine were collected in the countryside, dried by hanging in a warm room, and cut into small pieces to facilitate infusion in hot water, the resulting liquor being consumed as a dose. Mid-twentieth century saw the introduction of fluid extracts where 1 ml of finished liquid preparation represented 1 g of the starting material (dried herb). The industrial process to make these incurred considerable use of heat, very deleterious to fragile plant chemistry via hydrolysis, and/or oxidation. The last quarter of the century saw a substantial swing to the use of tinctures, aqueous-alcoholic preparations, made by steeping the dried herb in the liquor for a few weeks, cold, and pressing the residue to separate the liquid. Tinctures are typically 1:5, i.e., 1 g herb represented by 5 ml tincture. These products are very versatile for practitioner use, as the dose can be varied precisely from one prescription to the next, as the patient requires.

The herbal practitioner diagnoses and then writes his/her prescription. This will comprise a mixture of tinctures (and sometimes water) to make a 5-ml or 10-ml spoonful dose, using from 3 to about 25 per prescription. Experience teaches that there is a high degree of synergy accompanying the use of many together, with the advantage of an absolute minimum of adverse reactions as far less is used of each.

Some modern teachers have promoted the use of single plant extracts, standardized by assay of a single chemical constituent, dried to a powder, and formulated into tablets. This approach fails to appreciate the wide diversity of constituents in a plant (often several hundred), *all* of which work together therapeutically.

Significant behavior modifiers include hops (*Humulus lupulus*), scullcap (*Scutellaria lateriflora*), valerian (*Valeriana officinalis*), kava (*Piper methysticum*), and vervain (*Verbena officinalis*), as sedatives and hypnotics; St. John's wort (*Hypericum perforatum*) and Rhodiola rosea as antidepressants; and the ginsengs, Chinese or Korean (*Panax ginseng*) and Siberian (*Eleutherococcus senticosus*). The latter group acts as stamina providers, called adaptogens, because of their ability to improve the body's response to stress. Most of the named plants have added support from orthodox clinical trials. There are other less-studied herbs also in this area of therapeutics.

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Heritability

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Definition

Heritability, or h^2, is the fraction of the total variation in a phenotype that can be explained by genetic factors. Heritability is not about cause.

Description

Interpretation

Heritability has values between 0 and 1. A heritability of 0 would be a trait where none of

the phenotypic variance is due to genetic factors, whereas a heritability of 1 would be a trait where all of the variance can be explained by genetic factors.

Heritability is based on measuring the correlations of phenotypes within families. Families share more than their genes; they also share environments. Therefore, the correlations may not simply reflect their shared genetic profiles but also environmental (behavioral) exposures. Heritability can also be affected by other factors such as the population (ethnicity, age, gender, location, etc.) from which it was measured (measurement error) and their environmental exposures.

Calculations

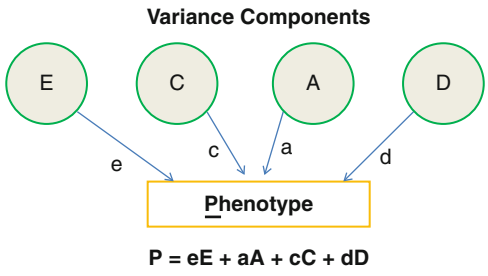
Twin studies are commonly used to measure heritability, as well as family studies. In the classic twin model, heritability is calculated by:

$$h^2 = 2(r_{mz} - r_{dz}),$$

which is the difference between intraclass correlations of a quantitative trait between monozygotic twins and dizygotic twins.

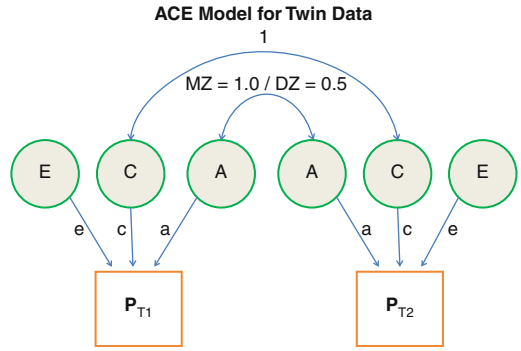
Intraclass correlations are subject to low power and large standard errors, necessitating the development of newer methods which take advantage of variances and covariances.

Path analysis or structural equation modeling estimates the genetic and environmental parameters that give the best fit of the variances and covariances observed in twins or families.



where E is the unique environment of each twin, C is the shared environment within the twin pairs, A is the additive genetic effects, and D is the dominant genetic effects.

Path analysis, or the ACE model, allows a diagrammatic representation between the linear models of the relationships between variables.



where MZ is monozygotic twins that share 100% of their genomes, and DZ is dizygotic twins that share 50% of their genomes, on average. It is assumed that both of the twins in a pair share 100% of their shared environment.

The ACE model can parse the variance of a trait into additive genetic, shared environmental, and non-shared environmental influences on the basis of the trait covariance observed among both monozygotic and dizygotic twin pairs. This model can accommodate hypothesis testing, both continuous and categorical variables, multiple variables, and more complex pedigrees or questions.

Cross-References

- ▶ Dizygotic Twins
- ▶ Monozygotic Twins

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Herpes Simplex Virus (HSV) Infection

▶ Genital Herpes

Heterogeneity

▶ Diversity

Heterozygous

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Definition

Humans have two alleles on homologous chromosomes for each gene or trait, one from their mother and one from their father. If the allele inherited from the mother is different from the allele inherited from the father, then the individual is heterozygous for that trait or gene. Alleles are variants of a gene and can also lead to a differential manifestation of the trait or disease they codify. Recessive alleles are not observed in the phenotype of heterozygous individuals.

The degree of heterozygosity (proportion of genes that are heterozygous by individual and by population) is used as a measure of genetic diversity in populations. The higher the mean heterozygosity is across the genome and across individuals in one population, the higher the genetic diversity.

Cross-References

▶ Allele
▶ Dominant Inheritance
▶ Gene
▶ Genotype
▶ Homozygous
▶ Recessive Inheritance

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Hidden Variable

▶ Latent Variable

Hierarchical Linear Modeling (HLM)

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Synonyms

Covariance components model; Linear mixed-effects model; Multi-level analysis; Random-coefficient model

Definition

Hierarchical linear modeling (HLM) is a particular regression model that is designed to take into account the hierarchical or nested structure of the data. HLM is also known as multi-level modeling, linear mixed-effects model, or covariance components model (Leyland & Goldstein, 2001).

Description

HLM has historically been used in educational research where hierarchies occur naturally: students nested within classrooms, classrooms nested within schools, and schools nested within districts (Sullivan, Dukes, & Losina, 1999). Recent advances in statistical computing capabilities have made this model more available to researchers across a variety of disciplines. For example, in organizational psychology research, data from individuals must often be nested within teams or other functional units. For repeated measures or longitudinal data, time can be considered as another level which occurs within subjects (Fitzmaurice, Laird, & Ware, 2004). For assessing differences in mortality rates across hospitals relative to a specific condition or procedure, data are collected on random samples of patients nested within each hospital. In this application, it might be appropriate to adjust for covariates at both the patient level (such as patient age, patient gender, and the severity of the disease) and at the hospital level (such as hospital size and hospital teaching status) (Austin, Yu, & Alter, 2003). These hierarchical data structures are often seen in many medical research applications.

HLM is a more advanced form of traditional linear regression models which were developed making certain assumptions about the nature of the dependency structure among the observed responses. For example, in a simple linear regression model, $y_i = b_0 + b_1 x_i + e_i$, where y_i is the response for individual i; the standard assumption is that the y_i given the covariate x_i is independently identically distributed, that is, the error term e_i is independent among individuals. In many real-life situations, however, one has data structures, whether observed or by design, for which this assumption does not hold.

Suppose, for example, that the response variable is the birth weight of a baby and the predictor is maternal age and data are collected from a large number of maternity units located in different physical and social environments. One would expect that the maternity units would have different mean birth weights so that knowledge of the maternity unit already conveys some information about the baby. A more suitable model for these data is

$$y_{ij} = b_0 + b_1 x_{ij} + u_j + e_{ij},$$

where another subscript j was added to identify the maternity unit and a unit-specific effect u_j was included to account for mean differences among units. If one assumes that the maternity units are randomly sampled from a population of units, then the unit-specific effect is a random variable and the above model becomes a simple example of a two-level model. Its complete specification, assuming normality for random variables, can be written as follows:

$$y_{ij} = b_0 + b_1 x_{ij} + u_j + e_{ij},$$

$$u_j \sim \mathrm{N}\left(0, {s_u}^2\right),\ e_{ij} \sim \mathrm{N}\left(0, {s_e}^2\right),$$

$$\mathrm{cov}\left(u_j, e_{ij}\right) = 0,$$

where ${s_u}^2$ represents the degree of heterogeneity among maternity units (between-unit variance) and ${s_e}^2$ is the pure random error variance (within-unit variance). In this model, the correlation among birth weights in the same unit can be written as ${s_u}^2/({s_u}^2 + {s_e}^2)$, which is known as an intra-cluster correlation. This lack of independence, arising from two sources of variation at different levels of the data hierarchy (births and maternity units), contradicts the traditional linear model assumption, and an HLM is a suitable regression model for this situation.

The above most simple HLM can be elaborated in a number of directions, including the addition of further covariates (both births and units levels) or levels of nesting. An important direction is where the coefficient of covariates is also allowed to have a random distribution, for example, the age relationship can vary across units. This is particularly important for assessing the covariate-by-unit interaction. The regression coefficients b are usually referred to as fixed effects parameter, and the unit-specific effects u_j are referred to as random effects parameter, and the above model is often referred to as a mixed-effects model or random coefficient model.

The parameters of fixed effects and variance components (s_u^2 and s_e^2) in HLM are obtained via a restricted maximum likelihood (REML) estimation method (Cnaan, Laird, & Slasor, 1997; Leyland & Goldstein, 2001). The random effects are estimated using shrinkage estimators, which is referred to as an empirical Bayes estimator or a best linear unbiased predictor (Fitzmaurice et al., 2004). This estimator is essentially an optimally weighted linear combination of the estimated overall mean and unit-level mean. The degree of shrinkage toward the overall means depends on the magnitude of the relative magnitude of s_u^2 and s_e^2. When s_e^2 is relatively large and the within-unit variability is greater than the between-unit variability, more weight is assigned to overall mean. On the other hand, when the between-unit variability is large relative to the within-unit variability, more weight is given to the unit-specific observed response.

There are two major specialized software packages for conducting HLM analysis, one is MLwiN and the other is HLM. MLwiN (version 2.21) can be found via the links provided on the Centre for Multilevel Modelling (CMM), which is a research center based at the University of Bristol, with Internet address http://www.cmm.bristol.ac.uk/. HLM (version 6.08) is available from Scientific Software International, Inc., where HLM has its homepage with Internet address http://www.ssicentral.com/hlm/index.html. HLM analysis can also be conducted using general purpose packages such as SAS or SPSS. SAS (version 9.2) has full-fledged possibilities for HLM analysis, using PROC MIXED or PROC GLIMMIX.

Cross-References

► Multilevel Modeling

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Hierarchy of Evidence

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Synonyms

Evidence hierarchy

Definition

The concept of the "hierarchy of evidence" refers to a tabular representation (sometimes presented as a pyramid) of the relative strengths of various investigational methodologies in providing the evidence that is used in evidence-based medicine and evidence-based behavioral medicine.

Byar (cited by Piantadosi, 2005) listed different types of medical studies in terms of the (increasing) strength of the evidence provided by them: case report, case series, database analysis, observational study, controlled clinical trial, and replicated clinical trials (independent verification of treatment efficacy estimates), referring to this progression as a "hierarchy of strength of evidence."

While the concept is simple, there can be disagreement on the order of some methodologies,

particularly toward the top of the pyramid. Randomized controlled clinical trials have typically been placed at the top, and many researchers still agree with this positioning. Others regard systematic reviews and meta-analyses as providing stronger evidence since they combine information garnered from more than one study (although many authors note the considerable care that is necessary when publishing a systematic review or meta-analysis). One such order, therefore, might be:

- Systematic reviews and meta-analyses
- Randomized controlled trials
- Cohort studies
- Case-control studies
- Cross-sectional surveys
- Case reports
- Expert opinions

More detailed accounts of several of these methodologies (listed in the "Cross-References" section) can be found in the encyclopedia.

Cross-References

▶ Case-Control Studies
▶ Meta-Analysis
▶ Randomized Clinical Trial

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High Blood Pressure

▶ Blood Pressure, Elevated
▶ Hypertension

High Blood Pressure Medications

▶ Antihypertensive Medications

High Cholesterol

▶ Dyslipidemia

High-Risk Drinking

▶ Binge Drinking

HIPAA

▶ Confidentiality

Hispanic Community Health Study/Study of Latinos

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Synonyms

Epidemiological studies; Ethnicity

Description

Behavioral, psychosocial, and sociocultural factors pose adverse cardiovascular disease (CVD) risks similar in magnitude to those caused by traditional risk factors such as smoking, high cholesterol, or hypertension. Little research has examined the risk and protective factors that contribute to CVD outcome of Hispanics/Latinos in the United States or that may lead to differential effects across subpopulations of Hispanics/Latinos. Consequently, the Hispanic Community Health Study/Study of Latinos (SOL), sponsored by the National Institutes of Health, was initiated to provide the largest, comprehensive, multicenter, community-based, longitudinal cohort study

of Hispanic/Latino health ever conducted in the continental United States. Objectives of the study include: (a) characterizing the health status and disease burden of the largest minority population in the United States; (b) describing the positive and negative consequences of their immigration and acculturation to the mainstream United States in relation to lifestyle, environmental factors, and access to health care; and (c) identifying likely causal factors of disease in this diverse population.

By June 2011, 16,000 men and women, 18–74 years of age, who self-identified as being Hispanic or Latino completed a 6.5 h baseline clinical exam. The participants were recruited from a stratified random sample of households in defined communities in the Bronx, Chicago, Miami, and San Diego. These communities were chosen so that the overall sample would include adequate representation from Central and South American, Cuban, Mexican, Puerto Rican, and Dominican ancestral backgrounds.

The study assessed risk factors for and prevalence of heart, lung, blood, and sleep disorders, kidney and liver dysfunction, diabetes, cognitive impairment, dental problems, and hearing disorders. Among the physical exam procedures employed were electrocardiogram, blood pressure, comparison of ankle and arm blood pressures to detect evidence of peripheral artery disease, evaluation of pulmonary function, physical activity assessed by activity monitors worn for a week by participants, and disordered breathing overnight to evaluate sleep interruption due to sleep apnea. Questionnaires assessed health histories of participants and their families, information about acculturation, social variables, education, occupation, smoking, nutrition, alcohol consumption, sleep, physical activity, and prescription and nonprescription drug use.

During annual follow-up phone calls, deaths, hospitalizations, and emergency department visits that occurred since the baseline examination are identified and followed up by examining hospital charts and death certificates. These records include documentation of acute myocardial infarction, heart failure, resuscitated cardiac arrest, cardiac revascularization, stroke, transient ischemia attacks, and asthma.

In general, SOL is designed to inform health care providers, the public health community, and the Hispanic/Latino population about the prevalence of impaired health in that population, the likely causes of such impairments as well as the measures needed to improve Hispanic/Latino health in the United States. In addition to the SOL parent study, the NIH has also sponsored ancillary studies that allow expansion of the major objectives of SOL. Thus, for example, an ancillary sociocultural study based on 5,280 of the original SOL participants is examining the influence of such factors as socioeconomic status (e.g., effects of cumulative deprivation during the lifespan), sociocultural factors (e.g., ethnic identity, fatalism), psychosocial risk (e.g., stress, negative emotions), and protective factors (e.g., social resources, family cohesion). In summary, SOL is providing the most comprehensive study of Hispanic/Latino health that has ever been conducted in the continental United States.

Cross-References

- Cardiovascular Disease
- Cardiovascular Disease Prevention
- Cultural Factors
- Epidemiology
- Ethnicity
- Health Disparities
- Hispanic/Latino Health

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Hispanic Health

▶ Hispanic/Latino Health

Hispanic/Latino Health

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Synonyms

Hispanic health

Definition

The terms *Hispanic* and *Latino/a* are used to collectively refer to people from Mexico, Central and South America, Spain, and Spanish-speaking or influenced countries. According to the 2010 Census, Latinos comprised 16.3% of the total US population, making them the largest racial/ethnic minority group in the USA (Pew Hispanic Center, 2011). Like other minorities, Latinos have a significant health risk factor profile marked by educational, economic, and disease challenges. Despite these disparities, Latino's appear to live longer than non-Hispanic Whites, an epidemiological phenomenon commonly referred to as the *Hispanic or Latino Mortality Paradox*.

Description

Background

Defining Latinos in the USA

Latinos in the USA are a heterogeneous group representing more than 23 nationalities and speaking at least 12 languages and dialects (Pew Hispanic Center, 2010). Approximately two-thirds (66%) of US Latinos are of Mexican descent followed by Puerto Ricans (8.9%), Cubans (3.5%), Salvadorans (3.3%), and Dominicans (2.8%). Latinos are largely defined and differentiated from other groups by cultural factors. In particular, Latino culture is *collectivistic*, meaning they place an emphasis on the needs of the group over those of the individual. Several specific values including the valuing family (*familismo)* valuing and warm interpersonal relationships (*personalismo)* and valuing interpersonal harmony (*simpatico*) facilitate strong social cohesion (Zea, Quezada, & Belgrave, 1994).

Demographics

Latinos account for approximately 16.3% of the total US population, accounting for 56% of the nation's population growth over the last decade (Pew Hispanic Center, 2011). By the year 2050, the Latino population is projected to triple in size to approximately 29% of the US population. Of the 50.5 million Latinos currently residing in the USA, an estimated 38% are foreign-born (Pew Hispanic Center, 2010). Importantly, foreign-born should not be equated with illegal immigration as unauthorized immigrants account for only 4% of the US population and, thus, a small percentage of the Latino community. Rather, native births are by far the largest source of growth in the US Latino population. Nearly one in five children in the United States is of Latino descent with approximately 89% of those children born in the USA, making them citizens by birth (Fry & Passel, 2009). It is estimated that by the year 2025, 30% of all children born in the USA will be of Latino descent.

The demographic profile of US Latinos differs significantly from other racial/ethnic groups. For example, Latinos are by far the youngest group with a median age of 27 years compared to 41 years for non-Hispanic Whites, 32 years for non-Hispanic Blacks, 36 years for non-Hispanic Asians, and a median age of 36 years for the total US population (Pew Hispanic Center, 2010). Latinos are less likely to have ever been married compared to non-Hispanic Whites (66.3% vs. 76.7%) but more likely to live in a "household" or family unit (81% vs. 69%). Latino households tend to be larger than non-Hispanic White households, and US-born Latinos are more likely to

live with family members than non-Hispanics of any race (Pew Hispanic Center, 2010).

Socioeconomic Profile

Socioeconomic status (SES) is among the most robust psychosocial modifiers of physical health and mortality. Similar to other racial/ethnic minorities, Latinos experience significant SES disparities relative to non-Hispanic Whites and the US population in general. For example, nearly one in five (19.5%) Latinos is classified as living in poverty, a rate comparable to the percentage of non-Hispanic Blacks (21.9%) and significantly higher than non-Hispanic Whites (8.2%; Pew Hispanic Center, 2010). Latinos have the lowest median income for full-time, year-round work relative to other racial/ethnic groups, which may be due in part to their overrepresentation in manual labor jobs and underrepresentation in management and technology (U.S. Bureau of Labor Statistics, 2009).

Education is an important determinant of future earning potential and is an integral part of the conceptualization of SES. Latinos are at a significant educational disadvantage relative to other racial/ethnic groups including other minorities. For example, nearly 40% of Latinos have less than a high school diploma, a rate that is twice that of any other racial/ethnic group and markedly higher than the national average of 15.1%. In addition, the college graduation rate for Latinos is just 12.9% compared to 17.7% of non-Hispanic Blacks, 30.7% of non-Hispanic Whites, and the national average of 27.7%. Together with the economic disparities, Latinos are at or near the bottom of most of the major SES indicators – a profile associated with significant health risk.

Latino Health Status

Perceived Health

Latinos' self-reported health ratings are typically lower than those from non-Hispanic Whites (Perez-Stable, Napoles-Springer, & Miramontes, 1997). For example, in a large survey of California residents, Latinos were less likely than non-Hispanic Whites to rate their health as "excellent" or "very good" (50.2% vs. 30.2%) and more likely to rate their health as "fair" (9.9% vs. 27.5%). This finding is consistent with the challenges of lower SES, although some data suggests that SES does not explain the entire effect.

Diabetes Diabetes is amongst the most common chronic diseases in the USA with an incidence rate of approximately 8.3% and rising (Center for Disease Control [CDC], 2011). Racial and ethnic minorities suffer a disproportionate burden of the diabetes epidemic (CDC, 2011). In general, Latinos are nearly 1.7 times as likely to have diabetes as compared to non-Hispanic Whites (11.8% vs. 7.1%) and are at 50% greater risk of dying from diabetes-related complications. There is considerable heterogeneity in diabetes risk among Latino subgroups with persons of Puerto Rican and Mexican descent twice as likely to have diabetes compared to non-Hispanic Whites (13.8% and 13.3%, respectively) whereas individuals of Cuban descent experience prevalence rates more similar to non-Hispanic Whites (7.6%). End-stage renal disease (ESRD) is a major complication arising from diabetes. Latinos are nearly 1.7 times more likely to initiate ESRD treatment compared to non-Hispanic Whites.

Cardiovascular Disease Cardiovascular disease (CVD) is the leading cause of death in the United States, accounting for approximately 34.3% of all deaths annually (American Heart Association, 2010). Race-related disparities in CVD incidence and mortality are well documented. The CDC and the Office of Minority Health report that African Americans are 1.5 times more likely to have hypertension (high blood pressure) and 1.3 times more likely to die from heart disease compared to Whites. Although far less Latino comparative health data is available, there are important differences to consider. Latinos are more likely to be overweight and have uncontrolled diabetes, two robust risk factors for heart disease. However, the age-adjusted mortality rate from heart disease is significantly lower for Latinos compared to non-Hispanic Whites (165.0 vs. 239.8 per 100,000). Emerging

data suggests that although the incidence rate of heart disease is similar between Latinos and non-Hispanic Whites, the rate at which the disease progresses to critical endpoints, such as heart attacks, is significantly slower for less acculturated Latinos.

Cancer Overall cancer incidence is approximately 30% lower for Latinos relative to non-Hispanic Whites (U.S. Cancer Statistics Working Group, 2011). This advantage is evident among both men and women although there is heterogeneity by cancer type. For example, Latina women are at 33% lower risk for breast cancer and Latino men are 23% less likely to have prostate cancer relative to their non-Hispanic White counterparts. However, Latinos are twice as likely to develop stomach and liver cancer relative to non-Hispanic Whites. In addition, Latina women have an 80% greater risk of being diagnosed with cervical cancer than non-Hispanic women. Unfortunately, the relative survival rate of Latinos following diagnosis is unclear due to a lack of available data. What is known is that relative mortality rates mirror incidence rates with Latinos more likely to die from stomach and liver cancers compared to non-Hispanic Whites.

Infectious Conditions Latinos suffer disproportionate rates of a range of infectious conditions. For example, Latinos account for approximately 75% of newly diagnosed tuberculosis cases and are at twice the risk of hepatitis A, twice the risk for gonorrhea and syphilis, and three times the risk for chlamydia compared to non-Hispanic Whites (CDC, 2009a). With respect to HIV/AIDS, Latinos accounted for approximately 17% of all HIV/AIDS cases despite accounting for less than 15% of the US population in 2008 (CDC, 2009b). Comparatively, Latinos are 3 times as likely to be diagnosed with HIV/AIDS relative to non-Hispanic Whites. Although disparities exist for both sexes, Latina women are 5 times more likely to be diagnosed with HIV compared to non-Hispanic White women. The AIDS-related mortality rate for Latino men is approximately 2.5 times, and for Latina women, 3.6 times the rate of non-Hispanic White men and women, respectively.

Despite their lower SES profile, infant/childhood immunization rates are comparable between Latinos and non-Hispanic Whites (CDC, 2009c). However, there are critical differences in the vulnerable 65 and older age category. At a time when risk increases, Latinos are 20% less likely to get the annual influenza (flu) vaccination and 40% less likely to be immunized for pneumonia (CDC, 2010). Such differences may increase vulnerability disparities in later life.

The Hispanic Mortality Paradox Substantial evidence suggests Latinos live longer than non-Hispanic Whites, an epidemiological paradox given the relative differences in SES and mortality risk factors. This phenomenon was first identified by Markides (1983) who noticed that among community samples in the Southwestern United States, Latino mortality rates were more similar to non-Hispanic Whites than to non-Hispanic Blacks despite substantial SES differences. Subsequent national cohort data consistently demonstrates a Latino mortality advantage relative to other racial/ethnic groups, including non-Hispanic Whites. For example, the 2006 CDC report on mortality by race/ethnicity indicates that the age-adjusted death rate for Latinos is approximately 27.4% lower than for non-Hispanic Whites and 43.7% lower than for non-Hispanic Blacks (Heron et al., 2009). Hence, the Hispanic mortality paradox refers to the epidemiological finding that Latinos appear to live longer than non-Hispanic Whites despite the lower SES of the former relative to the latter.

Multiple explanations have been postulated to explain these paradoxical findings. The most common criticism concerns data reliability. Critics argue that the national cohort data is highly suspect as it is based on representative statistics. For example, the Latino mortality rate is estimated by entering the annualized Latino mortality (determined by number of death certificates indicating Hispanic ethnicity in a given year) in the numerator and the corresponding Latino population size (determined via census estimates) into the denominator. The major

concern is that the numerator relies on accurate recording of Hispanic ethnicity on death certificates and that underreporting biases a more favorable ratio. However, recent data suggests that ethnic misclassification is rare and comparable for Latinos and non-Hispanics (Arias, Eschbach, Schauman, Backlund, & Sorlie, 2010). In addition, two general explanatory hypotheses, the *salmon bias hypothesis* (i.e., Latinos possibly returning to their ancestral country of origin prior to death) and the *healthy immigrant hypothesis* (i.e., biased Latino health in the USA due to better health among those who successfully migrate) have been largely debunked by available data.

An alternative to representative estimates is to examine longitudinal studies where individuals are tracked over time. A recent systematic review and meta-analysis of the longitudinal literature further supports the validity of the paradox (Ruiz, Steffen, & Smith, 2011). Across 58 identified studies involving over 4.6 million participants, Latinos were found to have a 17.5% lower risk of mortality compared to other racial groups. The advantage was most evident in initially healthy samples and in the context of cardiovascular disease. In conjunction with the consistent national cohort data, it may be time to turn the corner on questioning the validity of the paradox and direct efforts to identifying the mechanisms leading to these surprising outcomes.

It is important to note that a mortality advantage is not synonymous with better health. As the risk factor data illustrates, Latinos experience high rates of diabetes, infectious diseases, and several types of cancer. They are less likely to have access to regular medical care, and when they do seek care, it is often in more emergent conditions. Hence, Latinos may live longer but suffer a lower health-related quality of life.

Future Directions

The Hispanic mortality paradox illustrates the heterogeneity in minority health. Despite a risk profile similar to African American/Blacks, Hispanics have significantly different disease incidence, burden, and mortality outcomes. Such differences necessitate research specifically on the health and disease course of Latinos. It is possible that traditional risk factors simply are not the best predictors for disease and mortality among Latinos. It is also possible that Latinos may have resilience advantages at important time points in the disease course such as resistance to initial incidence, slower disease progression, or the ability or resources to more effectively recover from acute incidences. Additional research is also needed to understand the moderating and mediating factors specific to Latinos. Such studies may encompass the role of individual level factors (e.g., personality, experiences of stress), social (e.g., social network size and function) and cultural factors (e.g., cultural values, collectivism), as well as genetic/biological differences that may influence health outcomes. Finally, models are needed to understand the interplay between diverse factors and their impact on Latino health.

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▶ Health Disparities

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Histamine

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Synonyms

Substance H

Definition

Histamine is a biogenic amine which is involved in a number of biological processes including immune system, gastrointestinal, and nervous system functioning. Histamine is typically released in response to a pathogen or allergen and serves to increase capillary permeability allowing fluid and other immune system cells to leave the capillaries and enter tissues. The fluid will contain white blood cells which can attack and minimize the effect of the pathogen. In response to an allergen, histamine results in typical allergy symptoms of runny nose, congestion, sneezing, and watery eyes. Antihistamines are medications which block receptor sites for histamine and can reduce such allergy symptoms. Histamines are also thought to be implicated in memory, learning, and sleep regulation.

Cross-References

▶ Immune Function
▶ Immunity

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HIV Infection

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Synonyms

AIDS: Acquired immunodeficiency syndrome; Sexually transmitted disease/infection (STD/STI)

Definition

HIV infection can be characterized as a condition caused by the human immunodeficiency virus (HIV) (National Center for Biotechnical Information [NCBI], 2010a). HIV is a retrovirus that weakens the body's immune system by infecting and replicating in CD4 + T-cells (Morris & Cilliers, 2008). CD4 + T-cells stimulate lymphocytes that assist the body in attacking antigens and fighting off disease. When HIV infection occurs, the virus gains entry to host T-cells by attaching to CD4 receptors via glycoprotein (gp120). gp120 allows HIV to bind to CD4 receptors and kill off helper T-cells. During this process, HIV's genetic material integrates its viral DNA into the cell's host DNA and allows the virus to replicate in the immune system (Morris & Cilliers, 2008).

Description

HIV Infection: Prevalence

Since its co-discovery by Drs. Luc Montagnier and Robert Gallo in 1983, millions of people have been infected with HIV. Current prevalence estimates reveal that there are approximately 33.3 million people living with HIV worldwide (UNAIDS, 2010). HIV prevalence varies greatly by region: HIV disproportionately affects sub-Saharan Africa, a region that accounts for 68% of the global total of persons living with HIV (22.5 million); HIV prevalence has nearly tripled in Eastern Europe and Central Asia (1.4 million) and has risen in North America (1.5 million), Western and Central Europe (820,000), Oceania (57,000), and in the Middle East and North Africa (460,000); HIV prevalence remains relatively stable in South and Central America (1.4 million), the Caribbean (240,000), and Asia (4.9 million); however, there is considerable variation in HIV prevalence within these regions (UNAIDS, 2010).

HIV Infection: Incidence

Advances in HIV prevention have markedly reduced the number of new HIV infections. However, there are noticeable differences in HIV incidence trends worldwide, and incidence patterns vary within countries. Many new cases of HIV infection are concentrated among people who inject drugs, commercial sex workers, and men who have sex with men (UNAIDS, 2010). HIV incidence is particularly high in developing countries (Merson, 2006). However, countries with higher incomes are not immune from the HIV epidemic. In the United States, the epidemic disproportionately affects racial and ethnic minorities and men who have sex with men (MSM), especially in urban areas of the Northeast, West Coast cities, and small towns in the South (El-Sadr, Mayer, & Hodder, 2010). Higher HIV incidence in Western Europe can also be attributed to increases in risky sexual behavior among MSM. Concentrated epidemics, particularly among people using injection drugs and sex workers, contribute to the growing epidemic in Eastern Europe and Central Asia (UNAIDS, 2010). HIV incidence by region is as follows (UNAIDS, 2010):

- Sub-Saharan Africa: 1.8 million
- South and Southeast Asia: 270,000
- Eastern Europe and Central Asia: 130,000
- Central and South America: 92,000

- East Asia: 82,000
- Middle East and North Africa: 75,000
- North America: 70,000
- Western and Central Europe: 31,000
- Caribbean: 17,000
- Oceania: 4,500

Stages of HIV Infection

Within 2–4 weeks after HIV infection, individuals may experience acute retroviral syndrome (ARS) or acute HIV infection. Acute HIV infection can appear flu-like and may be accompanied by a number of symptoms such as sore throat, headache, fever, and swollen lymph glands (National Center for Biotechnical Information [NCBI], 2010b). It may take several months for individuals to exhibit ARS symptoms; however, many people never develop ARS or exhibit symptoms associated with the syndrome. During this phase, the virus is actively replicating and is highly transmissible (NCBI, 2010b). After primary HIV infection, the illness may be asymptomatic in which there are no major symptoms of HIV. Individuals vary in their detectable HIV viral load but can still transmit the virus to others. The asymptomatic stage can last up to 10 years. Over time, the body's immune system weakens from fighting the virus and transitions into symptomatic HIV infection. In this period, individuals exhibit symptoms typical of chronic HIV infection (e.g., diarrhea, fatigue, fever, weight loss). Acquired immunodeficiency syndrome (AIDS) is the final stage of HIV and is defined by <200 CD4 + T-lymphocyte cell counts per microliter of blood (CDC, 1993). The body's immune system is severely damaged and is susceptible to opportunistic infections (e.g., Kaposi's sarcoma). Individuals vary in their progression through these phases. Adherence to antiretroviral medications plays a vital role in suppressing HIV viral replication and the progression of AIDS. There are two types of HIV infection – HIV-1 and HIV-2: both types of HIV weaken the body's immune system; however, HIV-2 has a longer asymptomatic phase with slower and milder immunodeficiency and is predominately found in West Africa (CDC, 2010).

Two classification systems are currently used to assess the progression of HIV infection: the Centers for Disease Control (CDC) classification system and the World Health Organization (WHO) Clinical Staging and Disease Classification System.

The CDC classification system (CDC, 1993) categorizes the progression of HIV infection into three CD4 + T-lymphocyte categories and three clinical categories. These categories are accompanied by particular illnesses or conditions. The corresponding CD4 + T-lymphocyte categories are as follows:

1. Category 1: ≥500 cells/mL
2. Category 2: 200–499 cells/mL
3. Category 3: <200 cells/mL

These categories correspond to CD4 + T-lymphocyte counts per microliter of blood in which an acute HIV infection (≥500 cells/mL) progresses to acquired immunodeficiency syndrome (AIDS) (<200 cells/mL). The clinical categories of HIV infection and corresponding illnesses are as follows:

1. Category A (asymptomatic/acute HIV): Asymptomatic or acute retroviral syndrome/acute HIV infection (acute HIV infection occurs 2–4 weeks after HIV infection, and its symptoms include fatigue, fever, headache, sore throat, and decreased appetite).
2. Category B (symptomatic conditions): Conditions include, but are not limited to, cervical dysplasia, shingles, and pelvic inflammatory disease.
3. Category C (AIDS-indicator conditions): Presence of opportunistic infections such as Kaposi's sarcoma and *Mycobacterium tuberculosis*.

In settings with limited capability to test for CD4 viral loads, the WHO Clinical Staging and Disease Classification System (WHO, 2007) may be used to determine the progression of HIV infection and the appropriate antiretroviral therapy. This classification system is categorized into four stages ranging from primary HIV infection to AIDS-indicator illnesses. The stages of HIV infection and some accompanying illnesses are as follows:

1. Clinical stage 1 (asymptomatic): Asymptomatic, persistent generalized lymphadenopathy

2. Clinical stage 2 (mild symptoms): Unexplained weight loss, fungal nail infections
3. Clinical stage 3 (advanced symptoms): Unexplained severe weight loss, oral hairy leukoplakia
4. Clinical stage 4 (severe symptoms): HIV wasting syndrome, Kaposi's sarcoma

HIV Transmission

HIV is primarily spread through the following modes of transmission (CDC, 2010): unprotected sexual contact with a person who has HIV infection (unprotected oral sex is lower risk for transmitting HIV than vaginal or anal sex; unprotected anal sex is riskier than vaginal sex; unprotected receptive anal sex is riskier than unprotected insertive anal sex; having multiple sex partners also increases risk of HIV infection, especially in the presence of other sexually transmitted diseases during sex), sharing needles or syringes used to prepare illicit drugs for infection, and being born to an infected mother (HIV can be passed from mother to child through pregnancy, birth, or breast-feeding). Less common modes of HIV infection are through blood transfusions. These modes of HIV transmission occur by the transfer of semen, blood, vaginal fluid, or breast milk.

HIV Diagnosis

HIV is typically diagnosed by a set of blood tests (HIV ELISA) that detect the presence of HIV antibodies but can also be diagnosed by tests that identify HIV's genetic material (CDC, 2010). The presence of HIV antibodies marks the body's response to HIV infection. A "window period" between HIV infection and seroconversion (presence of detectable HIV antibodies) can range from 2 weeks to 3 months, in which an individual's immune system may not produce enough antibodies for the antibody test to detect. HIV is highly transmissible during this period even if the virus is not yet detectable. Most people will develop detectable antibodies within 2–8 weeks of HIV infection, and HIV test should be considered positive after a second confirmatory HIV test (CDC, 2010; Puren, 2008).

HIV Treatment

There is no cure for HIV infection. However, antiretroviral therapy can suppress HIV viral replication and the progression of AIDS. These medications help people living with HIV to lead longer and healthier lives. Inconsistent adherence allows HIV to acquire resistance to antiretroviral therapy. There are considerable barriers in HIV prevention and treatment, but global efforts continue to address these challenges.

Cross-References

▶ HIV Prevention
▶ Sexual Risk Behavior

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HIV Prevention

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Synonyms

AIDS prevention

Definition

HIV prevention is the science of designing, implementing, and evaluating the effectiveness of interventions, programs, and services that aim to prevent an individual from acquiring HIV.

Description

The acquired immune deficiency syndrome (AIDS) was first recognized in 1981 and is caused by the human immunodeficiency virus (HIV) (CDC, 2010). Since the 1980s, science has significantly increased our knowledge about HIV and AIDS. We now know how HIV is transmitted from one person to another, the physiological processes of how the virus replicates once acquired, which medications help stop the virus from replicating, why individuals are more susceptible to opportunistic infections, and how best to prevent HIV transmission between two individuals. As a result, the field of HIV prevention science has emerged to better understand how individuals can help protect themselves from acquiring HIV, and among those who are living with HIV, how best to improve their adherence to treatment and reduce the transmission of HIV to others.

Efforts to reduce new HIV cases are based on how the virus is transmitted. There are three primary modes of transmission, including sexual transmission, transmission through blood, and mother-to-child transmission (Avert, 2011). In detail, an individual may acquire HIV from infected donated blood and organ products, vertically from mother-to-child during labor and/or from feeding the infant breast milk, sexually from having unprotected anal or vaginal intercourse, and from sharing contaminated works when injecting drugs or being stuck with a contaminated needle. Accordingly, HIV prevention interventions, programs, and services are designed to target specific populations based on their primary risk factor(s) for acquiring HIV. For example, self-identified heterosexual individuals who may be more likely to have unprotected intercourse, otherwise known as "high-risk heterosexuals," would be targeted for a particular HIV prevention program that was specifically designed for them. Another HIV prevention program may target HIV-negative men who have sex with men (MSM) to help them sustain from having unprotected anal intercourse (UAI) with multiple sex partners of HIV-positive and/or unknown serostatus. As such, HIV prevention interventions, programs, and services are typically designed and implemented to reach certain populations that engage in behaviors that put them at risk for acquiring HIV.

HIV prevention also has evolved to include individuals living with HIV. For instance, people living with HIV may want help with adherence to their medications, finding appropriate doctors for their treatments, access to care, assistance with disclosing their HIV serostatus, or learning how to help protect others from acquiring HIV from them. In sum, HIV prevention interventions,

programs, and services include two broad types: (1) primary prevention which aims to prevent individuals from acquiring HIV and (2) secondary prevention which helps individuals living with HIV to maintain their health and to reduce the possibility of transmitting HIV onto others (Avert, 2011).

Moreover, the implementation of these interventions, programs, and services is further divided based on the context of the implementation and how many individuals will be targeted. Specifically, interventions are classified as individual-level, group-level, or community-level interventions. Each of these levels describes the population context of the intervention for the primary or secondary HIV prevention services that would be provided to the participants. For example, an individual-level, primary HIV prevention intervention would focus on reducing HIV risk for each individual who participates in that particular intervention.

Many interventions, programs, and services of HIV prevention include an aspect of psycho-behavioral modification and/or a biomedical approach. Several theories of behavior change and models have been developed or modified for the prevention of HIV. For example, many studies and interventions aimed at preventing HIV have used constructs from the health belief model (Rosenstock, Strecher, & Becker, 1994), social cognitive theory (Bandura, 1997), theory of reasoned action (Fishbein & Ajzen, 1975), theory of planned behavior (Ajzen, 1991), transtheoretical model (Prochaska, DiClemente, & Norcross, 1992), AIDS risk reduction model (Catania, Kegeles, & Coates, 1990), and the information-motivation-behavioral skills model (Fisher & Fisher, 1992). Constructs from these theories and models are used to help participants and their networks change their beliefs, attitudes, perceived ability, and norms to lower their risk for acquiring HIV. Scientists and other researchers also use these constructs to better understand individuals' thoughts, actions, and feelings about HIV and/or risk behaviors.

In contrast, the biomedical approach uses a "test and treat" approach that may or may not include a psycho-behavioral component to it. The biomedical approach emphasizes the importance of being tested for HIV. The frequency of being tested for HIV will depend on the individual needs, her or his risk, and access to receiving the HIV test. Because an individual's viral load is highest after initially being infected, the premise of the approach is to link HIV-positive individuals with care as soon as possible and, preferably, before the possibility of transmitting the virus to others. Regardless of which HIV prevention approach is used, only correct and consistent condom use for anal and vaginal intercourse, being in a long-term mutually monogamous relationship with an uninfected partner, or abstaining from anal and vaginal intercourse will significantly prevent an individual from contracting HIV (CDC, 2010).

In addition to testing, consistent condom use, and modifying behaviors to reduce HIV risk, new advances with treatments have led to the development of other promising HIV prevention methods. Current studies are assessing the efficacy and cost-effectiveness of administering a pill or inserting a microbicide (i.e., gel or foam) inside an anus or vagina as additional modes of preventing HIV transmission (AVAC, 2011). These new biomedical advances are still being studied in a variety of populations around the world.

The success of the psycho-behavioral and biomedical approaches to preventing primary and secondary HIV infections is not without debate. Evaluation of HIV prevention interventions is crucial for determining what works best in HIV prevention. For example, the US Centers for Disease Control and Prevention (2007) provides a list of interventions that have been shown to prevent HIV among a variety of at-risk populations. These interventions are called evidence-based interventions (EBIs). Further, new studies that examine the different contexts of HIV are constantly needed in order to address how the epidemic changes with time across the world. The Further Readings section provides additional information about HIV prevention.

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HIV Status

► Serostatus: Seronegative and Seropositive

HIV Wasting

► Cachexia (Wasting Syndrome)

HMG-CoA Reductase Inhibitors

► Statins

Holistic Medicine

► Integrative Medicine

Home Health Care

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Definition

Home health care is the rendering of predominantly medically related services to patients in a home setting rather than in a medical facility. The goals of home health care include helping patients recover from an injury or illness and restoring, maintaining, or increasing their ability to tend to their everyday needs at home.

Description

There are a wide range of services that fall under the definition of home health care. These include more skilled care often focused on a particular condition such as wound care for pressure ulcers or surgical wounds, physical and occupational therapy, speech-language therapy, patient and caregiver education such as for daily insulin injections, other intravenous injections, and monitoring serious or difficult to control chronic illness such as diabetes and congestive heart failure. However, it may also include a broader range of services such as social services or assistance from a home health aide. Examples of home health aide services include help with basic daily activities like getting in and out of bed, dressing, bathing, eating, and using the bathroom, as well as help with light housekeeping, laundry, shopping, and cooking for the patient.

In addition to such formalized services, the term can also include informal health-care

services provided by spouses, family, friends, and neighbors often similar in scope to those done by paid home health aides. In some European countries, such informal care is partially funded, while in most other countries, it is typically unreimbursed. One estimate from the USA is that one of every five adults in the United States provides some sort of such informal home care to someone else.

Historically, most health care was traditionally provided in a patient's home. For centuries, midwives, medicine men, and traveling doctors all performed their services in a patient's home. Sometimes, sick people went to a doctor's office, which was usually an extension of the provider's home or a small clinic, but more often than not, the health-care specialists visited the sick in their home. It was only at the start of the twentieth century with the advancement in medical knowledge, treatments, and technology, not feasible to be provided in the home setting, that led to movement of health service delivery for very sick patients to a hospital setting. Of note, in many countries, particularly those with fewer resources where hospital growth and development has been more limited, many very sick patients are still not able to receive hospital-based care and remain in their home, often cared, to the extent possible, by family and friends.

In the latter half the twentieth century, patient preferences and rising hospital care costs have led to initiatives in many advanced countries to shift some care back away from hospitals to a patients home. Thus, governments and other payers began reimbursing for services aimed at fostering home health-care practices. At present, in the USA, where nearly 12 million persons receive home health care at an estimated cost of over $70 billion, the Center for Medicare and Medicaid services is the largest payer for home health care. CMS is also responsible for regulating most home health companies and ensuring compliance. Many states also have additional regulations, licensing and certification requirements, and compliance measures. However, determination of eligibility for coverage and length of services is often a challenge. For example, under CMS, to qualify for reimbursement for home health services, an individual has to be "confined to his home." Operationalizing this definition is often difficult as policy makers need to balance ensuring patients needing services are not denied home care while at the same time limiting potential fraud and abuse. Which services are covered (e.g., skilled or unskilled), for how many hours per day, and for how long are decisions that have tremendous implications and need to be balanced between need and available resources.

Given the extensive evidence that such care is often less costly and sometimes of higher quality than that delivered in an inpatient facility, there will continue to be large growth in the home health-care sector. Particularly given the aging of the population in many countries, governments and policy makers will need to continue to foster, develop, and strengthen innovative programs aimed at facilitating home-based care.

Cross-References

- ▶ Aging
- ▶ Disability
- ▶ Family, Caregiver
- ▶ Health Care
- ▶ Patient Care

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Homeostasis

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Synonyms

Equilibrium; Milieu interieur

Definition

Tendency and/or ability of a system to strive for and maintain stability of vital internal functions/parameters under unstable, external, or internal circumstances by means of negative or positive feedback.

Description

The necessity of a constant internal stability in face of external instability as a requirement of a free and independent life has first been stated by Claude Bernard, a French physiologist (1813–1878). Walter Bradford Cannon (1871–1945), physiologist at Harvard, then first coined the term "homeostasis," which he based on four propositions: (1) Constancy needs mechanisms to maintain constancy, (2) any change will be automatically resisted, (3) controlling mechanisms are interacting and cooperative, and (4) homeostasis is organized. Thus, homeostasis is the result of a complex interaction of multiple physiological – as well as psychological and behavioral – processes in order to secure and maintain set levels. Formally, the components of a homeostatic system encompass receptors that assess and relay information afferent to a central control which then send efferent signals to subordinated effectors, such as muscles, glands, organs, or systems. The responses are then monitored and controlled by positive and/or negative feedback. Examples for homeostasis can be seen in the regulation of body temperature by redistribution of blood flow (cold feet and hands), activation of muscular (shivers) and behavioral responses (put on a sweater), or in the regulation of blood glucose concentration (release of insulin and glucagon) as well as in more complex interactions between perceived challenges in social situations and emotional, behavioral, as well as neuroendocrine responses and their impact on the situational circumstances. The consequences of the regulatory responses are manifold and encompass permissive, stimulatory, suppressive, as well as preparative actions.

The concept of homeostasis have been extended by the concept of allostasis (McEwen, 1998), which states that stability is not only achievable through regulation, but also through change. Thus, external or internal circumstances are not only met by counterregulatory activation of the respective response but also lead to adaptive changes in the activity and reactivity of the respective system. For example, enduring stress not only leads to enhanced output of adrenocortical steroids, but also to changes in the negative feedback regulation of the affected neuroendocrine system, depending on the duration and the extent of the stressor (for review: Sapolsky, Romero, & Munck, 2000).

Cross-References

- ▶ Allostasis, Allostatic Load
- ▶ Neuroendocrine Activation
- ▶ Stress

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Homocysteine

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Synonyms

$HSCH_2CH_2CH(NH_2)CO_2H$

Definition

Homocysteine is an amino acid that acts as the intermediary during the synthesis of cysteine from dietary methionine in the human body.

Description

Homocysteine is an amino acid that acts as the intermediary during the synthesis of cysteine from dietary methionine in the human body. Homocysteine is not obtained from the diet but rather formed from the demethylation of methionine. It can then condense with the amino acid serine to form cystathionine, in a step catalyzed by the enzyme cystathionine β-synthase that requires vitamin B_6 as a cofactor. Cystathionine is then cleaved by cystathionine γ-lyase to produce cysteine and alpha-ketobutyrate. Alternatively, homocysteine can be converted back into methionine by donation of a methyl group from methyltetrahydrofolate, which is derived from folic acid. This reaction is catalyzed by the enzyme methionine synthase and requires vitamin B_{12} as an essential cofactor (Konkle, Simon, & Schafer, 2008).

In patients with the medical condition homocystinuria, deficiencies in key enzymes of homocysteine metabolism such as those described above lead to dramatically elevated levels of plasma homocysteine, which then spill over and are detected in urine. In addition to abnormalities of neurological and skeletal development, these patients also suffer from premature atherosclerosis and thrombophilia and have high incidences of venous thromboembolism, cerebrovascular disease, and peripheral vascular disease, beginning as early as the second and third decade of life. Because of this, it has been suggested that elevated homocysteine may play a causative role in promotion of atherosclerotic disease. Both in vitro and in vivo studies have tended to support this hypothesis, demonstrating that homocysteine can increase oxidative stress, cause endothelial dysfunction, promote inflammation, and induce smooth muscle proliferation (Ridker & Libby, 2008; Undas, Brozek, & Szczeklik, 2005). Furthermore, multiple large, prospective epidemiological studies have also concluded that even mildly elevated levels of homocysteine may carry increased risks for both initial and recurrent cardiovascular events (Homocysteine Studies Collaboration, 2002; Wald, Law, & Morris, 2002).

These observations have led to interest in homocysteine reduction as a strategy for cardiovascular disease prevention. Since B-complex vitamins (folic acid, vitamin B_6, and vitamin B_{12}) play a vital role in homocysteine metabolism and are often insufficiently obtained from dietary sources, several large, randomized clinical trials have been carried out to test the efficacy of B-complex vitamin supplementation in patients with cardiovascular disease. Despite uniform reduction of homocysteine levels in the intervention groups, however, none of the trials were able to demonstrate any decrease in incidence of cardiovascular events. Most recently, a meta-analysis was performed by the Cochrane Collaboration in 2009 that included eight randomized clinical trials with a total of 24,210 participants, confirming that homocysteine lowering with B-complex vitamin supplementation does not significantly reduce the risk of fatal or nonfatal myocardial infarction, stroke, or death from any cause (Martí-Carvajal, Solà, Lathyris, & Salanti, 2009). Given the inconsistency between these results and previous experimental and epidemiological data, there remains considerable debate on whether the homocysteine hypothesis is itself incorrect or whether there are other negative effects of B-complex vitamin supplementation

that may have countered the potential benefit of homocysteine reduction (Loscalzo, 2006).

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Homozygous

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Definition

An allele is one of the possible variants of a gene or position in the DNA sequence. All humans have two alleles on homologous chromosomes for each gene or trait. If these two alleles are identical, the person is considered to be homozygous for that trait or gene.

Cross-References

▶ Allele
▶ Dominant Inheritance
▶ Gene
▶ Heterozygous
▶ Recessive Inheritance

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Hooking Up

▶ Sexual Hookup

Hopelessness

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Definition

This is an important reaction to situations involving lack of, or reduced, control. The literature at times confuses helplessness with hopelessness, with the former included in the latter. Hopelessness refers to the belief that one lacks control over outcomes (response-outcome noncontingency or helplessness) and to the expectation of a negative future (negative outcome expectancies or pessimism), as conceptualized by Everson et al. (1996). Hopelessness includes thus two cognitions, rather than feelings. In the general

population, hopelessness is quite a stable phenomenon and is predicted by economic difficulties and unemployment. However, positive changes in one's living condition may protect people from becoming hopeless (Haatainen et al., 2003).

Multiple ways exist to assess hopelessness including a 2-item scale by Everson et al. (1996), the Beck Hopelessness Scale, and other measures.

Hopelessness is a major precursor or depression, particularly in people with a negative attribution style (attributing a negative event to an internal, stable, and global cause; Sweeney, Anderson, & Bailey, 1986). Hopelessness has also been found to be a significant predictor of suicide. Given the difficulty to prevent suicidal behavior, conducting preventative interventions among high-hopeless people may have important preventative value for public health.

Hopelessness is a risk factor of death from coronary heart disease and cancer and significantly predicts progression of carotid atherosclerosis. In Everson et al.'s (1996, 1997), Everson, Kaplan, Goldberg, Salonen, and Salonen (1997) studies, controlling for known cardiac risk factors had little effect on the prognostic role of hopelessness, leading them to conclude that other, not tested factors, could explain these relationships. One model proposes that hopelessness predicts poor prognosis in cancer by its link to brain and systemic interleukin-1 (IL-1) since blocking brain IL-1 prevents helplessness, since brain IL-1 promotes peripheral metastases, and since peripheral IL-1 at tumor sites promotes tumor cell proliferation, angiogenesis, and metastasis (Argaman et al., 2005). Thus, hopelessness could serve as an important predictor and therapeutic target in behavior medicine intervention trials since it is a risk factor of both mental and physical illnesses.

Cross-References

- ▶ Depression: Symptoms
- ▶ Perceived Control

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Hormone System

▶ Endocrinology

Hormone Theory of Aging

▶ Neuroendocrine Theory of Aging

Hormone Therapy

▶ Hormone Treatment

Hormone Treatment

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Synonyms

Hormone therapy; Menopausal hormone therapy

Definition

Serum concentrations of the female gonadal sex steroid hormones estradiol and progesterone decrease sharply after ▶ menopause due to the stop of ovarian hormone production. In order to counteract this decline, several forms of hormone (replacement) treatment (or therapy; HT or HRT) have been developed. It has been suggested that this hormone regime can treat menopausal symptoms and could potentially prevent several age-associated disorders. Women with an intact uterus can be treated with a combined regimen of estrogens and progestins. The progestin is added in order to prevent endometrial hyperplasia. Women with a hysterectomy can be treated with estrogen only preparations. Oral route of administration is still the most commonly used route as of today, but other approaches (e.g., transdermal applications) are available.

Sex steroid treatment after menopause has beneficial effects on hot flushes and urogenital atrophy. Moreover it has been shown to prevent osteoporosis, fractures, and ▶ diabetes. Adverse effects include venothrombotic episodes, stroke, and ▶ breast cancer. Negative effects are more pronounced when the combined treatment is administered, when patients are older, and when treatment is continued for longer periods of time (more than 5 years). For reviews see Maki et al. (2010) and Santen et al. (2010).

Due to the widespread action of sex steroids in the brain, beneficial effects of hormone therapy on mood and cognition have been postulated. These could be of relevance for the prevention and/or treatment of menopausal depression and ▶ dementia. Beneficial effects on cognition or mood might occur if treatment is started during or directly after menopause ("window of opportunity hypothesis"). However large randomized controlled clinical trials in this age group are still lacking. In contrast to the postulated beneficial effects, adverse effects were observed in the women's health initiative (WHI) study. In older women, the combined treatment with estrogens and progestins increased the risk for stroke and dementia. More clinical trials with cognitive and affective measures are needed before recommendations can be provided based on solid empirical evidence. For reviews see Maki et al. (2010) and Santen et al. (2010).

As of today, hormone therapy is recommended by the food and drug administration (FDA) for the treatment of menopausal symptoms only. The FDA recommends that hormone therapy be used at the lowest doses for the shortest duration needed to achieve treatment goals.

Cross-References

- ▶ Breast Cancer
- ▶ Depression
- ▶ Diabetes
- ▶ Menopause

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Hormones

- ▶ Leptin
- ▶ Neuropeptide Y (NPY)

Hospice

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Synonyms

Hospice care; Hospice programs; Hospice services

Definition

Hospice is a specialized form of palliative care; however, whereas palliative care can be provided at multiple stages of illness, hospice care is reserved for patients who are estimated to have less than 6 months to live. The philosophy of hospice care is to provide comfort and control pain symptoms for patients in the last phases of incurable disease while holistically providing for the psychological, social, and spiritual needs of the patient and their loved ones. Hospice care also involves some components of home health care, which focuses on patient rehabilitation and medical management. Home health care focuses on the patient and their physical needs while hospice includes a focus on the family and also addresses emotional and spiritual needs.

Description

History

The word "hospice" stems from the Latin word "hospitium" meaning hospitality, lodging, or guesthouse. The concept of a hospice can be traced back to medieval times when it referred to a place of shelter and rest for weary or ill travelers on a long journey. There is evidence of facilities like modern hospices dating back to the 1800s. For example, Jeanne Garnier founded several hospices in France as early as 1842 and the Irish Sisters of Charity opened hospices in Ireland and England in the 1800s to provide care for dying or incurable patients. The modern hospice movement began in the 1960s when Dr. Cicely Saunders established St. Christopher's Hospice near London to provide pain management and compassionate care for the dying. The hospice movement was propelled by Dr. Elisabeth Kubler-Ross' book, *On Death and Dying*, which was published in 1969 and provided a personal glimpse into the lives of terminally ill patients. During the 1970s the Hospice movement gained attention in the United States when Kubler-Ross testified at the first national hearings on the subject of death with dignity (1972); the first hospice organization was established in the United States in New Haven, Connecticut (1974); and the first hospice legislation was introduced by Senators Frank Church and Frank E. Moss (1974). It was not until 1983 that the Medicare hospice benefit was officially introduced and in 1986 was made permanent by Congress. In the past two decades, hospice organizations have continued to evolve from volunteer-based, grassroots organizations into health care companies with paid staff and quality practices. Today there are more than 4,000 hospice programs in the United States and over one million people in the United States receive hospice services each year. Hospice organizations are also widespread internationally.

Goals and Services

People choose hospice when the disease is not responding to treatment (incurable) or when the treatment has detrimental effects on quality of life. While many hospice patients are diagnosed with cancer (approximately 40%), hospice services are also available to patients with pulmonary disease, heart disease, neurological disorders, Alzheimer's disease, AIDS, and other life-limiting illnesses. The majority of patients who receive hospice services are over the age of 65 (approximately 80%). In the United States, hospice services have historically been more widely utilized by Caucasian patients (greater than 80% of hospice patients in 2005); however, hospice utilization among ethnic and racial minorities has been increasing in recent years. On average, patients spend 69 days on the hospice service before dying (median length = 21 days). In the United States, the majority of hospice services are funded through the Medicare hospice benefit (84% of hospice stays), although private insurers and other medical programs (e.g., Veteran's Administration) offer hospice benefits.

To qualify for the Medicare hospice benefit, a physician and the hospice medical director must certify that the patient has less than 6 months to live if the disease were to run its normal course. Then the patient or a durable medical power of attorney signs a statement declaring that they would like to be admitted to hospice. By signing the statement, the patient agrees to no longer

receive curative treatment related to their terminal illness. Medicare will still pay for covered benefits for any health needs that are not related to the patient's terminal illness. A terminally ill patient may receive hospice care for as long as necessary when a physician certifies that he or she has a life expectancy of 6 months or less; however, recertification of life expectancy is required at regular intervals (approximately 60 days). At any time, patients or their family members have the right to revoke the hospice benefit and resume Medicare coverage of the benefits that were waived when hospice care was elected. The patient can reelect to return to receiving hospice coverage at a later date. The hospice agency can discharge a patient if their health improves and they no longer have a 6-month prognosis.

Hospice services can be provided in multiple settings. More than 90% of the hospice services provided in the United States are based in patients' homes. Home hospice typically requires that a family member or loved one be established as the primary caregiver and be home with the patient at all times. Members of the hospice service will have regularly scheduled visits with the patient and caregiver as well as being available 24 hours a day, 7 days a week by phone to handle emergencies or questions. Hospice services can also be provided in hospitals and nursing homes or other long-term care facilities. These services can be provided on specialized hospice units, by trained nursing staff that can care for hospice patients, or through arrangements made with independent community-based hospice services that provide care inside of the hospital or nursing home facilities. These services can be a good option for patients who want hospice care but do not have primary caregivers to take care of them at home. Finally, many communities have free-standing, independently owned hospices that feature inpatient care facilities as well as home care hospice services.

Hospice affirms the concept of palliative care as an intensive program that enhances comfort and promotes the quality of life for individuals and their families. When cure is no longer possible, hospice recognizes that a peaceful and comfortable death is an essential goal of health care. Hospice philosophy recognizes that death is a natural part of the life cycle and that people have a human right to die in comfort and with dignity. The goal of hospice is not to prolong life or hasten death, but to allow people to achieve high levels of quality of life at the end of their life. Hospice care is also holistic and aims to treat the whole person rather than just focusing on the disease. The expected outcome is relief from distressing symptoms, lower levels of pain, and/or enhanced quality of life. Hospice philosophy is a family-centered approach and aims to assist the family and loved ones with the dying process as much as the patient. Patients and family members are encouraged to be in control of decision making and treatment planning. Finally, hospice philosophy promotes the idea that palliative care should be available to all individuals and their families without regard to age, gender, nationality, race, creed, sexual orientation, disability, diagnosis, availability of a primary caregiver, or ability to pay.

To provide holistic care, hospice services are provided by an interdisciplinary team of doctors, nurses, social workers, counselors, home health aides, clergy, physical and occupational therapists, and trained volunteers among others. Hospice organizations typically become the primary care coordinators during end-of-life care. Services are provided during the last stages of illness, throughout the dying process, and to family members during the first year after the patient's death. Hospice services provide medical treatment to relieve pain and to control other physical symptoms. Patients on hospice can continue to receive palliative radiation and chemotherapy that are designed to reduce pain. Hospice services provide needed medications, medical supplies, and equipment, as well as special services such as speech therapy, physical therapy, or nutritional consultation when indicated. Hospice services also provide assistance with basic needs of daily living by providing limited home health aide and supervision/training for family members. Family coaching and family conferences are common to help patients and their family members understand the illness, how to care for the patient, and improve communication about end-of-life issues.

Hospice staff may assist the patient with unfinished legal or financial business and in making funeral arrangements. Patients who need additional care are able to receive inpatient care as well as brief periods of respite care (up to 5 days), so that caregivers can attend special events or get rest from caregiving responsibilities. Counselors and clergy provide emotional support and address the spiritual needs of patients and their family members. Family members are able to receive bereavement care up to 13 months after the patient's death, which might include counseling, supportive phone calls, referrals to community resources, support groups, and/or memorial services.

Cross-References

- ▶ End-of-Life Care
- ▶ Home Health Care
- ▶ Palliative Care

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Hospice Care

- ▶ Hospice

Hospice Programs

- ▶ Hospice

Hospice Services

- ▶ Hospice

Hospices

- ▶ Palliative Care

Hospital Anxiety

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Synonyms

Fear of hospitals; Hospital stress; Nosocomephobia; Operative anxiety

Definition

Hospital anxiety refers to the anxious response and unusual preoccupation about noxious consequences (real or not) that can result from visiting hospitals or from undergoing medical procedures in a hospital.

Description

Hospital anxiety and related concepts (e.g., "fear of hospitals" and "hospital stress") have been widely examined in studies investigating barriers to both seeking medical care (Tod, Read, Lacey, & Abbott, 2001) and blood and organ donation

(e.g., Boulware et al., 2002), and preparation for medical procedures (Johnston, 1980). These fears, if irrational, can lead individuals to avoid appropriate medical care when needed or to disregard medical instructions. Individuals with excessive fear of hospitals (i.e., nosocomephobia) often avoid hospitals which results in skipping or missing appointments scheduled at hospitals and avoid visiting friends/relatives who are hospitalized. These fears are often founded on the belief that visiting a hospital may result in serious illness and even death. Original work focused on nonspecific anxious responses, but recent work has expanded the study of "hospital anxiety" by focusing on specific triggers such as blood, needles, injuries, and medical personnel.

Early studies examined the affective responses that both adult and pediatric patients experienced in hospital settings (e.g., hospital stress). The aim of this work was to understand the effects of anxiety and stress experienced before and/or after undergoing a medical procedure on medical outcomes (e.g., slow recovery post-surgery, increased pain and physical symptoms). Pre- and postoperative anxiety was found to disturb sleep patterns (Kain & Caldwell-Andrews, 2003), reduce adherence to medical recommendations (Mathews & Ridgeway, 1981), and increase pain sensitivity (Chapman & Cox, 1977). Among children, self-report data also showed an association between preoperative anxiety and postoperative negative behaviors (e.g., eating/sleeping problems) (Melamed, Dearborn, & Hermecz, 1983).

Although it is well established that anxiety levels are indirectly and directly associated with physiological and psychological factors that influence health outcomes, the mechanisms are poorly understood (Kiecolt-Glaser, Page, Marucha, MacCallum, & Glaser, 1998). For example, psychoneuroimmunological data suggest that anxiety may delay wound healing by indirectly influencing immune function. Specifically, it has been proposed that individuals who report high levels of anxiety when undergoing medical procedures tend to experience increased pain and may require more anesthesia during surgery and more analgesics during recovery than low anxiety individuals (Maranets & Kain, 1999). This increased use of anesthesia and analgesics, in turn, can result in endocrinological and immunological changes that delay wound healing. Similarly, postoperative anxiety can delay recovery from surgical procedures by influencing adherence to medical recommendations. Existing data suggest that specific anxiety and fear (e.g., fear of pain, cardiophobia, fear of injury) and not general anxiety are the key determinants of maladaptive behaviors (Berlin et al., 1997).

Several individual and situational factors have been implicated as antecedents of hospital anxiety. Although the specific genetic contribution to hospital anxiety is currently unknown, twin studies suggest that genetic predisposition accounts for an important proportion of the variance in anxiety disorders (Hettema, Neale, & Kendler, 2001). Personal experience and learning may lead to specific anxiety/fear. For instance, needle phobia is often the result of an aversive experience with needles involving doctors and/or dentists. These fears can also be the result of watching another individual experience a negative reaction to the medical procedures (Willemsen, Chowdhury, & Briscall, 2002). Other individual factors associated with increases in hospital anxiety include sex (being female), moderate to intense levels of depressive symptoms, and a history of chronic medical conditions (Caumo et al., 2001). Adults undergoing surgical procedures also indicated that they experienced anxiety surrounding uncertainty of the outcomes of the procedure (Caumo et al.). Among situational factors, poor or lack of communication with hospital personnel and health care providers, as well as loss of autonomy and control, are considered some of the most stressful aspects of hospitalization that could lead individuals to experience anxiety (Volcier, 1974).

Assessments of hospital anxiety and related concepts can be conducted in multiple ways. Researchers often explore three main aspects of anxiety, patients' cognitions, emotional state, and situation-specific anxieties. The most commonly used self-report measures of anxiety for adults include Spielberger's State-Trait Anxiety

Inventory and the Hospital Anxiety and Depression Scale (HADS). Spielberger's State-Trait Anxiety Inventory for Children and the Children's Manifest Anxiety Scale have been widely used to assess anxiety among children. Observational tools used often include the Clinical Anxiety Scale, Yale Preoperative Anxiety Scale, and the Palmar Sweat Index. Other instruments that assess specific aspects of hospital anxiety (e.g., the Injection Phobia Scale Anxiety, Blood Injection Symptom Scale, Medical Fears Survey, and the Disgust Scale) can provide important information about the specific triggers that lead to patients' anxious responses.

Interventions to reduce hospital anxiety have a long history. Melamed and collaborators used behavioral modeling to reduce pre- and postoperative anxiety and negative behaviors among children (Melamed & Siegel, 1975). Initial work with adults focused on the reduction of pain and anxiety caused by stressful and painful medical procedures (e.g., endoscopy). Work with adults conducted by Johnson and Leventhal (1974) demonstrated that accurate sensory and procedural information successfully reduced anxiety and pain caused by medical procedures. This type of intervention has been shown to be effective across various types of medical procedures (Suls & Wan, 1989). Principles from these earlier works are at the core of more recent and innovative psychological interventions to reduce pre- and postoperative anxiety. Exposure techniques have been associated with reduction in fear of blood-injection illness in adults (Olatunji, Smits, Connolly, Willems, & Lohr, 2007); whereas behavioral therapies (e.g., modeling) can reduce anxiety levels in children with needle phobias (Willemsen et al., et al., 2002).

Cross-References

- ▶ Anxiety
- ▶ Fear and Fear Avoidance
- ▶ HADS
- ▶ Health Anxiety
- ▶ Trait Anxiety

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Hospital Anxiety Depression Scale

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Synonyms

HADS

Definition

The Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) is a 14-item measure designed to be used as a brief screen for depression (seven items) and anxiety (seven items) disorders among nonpsychiatric, medically ill, outpatient populations. As a screening instrument, the HADS does not provide definitive diagnosis, but is the first step in a multistage process of selecting individuals at elevated risk for disorder to be evaluated through clinical interview. For example, the HADS does not assess specific symptoms of major depressive disorder (e.g., suicidality, guilt) thought by its authors to be less prevalent in nonpsychiatric populations, functional impairment, or frequency of symptoms, and the time frame assesses the previous 7 days, rather than 2 weeks of mood disturbance. Moreover, five of the seven items in the Depression subscale assess anhedonia-related experiences, and there is no question specifically addressing low mood. The HADS attempts to improve specificity of measurement by reducing physical symptoms that confound physical and psychological disorder (e.g., fatigue, insomnia) and focusing more on behavioral and affective symptoms. Anxiety and Depression subscales can range in score from 0 to 21, with scores of 11 or higher indicating probable caseness for clinical disorders and scores of 8–10 being suggestive of caseness. However, numerous cut-points have been suggested in the literature, as have total rather than subscale scores, though this has not been recommended by the scale authors. The HADS has been used as a measure of symptom severity among individuals with an anxiety or depressive disorder to allow tracking of therapeutic response over time. Both Anxiety and Depression subscales have demonstrated adequate internal consistency (Cronbach's alpha $\geq$0.67 and $\leq$0.93) across numerous translated versions, and adequate sensitivity and specificity for disorder using a score of $\leq$8 (both approximately 0.80; Bjelland, Dahl, Haug, & Neckelmann, 2002), though positive predictive values have been shown to be substantially lower (e.g., 0.17 for depressive disorder) in

medical patients (Silverstone, 1994, cited in Parker & Hyett, 2010).

Cross-References

- ▶ Anxiety and Its Measurement
- ▶ Depression: Measurement
- ▶ Screening

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Hospital Stress

- ▶ Hospital Anxiety

Hostile Affect

- ▶ Trait Anger

Hostility

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Synonyms

Affective hostility; Aggression; Anger; Cynical distrust; Cynical hostility; Mistrust

Definition

Hostility is a multidimensional personality trait with distinct cognitive, affective, and behavioral features. The cognitive component of hostility is evident in the habitual patterns of cynical mistrust and negative, suspicious attitudes and beliefs that hostile individuals have toward their interpersonal network and the community at large. The affective (emotional) component reflects the internal and external expression of anger and contempt, which may vary in degrees from moderate to high. The behavioral component may manifest through mannerisms and actions that perpetuate interpersonal conflicts, such as aggression and irritability. Although distinctions can be made among the cognitive, affective, and behavioral components of hostility, they often are interrelated and co-occur.

Description

The role of hostility and anger in cardiovascular disease (CVD) and more general health and well-being has a long history. For centuries, anecdotal reports and historical observations suggested that strong negative emotions, and particularly feelings of anger, aggressive tendencies, and hostile attitudes, were related to poor health (Everson-Rose & Lewis, 2005; Gallo & Matthews, 2003). These beliefs were supported in early psychoanalytic and psychodynamic reports that identified such personality characteristics in patients with hypertension and heart disease (Everson-Rose & Lewis, 2005). In the mid-1900s, this literature and related observations prompted work on identifying and assessing "coronary-prone behavior"; indeed, hostility, anger, and aggression were identified as critical tenets of coronary-prone behavior, which later came to be redefined as Type A behavior pattern (Friedman & Rosenman, 1971). Type A individuals are described as highly ambitious, competitive, time-urgent, impatient, quick-tempered, and tightly wound. The seminal work by Drs. Rosenman and Friedman in the Western Collaborate Group Study provided clear evidence

identifying Type A behavior as a risk factor for coronary heart disease (CHD) (Rosenman et al., 1975). Indeed, this study and initial empirical confirmation of the study findings prompted the medical community to accept Type A as the first psychosocial factor clearly identified as a risk factor for CHD (Cooper, Detre, & Weiss, 1981). However, the reproducibility of these findings was limited and negative findings on Type A and CHD risk began to appear in the literature. Consequently, researchers began to consider whether one of the components of Type A was driving the adverse effects previously identified for the broader Type A construct. Strong empirical evidence suggests that hostility is the "toxic" component of Type A behavior pattern (Williams et al., 1980), and an independent predictor of CVD (Miller, Smith, Turner, Guijarro, & Hallet, 1996).

Measurement

The development of valid and robust tools for measuring hostile characteristics and traits enabled systematic investigations of the impact of hostility on health. The Cook and Medley Hostility scale (CMHS) (Cook & Medley, 1954) was derived from the MMPI nearly 60 years ago and remains one of the most commonly used measures of hostility. It originally was devised to identify teachers who had poor rapport with their students, but subsequently was adopted as a more general indicator of hostility following its reported association with angiographic evidence of CHD (Williams et al., 1980), and CVD events and mortality (for reviews, see Everson-Rose & Lewis, 2005, and Hemingway & Marmot, 1999). The CMHS consists of 50 true-false items, where 1 point is assigned for each "hostile" response and higher scores indicate greater hostility. Several subsets of items on the CMHS have been used to measure specific components of hostility, the most common among them being cynicism. Other commonly used measures include the Buss-Durkee Hostility Inventory, Multidimensional Anger Inventory, and the Potential for Hostility measure derived from the Type A Behavior Pattern structured interview. Each measure is designed to broadly assess hostility and expressions of anger; however, these scales are not completely overlapping in terms of the dimensions of hostility that they capture (e.g., cynicism, hostile affect, anger expression, anger suppression). The potential for these measures to tap into different components of hostility should be taken into consideration when relating them to health outcomes.

Hostility and Health

The literature on the relationship between hostility and health is expansive, and spans across several disciplines. Most of these observations focus on the relationship between hostility (and its components) and the risk for poor cardiovascular outcomes. The literature relating hostility to non-cardiovascular diseases remains scarce – limited evidence has tied hostile traits to cancer (Tindle, et al., 2009), cognitive function (Barnes et al., 2009), and general health (Adams, 1994). In contrast, numerous epidemiological studies have investigated hostility in relation to various indicators of CVD. Literature reviews summarizing this work (Everson-Rose & Lewis, 2005; Gallo & Matthews, 2003; Miller et al., 1996) highlight significant inconsistencies between study findings. Some of the studies reviewed found that higher levels of hostility are associated with CHD incidence, presence and progression of atherosclerosis, hypertension, peripheral arterial disease, and all-cause and CVD mortality. However, the literature is not unequivocal – other studies have reported marginal and/or null findings between hostility and risk for CHD or CVD (Hemingway & Marmot, 1999). Miller et al. (1996) conducted a meta-analysis of 45 studies relating hostility to CHD, and suggested that variations in the methodology used by these studies may account for the mixed findings in the literature, such as the type of hostility measure, the study design, and the sociodemographics of the study population. Nevertheless, Miller et al. concluded that hostility is indeed an independent predictor of CHD and all-cause mortality. More recent studies, particularly

population-based studies, provide further support for hostility as a significant risk factor for CVD (Everson-Rose & Lewis, 2005); however, among patients with documented CHD, there is limited evidence that hostility or anger predict recurrent coronary events or mortality (Hemingway & Marmot, 1999).

Sociodemographic Characteristics

Hostility is related to a number of sociodemographic characteristics, with some support for differences between groups. Consistent evidence has shown an inverse relationship between socioeconomic status (SES) and hostility. This effect is linear, and suggests that those with the lowest education, income, and occupational status are more likely to express hostile characteristics. Furthermore, there is evidence that hostility may mediate the relationship between SES and CVD (Gallo & Matthews, 2003). In addition to social and economic differences in the expression of hostility, some studies show that hostile traits may vary by race and sex. For instance, growing evidence suggests that blacks and men report higher levels of hostility than their white and female counterparts (Gallo & Matthews); other studies indicate that hostility is higher in younger adults and the elderly and lower in mid-aged adults (Barefoot, Beckham, Haney, Siegler, & Lipkus, 1993). The same study also found that education may moderate the relationship between race and hostility, with more educated blacks reporting lower levels of hostility compared to less educated blacks, and more educated whites reporting higher levels of hostility than less educated whites. Nevertheless, the current literature lacks clear evidence that differences in the expression of hostility by race or sex translate into disparities in the risk for CVD. Several studies have tested this hypothesis and found no association (Everson-Rose et al., 2006) and/or lacked the sampling power necessary to test for these interactions (Iribarren et al., 2000). Future research should include large, balanced, and diverse samples in order to further investigate potential sociodemographic differences in the risk of CVD related to hostility.

Hostility and Pathways to CVD

Hostility may confer independent risk for CVD but also may operate through indirect pathways via its relationship with a number of other psychosocial and biobehavioral risk factors. Hostility is positively correlated with negative emotional states, including depression, anger, and anxiety, and inversely associated with positive psychosocial factors and personality traits, including social support and optimism (Gallo & Matthews, 2003; Tindle et al., 2009). Additionally, a number of studies have identified behavioral risk factors related to hostility. For example, a recent meta-analysis of 27 studies found that the CMHS was significantly associated with several traditional CVD-related risk factors (Bunde & Suls, 2006), including obesity (i.e., body mass index, waist-hip ratio), alcohol consumption, and smoking. Other studies link hostility to physical inactivity and dietary/caloric intake (Scherwitz et al., 1992). Moreover, several studies have identified biological and physiological pathways that may link hostility to disease. For instance, hostility is related to metabolic dysregulation, including impaired glucose metabolism, insulin resistance, and metabolic syndrome (Bunde & Suls, 2006; Goldbacher & Matthews, 2007). Hostile attitudes, emotions, and behaviors are associated with physiological response patterns that reflect activation of several biological systems in the body. Indeed, the physiological responses induced by hostility are characteristic of the "fight or flight" response, including prolonged activation of the hypothalamic-pituitary adrenal (HPA) axis and autonomic nervous system (ANS). For example, research shows that hostile individuals tend to have higher systolic and diastolic blood pressure, higher heart rates, as well as increased levels of stress hormones (i.e., cortisol), catecholamines (i.e., epinephrine, norepinephrine), and pro-inflammatory cytokines (i.e., IL-6 and IL-1) (Everson-Rose & Lewis, 2005). Furthermore, hostility is related to increased platelet activation and aggregation, which together with the other physiological risk factors contributes to increased atherosclerosis, and thrombus and plaque

formation (Markovitz, Matthews, Kiss, & Smitherman, 1996). On the whole, these observations support the fact that hostility clusters with other psychosocial and biobehavioral factors and may increase risk for CVD and other poor health outcomes through both direct and indirect pathways.

Summary

This entry provides a general insight into the history and research relating hostility to CVD and other health outcomes. Hostility is defined by cynical and suspicious attitudes, anger, bitterness, and varying levels of aggression and opposition. The role of hostility in health emerged from the shadow of the classic Type A behavior pattern and has gained momentum as one of the most pathogenic components of Type A. Instruments created to assess hostility have been vital to measuring the impact of hostility on health, and the CMHS stands out as a valid and reliable assessment tool. The literature on the role of hostility in cardiovascular health is somewhat mixed: evidence is more limited for a role of hostility in recurrent events or mortality among persons with existing heart disease, whereas evidence from methodologically sound, population-based studies generally provides support for the hypothesis that increased hostility is associated with excess risk of CVD. Research targeted at addressing these inconsistencies has investigated a number of potential pathways and mechanisms underlying the relationship between hostility and CVD. As a result, it is becoming more apparent that the impact of hostility on health may vary based on an individual's sociodemographic profile, and that hostility may operate through other psychosocial, behavioral, and physiological factors to influence CVD outcomes.

Cross-References

- ▶ Anger, Measurement
- ▶ Hostility, Cynical
- ▶ Hostility, Measurement of
- ▶ Interpersonal Circumplex
- ▶ Negative Thoughts
- ▶ Trait Anger

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Hostility, Cynical

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Synonyms

Cynicism; Cynical distrust; Cynical hostility

Definition

Hostility is a relatively stable personality trait that is typically characterized as a multidimensional construct with significant affective (e.g., anger), cognitive (e.g., attitudes), and behavioral (e.g., aggression) components. Hostile individuals have a suspicious, mistrustful attitude and often disparaging view of others and generally have a cynical worldview of their environment and social interactions. Thus, this type of personality disposition is often referred to "cynical hostility."

An expansive literature on personality and disease processes and health risks has developed over the past 50–60 years. Hostility has featured prominently in this literature, particularly with regard to cardiovascular disease (CVD) risk (Miller, Smith, Turner, Guijarro, & Hallet, 1996; Everson-Rose & Lewis, 2005). Though some negative studies have been reported, on balance, the available evidence from methodologically strong, population-based studies suggests that hostility is a significant risk factor for myocardial infarction and cardiovascular mortality in healthy populations (Barefoot, Dodge, Peterson, Dahlstrom, & Williams, 1989; Barefoot, Dodge, Dahlstrom, Siegler, Anderson, & Williams, 1991; Everson et al., 1997). Less support has been shown for the role of hostility in recurrent CVD or mortality in patients with diagnosed heart disease (Hemingway & Marmot, 1999). Recent studies in older adult populations have looked at hostility in relation to other health outcomes. Accruing evidence shows that hostility levels are associated with worse metabolic function and glucose regulation (Niaura, Todaro, Stroud, Spiro, Ward, & Weiss, 2002; Shen, Countryman, Spiro, & Niaura, 2008), higher levels of inflammation (Graham et al., 2006), poorer lung function (Kubzansky, Sparrow, Jackson, Cohen, Weiss, & Wright, 2006), and lower levels of cognitive function (Barnes et al., 2009).

Cross-References

▶ Anger, Measurement
▶ Hostility, Measurement of

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Hostility, Measurement of

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Synonyms

Cynical distrust; Cynical hostility; Cynicism

Definition

Hostility is defined by a suspicious, mistrustful attitude and cynical disposition toward others. Considered an enduring personality characteristic, hostility is characterized by cognitive, behavioral, and affective or emotional dimensions.

Description

Measurement of hostility can be based on structured interviews, with interviewer ratings of behavioral dimensions and verbal expressions of hostility based on participants' actions and responses within the interview setting. The classic example of this method of assessing hostility is the structured interview for Type A behavior, which was developed by Drs. Meyer Friedman and Ray Rosenman, the two cardiologists who first coined the term "Type A" to describe what they perceived to be coronary-prone behavior (e.g., hostility, aggressiveness, time-urgency, and a need to be hard-driving) among their heart patients (Friedman & Rosenman, 1971). The Type A interview is administered by carefully trained interviewers using a structured format in which scenarios are presented that attempt to elicit behavioral responses consistent with Type A. For example, at varying points during the interview, the interviewer will interrupt the respondent, challenge his or her responses, abruptly change topics, and also appear to be distracted. Interviews are audiotaped for later scoring. A scoring system is used that characterizes respondents on "Potential for Hostility" (PH), among other Type A characteristics, and codes on three dimensions of hostility, including intensity of response, hostile content, and style of interaction (Dembroski & Costa, 1987). As empirical support for the Type A construct in relation to risk for coronary heart disease faltered, investigators shifted focus to what were considered the most toxic components of Type A, and in particular, emphasis was placed on hostility, anger, and aggressiveness. Despite the seeming advantages of having objective ratings of hostile behaviors made by trained observers, such as PH

ratings obtained within the context of the Type A interview, such ratings are uncommon in the current literature on personality and health. Indeed, structured or semi-structured interview formats are rarely used anymore, especially in large community- or population-based studies of health because of the considerable time and expense involved in conducting such interviews. In contrast, a vast majority of such studies typically rely on self-administered or interviewer-administered questionnaires or surveys (i.e., "self-report") to assess hostility and other personality characteristics. Among the most commonly used measures of hostility is the 50-item Cook-Medley Hostility Scale (CMHS; Cook & Medley, 1954), which was derived from the original Minnesota Multiphasic Personality Inventory (Hathaway & McKinley, 1940). Items include such statements as "It is safer to trust nobody," "Most people make friends because friends are likely to be useful to them," "I think most people lie to get ahead," and "Most people are honest chiefly through fear of being caught." Several shortened versions of the CMHS are commonly used as well; most of these versions focus on a subset of 8–13 items that specifically assess interpersonal attitudes characterized by cynicism, mistrust of others' intentions, and anger responses (Barefoot, Dodge, Peterson, Dahlstrom, & Williams, 1989; Greenglass & Julkunen, 1989; Everson et al., 1997). The original CMHS employed a "true/false" response format with 1 point assigned for each "true" response and a hostility score was then calculated by summing across items. Alternate response formats have been used with some of the shortened versions of the CMHS, including a 4-point Likert scale in which respondents are asked to indicate the extent to which they agree or disagree with each statement from completely agree (0) to completely disagree (3). With this format, item responses are reverse-coded and summed to create a hostility score.

Other validated self-report instruments used to assess hostility include the Buss-Durkee Hostility Inventory (BDHI; Buss & Durkee, 1957) and the Multidimensional Anger Inventory (Siegel, 1986). The BDHI includes 75 true/false items; though the items were written to assess seven aspects of hostility, as defined by the creators of the scale, studies show that responses generally reflect two broad categories related to overt verbal and physical expressions of anger and hostility and to angry or hostile thoughts and emotions. The BDHI commonly has been used by psychologists but has rarely been used in epidemiological studies assessing personality or psychosocial factors and health outcomes, largely due to its length and the availability of several shortened versions of the CMHS. Similarly, the Multidimensional Anger Inventory consists of 25 self-report items that tap into four dimensions, only one of which that is specific to hostility and the remainder which are more specific to anger. The dimensions assessed include hostile outlook, mode of anger expression (anger-in and anger-out), anger arousal, and range of anger-eliciting situations. This scale was developed in a convenience sample of college students and though also validated in a community sample of male factory workers, it has not been used widely in population- or community-based studies.

Cross-References

- ▶ Anger, Measurement
- ▶ Hostility, Cynical

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Hostility, Psychophysiological Responses

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Synonyms

Aggression; Anger; Antagonism; Cynical distrust; Cynical hostility; Cynicism

Definition

Hostility has distinct cognitive components, affective or emotional components, and behavioral features. This multidimensional construct most commonly is defined by a mistrustful and suspicious attitude, cynical perceptions of others and their motives, and a negative interactional style characterized by anger, resentment, contempt, antagonism, and suspiciousness. Behavioral expressions of hostility typically include verbally and/or physically aggressive actions. The physiological consequences of hostility and its components contribute to over-activation of neurochemical and biological pathways, consistent with the stress response system, that may contribute to atherogenesis, alterations in glucose metabolism and other bodily systems, and which are harmful to cardiovascular health.

Description

A wealth of research has investigated physiologic responses related to hostility (Everson-Rose & Lewis, 2005). The emotional, behavioral, and cognitive manifestations of hostility are interrelated, although they may operate via different biologic pathways to influence health. This may well be why the literature on hostility's impact on physiological functioning reveals effects on multiple biologic systems, including documented effects on heart rate (HR), heart rate variability (HRV), systolic and diastolic blood pressure (SBP; DBP). One of the well-tested hypotheses that was formulated nearly 30 years ago to understand the linkages of psychosocial risk factors, such as Type A behavior, with coronary heart disease, focused on individual differences in physiologic responding. What came to commonly be called the cardiovascular reactivity hypothesis (Treiber et al., 2003; Smith & Ruiz, 2002) postulated that exaggerated autonomic nervous system and/or neuroendocrine activation occurred under conditions of interpersonal stress in persons with particular psychological traits or under certain emotional states. Hostility is one such psychological trait that has been a focus of the cardiovascular reactivity literature. Although not unequivocal (Suls & Wan, 1993), the vast majority of studies testing this hypothesis with regard to hostility show that, indeed, hostile persons tend to have higher HR (Jamner, Shapiro, Goldstein, & Hug, 1991), reduced HRV (Sloan et al., 1994), higher SBP and DBP (Smith & Allred, 1989; Suarez & Blumenthal, 1991), increased levels of epinephrine and norepinephrine (Zhang et al., 2006), and alterations in cortisol secretion or patterning (Pope & Smith, 1991) particularly under conditions of challenge and/or in ambiguous conditions, which are thought to be interpreted as adversarial by hostile persons (Everson, McKey, & Lovallo, 1995). Furthermore, prolonged and frequent physiological arousal experienced by hostile individuals increase the risk for endothelial injury and damage, a precursor of inflammation, platelet formation, thrombogenesis, and atherogenesis. Nonetheless, the literature on hostility and

risk for cardiovascular disease morbidity and mortality has not clearly shown that this exaggerated cardiovascular reactivity evident among hostile individuals is in fact the primary pathway by which hostility increases cardiovascular risk.

Other biologic pathways are important and also have been associated with hostility. For example, several studies have linked hostility with altered glucose metabolism, central adiposity, and metabolic dysregulation, which additionally may serve as mediators between hostility and CVD and related risk factors. In a recent review of the literature, Goldbacher and Matthews (2007) concluded that negative psychosocial factors, including depression, hostility, and anger, were important contributors to the risk of metabolic syndrome. Specifically in regards to studies investigating the role of hostility on metabolic syndrome, Goldbacher and Matthews found that hostility and its components (i.e., cynicism, aggression) related to increased risk of metabolic syndrome over time, and found longitudinal and cross-sectional evidence supporting the positive association between hostility and visceral adipose tissue, waist-hip ratio, and insulin. For example, one of the studies reported from Niaura and colleagues (2000) found that men high in hostility, as assessed by the Cook-Medley Hostility Scale, have increased insulin resistance and higher fasting insulin levels in the presence of high stress levels, and impaired glucose metabolism than men who are low in hostility. Moreover, there is evidence that ties the combined effects of high hostility and metabolic syndrome to increased risk of myocardial infarction (Todaro et al., 2005). Even so, the review of the literature revealed studies that find no relationship between hostility and metabolic syndrome (Goldbacher & Matthews, 2007), and although steps have been taken to address and conceptualize some of these inconsistencies (Zhang et al., 2006), this work is limited.

Studies also have examined neuroendocrine and genetic biomarkers related to hostility, especially those involved in serotonergic function. Williams (1994) proposed that reduced serotonin levels in the central nervous system may constitute an important brain mechanism by which hostility and related biobehavioral risk characteristics influence risk for cardiovascular diseases. In his hypothesis, he suggested that serotonin dysfunction was the underlying cause of harmful traits (e.g., hostility, anger), behaviors (e.g., excessive alcohol use, smoking), and physiological responses (e.g., heightened sympathetic arousal) which seem to cluster in certain individuals. Justification for his theory is supported by a growing number of studies that identify genetic markers and environmental stimuli that regulate serotonin levels and shape the expression of hostile traits and behaviors. Indeed, more recent studies have provided scientific evidence that individuals with alleles that code for lower CNS serotonin function are more likely to express hostile-like traits (Manuck, Flory, Ferrel, Mann, & Muldoon, 2000; Lesch et al., 1996), including aggression and neuroticism, and other negative psychosocial characteristics like depression (Siegler et al., 2008). In addition, there is some evidence that these genes may vary by race and/or sex; however, this work is limited and requires further investigation. More work is needed to understand the development of hostility and potential biomarkers that may predispose individuals to hostile traits and characteristics.

Hostility is a multidimensional personality trait that functions through several biological pathways to influence health and well-being. The expression of hostile attitudes, emotions, and behaviors are stress inducing, and particularly cardio-reactive. Indeed, the body uses autonomic and neuroendocrine pathways to interpret hostile experiences, which may manifest as heightened cardiovascular stimulation or metabolic dysregulation. In addition, the tendency for hostility to cluster with other negative psychosocial factors and harmful behaviors, led to the growing theory that low CNS serotonin function may predispose individuals to these disease-inducing traits and behaviors. In order to fully conceptualize the mechanisms that shape the relationship between hostility and disease, further research will be required that supports physiological mediators.

Cross-References

- ▶ Anger, Measurement
- ▶ Hostility, Cynical
- ▶ Hostility, Measurement of

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Household Income

- ▶ Family, Income

HPA Axis Negative Feedback Testing

- ▶ Dexamethasone Suppression Test

HPA Axis Stimulation Tests

- ▶ Pharmacological Stress Tests

HPV

▶ Human Papillomavirus (HPV)

HR_{max}

▶ Maximal Exercise Heart Rate

$HSCH_2CH_2CH(NH_2)CO_2H$

▶ Homocysteine

HSV-1

▶ Genital Herpes

HSV-2

▶ Genital Herpes

Human Factors/Ergonomics

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Synonyms

Applied cognitive psychology; Applied experimental psychology; Engineering psychology; Ergonomics

Definition

Human factors/ergonomics can be broadly defined as optimizing the relationships between systems and the humans using them. More specifically, it is defined as follows:

"...the understanding of interactions among humans and other elements of a system, and the profession that applies theory, principles, data, and other methods to design in order to optimize human well-being and overall system performance" (Human Factors and Ergonomics Society: About HFES, n d).

In health care, the goal of human factors is to improve patient outcomes by supporting provider performance and by increasing patient safety through the reduction of iatrogenic error. In the context of behavioral medicine, the specific goal of human factors is to promote effective clinical interventions for disease prevention and treatment and to facilitate positive changes in patient behavior. The application of human factors to health care is discussed in more detail below.

Description

In the USA, an estimated 98,000 patients die each year due to preventable medical errors (Institute Of Medicine, 1999). Generally, health care has lagged far behind other industries, notably aviation, for improvements in safety (Leape, 1994). However, some specialties in medicine (e.g., anesthesiology) have made tremendous progress in patient safety over the last decades. For example, by the late 1990s, the risk of death from anesthesia was 1/60th the mortality rate in the 1980s, which translates into a 60-fold decrease in the number of annual deaths in the USA (Kohn, Corrigan, Donaldson, & Institute of Medicine U.S. Committee on Quality of Health Care in America, 2000). This phenomenal accomplishment in anesthesiology patient safety resulted from a combination of changes, most based on the application of human factors principles. Among the changes that increased anesthesia safety were steps toward optimization and standardization of system components (equipment

and training), establishment and implementation of practice guidelines which were widely accepted by providers, hospitals, and professional organizations, and significant advances in technology (Gaba, 2000). In contrast to anesthesiology, other areas in health care have not made as much progress in patient safety. For example, approximately half to two-thirds of all annual patient deaths in the USA are caused by a preventable systemic infection that is associated with a central line (a catheter that is used to draw blood and deliver fluids and medication) placed in a patient (Institute of Medicine, 1999).

A human factors approach that pursues the goal of improving patient safety focuses not only on individual components of the system in isolation, but also takes into account the interactions of these elements with each other. Only such an integrative perspective will result in sustainable change of clinical practice and improvements in patient outcomes and patient safety.

Socio-Natural Systems Perspective of Health Care

Comprehensively addressing patient safety requires considering human performance in a range of different systems while acknowledging the differences between such systems (Durso & Drews, 2010). The safety improvements in anesthesiology were successful because human factors principles were applied to multiple elements (optimization and standardization of equipment and tasks with practice guidelines adopted by providers and organizations), widespread implementation of simulator-based training with feedback, and patient monitoring technology was improved (Gaba, 2000). This combination of interventions improved the transparency of patient outcomes through feedback with better equipment and structured training and education.

Clearly, all of the multiple system components (patients, providers, physical environment, organizational environment, tasks, and equipment/tools; adapted from Carayon, Alvarado, & Hundt, 2007) that are involved in health care need to be included in a comprehensive systemic intervention. In addition, to maximize improvements in performance and safety, it is important to acknowledge the differences and similarities between different systems (e.g., technical systems in aviation vs. socio-natural systems in health care).

For example, health care can be conceptualized by applying a socio-natural systems perspective: patients are, at the core, biological or natural systems, interacting with providers in an organizational environment that constitutes a social system, where often equipment/tools are used to accomplish a goal with the equipment/tools constituting the technical aspect of the system (Durso & Drews, 2010). Interventions to improve safety in aviation and health care are frequently compared, and there are important similarities between the two systems that allow adoption. However, the comparison faces limitations when focusing on a key distinction between the relevant system components in each domain at the microlevel: in health care, tasks are performed on a patient, a natural system. Consequently, feedback on a task, such as treating a patient, is often delayed or not accessible because causality is not directly observable. At the microlevel, tasks in aviation are performed on an aircraft, an engineered system that is designed to provide continuous feedback, making it largely transparent and highly predictable (Durso & Drews, 2010).

In health care, transparency (observation and understanding of a system) of the system component (patients) is partial, at best. One challenge is that the lack of transparency limits the opportunities for learning and reduces the ability to predict the consequences of actions. Another difference between health care and aviation can be found in the approach toward training and distinction in microlevel variability between patients and aircraft. Due to the lack of standardization in health care (Timmermans & Berg, 2003), providers are unlikely to receive detailed training and practice with particular equipment and frequently need to perform the same procedure using different brands of equipment with distinct designs. Whereas in aviation, pilots train and become certified on a specific aircraft, thus they do not encounter differences between aircraft. Furthermore, tasks in aviation are highly structured and therefore predictable, with

checklists (protocols) for routine activities and emergency situations (Wickens, Gordon, & Liu,, 1998). In health care, protocols for every possible situation and patient simply do not exist. Thus, providers perform tasks based on their training, local practices, and the specific needs of patients, because the inter-individual variability of patients is significant. This situation can be contrasted with aviation, where it is one of the most important goals of quality control in aircraft manufacturing to minimize the variability between aircraft.

Other differences between health care and aviation extend to the physical and organizational environment. The work environment in health care has the potential to negatively impact cognition because of frequent error-producing conditions: interruptions, multitasking, suboptimal equipment, and inadequate staffing (Carayon et al., 2007; Nolan, 2000). The work environment in health care sharply contrasts with aviation, where during critical phases of a flight (e.g., takeoff, landing), nonessential conversations in the cockpit are prohibited (sterile cockpit), minimizing interruptions and distractions. Finally, health care is only beginning to establish a safety culture such as that successfully established in aviation. A consequence of this early stage of safety culture adoption is that errors and "near misses" generally go unreported (Leape, 1994), limiting the opportunities for learning because reoccurring patterns are not identified.

To improve performance in a complex system, components such as equipment and training need to be optimized and standardized by applying human factors principles (Nolan, 2000, Wickens et al., 1998). Specifically, this involves reducing the cognitive demands of tasks that are performed. For example, high reliance on perception, memory, attention, and decision-making when performing a task is likely to increase the probability of suboptimal actions and do not promote optimal performance (Wickens et al.). Consequently, reducing the cognitive demand of a task can provide defenses against human error (Wickens et al.). To illustrate the application of human factors in health care, two examples will be discussed in more detail: the development of a drug display (a visual monitor) to improve medication delivery in anesthesiology and tools to improve medication adherence.

Drug Display for Medication Delivery

Delivering correct drug concentrations and doses over the course of administering anesthesia is critical to patient safety. This task has significant cognitive demands because the anesthesiologist must remember the times and amounts of past drug administrations, then use this recalled information to calculate changes in concentration over time, and finally, determine the difference between the calculated current concentrations and the appropriate levels to determine present dosages. The consequences of high cognitive demands are ample opportunities for errors.

Consequently, anesthesia drug delivery performance can be improved with a drug display consistent with human factors principles. Drews, Syroid, Agutter, Strayer, and Westenskow (2006) demonstrated the positive impact of such technology optimized by application of human factors by comparing the display to the standard approach of delivering anesthesia without such information available to the anesthesiologist. The success of this approach can be attributed to the fact that the drug display creates defenses against error by providing "knowledge in the world" which reduces demands on cognition (Norman, 2002). More specifically, the human factors principles that were implemented in the drug display are as follows (Norman, 2002; Wickens et al., 1998):

1. Affordances (perceived functions of objects: intuitive representations of medication doses over time)
2. Chunking (meaningful grouping of related items: different drugs separated, current dose levels for all drugs together)
3. Reduced effort (mental/physical task demands to perform actions: information is provided on the monitor rather than requiring cognition)
4. Structure/guidance (design, layout, and sequence: e.g., time in Western cultures goes from left to right, chunking: information in meaningful units that can be maintained in memory)

5. Visual feedback (visibility: graphical representation depicting patterns of change over time for past, current, and predicted future drug concentration levels)

The drug display is an example of the application of human factors principles to equipment design (or tool design) to increase clinical task performance and reduce human error. However, equipment and tasks are only two of many additional components that are part of health care. Macroergonomics emphasizes such a broad system-based perspective when planning interventions for improvement in performance.

Medication Adherence Tools

A socio-natural systems perspective can be applied to a problem that is highly relevant for behavioral medicine: medication adherence. Lack of medication compliance has negative effects on patient health and produces significant economic costs (Osterberg & Blaschke, 2005). There are a number of causes that can lead to nonadherence, for example, the patient does not understand how to weigh medication benefits compared to the side effects, high access costs (high purchase costs of the medication, difficulty accessing a pharmacy, and other cognitive barriers such as challenges in the complexity of coordinating the timing and dosing of a number of medications) (Osterberg & Blaschke, 2005).

One human factors approach to improve medication adherence is providing cognitive aids or tools to schedule times to take medications (Waicekauskas et al., 2010). Participants scheduled the times to take several medications using a paper or electronic daily calendar, working in pairs (one participant was the "patient" and the other participant was the "provider"). Together the dyads solved examples of scheduling simple and complex sample medication problems. Complexity was increased by adding scheduling constraints, for example, fewer possible times to take a medication because of routine activities on the calendar, requiring the medication to be taken on an empty stomach. Both, the paper and electronic versions of the tool provided support to creating more accurate medication schedules than no aid.

The medication scheduling tool may serve as an example for how human factors approaches can help develop effective cognitive tools that reduce memory and other cognitive demands in coordinating the administration of medications and may also lead to increases in compliance with medication scheduling. However, such a cognitive aid is a technical system component that will likely only raise adherence if the causes for noncompliance were either forgetting to take medication or difficulty in figuring out when and how to take medication. The limitation of this approach is clearly associated with the fact that no improvement in medication compliance can be expected for patients that do not understand how to weigh the benefits of medication versus the medication side effects. For example, blood pressure medication has significant long-term health benefits, but the benefits are not directly visible to most patients, and there are frequent negative side effects (e.g., Osterberg & Blaschke, 2005). The tradeoff between the potentially invisible or less salient therapeutic benefits of medication and noticeable side effects from the perspective of the patient illustrates the challenges associated with a natural system that lacks transparency.

Nevertheless, the efficacy of a medication scheduling tool may be further enhanced by addressing other factors that contribute to noncompliance. For example, a clinical decision support system (a system designed to improve quality of care by providing cognitive support) could be implemented in electronic medical records or paper and applied to the problem of medication compliance (e.g., Drews et al., 2010). Implementing such a support system could prompt providers to: (a) remind patients about benefits versus side effects, (b) discuss medication cost and possible alternatives, and (c) provide pharmacy locations, etc. Although applying human factors to a single system can improve patient care, the example of medication compliance demonstrates both the challenges that are associated with applying an integrative human factors approach to health care and the greater benefits to patients that come with the application of this comprehensive approach.

Cross-References

- ▶ Adherence
- ▶ Cognitions
- ▶ Risk Perception

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Human Genome Project

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Definition

The Human Genome Project (HGP) was an international scientific research project to map and sequence the entire human genome. The project goals were to identify all 20,000–25,000 genes and determine the sequences of the three billion base pairs that make up the haploid human genome. The project was started in 1990 and coordinated by the US Department of Energy and National Institutes of Health, and in partnership with the Wellcome Trust (UK). In 1998, Celera Corporation launched a parallel private effort to sequence the human genome. Most of the government-sponsored sequencing was performed in universities and research centers from the USA, the UK, Japan, France, Germany, and China. The public and private efforts succeeded, with a draft sequence published in 2001 (International Human Genome Sequencing Consortium, 2001; Venter et al., 2001), and completion of the HGP in April 2003 (Collins, Green, Guttmacher, & Guyer, 2003; Collins, Morgan, & Patrinos, 2003; Frazier, Johnson, Thomassen, Oliver, & Patrinos, 2003). The HGP also included parallel efforts to sequence select model organisms, such as the bacterium *Escherichia coli*, the fruit fly *Drosophila melanogaster*, and the mouse (*Mus musculus*). Sequencing model organisms served to both help to develop the technology and interpret human gene function. Data from the project are freely available to researchers.

Cross-References

- ▶ DNA
- ▶ Gene

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Human Herpesvirus-4 (HHV-4)

- ▶ Epstein-Barr Virus

Human Immunodeficiency Virus (HIV)

- ▶ AIDS: Acquired Immunodeficiency Syndrome

Human Papillomavirus (HPV)

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Synonyms

HPV

Definition

The human papillomaviruses (HPVs) are members of the Papillomaviridae. These viruses (Papillomaviridae) are specie specific, so they can only infect humans. They are non-enveloped viruses that carry their genetic information on circular, double-stranded DNA. The virus measures approximately 55 nm in diameter. Nearly 200 types of HPV have been identified. While many of them do not produce any symptoms in humans, some strains of the virus can cause warts and in a minority of cases can lead to cancer (Bonnez & Reichman, 2009; Giuliano et al., 2011).

Description

HPV tends to infect skin or mucosal tissue, and while many HPV subtypes do not cause any symptoms, the subtypes that are known to produce symptoms can cause a multitude of skin diseases (e.g., plantar warts, common warts, epidermodysplasia verruciformis, genital warts, cervical and anal carcinoma). Each virus subtype tends to be associated with a specific pathology, for example, plantar warts tend to be caused by HPV-1 or HPV-2, while cervical carcinoma is frequently caused by HPV-16 and HPV-18. However, other HPV subtypes may cause the same diseases, although less commonly (Bonnez & Reichman, 2009).

HPV is easily recognized when it causes symptoms (i.e., clinically apparent infections); however, subclinical and asymptomatic infections are more common. In addition, individuals with past HPV infections represent an even larger group of affected persons. These three types of infections (i.e., clinical, asymptomatic, and past infections) and their relationship to each other make difficult their epidemiological study (Bonnez & Reichman, 2009). HPV is also classified by the location of the infection. For example, a significant distinction is made between infections in genitals and mucosal tissue, and non-genital infections (cutaneous infections).

In the United States, 20 million people are currently estimated to be infected and contagious

(Bonnez & Reichman, 2009), while an additional 6.2 million people between the ages of 15 and 44 become infected with genital HPV annually (Richman, Whitley, & Hayden, 2009). HPV transmission requires skin to skin contact; therefore, most genital infections are transmitted through sexual activity (Giuliano et al., 2011; Richman et al., 2009). Anal HPV infections happen most frequently through anal intercourse, although it is possible to spread the infection from one area to another by contact with hands or objects that have been exposed to infected areas (Giuliano et al., 2011). Nonsexual infections are rare. Most HPV infections (70%) resolve within a year, up to 90% within 2 years. However, individuals who do not clear their HPV infection are at risk for cervical or anal cancer, though the progression to cancer can take 10–20 years (Giuliano et al., 2011).

The three most observed cutaneous manifestations of HPV are *common warts* (71% of all cutaneous infections), *plantar warts* (34%), and *juvenile or flat warts* (4%). Common warts and flat warts are more common in children, while plantar warts are more common in adolescents. The prevalence of common warts in school-age children is estimated to be 4–20% (Bonnez & Reichman, 2009). Since contact with infected skin cells is necessary for transmission, two other vehicles of transmission besides sexual contact are possible, though rare: mother-to-child transmission and exposure to contaminated objects (Giuliano et al., 2011). Infection during birth is rare, but may lead to juvenile-onset recurrent respiratory papillomatosis (JORRP) – an infection that results in warty growths in the upper respiratory tract and can lead to obstruction of the airway in severe cases. JORRP has an incidence of 2 per 100,000 children in the United States (Giuliano et al., 2011). The other uncommon route of transmission is through contact with an object that has residual infected skin cells or with infected hands with a common wart or carrying infected cells (e.g., after scratching) (Giuliano et al., 2011). This means of transmission is more likely if there is trauma to the receiving skin, which may explain the higher frequency of cutaneous warts seen among meat handlers (Bonnez & Reichman, 2009).

Currently, there is no treatment to specifically eradicate an HPV infection; infections have to be cleared by the immune system of the infected person. Treatment of HPV focuses on removing the lesions by chemical or physical means, or by local stimulation of cytokines, to reduce or eliminate the symptoms of the infection (Bonnez & Reichman, 2009). There are two potential methods of preventing HPV transmission. Avoiding skin exposure to infected areas can reduce the likelihood of transmission. However, since HPV can be asymptomatic or subclinical (i.e., an infected individual may not have any visible skin lesions), avoiding contact with infected skin can be difficult or impossible. Condoms provide only limited protection from HPV since they protect only skin that is covered by the condom (Giuliano et al., 2011). A second method of prevention is vaccination against HPV infection, which has been proven to be quite effective (Bonnez & Reichman, 2009; The FUTURE II Study Group, 2007; Trotter & Franco, 2006). The available HPV vaccinations target only up to four HPV subtypes. However, since these HPV subtypes cause the majority of cervical cancers and genital warts, the vaccines can prevent the vast majority of clinically important and cosmetically unpleasant HPV infections (Bonnez & Reichman, 2009).

Cross-References

- ▶ Infectious Diseases
- ▶ Immune Function

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Human Subject Protections

▶ Protection of Human Subjects

Human Subjects Committee

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Synonyms

Institutional review board (IRB); Research ethics committee

Definition

Ethical conduct of research requires an objective and prospective review and approval by a committee established for the purpose of protecting human subjects involved in research. In the United States, these human subjects committees are usually called Institutional Review Boards (IRBs). They may also be known as Ethics Review Committees.

The main responsibility of the IRB is to conduct a comprehensive scientific and ethical review of the planned research activities. The purpose of this review is to ensure the proposed research meets the federal requirements for ethical research. It determines study feasibility, scientific merit, ethical soundness, strength of study design, balance of risk and benefits, and adequacy of informed consent. The review ensures that the design is scientifically sound and does not expose subjects to unnecessary risk. A sound scientific design is essential, as it is not ethically justifiable to expose subjects to any risk, discomfort, or inconvenience if the research is poorly designed and not likely to obtain meaningful information (Council for International Organizations of Medical Sciences, 1993). The IRB is also responsible for identifying, minimizing, and eliminating conflicts of interests.

Key components of the system include the federal government's regulations, IRBs, the sponsor, and the principal investigators (PIs) conducting the research. Additionally, all investigators and members of the research team and community are charged with protecting human subjects. The federal regulations, issued by the Department of Health and Human Services (DHHS) and the Food and Drug Administration (FDA), are the cornerstone in this system of protection. The DHHS regulates all research it funds and conducts, and the FDA regulates research involving drugs, biologics, and clinical devices.

The criteria for IRB approval are that risks to research participants are minimized; risks are reasonable in relation to anticipated benefits and the knowledge that may result; selection of participants is equitable; informed consent will be sought from each participant or their legally authorized representative and appropriately documented, as required; adequate plans for monitoring data to ensure participant safety are in place; adequate provisions are made to protect the privacy of the participant and the confidentiality of the data; and additional safeguards are provided to protect the welfare and safety of vulnerable participants such as children, prisoners, pregnant women, mentally disabled persons, or economically or educationally disadvantaged individuals (U. S. Department of Health and Human Services, 2009a).

IRBs are responsible for conducting comprehensive prospective and ongoing review of all research activities at least annually. The level of scrutiny and frequency of continuing review depends upon the level of risk posed by the research activities. To best protect human

subjects, an objective review is required by a diverse group of individuals with no direct involvement in the planned research. The Federal Regulations stipulate that the IRB is composed of at least five members. The members must include a scientist, nonscientist, and at least one person who is not affiliated, and is not a family member of an affiliated person, with the institution conducting the research (U. S. Department of Health and Human Services, 2009b). These are the minimal requirements established by the federal guidelines; however, the size and composition may vary depending on the institutional requirements and resources. On occasion, it may be necessary for the IRB to invite an individual to provide expertise in a specific area of research.

The responsible and ethical conduct of research requires that the PI and all team members are appropriately trained and well qualified to conduct research with humans. Responsible for the conduct of the study, PIs must consider the participants' safety and welfare at every phase of the study including initiation, implementation, closeout, and dissemination. Fundamental in providing these protections is ensuring subject privacy and confidentiality (Steneck, 2007). Conducting research in a manner consistent with the established ethical principles is of paramount importance to protect human participants in research. Providing adequate protections requires a broad range of knowledge and a systems approach. Such a system is essential to accomplish comprehensive reviews of planned research activities and to ensure sound ethical interactions between the investigator and participant, especially in the informed consent process. Also important in the system of protection is oversight, including safety monitoring and a robust quality improvement and compliance program (Federman, Hanna, & Rodriguez, 2002).

The Belmont Report and the Protection of Human Subjects

Human subjects who participate in research benefit society by contributing to the development of new drugs, medical procedures, and a greater understanding of how we think and act (Steneck, 2007). Individuals who assume the inherent risks of participating in research deserve to be treated with dignity and respect and to have their rights as humans protected. The federal regulations just discussed are founded upon ethical principles written in the Belmont report. The ethical principles of respect for persons, beneficence, and justice are tightly integrated in the regulations. The principle of respect for persons states that the protection of autonomy and personal dignity requires informed consent of each person before they are involved in research. Special protections, to prevent coercion and undue influence, are required for individuals who are unable to make autonomous decisions, such as children, prisoners, and those with compromised decision-making capacity. Securing the well-being of research participants by minimizing risk and maximizing benefits is essential to ensuring beneficence and protecting subjects from harm. Justice requires equitable subject selection to reduce or prevent overburdening certain populations and providing access to research opportunities for others (The Belmont Report, 1979). While the federal government provides the overarching governance for research in humans, oversight and implementation of the regulations are conducted by the IRB.

Cross-References

- ▶ Ethics Committee
- ▶ Human Subjects Committee
- ▶ Informed Consent
- ▶ Institutional Review Board (IRB)

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Human Subjects Protections

► Protection of Human Subjects

Hybridoma Growth Factor

► Interleukins, -1 (IL-1), -6 (IL-6), -18 (IL-18)

Hybridoma Plasmacytoma Growth Factor

► Interleukins, -1 (IL-1), -6 (IL-6), -18 (IL-18)

Hypercholesterolemia

► Dyslipidemia

Hyperglycemia

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Synonyms

Control; Glycemia; Load-high

Definition

Hyperglycemia is defined as a state of elevated blood plasma glucose ($\geq$7.0 mmol/L, or 120 mg/dL, before meals) and is a hallmark feature of all diabetes subtypes. Hyperglycemia results from disruptions in blood glucose metabolism, insufficient insulin production (in the case of type 1 diabetes), or the cellular resistance to the endogenous insulin produced by the pancreas (in the cases of type 2 diabetes and gestational diabetes). Hyperglycemia can be assessed at home using a portable glucometer, or in the laboratory setting by drawing a blood sample for analysis. Symptoms include excessive thirst, excessive urination, fatigue, itchy skin, and, over time, weight loss. In mild forms, individuals are often unaware of blood glucose elevations. However, in more flagrant episodes, individuals may self-detect these aforementioned symptoms. Hyperglycemia may be managed by a number of self-management behaviors including changes in dietary intake and physical activity. Among individuals with diabetes who are receiving insulin therapy, they may also administer an injection of short-acting insulin. These behaviors, in isolation or combination, will help to restore normal blood glucose levels. However, if unmanaged, extreme hyperglycemia can precipitate a state of diabetic ketoacidosis (DKA), which results in both the production of ketones and metabolic acidosis. DKA leads to excessive urination and loss of both fluid and electrolytes that can result in myocardial infarction and death. Repeated episodes of DKA may be evidence

of poor metabolic control, which potentiates morbidity and premature mortality. Precipitants for DKA include infection, abdominal crises (e.g., gastrointestinal bleeding, pancreatitis), physical trauma, and insulin omission. If unmanaged, repeated episodes of hyperglycemia lead to the development of serious vascular pathology resulting in blindness, renal failure, pain and loss of sensation in the extremities, myocardial infarctions, cerebrovascular accidents, and amputations.

Cross-References

- ▶ Diabetes
- ▶ Glucose
- ▶ Glucose: Levels, Control, Intolerance, and Metabolism
- ▶ Glycemia
- ▶ Glycemia: Control, Load-High

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Hyperinsulinemia

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Synonyms

Insulin Resistance; Metabolic syndrome X; Prediabetes

Definition

Hyperinsulinemia is an indication of an underlying problem controlling blood glucose levels and is characterized by an excess of insulin circulating in the blood (Shanik et al. 2008). Insulin is produced by the pancreas and helps regulate blood glucose. Hyperinsulinemia may be caused by insulin resistance, a condition in which an individual's body becomes resistant to the effects of insulin and the pancreas compensates by producing additional insulin. Increases in insulin result in a decrease in circulating blood glucose (Buse, Polonsky, & Burant, 2008). Symptoms of hyperinsulinemia are often absent unless hypoglycemia (abnormally low glucose levels) occurs. When present, symptoms may include temporary muscle weakness, brain fog, fatigue, visual problems, headaches, shaking, and/or thirst. Treatment of hyperinsulinemia is directed at the underlying problem and typically focuses on diet and exercise (Diabetes.co.uk Team, 2011). Dieticians recommend a nutritional regimen that is low in sugar and carbohydrates, while high in protein. Additionally, regular monitoring of weight, blood glucose, and insulin is advised. In some cases, typically where obesity is present, treatment with metformin (i.e., an oral anti-diabetic drug) may be used to reduce insulin levels (Glueck, 2007). Though it is often mistaken for diabetes or hypoglycemia, hyperinsulinemia is a separate condition. Left unmonitored and untreated, however, it can develop into type 2 diabetes mellitus. A significant risk factor of hyperinsulinemia is the increased likelihood of developing a cluster of closely related abnormalities known as the metabolic syndrome (Reaven, 2005). Within this syndrome, it is believed that the more insulin resistant a person, the more likely he or she will develop some degree of glucose intolerance, high triacylglycerol and low HDL concentrations, essential hypertension, and procoagulant and proinflammatory states (Reaven, 2006), all of which are known to increase the chance of cardiovascular disease.

Cross-References

- Blood Glucose
- Diabetes
- Glucose: Levels, Control, Intolerance, and Metabolism
- Hypoglycemia
- Insulin
- Insulin Resistance (IR) Syndrome
- Metabolic Syndrome
- Type 2 Diabetes Mellitus

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Hyperlipidemia

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Synonyms

Lipid disorder

Definition

Hyperlipidemia simply means high blood lipid levels. Lipids refer to cholesterol, cholesterol compounds, triglycerides, and phospholipids. Lipids are carried in the blood by lipoproteins (e.g., low-density lipoproteins (LDL) and high-density lipoproteins (HDL)) (American Heart Association, n.d.).

Description

Hyperlipidemia increases the risk for atherosclerosis (i.e., hardening of the arteries, which is a buildup of plaque in the arteries). Atherosclerosis leads to narrow and stiff arteries that provide a reduced blood flow (Society for Vascular Surgery, 2010) and ultimately an increased risk for high blood pressure, heart disease, stroke, and poor lower leg circulation (Society for Vascular Surgery; U.S. National Library of Medicine & National Institutes of Health, 2011a, 2011b).

Risk Factors

Hyperlipidemia has several risk factors and many are modifiable. Some risk factors include (Mayo Clinic, 2010b; Society for Vascular Surgery, 2010; U.S. National Library of Medicine & National Institutes of Health, 2011b):

- Having certain genetic disorders; for example, familial hypercholesterolemia or familial dysbetalipoproteinemia
- Having a (nuclear) family history of early heart disease (before age 55)
- Age (men >45, women >55)
- Having certain diseases such as diabetes, high blood pressure, kidney disease, hypothyroidism, Cushing syndrome, polycystic ovary syndrome
- Having a body mass index over 25 (also defined as being overweight or obese)
- Drinking alcohol excessively
- Eating diets high in unhealthy fats (i.e., saturated and trans fat)
- Eating diets high in dietary cholesterol
- Lack of exercise

Hyperlipidemia, Table 1 Cholesterol screening summary

	Why the test is important	Desired or optimal value[a]	Some possible modifiable reasons why value is not at desired or optimal value
Total cholesterol	Tells the total amount of cholesterol in blood	<200 mg/dL	Being overweight
			Eating a diet high in saturated and/or trans fats
LDL (bad) cholesterol	When LDL cholesterol is too high, plaque can form in the arteries, which can increase risk for cardiovascular disease	<100 mg/dL	Being overweight
			Eating a diet high in saturated and/or trans fats
HDL (good) cholesterol	High levels can be protective whereas low levels (<40 mg/dL for men and <50 mg/dL for women) are a significant risk factor for heart disease. HDLs also help remove LDL cholesterol	≥60 mg/dL	Smoking cigarettes
			Being inactive
			Being overweight
			Eating a diet high in trans fats
Triglycerides	Unused calories are converted to triglycerides (fat that is circulating in the blood before it is stored)	<150 mg/dL	Eating more calories than working off
			Eating too many sweets
			Drinking alcohol excessively
			Being overweight
			Having diabetes with elevated blood sugar

[a]These are general guidelines and results should be discussed with primary care provider; the table was compiled with information from American Heart Association 2010, 2011; Centers for Disease Control and Prevention, 2010; Mayo Clinic, 2011; National Heart, Lung and Blood Institute, n.d.; U.S. National Library of Medicine & National Cancer Institute, 2011

- Using certain medications such as estrogen, birth control pills, corticosteroids, beta blockers, some types of diuretics, and some types of antidepressants
- Smoking cigarettes

Screening

Hyperlipidemia causes no symptoms; therefore, screening is important (Mayo Clinic, 2010c). To screen for hyperlipidemia a blood sample is obtained and a lipid panel (e.g., total cholesterol, LDL, HDH, triglycerides) is done (Mayo Clinic, 2011). Table 1 summarizes why the test is done, the desired or optimal value, and some possible modifiable reasons why the test is not at the desired or optimal value (American Heart Association 2010, 2011; Centers for Disease Control and Prevention, 2010; Mayo Clinic, 2011; National Heart, Lung and Blood Institute, n.d.; U.S. National Library of Medicine & National Cancer Institute, 2011).

Screening Recommendations

Recommendations for when hyperlipidemia screening should begin differ across organizations but generally testing is recommended to begin for men between ages 20–35 and 20–45 for women and then checked every 5 years. Additionally, screening is recommended when someone develops certain diseases (e.g., diabetes). Guidelines for cholesterol screening for children and/or adolescents vary due to limited evidence. However, some experts say those under age 18 should be screened if risk factors are present (e.g., family history of high cholesterol) while some recommend screening all children (U.S. National Library of Medicine & National Institutes of Health, 2011a).

Treatment

There are two common treatment methods for addressing an abnormal lipid panel. Sometimes only a lifestyle change is needed. A lifestyle

change is often the first step in treating hyperlipidemia. However, if a lifestyle change is not effective (especially if LDLs are still elevated), a combination of lifestyle modification and cholesterol medication may be recommended (Society for Vascular Surgery, 2010).

Possible lifestyle recommendations (Centers for Disease Control and Prevention, 2011; Mayo Clinic, 2010a; Society for Vascular Surgery, 2010; U.S. National Library of Medicine & National Cancer Institute, 2011):

- Eating the recommended amount of fruits and vegetables
- Limiting saturated (e.g., butter, cheese, whole milk) and trans (e.g., processed foods, fried foods, commercially baked foods) fats and replacing them with monounsaturated (e.g., canola oil, olive oil, avocados) and polyunsaturated (e.g., canola oil, walnuts, salmon) fats
- Limiting dietary cholesterol intake
- Eating whole grains
- Engaging in regular exercise
- Losing weight (if overweight)
- Quit smoking cigarettes
- Limiting alcohol consumption

There are five major classes of cholesterol lowering medication, which include: (Agency for Healthcare Research and Quality, 2009)

- Vitamins and supplements
- Statins
- Bile ace binders
- Cholesterol absorption inhibitor
- Fibrates

Cross-References

- ▶ Cholesterol
- ▶ Dyslipidemia
- ▶ Lipid Abnormalities
- ▶ Lipid Metabolism
- ▶ Plasma Lipid

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Hypertension

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Synonyms

High blood pressure

Definition

Hypertension has been variously defined over the years. However, systolic (i.e., peak) bloodpressure ≥140 mmHg and diastolic blood pressure ≥90 mmHg are now considered indicative of hypertension and of meriting treatment. Blood pressure ca 120/80 mmHg is deemed to be normal (normotensive). Although hypertension may be a secondary consequence of problems elsewhere, e.g., renal failure, the vast majority of cases of hypertension have no proximal and discrete cause. This is called essential hypertension and is a major focus of research in behavioral medicine. This is easy to understand given the prevalence and health consequences of hypertension. It is estimated to affect 15% and 20 % of the adult population in Western countries, with worldwide prevalence being around 10%. Concern with hypertension also reflects its association with coronary heart disease and stroke; the results of many studies testify that as blood pressure rises, so life expectancy decreases. For example, at the outset of the famous Framingham study, some 5,000 of the citizens of that small community in the USA had their blood pressures recorded; around 20% of them were hypertensive. Those with hypertension were three times more likely to go on to have a heart attack and eight times more likely to have a stroke. Given the clinical importance of hypertension, it is perhaps hardly surprising that there are several dedicated scientific journals and many texts devoted to hypertension. My favorite, however, remains Beevers and MacGregor (1995).

Cross-References

- ▶ Blood Pressure, Measurement of
- ▶ Blood Pressure
- ▶ Hypertrophy

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Hypertriglyceridemia

- ▶ Dyslipidemia

Hypertrophy

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Synonyms

Cardiovascular disease; Hypertension

Definition

Hypertrophy is a structural response of an anatomic chamber, particularly the right and left cardiac ventricles, to chronically increased volumes or pressures. Left ventricular hypertrophy has been shown to be independently predictive of coronary heart disease onset and is associated with common conditions such as hypertension (high blood pressure) and cardiac valvular dysfunction (Maron, Ridker, Grundy, & Pearson, 2010). While hypertrophy is initially an adaptive mechanism for the heart, the chronic enlargement leads to unsustainable pressures and deformation that impedes contraction and relaxation. Serial

measurements of hypertrophy can be helpful in primary and secondary prevention for cardiovascular events.

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Hypochondriasis

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Synonyms

Health anxiety

Definition

Hypochondriasis is defined by the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR) (American Psychiatric, 2000), as preoccupation with fear of having or the belief that one has a serious health condition. Criteria for the diagnosis include that the belief: is often based on the misreading of physical symptoms, is not of intensity better described as delusional, leads to clinically significant impairment in functioning, is of 6 months duration or longer, and is not better accounted for by a medical or psychiatric disorder. A specifier of "with poor insight" may be added to the diagnosis if the individual does not have recognition that the fears of illness are unwarranted and unfounded.

Description

Epidemiology

The prevalence of hypochondriasis is estimated to be between 1% and 5% in the general population. However, the prevalence rates increase to 2–7% within primary care outpatient populations, which is not unexpected given that this population is likely to seek treatment (American Psychiatric, 2000). Hypochondriasis may manifest at any age, but occurrences are commonly noted in early adulthood, with no gender differences noted. The course of hypochondriasis is typically described as chronic in greater than 50% of cases, and likelihood of chronicity is increased by when the individual experiences a number of bodily sensations, believes that one has a serious illness, or has other comorbid psychiatric diagnoses (Taylor & Asmundson, 2006). Cross-cultural differences have been noted among patients diagnosed with hypochondriasis; (Taylor & Asmundson; Asmundson, Taylor, & Sevgur, 2001) for example, gastrointestinal sensations are predominant in the United Kingdom, pulmonary complaints are common in Germany, and concerns surrounding immunology and chemical sensitivities frequently occur in the USA and Canada (Taylor & Asmundson, 2006).

Evaluation

Essential features of an evaluation of hypochondriasis include a thorough medical examination to rule out any potential physiological explanation for the reported symptoms (Taylor & Asmundson, 2006). Following this evaluation, a detailed psychiatric interview should be conducted and include any recent psychosocial stressors and previous history of health-related difficulties of the patient, family member, or significant others. Interviewers should consider in the evaluation that hypochondriasis typically manifests in the forms of disease phobia, bodily preoccupation, and disease conviction. For example, a patient may present with the belief that they have cancer (disease conviction) or may have a preoccupation with gastrointestinal complaints (bodily preoccupation) and insists on laboratory tests and radiological imaging. Although a person

with hypochondriasis may exhibit symptoms in all three of these categories, symptoms that fall within a predominant category can impact patient presentation and treatment (Stewart & Watt, 2001). Adjunctive standardized assessments may be utilized but with cautious interpretation. For example, the Minnesota Multiphasic Personality Inventory-2 (MMPI-2), which has a hypochondriasis scale, does not necessarily measure the fear associated with health complaints but rather measures the degree to which one experiences physical symptoms (Taylor & Asmundson, 2006). Other measures, such as the Structured Clinical Interview for the DSM-IV (SCID), the Structured Diagnostic Interview for Hypochondriasis (SDIH), and other self-report hypochondriasis measures (e.g., Whiteley Index, Health Anxiety Questionnaire, Illness Attitudes Scale, Somatosensory Amplification Scale and Illness Behavior Scale), may be more appropriate tools in gathering diagnostic information within this patient population (Speckens, 2001; Stewart & Watt, 2001).

Treatment/Best Practices

Although there is no evidence to date on combined treatments, there is consistent evidence suggesting that cognitive behavioral therapy (CBT) and medication-based treatments independently for hypochondriasis are effective (Taylor & Asmundson, 2006). Specifically, CBT interventions that focused on exposure and response prevention, psychoeducation, and cognitive therapy were found to improve symptoms in patients with hypochondriasis, and treatment gains continued to be present in a 12-month follow-up (Asmundson, Taylor, & Sevgur, 2001; Fallon & Feinstein, 2001; Taylor & Asmundson, 2006). As an example of CBT with exposure and response prevention for hypochondriasis, a patient with a bodily preoccupation of skin rashes may be exposed to anxiety associated with thoughts of having a skin rash, and resist the typical subsequent responses to that anxiety that would usually follow (e.g., calling a doctor, asking numerous questions about the rash, researching information on the internet/medical books, etc.) until the anxiety is relieved. Finally, while there are self-help and internet resources available, there is a paucity of evidence on the effectiveness of these resources utilized independently and/or as an adjunct to other therapies described (Taylor & Asmundson, 2006).

With regard to pharmacotherapy, selective serotonin reuptake inhibitors (SSRIs) and imipramine, a tricyclic antidepressant, have been shown in both head-to-head trials and randomized control trials to be useful in patients with hypochondriasis. Of the SSRIs studied (paroxetine, fluoxetine, fluvoxamine, and nefazadone), fluoxetine appears to have greater potential for desired treatment effects. However, there are no recent studies on long-term use of these medications with persons diagnosed with hypochondriasis (Asmundson et al., 2001; Enns, Kjernisted, & Lander, 2001; Taylor & Asmundson, 2006).

In addition to these treatment choices, environmental factors may impact treatment effectiveness. For example, the relationship between the patient and treating provider may impact treatment and exacerbation of symptoms (Lipsitt, 2001). If the provider is perceived by the patient as dismissing of symptoms or as not addressing the patient's concerns, the patient may choose to move from provider to provider until such symptoms are addressed. It is also suggested that overreassurance can exacerbate symptoms because the patient may perceive that their symptoms are being dismissed, leading to patient dropout (Starcevic, 2001). Optimally, providers who communicate clearly about guidelines on when to seek treatment and who have regularly scheduled follow-ups may reduce unnecessary treatment visits. Furthermore, patient preference should be weighted heavily into the decision to utilize pharmacotherapy, psychotherapy, or combined treatments (Taylor & Asmundson, 2006).

Cross-References

▶ Health Anxiety
▶ Somatoform Disorders

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Hypoglycemia

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Synonyms

Insulin shock; Low blood glucose

Definition

Hypoglycemia is characterized by an abnormally low level of blood glucose concentration, designated as less than or equal to 70 mg/dL. Blood glucose serves as the body's main energy source (Pub Med Health, 2011). With hypoglycemia, there is an inadequate supply of glucose to the brain, resulting in impairment of function that requires immediate attention to prevent organ or brain damage, especially when blood glucose is extremely low (Cryer, 1997). Hypoglycemia occurs when an individual's glucose is metabolized too quickly, when glucose is released into the bloodstream too slowly, and when too much insulin is released into the bloodstream (Cryer, Davis, & Shamoon, 2003). Hypoglycemia is relatively common among people with type 1 diabetes, and may occur when too much insulin or diabetes medication is taken, when insufficient amounts of food are ingested, or with sudden increases in exercise. Hypoglycemia is characterized as mild (below 70 mg/dL), moderate (below 55 mg/dL), or severe (below 35–40 mg/dL); and common symptoms include: trembling, nausea, sweating, dizziness, irritability, confusion, seizures, fainting, and coma. Immediate treatment involves quick steps to return blood glucose levels to a normal range. Mild-to-moderate hypoglycemia is treated by ingesting carbohydrates, while glucagon injections, a hormone that blocks insulin and raises blood glucose, are used to treat severe cases (Wysocki, Greco, & Buckloh, 2003). People with type 1 diabetes suffer an average of two episodes of symptomatic hypoglycemia per week, and approximately one episode of severe, at least temporarily disabling, hypoglycemia per year (Cryer, 2010). Careful monitoring of blood glucose levels can help predict or prevent moderate to severe hypoglycemia in at-risk individuals.

Cross-References

- Blood Glucose
- Diabetes
- Glucose
- Glucose: Levels, Control, Intolerance, and Metabolism
- Glycemia: Control, Load-High
- Insulin
- Type 1 Diabetes Mellitus

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Hypothalamic Nuclei

► Hypothalamus

Hypothalamic-Pituitary-Adrenal Axis

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Synonyms

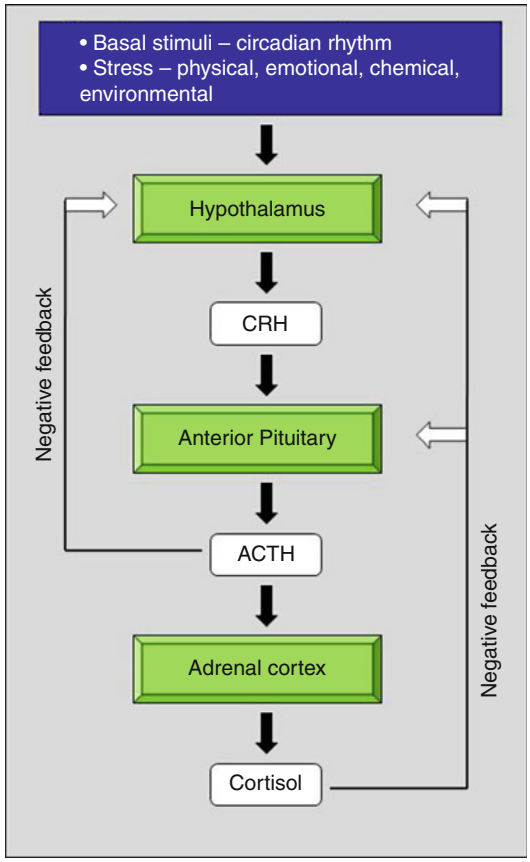
Pituitary-adrenal axis

Definition

The hypothalamic-pituitary-adrenal axis (HPA axis) is an interactive neuroendocrine unit comprising of the hypothalamus, the pituitary gland, and the adrenal glands. The hypothalamus is located in the brain and the pituitary at the base of it, whereas the adrenals are on top of the kidneys.

The HPA axis plays key roles in basal homeostasis and in the body's response to stress. The major pathway of the axis results in the production and secretion of cortisol. The hypothalamus responds to basal neural input which follows a circadian rhythm and input as a result of stress by increasing the secretion of corticotrophin-releasing hormone (CRH) from the hypothalamus. This increase in CRH acts upon the anterior pituitary gland to secrete adrenocorticotropic hormone (ACTH), which in turn circulates to the adrenal cortex to stimulate the release of cortisol into the bloodstream. The HPA axis is an example of a negative feedback loop; cortisol can reduce its own secretion via feedback to the anterior pituitary to reduce ACTH and the hypothalamus to limit the secretion of CRH. ACTH also provides negative feedback limiting its secretion via CRH.

The importance of the HPA axis to homeostasis is illustrated through the wide range of functions that cortisol is involved in, including metabolism, vascular activity, and immune and inflammatory responses. Therefore, even in the absence of stress, the production of cortisol via the HPA axis performs vital roles within the human body. In the presence of stress, be it physical, emotional, chemical, or environmental, the HPA axis governs the body's response. An increase in the production of cortisol enhances vascular activity, reduces immune responses, limits inflammation, stimulates gluconeogenesis, and inhibits nonessential functions. These effects serve to protect the body from potential damaging effects of stress, e.g., excessive immune and inflammatory response, but also act to increase resources to cope during the period of stress through changes in metabolism. While the role of cortisol in the stress response is initially protective, long-term stimulation of the HPA axis and over exposure to cortisol can be damaging, leading to immunosuppression and excessive catabolism of body tissue. Dysregulation of the HPA axis has been associated with various illnesses and disorders, both physiological and psychological. More detail on the HPA axis can be found from the following sources: Widmaier et al. (2004) and Greenspan and Forsham (1983) (Fig. 1).



The hypothalamic-pituitary-adrenal axis
CRH: Corticotrophin releasing hormone
ACTH: Adrenocorticotrophic hormone

Hypothalamic-Pituitary-Adrenal Axis, Fig. 1 The hypothalamic-pituitary-adrenal axis. *CRH* Corticotrophin releasing hormone, *ACTH* Adrenocorticotrophic hormone

Cross-References

- ▶ ACTH
- ▶ Cortisol
- ▶ Pituitary-Adrenal Axis

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Hypothalamus

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Synonyms

Anterior hypothalamic area; Arcuate nucleus; Dorsal hypothalamic area; Dorsomedial nucleus; Hypothalamic nuclei; Lateral mammillary nucleus; Lateral nucleus; Lateral preoptic area; Medial mammillary nucleus; Medial preoptic area; Paraventricular nucleus; Posterior hypothalamic area; Suprachiasmatic nucleus; Supraoptic nucleus; Ventromedial nucleus

Definition

The hypothalamus is a subcortical collection of nuclei that monitor, modulate, and regulate physiology and behavior including feeding, thirst, reproduction, temperature regulation, sleep, and emotional behavior such as fear and aggression.

Description

This entry describes the hypothalamus in humans. All vertebrates have a hypothalamus, and while there is a high degree of conservation across species and especially in mammals, readers should refer elsewhere for phenotypic and functional details regarding other animals.

The hypothalamus (Latin: hypo: "below" the thalamus) is located below the thalamus on the base of the brain on both sides of the ventral portion of the third ventricle. Three main divisions of the hypothalamus are the anterior, medial

(or tuberal), and posterior regions, each made up of several nuclei. These clusters of neurons coordinate autonomic, endocrine, somatic, and behavioral information to maintain homeostasis and motivate behavior in both the short and long term. Via bidirectional connections throughout many areas of the brain, the hypothalamus regulates a host of physiological processes and related reflexes including feeding, drinking, reproduction, temperature regulation, sleep, and emotional behavior such as fear and aggression.

The hypothalamus receives sensory information from the entire body about conditions in the external world including smells and light cycles and internal information from the visceral somatosensory system. Other sensory cells within hypothalamic nuclei monitor and respond to changes in internal states and biological set points such as temperature, salinity, and glucose, in addition to other factors outside the blood-brain barrier via connections with circumventricular organs such as the area postrema. A change in homeostasis (e.g., increased body temperature) triggers a variety of responses aimed at body temperature reduction, all of which are regulated by the hypothalamus. These include moving blood to the surface of the skin, increased sweating, and increased water conservation at the kidneys.

The hypothalamus controls the endocrine system via the anterior and posterior pituitary gland. The hypothalamus is neural tissue, but it also has specialized neural cells that secrete neurohormones from nerve cell terminals into blood vessels in the pituitary gland. Although the output of these neurosecretory cells is endocrinological, they are morphologically similar to typical neurons and serve to bridge the neural and endocrine systems. Oxytocin and vasopressin are peptides produced in neuroendocrine cells in the paraventricular and supraoptic nuclei of the hypothalamus and released into general circulation directly via projections into the arterial blood within the posterior pituitary gland (also called the neurohypophysis). Other hypothalamic nuclei indirectly control the endocrine system through the production of neuroendocrine factors that inhibit or increase release of various hormones from the anterior pituitary gland (also called the adenohypophysis). For example, corticotropin-releasing factor and growth hormone-releasing factor regulate the release of adrenocorticotropin and growth hormone from the anterior pituitary respectively.

Finally, bidirectional communication between higher cortical regions and the hypothalamus occurs via the limbic system. In the temperature regulation example above, information from the hypothalamus to other limbic areas and the cortex can regulate complex behavior and motivation such as acting on the environment to generate cooling or mobility to a cooler environment. Cognition and emotion related to the situational context can also modulate hypothalamic responses.

References and Readings

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Hypothesis Testing

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Synonyms

Inferential statistical testing

Definition

Hypothesis testing is the core component of inferential statistics, a branch of statistics that is

employed to provide compelling evidence that two or more groups of numbers (data) differ from each other in a meaningful manner. It is therefore a strategy that facilitates provision of compelling evidence of the efficacy of a new behavioral medicine intervention or treatment.

Description

One of the most efficient ways to acquire new knowledge about any topic (including new behavioral medicine interventions) is to devise questions that guide our investigations. While relatively loosely formed questions can be helpful in early stages of the knowledge acquisition process, refining our knowledge is facilitated by asking more specific questions, and, in turn, acquiring more specific information and knowledge.

The scientific method is one particular method of acquiring new knowledge. In scientific research, our questions need to be asked in a particular manner. These questions are called research questions, and they lead to the development of two research hypotheses in each case. Turner (2007) provided an operational definition of a useful research question:

- It needs to be specific (precise).
- It needs to be testable.

Consider a new behavioral medicine intervention for pain management. To test its efficacy against a standard (existing) treatment, it is planned to conduct a randomized, controlled clinical trial. The research question of interest is:

- Is the new treatment statistically significantly more effective than the standard treatment?

Actually, the "pure" statistical version of this question asks whether there is a statistically significant difference in the efficacy demonstrated by the two treatments. This question allows for the fact that the new treatment could theoretically be statistically significantly ***less*** effective than the standard treatment as well as being significantly more effective. That is, use of a two-sided approach (hypothesis) is, in reality, appropriate here. However, we will proceed here with interest focused on just one of the two directions of differential performance of the new treatment.

The research hypotheses are commonly called the null hypothesis and the alternative hypothesis. In this author's opinion, the term "research hypothesis" is more informative than the term "alternative hypothesis," and so it is used here. The null hypothesis is the crux of hypothesis testing. For our research question, the null hypothesis would be:

- The new treatment is not statistically significantly more effective than the standard treatment.

The accompanying research hypothesis is:

- The new treatment is statistically significantly more effective than the standard treatment.

It is important to note that, whatever the outcome of the trial, the following statements are true:

- It is never the case that neither hypothesis is correct.
- It is never the case that both hypotheses are correct.
- It is always the case that one of them is correct, and that the other is not correct.

A helpful way of remembering which hypothesis is which, i.e., which form the null hypothesis takes and which form the research hypothesis takes, is to conceptualize that the research hypothesis states what you are "hoping" to find, and the null hypothesis states what you are not hoping to find (researchers who have developed a new treatment have an understandable tendency to want it to be better than other treatments). It must be emphasized here, however, that, while helpful, this conceptualization skates on very thin scientific ice. In strict scientific terms, hope has no place in experimental research. The goal is to discover the truth, whatever it may be, and one should not start out hoping to find one particular outcome. In the real world, this ideologically pure stance is not common for many reasons (financial and personal recognition reasons being not the least of them).

Statistical analysis of the data acquired in the trial conducted to compare the two treatments will enable us to decide between the two mutually exclusive hypotheses, i.e., the null hypothesis and the research hypothesis. A measure of pain reduction will be obtained for each person receiving

the new treatment (the test treatment group), and will also be obtained for each person receiving the standard treatment (the control treatment group). The mean (average) pain reduction will be calculated for each treatment group. The focus of attention can be captured by a treatment effect associated with the new treatment, which is calculated as follows: Treatment effect = Mean pain reduction in the test treatment group minus mean pain reduction in the control treatment group.

The concept of statistical significance becomes important here because it is possible for the mean pain reduction in the test treatment group to be numerically greater than mean pain reduction in the control treatment group, but for the difference not to attain statistical significance. To provide compelling evidence that the new treatment is "better" than the standard one, there must be a statistically significant treatment effect. The statistical methodology involved in determining the presence or absence of a statistically significant treatment effect is formulaic: The treatment effect is either of statistically significant magnitude or it is not. If it is, the appropriate test will ultimately produce a result in the form "$p < 0.05$" (see the ▶ Probability entry). If so, given that a randomized study design has been used, this evidence would allow the statement that the difference in magnitude between the mean response in the test treatment group and that in the control group (the treatment effect) is unlikely to have been due to chance, i.e., it is sufficiently great to be declared statistically significant. In this case, the null hypothesis is rejected in favor of the research hypothesis. If the result is "$p \geq 0.05$" statistical significance is not achieved. This result allows the statement to be made that the degree of difference in pain reduction between the two treatment groups could well have arisen by chance alone. In this case, we fail to reject the null hypothesis.

Statistical methodology necessitates a choice being made here: It is a forced choice paradigm. It is always the case that the process of hypothesis testing will result in one, and only one, of these two mutually exclusive actions.

The connection between the terms "hypothesis testing" and "inferential statistics" is a result of the following statement. The ultimate purpose of the results from a single clinical trial (or a group of related trials) is not to tell us precisely what happened in that trial, but to allow us to gain insight into, i.e., to infer in an educated manner, what is likely to happen in a much larger group of patients should the treatment make it into widespread clinical practice.

Cross-References

▶ Probability

References and Readings

Turner, J. R. (2007). New drug development: design, methodology, and analysis. Hoboken, NJ: Wiley.

Hypothetical Construct

▶ Latent Variable

Hypothetical Variable

▶ Latent Variable