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Decision Making

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Synonyms

Choice; Inference

Definition

Decision making is the process by which a course of action is chosen from among two or more alternatives. This definition is broad enough to span different decision types and domains, from fast, habitual decisions to complex, life-changing ones. The extent to which such disparate decisions share or not the same underlying cognitive processes and are differentially affected by age-related change is an ongoing topic of research.

Historical Background

There are a number of different traditions in psychology, economics, and related disciplines to describe and formalize decision-making processes. The perhaps most prominent approach comes from expected value theory and related views, which describe decision making as the computation of expected value, that is, the weighting of the value of possible outcomes by their probability of occurrence (Bernoulli 1954). Variations of this principle introduce the idea of subjective value/utility and probability functions that may vary as a function of individual characteristics or situations, but such approaches typically do not make reference to the specific cognitive architecture underlying these calculations. One example of such a theory is cumulative prospect theory (Tversky and Kahneman 1992). A second approach, strongly anchored in cognitive science and psychology, tries more explicitly to link descriptions of decision processes to cognitive functions of attention, learning, and memory. The models associated with such an approach may also describe the computation of value and probability but often will make stronger assumptions about information search and updating of information. For example, sequential-sampling and reinforcement-learning models make assumptions about information sampling and updating to describe decision making. Other related approaches attempt to describe decision making at the algorithmic level by making assumptions about the series of computational steps required for search, stopping, and deciding (Gigerenzer and Brighton 2009). Third, and finally, a more recent approach investigates the neural basis of decision making, often making links to some of the formal and computational theories described above (Glimcher and Fehr 2014).

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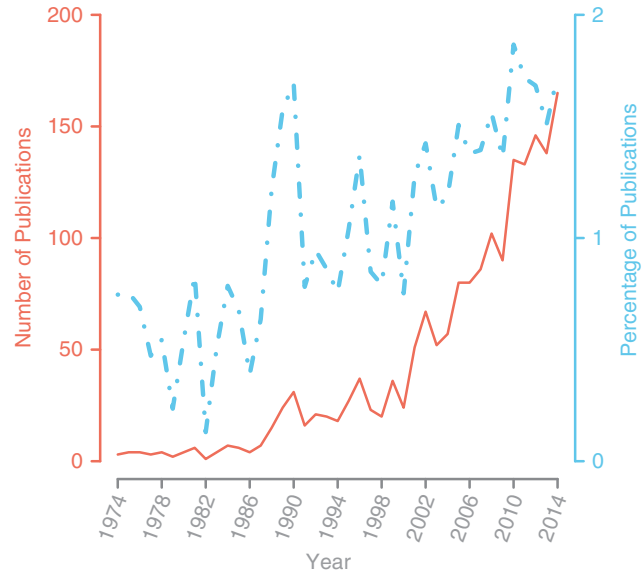

Decision Making, Fig. 1 Depiction of an estimate of the number of publications on aging and decision making as well as the corresponding percentage relative to all publications on decision making in the past 40 years. The search was conducted using the terms "aging" and "decision making" in *all fields* in PubMed

The question of how decision making changes across the life span and, in particular, with aging, has received some attention. Yet, the field of aging and decision making represents less than 2% of all work in decision making, albeit there has been an increase in the relative proportion of decision research focusing on aging (see Fig. 1).

Key Research Areas

There are a number of reviews on aging and decision making that emphasize how different aspects of age-related change can influence decision making, including affective and motivational (Samanez-Larkin and Knutson 2015), as well as cognitive and ecological, factors (Mata et al. 2012a). A complete understanding of the impact of aging on decision making will likely require the integration of different perspectives (Hess et al. 2015). Crucially, the ecological perspective suggests that there is no domain-general answer to the question of how changes in motivational or cognitive capabilities impact decision making. For example, the impact of age-related cognitive decline should depend strongly on the demands of specific task environments, such as memory demands. In other words, the quality of decisions made by people of all ages is the result of how task demands and affordances interact with particular cognitive strategies. To better understand this interaction, one needs to describe the structure of decision environments and the cognitive or affective components that such environments exploit (Mata et al. 2012a). In what follows, the specific key research areas that have received most attention in the past are described with the goal of exemplifying the importance of age-related changes in cognitive and motivational components and the moderating role of ecological (i.e., task) characteristics on decision making.

Perceptual Decision Making

Perceptual decision making refers to low-level decisions about immediately presented stimuli, with most research on aging having been carried out on visual discrimination, such as discrimination between different letters (E vs. F) or varying levels of brightness in stimuli presented very briefly on a computer display (e.g., for less than 1 s). The state-of-the-art approach is to use sequential-sampling models (e.g., diffusion models) to describe both reaction times and accuracy of responses simultaneously and account for possible speed–accuracy trade-offs. The latter models are able to distinguish between different components, such as motor components, response criteria components, and evidence accumulation

components, because each makes different predictions about the shape of the reaction time distribution of correct and error responses. Key results in this area are that older adults do seem motivated to perform well and show motivational adaptations by adopting more conservative decision criteria than the younger adults. Older adults are also overall slower in noncognitive (e.g., motor) components. However, the quality of evidence accumulation driving the decision process is significantly lower for older relative to younger adults in some but not all tasks (Ratcliff et al. 2007), suggesting that task characteristics, such as the nature of the stimuli, can be crucial in engendering age differences in perceptual decision making. The extent to which the results from perceptual decision making can be directly translated or are correlated to performance in higher-level decisions is yet to be investigated.

Multiple-Cue Decision Making

The bulk of decision-making research has focused on problems in which decision makers have to integrate different pieces of information (i.e., cues, attributes) and deliberately decide between two or more options. The nature of information presentation may vary dramatically from those cases in which it is conveniently summarized in a table or, alternatively, needs to be retrieved from memory. A major distinction in this field concerns the existence of an objective criterion that determines the correctness of the decision, such that decisions amount to inferences, and those cases for which no objective criterion exists – decisions thus represent expressions of individual preferences. Some work on aging and multiple-cue decision making has artificially created objective criteria and examined the strategies selected by younger and older adults in inferential decision making (Mata et al. 2007). Strategies can be defined as sequences of operations or processes that are goal directed, that is, are aimed at accomplishing a particular task and, therefore, mediate task performance. The strategy concept has been used to describe cognitive processes and mechanisms of human cognition in many domains, including memory, arithmetic, and decision making. In particular, the strategy approach has a long tradition in decision research with various strategies having been proposed, each with its particular cognitive demands and domain of execution (Shah and Oppenheimer 2008). For example, some strategies, like take-the-best (TTB), ignore significant amounts of information because they infer which of the two alternatives has the higher value on a criterion by (a) searching through cues in order of validity (i.e., how much the cue is correlated with the criterion), (b) stopping search as soon as a cue discriminates between decision alternatives, and (c) choosing the alternative this cue favors. In turn, other strategies, like weighted-additive strategy (WADD), consider all information by (a) multiplying each cue value by the respective cue weight (i.e., a measure of how important this cue is to the prediction), (b) summing up the results for each alternative, and (c) choosing the alternative with the highest sum. TTB does well in environments with many redundant cues or in which search is costly, the opposite being true for WADD. In the inference domain, there is some inherent difficulty in distinguishing age-related changes in preferences from the impact of age-related cognitive decline on the selection and application of specific decision strategies that impact the search and integration of information. Overall, work on inferential decision making suggests that older adults tend to search for less information prior to making a decision (Mata and Nunes 2010) and use simpler strategies that ignore some information (Mata et al. 2007) or strategies that do not rely heavily on memory (Mata et al. 2012b) to integrate information, possibly due to age-related cognitive decline. There are, however, other aspects related to age-related changes in motivation and preferences that can affect decision making. The work on the impact of aging on preferential decisions is summarized below for a number areas in which age differences have been investigated, such as in the domains of risky, intertemporal, and social decision making.

Risky Decision Making

Conceptions of risk and risk taking abound, with economists viewing risk as the variance or probability of possible outcomes, whereas

psychologists and lay people often emphasize the link between risk and the possibility of losses. There are a number of measures and tasks that attempt to capture individual and age differences in the tendencies to decide for or against risky courses of action. Some behavioral tasks provide explicit information about outcome magnitudes, whether outcomes are positive (gains) or negative (losses), as well as their respective probabilities (decisions from description), but others require individuals to learn about probability and outcome information over time (decisions from experience). A review of the literature suggests that the pattern of age differences in risky decision making is heterogeneous and may depend heavily on task characteristics (Mata et al. 2011). In particular, in decisions from experience, age-related differences in risk taking seem to be a function of decreased learning performance: Older adults may be more (or less) risk seeking compared to younger adults depending on whether learning leads (or not) to risk-avoidant behavior. In decisions from description, younger and older adults may show more similar risk-taking behavior at least when the cognitive demands of the task are low. The exact decision strategies used by younger and older adults in these tasks have not been fully investigated, thus making it difficult to disentangle the role of age-related changes in risk preferences due to motivational factors and more cognitive factors, such as the changes in strategy use due to age-related cognitive decline (Depping and Freund 2011). One interesting avenue for future research is to investigate which behavioral tasks can best capture the underlying risk preferences of older adults as captured in commonly used self-report measures.

Intertemporal Decisions

Many important life decisions require trading off immediate rewards against future ones, such as the choice between spending and saving. This type of decisions is typically studied empirically using monetary decisions between smaller-sooner and larger-later amounts of money, for example, $10 today or $20 in 1 week. Overall, humans seem to deviate from the assumption of economic theory, which assumes exponential discounting (i.e., constant discounting per time period). Instead, humans typically show a present bias by showing valuations that fall rapidly for small delay periods, but slowly for longer delay periods. The empirical literature concerning age differences in intertemporal decisions is mixed (Rieger and Mata 2015; Samanez-Larkin et al. 2011). However, the majority of existing studies seem to suggest a pattern, whereby older adults are more patient, by showing choices indicative of either less steep discounting rates or increased neural responses to later rewards (Samanez-Larkin and Knutson 2015).

Social Decision Making

Aging is traditionally perceived as being associated with increased wisdom, including an increased ability to navigate the social world. But do older adults deal more or less strategically and prosocially relative to younger adults in social contexts? Standard economic theory assumes that people are, perhaps exclusively, motivated by material self-interest and thus do not care about the well-being of others. A number of studies have rejected this assumption and suggested that individuals of all ages have prosocial motivations (Engel 2011). The typical measures used to assess prosocial motivations consist of having individuals (i.e., players) make decisions in the context of groups in which other individuals may or not be anonymous. For example, individuals may be asked to allocate monetary amounts to themselves and others, with the amount assigned to the social partner being used as an indicator of prosocial motivation. The economic games include the dictator, ultimatum, and trust games, among many others (Rieger and Mata 2015; Engel 2011). The literature on age differences in social preferences in such games is relatively scarce and most work has only considered age as a nuisance variable. Some work does suggest an increase in prosocial motivations with increased age (Engel 2011), but there is evidence that such patterns may not hold across tasks or populations (Rieger and Mata 2015). More empirical studies investigating a more culturally and age-diverse set of participants are still needed to characterize age differences in such strategic social interactions.

Another area of research that has received some attention concerns decision making and problem solving in a social context. Such abilities are typically assessed by tallying the number and quality of solutions participants generate to deal with everyday problems, for example, the course of action to take under financial distress or how to handle a social conflict between members of a couple. A meta-analysis of such problems concluded that there is a decline in effectiveness of everyday decision making (Thornton and Dumke 2005), with a medium effect size difference between younger and older adults (Hedge's $g \approx 0.4$). However, moderator analyses revealed that age differences were reduced when problems were of a social nature (Hedge's $g \approx 0.2$). Overall, the literature suggests that there may be changes in social decision making, but it remains an open issue whether these are due to motivational or cognitive factors.

Future Directions

An important avenue for future work concerns evaluating the consequences of basic age-related changes in motivational and cognitive changes on real-world decisions. There is some evidence for an inverted-U-shaped function of age and financial decision making in the real world (Agarwal et al. 2009). Yet, the exact mechanism for this pattern remains unknown, and it could be a function of age-related changes in financial knowledge, numeracy, fluid cognitive abilities, motivation, or all of the above. Prospective and longitudinal studies of decision making that include real-world outcomes are yet to be conducted but are sorely needed to distinguish such different possibilities.

The area of consumer decision making provides an interesting future test-bed for different theories because it likely conflates different influences, including cognitive and motivational factors (Yoon et al. 2009). One important principle to keep in mind in such work is that age-related cognitive decline may not always be associated with poor choice outcomes. For example, simple strategies that ignore information can lead to satisfactory choices in many real-world consumer environments (Mata and Nunes 2010).

In sum, aging seems to be associated with changes in decision making, from simple perceptual decisions to complex social ones. The motivational and cognitive mechanisms leading to such changes still need to be uncovered, as do the task characteristics that foster or hinder successful choices by aging decision makers.

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Cross-References

- ▶ Aging and Strategy Use
- ▶ Decision-Making Capacity in Older Adults, Overview of
- ▶ Everyday Cognition
- ▶ Risk Taking in Older Adulthood
- ▶ Working Memory in Older Age

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Decision-Making Capacity in Older Adults, Overview of

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Synonyms

Assessment; Competency; Decision-making

Definition

The necessary cognitive and functional abilities required to perform a specific task or make a specific decision.

Introduction

The term *capacity* refers to a person's ability to perform a specific task or make a specific decision. Determinations of capacity have historically been made by clinicians in clinical settings. This is in contrast to the legal term *competency*, which is a determination made by the court. At times these terms have been used interchangeably; however, for the purposes of this section, we henceforth use the terms "clinical capacity" and "legal capacity."

Psychologists are increasingly called upon to make determinations of capacity. The reasons for this are multifactorial. It is widely cited that the number of older adults worldwide has grown exponentially. And while not all older adults develop dementia, they may experience physical and mental changes as they age that place them at risk for impaired capacity. There has also been shift of wealth from the World War II generation to the baby boomers and now to the Generation X, in increasingly diverse families that are separated geographically. Consequently, probate courts are seeing an increase in contested wills and guardianship proceedings (Moye and Marson 2007). The probate law has also shifted from a global and absolute view of capacity towards a more task- or decision-specific standard of capacity, recognizing a person can have capacity in one area but not another. Thus to meet the current legal standard of capacity, the capacity evaluation has also evolved to include neurocognitive, psychological, and functional assessments. Psychologists are often trained in these assessments thus are well suited to conduct capacity evaluations (Lichtenberg et al. 2015).

Yet while psychologists may have the appropriate training to address the functional, cognitive, and mental health components of the evaluation, they are often less familiar with the term capacity or the interventions available to persons with

diminished capacity. To further complicate the issue, professionals who often work with the probate laws surrounding capacity (i.e., lawyers and judges) may be less familiar with the unique needs of and challenges in working with older adults. So in 2003, the American Bar Association (ABA) and the American Psychological Association (APA) formed a workgroup to develop educational materials and handbooks for lawyers, judges, and psychologists. The intent of this workgroup was to provide a framework for professionals to draw upon in capacity determinations as opposed to more rigid standards of practice. The workgroup produced the first handbook, *Assessment of Older Adults with Diminished Capacity: A Handbook for Lawyers*, in 2005. The second handbook, *Judicial Determination of Capacity of Older Adults in Guardianship Proceedings: A Handbook for Judges*, was published in 2006. The final handbook, *Assessment of Older Adults with Diminished Capacity: A Handbook for Psychologists*, was published in 2008. These handbooks are available online at www.apa.org/pi/aging and www.abanet.org/aging (American Bar Association and American Psychological Association Assessment of Capacity in Older Adults Project Working Group 2008).

The following sections will detail the evolution of the legal capacity and guardianship laws; the core ethical principles inherent in decision-making capacity; the requisite functional abilities for determinations of capacity; the role of culture in capacity evaluations; a framework for capacity evaluations; and future directions for the field.

Evolution of Legal Capacity and Guardianship

Over the past 65 years, the legal aspects of capacity evaluations have undergone significant change in the United States and internationally. In essence, there has been a shift towards increased autonomy and limited guardianship, resulting in various legal reforms. Historically, the concept of capacity was global and absolute in that a person deemed incapacitated would have his or her legal rights revoked in a broad range of legal domains (American Bar Association and American Psychological Association Assessment of Capacity in Older Adults Project Working Group 2008). This has shifted over time and current legal practice uses the term "capacity" to refer to a person's ability to complete a specific task or decision (Bailar-Heath and Moye 2014), thus recognizing that a person can have capacity in one area (e.g., medical decision-making) but not another (e.g., driving). Another relatively recent change has replaced the term "incompetency" with the term "incapacity" as the determination of these have evolved to integrate clinical findings into legal findings in a multidisciplinary manner (American Bar Association and American Psychological Association Assessment of Capacity in Older Adults Project Working Group 2008). This shift is in keeping with the view of capacity as being decision relevant, which holds that judgments of capacity are for specific abilities at specific time in a specific context and relevant to a specific decision (Buchanan and Brock 1989).

The past few decades have yielded significant reform in legal practice pertaining to adult guardianship in the United States. The ABA defines guardianship as legal decision-making power given to an outside entity or person in response to a determination of incapacity. The term itself is often used interchangeably with "conservatorship" depending on the state or country in which the determination is being made and may be used in reference to guardianship of property specifically (American Bar Association and American Psychological Association Assessment of Capacity in Older Adults Project Working Group 2008; Bailar-Heath and Moye 2014). Criteria for guardianship fall under state and not federal regulation, and thus there is variability across states in how guardianship determinations are made. These decisions are defined by either statutory or case law and are transaction specific. Examples of transaction-specific capacity include testamentary capacity, donative capacity, contractual capacity, capacity to execute a durable power of attorney, and capacity to consent to medical care. Despite the inconsistency in legal definition, basic guidelines for determining diminished capacity include disabling condition, functional behavior, cognitive functioning, and consideration of the least restrictive alternative. These guidelines are commonly expanded upon by state law (American Bar

Association and American Psychological Association Assessment of Capacity in Older Adults Project Working Group 2008).

As part of the recent capacity reforms, 32 states have passed comprehensive reform bills, and 261 separate capacity laws have been passed. Currently, more than 30 states now require clinical evaluation for capacity to determine whether an adult may qualify for guardianship (Bailar-Heath and Moye 2014). Further, the majority of US states have done away with the global and absolute determination of incapacity in favor of the relatively recent model of limited guardianship. In the limited guardianship model, a guardian is appointed on for the areas in which the person has been deemed to lack capacity (American Bar Association and American Psychological Association Assessment of Capacity in Older Adults Project Working Group 2008). In determining whether a person is in need of guardianship, all states begin with the assumption of capacity and put the burden of proof on the party attempting to establish guardianship. In other words, every person is assumed to have capacity until established otherwise.

In contrast to the variability of laws pertaining to capacity determinations based on state jurisdiction in the United States, international law offers a more unifying standard. Since the 1960s, guardianship law has been of particular concern in many countries. In 2006, the Convention on the Rights of Persons with Disabilities (CRDP) marked a major international milestone in the legal rights of persons deemed to have diminished capacity. The CRDP brought significant changes to the laws of member nations to protect the legal rights, status and autonomy of incapacitated adults, and to provide support to this legally vulnerable population (Bailar-Heath and Moye 2014). Among other things, these changes mark an increase in the emphasis on autonomy and independence, cultural sensitivity, and the consideration of least restrictive alternatives (Doron 2002).

Within the broader legal standards outlined in the CRDP, the laws regarding capacity determinations vary by country. Canada, the United Kingdom, and Portugal define incapacity using the benchmark of an individual's ability to care for "person or property" (Bailar-Heath and Moye 2014). In Germany, the guardianship model has shifted focus towards a "care and assistance" model that allows the individual to be appointed with a caretaker who carries out specific tasks as defined by the court, protecting the incapacitated individual from losing his or her legal rights. Sweden offers two possibilities for legal support for incapacitated persons. In both, the individual is appointed an administrator, mentor, or trustee that is responsible for assisting him or her. Additionally, the person may forfeit legal capacity in specified domains only or may not lose any of his or her legal rights. Israel is gradually transitioning from guardianship laws that eradicate the legal rights of the individual in all domains, to the appointment of a guardian and retention of legal competence (Bailar-Heath and Moye 2014).

In Hong Kong, the courts that determine guardianship appointment are multidisciplinary panels made up of lawyers, someone who has personal experience with incapacitated individuals, and either physicians, psychologists, or social workers. The structure of the panels in Hong Kong illustrates the shift towards a psycholegal construct of capacity that has been the recent trend in numerous countries. Similarly, Australian guardianship tribunals include members of the community as well as legal professionals, and various provinces in Canada require collaboration of legal and clinical professionals (Bailar-Heath and Moye 2014).

The legal aspects of capacity evaluations are complicated by variance in laws according to country and state jurisdiction. However, recent reforms both in the United States and internationally point to a shift in consciousness towards providing those who are legally determined to be incapable of safely making decisions about their person or property the least restrictive guardianship and preserving many of their legal rights. By including clinical assessments in legal proceedings that determine capacity, the system is beginning to allow for a more holistic view of the individual's abilities as opposed to the historically broad revocation of legal rights.

Principlism in Health Care

The concept of capacity spans the fields of health care and law. Yet health-care ethics is a third area which is central to the concept of capacity. Principlism is system of ethics deployed in health care that based on four moral principles of: (1) respect for autonomy, (2) beneficence, (3) nonmaleficence, and (4) justice.

The principle of respect for autonomy, also referred to as self-determination, is the person's ability to make his or her own decisions. This principle is rooted in the longstanding belief of the importance of personal freedom and individualism. Health-care providers are tasked with ensuring that autonomous decisions are intentionally made, with substantial understanding, and free from coercion (Beauchamp 2007; Beauchamp and Childress 2011).

Beneficence may be viewed as a group of principles that both prevents harm and also provides benefits that outweigh costs and risks. This principle reflects the moral obligation to act for the benefit of others and is often considered a foundational value in health-care ethics. It could be argued that the obligation to promote patient welfare is of the utmost importance in medicine. Beneficence obligates health-care providers to assist older adults in furthering their interests, often by removing or minimizing risk and harm to the patient (Beauchamp 2007).

The principle of nonmaleficence prevents providers from causing harm to others, or put simply is the "do no harm" principle. While beneficence includes the prevention of harm or reduction of risk for the ultimate benefit of the patient, nonmaleficence is the intention to avoid unnecessary harm or injury to the patient. As noted by Beauchamp (Beauchamp 2007), nonmaleficence is one of the most frequently cited codes in health-care ethics. Some have advocated to place the greatest emphasis upon this obligation, even if that is to the detriment of other obligations, including the respect for autonomy. Nonmaleficence obligates providers to inflict the least amount of harm to achieve a beneficial outcome.

Finally, the principle of justice requires the fair distribution of benefits, costs, and risks (Beauchamp 2007). Put another way, this principle obligates providers to act on the basis of unbiased decision-making in the face of competing claims. Thus the principle of justice extends beyond equitable access to treatment, as it obligates providers to be aware of their own biases to ensure the fair distribution of health-care resources.

These four principles are not hierarchical; thus clinicians have an obligation to uphold each of them. While this is the ideal, ethical clinical practice dictates that the clinician must examine the balance of these principles by examining their respective weights on a case-by-case basis. Thus to weigh the respective weights is to carefully evaluate the risks involved in the situation. There are no hard and fast rules that dictate that one principle take precedence over another. To further complicate the matter, different professionals may place a greater emphasis on different principles. When faced with the same clinical case, providers may recommend different treatments based on their evaluation of the potential risks and benefits involved. For example, a provider may recommend an older adult with a history of falls and mild cognitive impairment be discharged to home with the assistance of home health aides in order to promote the respect for the patient's autonomy. However, another provider, when presented with the same clinical scenario, may recommend the patient be discharged to an assisted living facility in order to promote the principle of beneficence (i.e., prevent the patient from sustaining future harm). As is highlighted in this example, determinations of capacity are often a balancing act between these foundational principles of health-care ethics.

Essential Functional Abilities

Assessment of functional abilities is a core component of capacity evaluations. In the field of geropsychology, the concept of functional abilities often refers to a person's ability to perform activities of daily living (i.e., bathing, grooming, eating) and instrumental activities of daily living (i.e., shopping, bill payment, household chores). Yet in capacity evaluations, the legal concept of functional abilities also refers to the intact decisional abilities that are generally agreed to convey capacity (Lichtenberg et al. 2015). These abilities,

which are drawn from case law, include *understanding*, *appreciation*, *reasoning*, and *expressing a choice* (Grisso 2003; Smyer 2007).

In the context of capacity assessments, *understanding* refers to the ability to comprehend the nature of a proposed decision, including an awareness of its risks and benefits. The ability to adequately understand a proposed decision is impacted by several factors including the person's intelligence, educational level, and the method by which the information is presented to them. The risks and benefits of a decision must be presented to a person in a manner that promotes their understanding.

While there are different interpretations of *appreciation*, it is generally thought to refer to the ability to understand the relevance or applicability of a decision to the older adult. At the most basic level, older adults must recognize that a decision must be made, that they are the decision-maker, and it is their life that will be affected by the decision. Thus it is not surprising that appreciation is greatly impacted by the degree of patient insight, as well as the type of decision to be made and the complexity of that decision.

Reasoning entails the process of rationally comparing different treatment options or proposed solutions in a consistent manner. Older adults must demonstrate that they can weigh the risks and benefits of the proposed choices as well as the possible consequences. The ability to reason directly impacts understanding and appreciation. If a person cannot rationally reason or logically manipulate the presented information, it is not possible to fully understand or appreciate the issues in the decision (Grisso and Appelbaum 1998).

Older adults must also be able to *express a choice*; those who are unable to outwardly communicate a choice or who waver in their choice are seen as lacking capacity (Lichtenberg et al. 2015; American Bar Association and American Psychological Association Assessment of Capacity in Older Adults Project Working Group 2008; Grisso 2003). That choice should be consistent with the person's value or beliefs; however, it is accepted that a person's value and beliefs may change over time. The importance of expressing a choice should not be minimized as there are situations in which a person may be able to understand, appreciate, and rationally reason about a decision; however, due to a physical condition, such as stroke, is unable to express a choice. In situations such as this, it is impossible to know what that person's preference or desire would be.

In addition to these four functional abilities, *the role of values in the determination of capacity cannot be overstated.* The ABA and APA Assessment of Capacity in Older Adults Project Working Group (American Bar Association and American Psychological Association Assessment of Capacity in Older Adults Project Working Group 2008) defined values as "an underlying set of beliefs, concerns, and approaches that guide personal decisions." This definition is useful as it not only defines values but also highlights the relationship between values and decision-making. As highlighted in Moye (Moye 2007), the "extent to which a person's expressed choice is consistent with their values is an indicator of capacity"; thus, it is an essential component to the assessment of these functional abilities. An understanding of a person's values will also greatly assist in the development of appropriate treatment recommendations. Providers should also be aware of their own values so that any inherent biases regarding the decision at hand can be appropriately addressed.

The Role of Culture in Capacity

In addition to being one of the fastest growing segments of society, older adults are one of the most culturally diverse groups. That diversity is projected to continue to expand in coming years as evidenced by recent US census data. In 2014, 14% of the adults in the United States were age 65 or older. A closer analysis of this census data reveals that within this older segment of the US populace, approximately 1 in 7 (14%) sampled identified as a racial minority. That percentage of racial minorities is projected to steadily grow to 18% by 2030 and 23% by 2050 (U. S. Census Bureau 2014). As noted by Karel (2007), within these racial groups are further subgroups (denoted by their countries of origin) with their own values and beliefs. These values and beliefs are often the

foundation for their views on aging, health care, family and familial roles, finances, and end-of-life. Among older White Americans, there are further ethnic, regional, and religious subgroups. And not surprisingly, these subgroups have strong values and beliefs that influence their views on many of the abovementioned issues.

There are also cross-cultural differences within the aforementioned principles of health-care ethics. In Western cultures, the principle of respect for autonomy or self-determination is strongly valued. This is evident in the widespread use of advance care directives and durable powers of attorney, which are designed to foster patient autonomy in situations where patients are unable to make their own decisions. This emphasis on patient autonomy is unique to Western cultures, as other cultures encourage collective decision-making that involves the patient's community and family. In cultures that value beneficence, providers are obligated to encourage hope above all else. This is contrasted with those cultures that value nonmaleficence, in which providers protect patients from harm by not directly addressing seemingly negative outcomes such as death or end-of-life (Searight and Gafford 2007).

Conceptual Framework of Capacity

The ABA-APA Working Group on the Assessment of Capacity in Older Adults detailed a nine-part framework for conceptualizing capacity assessments (American Bar Association and American Psychological Association Assessment of Capacity in Older Adults Project Working Group 2008). The model builds off of the frameworks for guardianship as well as the framework for capacity assessment previously developed by Grisso (American Bar Association and American Psychological Association Assessment of Capacity in Older Adults Project Working Group 2008; Grisso 2003; Moye 2007). Components of the nine-part framework proposed by the ABA-APA work group includes the identification of: the relevant legal standards, functional abilities of capacity, relevant medical or psychiatric diagnoses contributing to incapacity, cognitive function, psychological and emotional factors, values and preferences, risks to the individual and of the situation, ways of enhancing capacity, and a clinical judgment of capacity.

While a capacity assessment is a clinician's opinion about a person's ability to perform a specific task or make a specific decision, that task or decision has a specific legal standard. A clinical judgment regarding a person's capacity can then have a direct impact on that person's legal rights henceforth. Therefore, a familiarity with the legal standard is a requisite initial step in the approach to a capacity assessment. The expectation here is not that a provider becomes an expert in the legal standards surrounding the capacity in question, but more that the provider becomes familiar with the legal standard. This can be accomplished through a review of a state's statutory or case law or through a consultation with an attorney. Information gleaned from this review or consultation should be then used to guide the selection of the assessment battery, so as to ensure all aspects of the legal standard are met (American Bar Association and American Psychological Association Assessment of Capacity in Older Adults Project Working Group 2008). It should be noted that from the legal perspective, all persons are presumed to have capacity until proven otherwise.

The ABA-APA framework builds off of the previous work by Grisso (1986) to expand the concept of "function" to also include the identification and evaluation of the functional elements essential to the questioned capacity. Capacity assessments should include a tailored evaluation of the specific task or specific decision which can be accomplished through specific questions in a clinical interview as well as through direct assessment or observation of the person's functioning (American Bar Association and American Psychological Association Assessment of Capacity in Older Adults Project Working Group 2008). This portion of the capacity assessment will vary based upon to the type of decision-making capacity being assessed. For instance, if the assessment is one of financial decision-making, the provider should include a structured assessment of financial decision-making. If the assessment were one of testamentary capacity, the provider should include specific questions in the clinical interview designed to demonstrate a person's ability to

describe a will, to describe the nature and extent of one's assets, to name potential heirs, and to describe plans for distribution of one's wealth. This focus on functional abilities specific to the task or decision to be made is a defining feature of the capacity assessment.

The purpose of establishing or documenting a diagnosis in the capacity assessment is to identify a possible "causal factor" for potential incapacity (Grisso 2003). Older adults are vulnerable to many physical and psychiatric illnesses that may impact capacity including dementia, delirium, neurodegenerative disease (e.g., Parkinson's, Alzheimer's), stroke, and many more. Yet these conditions can have markedly different long-term outcomes, thus it is important to recognize the role of the prognosis of the condition in judgments of decision-making capacity. For instance, Alzheimer's disease is a progressive neurodegenerative disorder for which there is no cure. This is contrasted with delirium, which is a life-threatening medical condition in which a person's cognition can rapidly fluctuate, though with medical intervention can fully resolve. In both of these conditions, patients will have impaired decision-making ability. Yet in the case of delirium, patients are often able to fully recover decision-making abilities while those patients with Alzheimer's disease are not likely to regain their decision-making ability (American Bar Association and American Psychological Association Assessment of Capacity in Older Adults Project Working Group 2008). While a diagnosis can serve as a causal factor for the impaired decision-making, it can also serve as a prognostic indicator as to if capacity is likely to be regained. Yet a medical or psychiatric diagnosis by itself is insufficient to establish a patient's decision-making capacity as patients with impaired cognitive function due to a medical or psychiatric disorder may still retain the ability to make some decisions. Thus the focus should not be on the presence of the diagnosis but on the influence of the diagnosis on the person's decision-making.

Most states include a comment on a person's cognitive function as a necessary element in the determination of capacity. The causative role that many diagnoses have on decision-making is often through their effect on cognitive functioning. Impaired cognitive functioning can result in impaired insight or impairment in the cognitive abilities necessary to perform a specific task or make a specific decision. This portion of the capacity assessment should include assessments designed to comment directly on the cognitive functions necessary to perform a specific task or specific decision, in addition to measures of overall cognitive function. For instance, assessments of financial capacity may include measures of written arithmetic whereas an assessment of driving capacity may include measures of visual attention and processing speed (American Bar Association and American Psychological Association Assessment of Capacity in Older Adults Project Working Group 2008). As with the determination of medical and psychiatric diagnoses, the purpose of the cognitive assessment is to characterize the level and nature of cognitive impairment and determine if (and how) the decision-making process is impacted by cognitive status.

Similar to the cognitive assessments, the purpose of the screening for symptoms of mental health disorders is to detect possible underlying factors that may impact a person's decision-making ability. Mental health disorders, like psychotic spectrum disorders and severe mood disorders, can impair a person's insight and ability to rationally weigh the risks and benefits of the proposed choices as well as the possible consequences. Many mental health disorders are amenable to intervention which presents with greater likelihood that the patient will regain decision-making ability (American Bar Association and American Psychological Association Assessment of Capacity in Older Adults Project Working Group 2008). It should be noted that many patients with clinically significant mental health symptoms are not captured by strict criteria-based diagnostic categories (Lyness et al. 2015), thus again the focus of these measures is not just diagnosis but to comment on the impact of the mental health symptoms on cognitive and functional abilities relevant to the questioned capacity.

As aforementioned, values are the beliefs, concerns, and experiences that directly inform one's decisions. The ABA-APA handbook

distinguishes values from preferences, as former refers to "preferred option of various choices that is informed by values." Assessment of values and preferences is an essential component to a capacity assessment as one of the requisite functional abilities is the expression of a choice that is consistent with a person's values. It should be noted that values and preferences can change over time thus a change in person's values may not represent impaired decision-making capacity (American Bar Association and American Psychological Association Assessment of Capacity in Older Adults Project Working Group 2008). In addition to conveying capacity, knowledge of a patient's values and preferences can assist in the development of effective treatment recommendations that are more likely to be accepted by the patient.

Some have argued that at its most basic, a capacity evaluation is a type of risk assessment (American Bar Association and American Psychological Association Assessment of Capacity in Older Adults Project Working Group 2008; Moye 2007; Ruchinskas 2005). The provider must consider all available data, including medical and psychiatric diagnoses, cognitive and functional impairment, and patient values and preferences, in the context of the risk of the situation. In addition to evaluating the patient in terms of the risk of the situation, the provider should also take account of social and environmental supports, as these may serve to mitigate or exacerbate the initial risk (American Bar Association and American Psychological Association Assessment of Capacity in Older Adults Project Working Group 2008; Moye 2007). For instance, the discharge of an older adult with limited mobility to independent living would carry more risk if that older adult lived in a two-level home and had no immediate family in the area to provide assistance. Those risks would be mitigated, however, if the older adult had the financial means to install a stairway lift and employ regular home health aides to assist him. Thus these risks were mitigated with effective interventions to enhance the older adult's capacity. All recommended interventions should match the level of risk in the situation so to ensure the deployment of the least restrictive means necessary (American Bar Association and American Psychological Association Assessment of Capacity in Older Adults Project Working Group 2008).

All capacity evaluations should include considerations of what can be done to maximize a patient's functioning. As noted by Moye (2007), many of these recommendations are practical in nature and include things such as hearing or visual aids or medication management systems. Other interventions may include work with occupational or physical therapy as well as additional training or counseling. Efforts to maximize patient functioning represent opportunities for potential clinically impactful interventions (American Bar Association and American Psychological Association Assessment of Capacity in Older Adults Project Working Group 2008).

In the final component of the ABA-APA framework, the provider takes into consideration all of the data gathered through the capacity evaluation and provides a clinical opinion regarding the patients questioned capacity. This clinical opinion is oft expected to be presented in the form of a dichotomous conclusion (e.g., yes or no). There will be situations in which the determination of capacity will be clear based upon the available information such as when a patient is grossly impaired across multiple cognitive and functional domains or is unable to express a choice due to significant neurological impairment. Other decisions are more complex due to varying levels of impairment across multiple domains. In situations such as these, providers are encouraged to review the available data in the context of the patient's values and preferences as well as any environmental supports or risks (American Bar Association and American Psychological Association Assessment of Capacity in Older Adults Project Working Group 2008).

Conclusion

The rapid global growth of older adults has compelled geropsychologists to gain the requisite knowledge and skills to address issues surrounding capacity. This topic is particularly relevant to geropsychologists as these professionals

understand the physical and mental changes that occur in late life and can often increase the risk of impaired capacity. Geropsychologists are also trained in the psychological, neurocognitive, and functional assessments that are included in the capacity assessment. Yet while geropsychologists have the clinical expertise, they are often less familiar with the legal standards required to determine capacity. The legal and health-care fields continue to evolve in their definitions of capacity as the focus has shifted towards one that recognizes capacity to be decision and domain specific as opposed to a global judgment of ability. Geropsychologists may find capacity evaluations to be a type of risk assessment that requires the balancing of the four moral health-care principles, which requires an understanding and appreciation of the role of culture on these principles. While there are no current "gold standards" for the assessment of capacity, there are conceptual frameworks as well as other assessment-specific tools available to assist those in evaluations such as these. Psychologists who work with older adults are encouraged to explore these frameworks and suggested assessments as they move towards achieving competency in assessment of decision-making capacity.

Cross-References

- ▶ Clinical Issues in Working with Older Adults
- ▶ Cognition

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Delirium

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Synonyms

Confusion; Derangement; Irrationality; Hallucination

Delirium is characterized by an acute change in an individual's mental state, marked by fluctuating patterns of confusion and inattention. It is unfortunately encountered by nurses, family, and physicians, both in hospital and home-based care settings. Delirium can affect individuals of any age, though it is more frequently experienced by older individuals. It raises the risk of mortality, causes distress to both patients and caregivers, and increases health care expenditures. It is an acute condition that may present independently or in combination with other dementia syndromes; therefore, accurate diagnosis and timely treatment are imperative. In this entry, the authors discuss delirium in the general hospitalized patient, then subsequently focused on postoperative delirium for major orthopedic and cardiac surgeries that have the highest rates of delirium-affected individuals.

Delirium Defined

Delirium is an acute and temporary change in orientation and cognition. The Diagnostic and Statistical Manual of Mental Disorders (DSM-V; American Psychiatric Association and American Psychiatric Association, DSM-5 Task Force 2013, www.dsm5.org) provides a description of delirium with subtypes. These include delirium due to an underlying medical condition (*delirium due to a medical condition*), medications (*substance-induced delirium*, *substance intoxication delirium*), or withdrawal from medications (*substance withdrawal delirium*). Delirium can also be multifactorial (*delirium due to multiple etiologies*) (see Table 1 for criteria and descriptor information; ICD-10 criteria are presented in Table 2). Delirium can be present at hospital admission and presurgically, although it is more often seen in postsurgically managed general medical units and most frequently in intensive care units (ICUs).

General Characteristics of Delirium

Delirium characteristics can vary by individual. Most common is fluctuating arousal with waxing and waning awareness of orientation. It is often accompanied by altered sleep-wake cycle and reversed night cycles. Hallucinations and delusions are common. Variability can be seen in activity levels; however, patients can present with hyperactive, hypoactive, or mixed hyper-hypo active cognitive and motor states. Hyperactive patients show increased psychomotor activity, such as rapid speech, irritability, and restlessness. These patients can be disruptive, time-consuming, and harmful to staff. They are therefore more readily identified and treated. Hypoactive patients, by contrast, typically show a calm appearance combined with inattention, decreased mobility, and have difficulty answering simple questions about orientation. Due to their calm appearance, these individuals are unfortunately less readily identified with delirium and may be inappropriately treated (Peritogiannis et al. 2015).

Significance of Delirium

Although a temporary condition, delirium is a medical and societal stressor from an economic and healthcare standpoint. Delirium occurs in at least 10–24% of the general patient population,

Delirium, Table 1 The following criteria are derived from the *Diagnostic and Statistical Manual of Mental Disorders*, 5th ed. All criteria (A-E) are required for diagnosis

DSM-V diagnostic criteria for delirium
(A) A disturbance in attention (i.e., reduced ability to direct, focus, sustain, and shift attention) and awareness (reduced orientation to the environment)
(B) An additional disturbance in cognition (e.g., memory deficit, disorientation, language, visuospatial ability, or perception)
(C) The disturbances in Criteria A and C are not better explained by another preexsisting, established, or evolving neurocognitive disorder and do not occur in the context of a severely reduced level of arousal, such as a coma
(D) There is evidence from the history, physical examination, or laboratory findings that the disturbance is a direct physiological consequence of another medical condition, substance intoxication, or withdrawal (i.e., due to a drug of abuse or to a medication), or exposure to a toxin, or is due to multiple etiologies
Specify whether:
Substance intoxication delirium
Substance withdrawal delirium
Medication-induced delirium
Delirium due to another medical decision
Delirium due to multiple etiologies
Specify if:
Acute: Lasting a few hours or days
Persistent: Lasting weeks or months
Specify if:
Hyperactive: The individual has a hyperactive level of psychomotor activity that may be accompanied by mood lability, agitation, and/or refusal to cooperate with medical care
Hypoactive: The individual has a hypoactive level of psychomotor activity that may be accompanied by sluggishness and lethargy that approaches stupor
Mixed level of activity: The individual has a normal level of psychomotor activity even though attention and awareness are disturbed. Also includes individuals whose activity level rapidly fluctuates

Delirium, Table 2 The following criteria are derived from the *2016 ICD-10 Procedure Coding System* (*ICD-10-PCS*)

ICD-10 diagnostic criteria
(A) Clouding of consciousness, i.e., reduced clarity of awareness of the environment, with reduced ability to focus, sustain, or shift attention
(B) Disturbance of cognition, manifest by both: (1) impairment of immediate recall and recent memory, with relatively intact remote memory; and (2) disorientation in time, place, or person
(C) At least one of the following psychomotor disturbances:
(1) Rapid, unpredictable shifts from hypoactivity to hyperactivity
(2) Increased reaction time
(3) Increased or decreased flow of speech
(4) Enhanced startle reaction
(D) Disturbance of sleep or the sleep-wake cycle, manifest by at least one of the following:
(1) Insomnia, which in severe cases may involve total sleep loss, with or without daytime drowsiness, or reversal of the sleep-wake cycle
(2) Nocturnal worsening of symptoms
(3) Disturbing dreams and nightmares which may continue as hallucinations or illusions after awakening
(E) Rapid onset and fluctuations of the symptoms over the course of the day
(F) Objective evidence from history, physical and neurological examination, or laboratory tests of an underlying cerebral or systemic disease (other than psychoactive substance related) that can be presumed to be responsible for the clinical manifestations in A–D
Comments: Emotional disturbances such as depression, anxiety or fear, irritability, euphoria, apathy or wondering perplexity, disturbances of perception (illusions or hallucinations, often visual), and transient delusions are typical but are not specific indications for the diagnosis
Use the fourth character to indicate whether the delirium is superimposed on dementia or not: F05.0 Delirium, not superimposed on dementia; F05.1 Delirium, superimposed on dementia; F05.8 Other delirium; F05.9 Delirium, unspecified

with reports up to 50% of hospitalized older adults (over 65) (Inouye et al. 2014). Indirect costs of delirium stem from lost work and personal productivity by patients and caregivers and have been estimated to total more than $164 billion in the USA alone (Inouye et al. 2014). Acute postoperative delirium has been shown to be an independent predictor of functional decline and morbidity after cardiac and orthopedic surgeries.

Assessing Delirium

To assist with diagnosis, a number of investigators have collaborated to develop and validate rapid bedside approaches to diagnose patients at the bedside and ICU. The Confusion Assessment Method (CAM; Inouye et al. 1990) is the most well-known measure for assessing delirium. The CAM assesses four features: acute onset and

fluctuating course (feature 1), inattention (feature 2), disorganized thinking (feature 3), and altered level of consciousness (feature 4). Delirium diagnosis requires the presence of features 1 and 2 and either 3 and 4 (note that memory impairment is not included, for this is sometimes absent in mild delirium, whereas it is present in other conditions, such as dementia (Inouye et al. 1990)).

The Society of Critical Care Medicine has strongly recommended routine evaluation of delirium in ICU patients. For critically ill patients where delirium prevalence ranges from 11% to 87% (Aldemir et al. 2001; Ely et al. 2001a), the CAM-ICU (Ely et al. 2001b) was designed. It assesses the same four features of the original CAM, but relies more on nonverbal responses. For this exam, patients are observed to assess the presence of acute mental status change, inattention, disorganized thinking, and altered levels of consciousness. We encourage you to review some excellent video introductions to the CAM-ICU available via the Internet.

The CAM has been translated into 20 other languages including Chinese, Dutch, German, and Spanish, with the CAM-ICU having high validity relative to other delirium scales available in those languages. Overall, the CAM and CAM-ICU's simple algorithms allow them to be useful for rapid identification of delirium by both physicians and staff nurses. After training, both scales should take approximately 2 min to administer.

Aside from the CAM instruments, other frequently encountered screening tools include the Delirium Rating Scale-Revised-98 (DRS-R-98; Trzepacz et al. 2001), the Memorial Delirium Assessment Scale (MDAS; Breitbart et al. 1997), and the Nursing Delirium Screening Checklist (NuDESC; Gaudreau et al. 2005). The DRS is a 10-item scale, rated by a clinician with psychiatry training and is based on a patient's behavior over a 24-h period. The DRS was later revised and renamed the DRS-R-98. The DRS-R-98 includes a 16-item clinician-rated scale, including 13 items assessing delirium severity and three diagnostic items. The MDAS is a 10-item clinician-rated scale, based on DSM-IV criteria, which assesses disturbances in arousal and level of consciousness, cognitive function, and psychomotor activity. The NuDESC is a measure used exclusively in surgical and recovery ward patients and can be administered by trained nursing staff. It consists of a 5-item scale assessing disorientation, inappropriate behavior and communication, hallucinations, and psychomotor retardation over a 24-h period. See Table 3.

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Prevalence of Delirium Type and Considerations for Risk Factors in General Medical and Surgical Populations

The prevalence of delirium is reported highest before hospital discharge, this being often associated with respiratory infections, cellulites, and urinary tract/kidney infections. Central nervous system disorders have the second most frequently reported delirium codes with this particularly seen among those having craniotomy, CNS neoplasms, degenerative nervous system disorders, strokes/transient and other seizures/headaches, and other nervous system disorders (Lin et al. 2010).

Delirium due to a medical condition is the most common type of delirium, with drug-induced delirium being the second most frequently reported (Lin et al. 2010). Patients with drug-induced delirium, however, are typically younger than the other delirium groups and had the lowest proportion of comorbidities. Drug-induced delirium was also most common in patients who have lower extremity orthopedic surgery (relative to comparison groups of patients with pneumonia, urinary tract infection, congestive heart failure) (Lin et al. 2010). Dementia-related delirium, by contrast, is associated with high rates of admission from long-term facilities and older adults. This subtype typically has a higher mortality rate and greater frequency of atrial fibrillation, pneumonia, and urinary tract infections. General delirium risk factors include male sex type, increasing age, and cerebrovascular risk factors.

Structural brain disease traits are additional considerations for delirium risk. Although studies are of variable quality with regard to imaging methods, studies show that delirium patients

Delirium, Table 3 Common delirium screening tools

Tool[a]	Pro	Con	Sensitivity/ specificity[b]
CAM	Based on DSM criteria; best in ED, postoperative and mixed inpatient settings; high interrater reliability	Potential for false negatives in postop population	46–100%/63–100%
CAM-ICU	Brief assessment (<2 min); can be used in nonverbal pts	Be used in nonverbal pts in postop population	46–100%/63–100%
MDAS	Best in postop settings; designed to track changes in delirium	Tested in modest number pts, limited generalizability; may only be assessed by physician	64.1%/100%
DRS	More useful than DRS-R-98 in more impaired patients	May only be assessed by psychiatric physician; not useful with repeated admin	91–100%/84–92%
DRS-R-98	Can assess severity of delirium; distinguishes delirium from dementia	May only be assessed by a trained nurse	91–100%/84–92% and <75%/<75% (in older adults)
NuDESC	Best sensitivity and specificity of tools for postsurgical populations; brief assessment	May only be used by a trained nurse	85.7%/86.8%

[a]*CAM:* Inouye et al. (1990); *CAM-ICU*: Ely et al. (2001b); *DRS*: Trzepacz et al. (1988); *DRS-R-98*: Trzepacz et al. (2001); *MDAS*: Breitbart et al. (1997); *NuDESC*: Gaudreau et al. (2005)
[b]Information adapted from De and Wand (2015). See article for further review

have preexisting brain differences. Patients with delirium are reported to have preexisting larger ventricle sizes, basal ganglia or caudate lesions/lacunes, white matter abnormalities in the periventriclar and deep regions of the brain, greater cortical and subcortical atrophy, and decreased regional and overall perfusion. An important caveat, however, is that almost all of these studies are confounded by age; delirium patients are significantly older in age than those without delirium. White matter abnormalities and atrophy increase with age for many individuals, particularly those with hypertension and hypercholesterolemia. See De Groot and Slooter (2014) for a more thorough review.

Functional MRI techniques may improve understanding of neural mechanisms for delirium. Choi and colleagues (2012) assessed the functional brain patterns of delirious subjects. They found that activity in the dorsolateral prefrontal cortex and posterior cingulate cortex were strongly correlated in patients during an episode of delirium, as compared with control subjects who demonstrated an inverse correlation between these regions. The authors also revealed that functional connectivity between the intralaminar thalamic and caudate nuclei were reduced during a delirious episode, but this connectivity recovered to normal function after resolution of delirium (Choi et al. 2012).

These studies lend further support to the hypothesis that there are predisposing demographic, comorbidity, and brain vulnerability factors contributing to the development of delirium. Attention will now shift to specifically discuss unique risk and treatment applications associated with two major surgery types: orthopedic and cardiac surgery.

Reducing Postoperative Delirium: Anesthetic Considerations and Perioperative Variables

Postoperative delirium typically presents around 24 h after surgery and resolves in most patients by 48 h; however, in rare cases it can last for up to months or even a year or more. Different risk factors for orthopedic and cardiac surgery have been discussed in the literature and are therefore reviewed separately below.

Specific to Orthopedic Surgery

Although delirium can be noted in elective orthopedic surgery, it is more prominent and concerning among urgent orthopedic surgeries such as hip fracture. Patients who develop delirium after hip fracture surgery have higher rates of mortality, are more likely to be diagnosed with dementia or mild cognitive impairment, and/or require institutionalization. There is an increased need to identify ways to reduce delirium in these patients. Recent randomized controlled studies suggest that analgesia and pain management and depth of general anesthesia are important modifiable factors for delirium prevalence after hip fracture surgery. A Cochrane review (Parker et al. 2004) compared outcome differences in hip fracture patients versus regional anesthesia. From five randomized controlled trials meeting inclusion criteria, there were more patients with postoperative confusion in the general anesthesia groups relative to the regional anesthesia groups. The authors concluded that with hip fracture surgery, regional anesthesia relative to general anesthesia results in a twofold reduction of acute delirium. Zywiel and colleagues (2014) found mixed results (Zywiel et al. 2014), however. They identified that patients who receive general versus regional anesthesia during a hip replacement surgery experience delirium at a greater frequency, though after a few days postsurgery, the differences are no longer significant.

Identifying and treating delirium risk factors in patients *prior* to surgery may also be a venue for reducing postoperative delirium. Investigators have recently lead a quasi-experimental intervention study (Bjorkelund et al. 2010) where preoperative patients with hip fractures admitted to the hospital were either treated with a multifactorial intervention program ($n = 131$) or served as a control group ($n = 132$). The multifactorial intervention program included: (1) the use of supplemental oxygen; (2) intravenous iv fluid supplementation and extra nutrition; (3) increased monitoring of vital physiological parameters (oxygen saturation, systolic blood pressure maintained >90–100 mmHg, red blood cell transfusion should be considered if hemoglobin <100 g/l, avoid hypo/hyperthermia); (4) adequate pain relief; (5) avoidance of delay in transfer logistics; (6) daily delirium screening; (7) avoidance of polypharmacia (sedatives/hypnotics with anticholinergic properties given in restriction); and (8) anesthesia recommendations (premedication with paracetamol, propofol and/or alfentanil iv on arrival to operating suite, spinal anesthesia with bupivacain, sedation with propofol) with systolic blood pressure maintained at >2/3 of baseline or >90 mmHG, red blood cell transfusion only when there is increased blood loss (>.3 l) or hemoglobin (<100 g/l), and postoperative analgesia with paracetamol as the first choice or in combination with an opioid. Findings showed less delirium in the intervention group relative to the control group, suggesting value of *multifactorial perioperative intervention approaches* rather than the use of one or two therapies alone.

Presurgical education may be an additional approach. One study (Krenk et al. 2012) showed that delirium did not occur for elective orthopedic surgery patients who were provided with more information about the anesthesia and surgical procedures, as well as prehabilitated by physiotherapists for appropriate exercise regimes commonly used after surgical intervention. The authors cite the need to engage patients in their own rehabilitation as well as consistent monitoring of cognitive changes. The study was limited, however, in that subjects were all cognitively well (Mini Mental State Exam >23) and may have experienced a protective factor in this regard.

Specific to Cardiac Surgery

Delirium after cardiac surgery has been reported for many years, but has recently been shown to be a strong independent predictor of mortality for up to 10 years postoperatively, even in younger individuals and in those without prior stroke. Coronary artery bypass graft or valve surgery is also associated with risk of functional decline at 1 month after discharge, with this outcome independent of comorbidity, baseline function, and cognition.

Operative risk factors include impaired left ventricular ejection fraction, time on cardiopulmonary bypass, high perioperative transfusion

requirement, and postoperative hypertension. Microemboli, common to all cardiac surgical procedures, has not, to date, been specifically associated with delirium although it continues to be considered a potential contributor when it occurs in combination with other risk factors such as hypoperfusion.

There may be specific modifiable risk factors for delirium after cardiopulmonary bypass (CPB). In a group of individuals receiving standardized surgery, anesthesia, and postoperative pain management protocols, and daily delirium evaluations, Burkhart and colleagues (2010) identified that delirium risk factors were: (1) the dose of fentanyl per kilogram of body weight administered during the operation, (2) the duration of mechanical ventilation, and (3) maximum value of C-reactive protein measured postoperatively (Burkhart et al. 2010). The authors pose that fentanyl, with questionable anticholinergic effects, may be a modifiable risk factor; alternatives such as remifentanil or other opioids may be worth considering for intervention trials. Duration of mechanical ventilation requires sedation and therefore may be a consequence of the specific drugs used to maintain sedation. Sedation depth may also be worth considering. Postoperative rates of C-reactive protein suggest a systemic inflammatory response to surgery. This may indicate a relationship to endotoxin, a common consequence of coronary artery bypass grafting (CABG) and trauma-induced intraabdominal infections. Buckhart and colleague's (2010) C-reactive protein findings coupled with others findings that cortisol levels also correlate with postoperative delirium suggest that these areas need further investigation. Clearly, there is a specific need for intervention trials with potentially modifiable risk factors in cardiac patients.

Considerations for Anticholinergic Medications and Anesthesia

Anticholinergic drugs and/or interaction of these drugs with anesthetic agents is a probable factor for postoperative delirium. Anticholinergic drugs compete for acetylcholine receptor subtypes (nicotinic and muscarinic). They impair memory performance by antagonizing the neurotransmitter acetylcholine and muscarinic receptors in the brain. High serum anticholinergic levels of anticholinergic drugs are associated with delirium and cognitive impairment. Unfortunately, these medications are commonly taken by older adults over the counter for sleep aids (any "*pm*" medication). Common anticholinergic medications include tricyclic antidepressants used to treat mood but also pain and sleep (i.e., amitriptyline), antihistamines, and antibiotics (e.g., cephalosporin, third generation). Anesthesiologists should identify patients on anticholinergic medications prior to surgery. One potential mechanism for anesthetic action is via the suppression of cholinergic cells (i.e., isoflurane and sevoflurane suppress acetylcholine release). Thus, there is potential for increased depletion of cholinergic activity. Randomized prospective studies are needed to identify the extent to which presurgery anticholinergic medications interact with anesthesia to increase vulnerability to delirium and even postoperative cognitive dysfunction. There have been attempts to prevent postoperative delirium with cholinesterase inhibitors (e.g., rivastigmine) in randomized treatment trials. Unfortunately, to date, these have been largely unsuccessful for both elective total joint replacement and cardiopulmonary bypass. Large intervention trials appear needed.

Considerations for Postsurgery Dementia Development or Progression

Threshold and Brain/Cognitive Reserve

The concept of a threshold and brain/cognitive reserve is often mentioned when attempting to explain why certain individuals may: (1) develop delirium or (2) proceed to dementia.

Martin Roth and colleagues (Roth 1971) first introduced the concept of a "threshold" in their postmortem Newcastle upon Tyne studies. These researchers observed patterns in senile plaque counts and measures of disease/dementia severity. For example, in Parkinson's disease, clinical Parkinsonism does not appear until 85% of the cells of the nigrostriatal system are depleted and

dopamine has declined in a similar proportion. A similar pattern has been reported in Alzheimer's disease with regard to neurofibrillary plaques and tangles.

Paul Satz contributed significantly to the concept of a threshold by formally providing some general properties for a threshold theory and reserve (Satz 1993). Satz outlines two postulates: A) how greater brain reserve (as measured by premorbid intellectual abilities, academic abilities, or current intelligence) serves as a protective factor to a lesion or pathology and B) how lesser brain reserve serves as a vulnerability factor to lesion or pathology. He also provides subpostulates discussing the effects of aggregate lesions, disease progression, and challenge. Satz' (1993) postulates apply to the topics of delirium.

Unfortunately, it is difficult to define and measure reserve and use the concept to predict risk for delirium. Some researchers propose that education is a surrogate marker for cognitive reserve. The concept of education is multifold, however. Education may signify more neuronal connections, but also it may simply mark better test taking abilities, better social networks, and healthcare. For these reasons, reserve has been extensively studied beyond that of education alone. According to Yacob Stern, Ph.D. at Columbia University, a leader on the topic of cognitive reserve, there are at least two forms of reserve. Reserve can be characterized: (1) as simply "brain reserve" (essentially brain structure) or (2) "as cognitive reserve" represented by neural reserve and neural compensation. Unfortunately, operationally defining both brain and cognitive reserve remains challenging and are topics worthy of longitudinal investigation. Familiarization with the topic of reserve subtypes, as well as discussions on reserve and postoperative disorders is encouraged.

Conclusion Statement

There is growing evidence that delirium can result in reduction of acute and long-term function for some individuals, especially older adults. Delirium can therefore be interpreted as representing an insult to the brain. The development of interventions to prevent and treat delirium and postoperative cognitive dysfunction is essential with our ever-increasing older adult population.

Cross-References

- ▶ Cognitive and Brain Plasticity in Old Age
- ▶ Dementia and Neurocognitive Disorders
- ▶ End of Life Care
- ▶ Frailty in Later Life
- ▶ Palliative Care
- ▶ Physiological Effects on Cognition

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Dementia and Neurocognitive Disorders

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Synonyms

Dementia and major neurocognitive disorder; Mild cognitive impairment and minor neurocognitive disorder

Definition

Dementia and neurocognitive disorder: In the latest edition of the Diagnostic and Statistical Manual Fifth Edition (DSM-5), the American Psychiatric Association panel subsumed the term "dementia" and its etiologies under the category "major neurocognitive disorder" (MND) or "minor neurocognitive disorder" (mND) based on disease severity (Ganguli et al. 2011; American Psychiatric Association 2013).

Introduction

The American Psychiatric Association retired the term "dementia" and introduced "major neurocognitive disorder" in the fifth edition of the Diagnostic and Statistical Manual (DSM-5) to emphasize the neurological origin of the degenerative disorders (i.e., presence of known structural or metabolic brain disease). Furthermore, authors proposed this amendment to differentiate neurodegenerative diagnoses from other illness with cognitive sequelae (such as psychiatric Axis 1 disorders), because a temporal relationship between psychiatric illness and cognitive deficits is an exclusion criterion for the diagnosis of dementia (Ganguli et al. 2011).

Recent advances especially in the field of genetics and neuroimaging modalities have generated a variety of potential biomarkers that may predict the presence of a neurodegenerative disease years before diagnosis or full manifestation of the clinical symptoms. Consequently, reformulation of diagnostic criteria for neurodegenerative disorders is underway. For example, in recognition of underlying disease biomarkers, the National Institute on Aging (NIA) and the Alzheimer's Association have proposed new syndromic stages of Alzheimer's disease (AD) including preclinical and prodromal stage of AD, which will be discussed in detail in this chapter (Jack et al. 2011).

This entry will include a review the general criteria for neurocognitive disorders (major and minor). The next section will discuss updates to research criteria based on biomarkers to delineate

between syndromic presentations (i.e., preclinical state, mild cognitive impairment [MCI], and dementia). Finally, a brief overview of etiology specific criteria for neurocognitive disorders is included. For detailed etiology (e.g., Alzheimer's disease) review, we encourage readers to refer to that entry in this encyclopedia (also cross-referenced with this entry).

Diagnostic Clinical Criteria

In the latest revision of the DSM (American Psychiatric Association 2013), "major neurocognitive disorder" (MND) replaced dementia while "mild neurocognitive disorder" (mND) was elevated from research criteria only to full clinical use. Etiologies related to the syndrome are included as specifiers for both MND and mND (e.g., due to Alzheimer's disease, Parkinson's disease, frontotemporal lobar degeneration, vascular disease, Lewy body disease, or traumatic brain injury). Delirium remains as a separate category under neurocognitive disorders (American Psychiatric Association 2013).

Minor Neurocognitive Disorder (mND)

The DSM-5 criteria (American Psychiatric Association 2013) are:

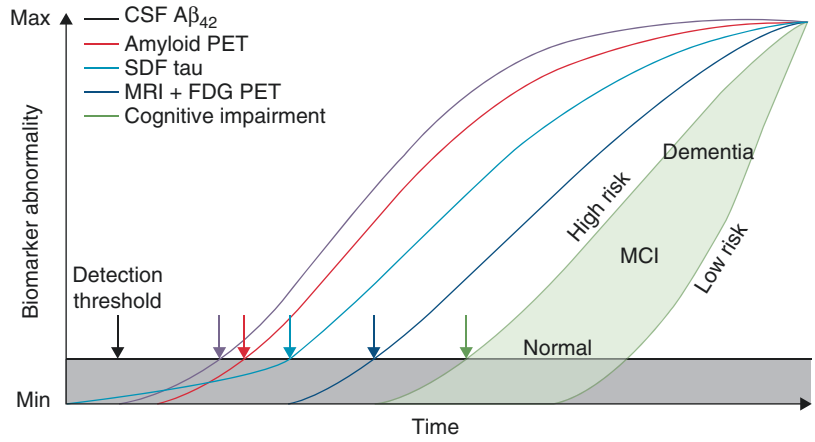
1. Evidence of *modest cognitive decline from a previous level of performance* in one or more cognitive domains (complex attention, executive ability, learning and memory, language, visual constructional-perceptual ability, or social cognition) based on both the criteria listed below.
   (a) Report or concern for possible cognitive decline by patient, a knowledgeable informant, or by the clinician
   (b) Quantifiable documentation of cognitive deficits, preferably with standard neuropsychological testing, typically 1.0–2.0 standard deviations (SD) below the mean (or 2.5th-16 percentile) based on a reference population (i.e., comparable with respect to age, gender, education, premorbid functioning, and cultural background)
2. The subtle but measurable cognitive deficit does not impede the individual's independence in instrumental activities of daily living (i.e., complex activities such as driving, medication and financial management, employment), but may require greater effort or compensatory strategies to maintain an independent level of functioning.
3. The cognitive deficits do not occur exclusively in the context of a delirium.
4. The cognitive deficits are not wholly or primarily attributable to another Axis I disorder (e.g., Major depressive disorder, schizophrenia).

Clearly, mND criteria comport with early suggestions for recognizing MCI (Smith et al. 1996; Petersen et al. 1999) but eliminates previously delineated subtypes: amnestic MCI-single domain (primary deficit in memory), amnestic MCI-multiple domains, nonamnestic MCI-single domain, and nonamnestic MCI-multiple domains based on the nature of the cognitive impairment (Petersen et al. 1999).

Major Neurocogntive Disorder (MND)

The DSM-5 criteria (American Psychiatric Association 2013) are:

1. Evidence of significant cognitive *decline from a previous level of performance* in one or more cognitive domains (complex attention, executive ability, learning and memory, language, visual constructional-perceptual ability, or social cognition) based on both the criteria listed below.
   (a) Report or concern for significant cognitive decline by patient, a knowledgeable informant, or by the clinician
   (b) Quantifiable documentation of cognitive deficits, preferably with standard neuropsychological testing, typically > 2.0 standard deviations (SD) below the mean (or below the 2.5th percentile) based on a reference population (i.e., comparable with respect to age, gender, education, premorbid functioning, and cultural background)
2. The documented cognitive impairments significantly interfere with the individual's ability to



Dementia and Neurocognitive Disorders, Fig. 1 Model integrating Alzheimer's disease biomarkers and immunohistology. *Aβ* amyloid β. *FDG-PET* fluorodeoxyglucose Positron Emission Tomography, *CSF* Cerebrospinal Fluid, *MCI* mild cognitive impairment. The *gray* area denotes abnormal pathophysiological changes below the biomarker detection threshold (*black line*). In this model, tau pathology precedes other markers at a subthreshold level. Aβ deposition occurs independently and rises above the biomarker detection threshold (*purple* and *red arrows*), which accelerates detection of tauopathy and CSF tau (*light blue arrow*). Later still, FDG PET and MRI (*dark blue arrow*) rise above the detection threshold. Finally, cognitive impairment becomes evident (*green arrow*) depending on the individual's risk profile (*light green*-filled area) (Reprinted from The Lancet Neurology, 12, Jack, Clifford R., et al. *Tracking pathophysiological processes in Alzheimer's disease: an updated hypothetical model of dynamic biomarkers*, 210(2013), with permission from Elsevier)

independently manage instrumental activities of daily living (ADLs) (i.e., complex activities such as driving, medication, and financial management).

3. The cognitive deficits do not occur exclusively in the context of a delirium.
4. The cognitive deficits are not wholly or primarily attributable to another Axis I disorder (e.g., Major depressive disorder, schizophrenia).

Research Criteria

The National Institute on Aging (NIA) and the Alzheimer's Association have spearheaded research criteria updates based on burgeoning information regarding utility of biomarkers in preclinical detection, tracking disease burden, and evaluating efficacy of treatment interventions in AD (Albert et al. 2011). While these updates have been made to AD research criteria, the pattern of differentiation between the syndromic presentations (preclinical, MCI, and dementia) will be common to most etiologies of dementia. The following section outlines the criteria for these syndromes. Figure 1 is a model demonstrating the temporal pattern of involvement of biomarkers across clinical diagnoses (Jack et al. 2013).

Preclinical Stage

It is now possible to identify the presence of biomarkers of neurodegenerative disease years before clinical detection of symptoms or syndromes. Biomarkers for AD include genetic, molecular, neuroimaging modalities, and neurocognitive assessment (Knopman 2013; Fields et al. 2011; Smith and Bondi 2013). For AD, genetic markers include causative genetic mutations (Sherrington et al. 1995), as well as susceptibility genes such as apolipoprotein E (APOE) gene (Knopman 2013). Neuroimaging biomarkers include positron emission topography (PET) for amyloid detection and phosphorylated tau accumulation in the brain (Knopman et al. 2013), MRI for hippocampal volume loss, and accumulation of a-beta42 in the cerebrospinal fluid are typically used in AD (Jack et al. 2011). However, presence of neuroimaging biomarkers

is not definitive for future cognitive impairment as shown in a population based sample where over 50% of older adults demonstrated neurodegenerative findings on neuroimaging but demonstrated cognitive normality (Knopman et al. 2013).

Mild Cognitive Impairment

The NIA-Alzheimer's Association work group on MCI (Albert et al. 2011) proposed core criteria for MCI followed by characterization of biomarker data to identity level of certainty for presence of AD etiology. The core MCI features are comparable to mND diagnostic criteria and include:

1. Concern or report of change in level of cognitive function by patient, a knowledgeable informant, or a skilled clinician.
2. Presence of decline from estimated premorbid level of functioning in one or more cognitive domains including memory, executive function, attention, language, and visuospatial skills. If serial cognitive evaluations are present, there must be a progressive decline in scores.
3. Preservation of independence in functional abilities. Patients with MCI may struggle with complex activities such as managing finances and preparing a meal but are generally able to function independently with minimal aids or assistance.
4. Absence of dementia: Observed changes should not significantly impede social or occupational activities.

Dementia

Similar to MCI core symptoms, the NIA-Alzheimer's Association work group provided diagnostic guidelines for core dementia criteria (McKhann et al. 2011):

1. Interfere with the ability to function at work or at usual activities
2. Represent a decline from previous levels of functioning and performing
3. Are not explained by delirium or major psychiatric disorder
4. Quantifiable impairment in two or more cognitive domains

The guidelines elucidate on criteria for prominent cognitive and behavioral symptoms observed in dementia (minimum of two of the following (McKhann et al. 2011)):

(a) Memory: Impairment in encoding and recall of recent information. Individuals may ask repetitive questions, frequently misplace belongings, forget appointments, or get lost on a familiar route.
(b) Executive function: Impaired reasoning and difficulty completing complex tasks. Individuals may demonstrate poor decision-making, poor understanding of safety risks, and may be unable to manage finances or plan complex activities.
(c) Visuospatial functioning: Individuals may have object agnosia, impaired face recognition, simultanagnosia and alexia, difficulty operating simple implements, or demonstrate difficulty finding objects despite good acuity.
(d) Language (speaking, reading, and writing): Individuals may have word retrieval difficulty while speaking, speech may be hesitant, and writing may involve spelling or grammatical errors.
(e) Changes in personality, behavior, or comportment – symptoms include: Individual demonstrates uncharacteristic mood fluctuations such as agitation, impaired motivation, initiative, apathy, loss of drive, social withdrawal, and decreased interest in previous activities, loss of empathy, compulsive or obsessive behaviors, and socially unacceptable behaviors.

Role of Neuropsychological Assessment

The strongest predictive power for progression to dementia is demonstrated by cognitive biomarkers (Fields et al. 2011). Neuropsychological assessments can provide measurable data regarding cognitive performance comparing the individual to a normative sample (ideally based on age, education, gender, and ethnicity) and accounting for confounding factors such as preexisting areas of cognitive weakness, preexisting mood disorder, and motivational factors. Neuropsychological evaluation can assist with diagnostic clarification

and to establish a baseline evaluation of cognitive function, should clinical features in the future warrant a reevaluation. These tests may be of greatest value in mild cognitive impairment or early dementia states as cognitive performance in most domains deteriorates due to eventual disease encroachment on neighboring neural structures and can be difficult to differentiate etiology at later stages of the disease.

Neurocognitive assessments may broadly use the heuristic "cortical" or "subcortical" to classify dementia syndromes based on typical pattern of cognitive impairment (Whitehouse 1986; Salmon and Filoteo 2007). A typical "cortical" dementia such as AD can be characterized by deficits in memory, language, and visuospatial and executive functioning. "Subcortical" dementias (vascular dementia or Parkinson's disease) typically present with motor dysfunction in addition to reduced processing speed and prominent early deficits in executive function, visuoperceptual and constructional abilities. However, from a neuropathological perspective, these profiles are often mixed as patients with "cortical" dementia will often demonstrate abnormal neuropathology in "subcortical" regions, which speaks to the potential presence of neuropathological biomarkers before clinical symptom presentation as seen in Fig. 1. Neurocognitive performance in frontotemporal dementia and dementia due to Lewy body disease (LBD) may demonstrate a mixed cortical/subcortical pattern.

Etiologies

Alzheimer's Disease

Majority of individuals diagnosed with dementia will demonstrate etiology consistent with AD. Neuropathology reveals neuronal loss associated with presence of neuritic plaques (deposition of amyloid) and neurofibrillary tangles (accumulation of tau abnormalities) (McKhann et al. 2011).

MCI or mND due to AD (Research Criteria)

The individual meets criteria for MCI or minor neurocognitive disorder as outlined previously (Ganguli et al. 2011; American Psychiatric Association 2013). A majority of patients with MCI due to AD demonstrate prominent impairment in episodic memory (i.e., amnestic MCI), but other patterns of cognitive impairment can also progress to AD over time (e.g., multidomain MCI, executive dysfunction/nonamnestic MCI, or visual spatial impairments in the posterior cortical atrophy variant of AD). Presence of a positive topographic (e.g., MRI evidence of medial temporal atrophy, or FDG PET evidence of age-adjusted temporoparietal hypometabolism) or molecular neuropathology of AD (e.g., lower CSF Aß-42 and raised CSF tau measures) when available can further characterize the pattern of MCI (Albert et al. 2011). To further classify patients based on level of certainty of etiology, the following research criteria for AD are proposed (Albert et al. 2011):

1. MCI of a neurodegenerative etiology: Low confidence of AD etiology
   (a) Core features of MCI are present.
   (b) Negative or ambiguous biomarker evidence (topographic or molecular biomarkers).
2. MCI of the Alzheimer type: Intermediate confidence of AD etiology
   (a) Core features of MCI are present.
   (b) Presence of one or more topographic biomarkers (MRI evidence of medial temporal atrophy or FDG PET pattern of hypometabolism in the temporoparietal region).
   (c) Absence of molecular biomarker information.
3. Prodromal Alzheimer's dementia: High confidence of AD etiology
   (a) Core features of MCI are present.
   (b) Presence of molecular neuropathology of AD (e.g., lower CSF Aß-42 and raised CSF tau measures).
   (c) Further increased certainty with presence of a topographic biomarker. However, absence or equivocal findings are still consistent with the highest level of certainty that the individual will progress to AD dementia over time.

Dementia due to AD (or MND Due to AD)

The most common syndromic profile of AD dementia is an amnestic presentation. The deficits should include impairment in learning and recall of recently learned information in addition to significant impairments in other cognitive domains as outlined in the dementia criteria described above. McKhann and colleagues (2011) also proposed levels of certainty in AD diagnosis characterized by neuropathological biomarkers, primarily used in research settings (McKhann et al. 2011).

1. Probable AD dementia:
   Meets clinical and cognitive criteria for dementia given above with primary amnestic presentation. There is no evidence of alternative diagnoses, specifically, no significant cerebrovascular disease. In these individuals, presence of any *one* of the three features increases certainty of AD:
   (a) *Documented decline*: Subsequent evaluations demonstrate progressive cognitive decline based on a knowledgeable informant or cognitive testing (brief mental status screens or neuropsychological testing).
   (b) *Biomarker positive*: Has one or more of the following supporting biomarkers.
      (i) Low cerebrospinal fluid Aβ42, elevated cerebrospinal fluid tau or phospho tau
      (ii) Positive amyloid PET imaging
      (iii) Decreased FDG uptake on PET in temporoparietal cortex
      (iv) Disproportionate atrophy on structural MR in medial temporal lobe (especially hippocampus), basal and lateral temporal lobe, and medial parietal isocortex
   (c) *Mutation carrier*: Meets clinical and cognitive criteria for AD dementia and has a proven AD autosomal dominant genetic mutation (PSEN1, PSEN2, and APP).
2. Possible AD dementia.
   (a) *Atypical course*: Evidence for progressive decline is lacking or uncertain but meets other clinical and cognitive criteria for AD dementia
   (b) *Biomarkers obtained and negative*: Meets clinical and cognitive criteria for AD dementia but biomarkers (CSF, structural or functional brain imaging) do not support the diagnosis
   (c) *Mixed presentation*: Meets clinical and cognitive criteria for AD dementia but there is evidence of concomitant cerebrovascular disease; this would mean that there is more than one lacunar infarct; or a single large infarct; or extensive and severe white matter hyperintensity changes; or evidence for some features of dementia with Lewy bodies (DLB) that do not achieve a level of a diagnosis of probable DLB.
3. Not AD Dementia
   (a) Does not meet clinical criteria for AD dementia.
   (b) Has sufficient evidence for an alternative diagnosis such as HIV, Huntington's disease, or others that rarely, if ever, overlap with AD.
4. *Pathologically proven AD dementia*. Meets clinical and cognitive criteria for probable AD dementia during life AND is proven AD by pathological examination.

Vascular Dementia

In 2011, the American Heart Association and American Stroke Association workgroup jointly published consensus definitions and recommendations for the vascular contributions to mild cognitive impairment and dementia (Gorelick et al. 2011). Vascular pathology includes ischemic and/or hemorrhagic cardiovascular disease (CVD), other cerebrovascular insults (subclinical brain infarction [SBI]), multiple small vessel disease, or cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL).

Vascular MCI

1. Probable VaMCI:
   (a) Meets core MCI criteria (Albert et al. 2011).
   (b) Presence of clear temporal relationship between a vascular event (e.g., clinical stroke) and onset of cognitive deficits.

(c) Onset of cognitive deficits or relationship in the severity and pattern of cognitive impairment and the presence of diffuse, subcortical cerebrovascular disease pathology (e.g., as in CADASIL).
(d) No history of gradually progressive cognitive deficits before or after the stroke that suggests the presence of a nonvascular neurodegenerative disorder.

2. Possible VaMCI:
   (a) Meets core MCI criteria (Albert et al. 2011).
   (b) Presence of cognitive impairment and imaging evidence of cerebrovascular disease.
   (c) *No* clear relationship (temporal, cognitive pattern or severity) between the demonstrated vascular disease (e.g., silent infarcts, subcortical small-vessel disease) and onset of cognitive deficits.
   (d) There is insufficient information for the diagnosis of VaMCI (e.g., clinical symptoms suggest the presence of vascular disease, but no CT/MRI studies are available).
   (e) Severity of aphasia precludes proper cognitive assessment. However, patients can be classified as probable VaMCI with documented normal cognitive function (prior cognitive evaluations) before the vascular event that resulted in aphasia.
   (f) There is evidence of other neurodegenerative diseases or conditions in addition to cerebrovascular disease that may affect cognition, such as:
      (i) A history of other neurodegenerative disorders (e.g., Parkinson disease, progressive supranuclear palsy, dementia with Lewy bodies).
      (ii) The presence of Alzheimer's disease pathology is confirmed by biomarkers (e.g., PET, CSF, amyloid ligands) or genetic studies (e.g., PS1 mutation).
      (iii) A history of active cancer or psychiatric or metabolic disorders that may affect cognitive function.

3. Unstable VaMCI:
   Subjects with the diagnosis of probable or possible VaMCI whose symptoms revert to normal should be classified as having "unstable VaMCI."

Vascular Dementia (VaD)

Individuals meet criteria for core features of dementia (decline in cognitive function and deficit in two cognitive domains) (McKhann et al. 2011) with sufficient severity to affect a person's ADLs. In addition, the impairments in ADLs are independent of the motor/sensory sequelae of a vascular event (Gorelick et al. 2011). Criteria for probable and possible VaD are similar to those stated for VaMCI, but these individuals demonstrate significant impairment in activities of daily living to meet criteria for dementia (vs. MCI criteria).

Lewy Body Disease (LBD)

Lewy bodies are intraneuronal inclusions primarily made of alpha-synuclein (McKeith et al. 2005). High concentration of inclusions in substantia nigra are associated with Parkinsonism (e.g., idiopathic Parkinson's disease), where subsequent onset of dementia is termed Parkinson's disease dementia (PDD). On the other hand, presence of inclusions in the cortex can lead to Lewy body disease (LBD), which can refer to any syndromic presentation of Lewy body (preclinical, MCI, and dementia). Dementia with Lewy Body (DLB) refers solely to the dementia syndrome due to LBD.

Mild Cognitive Impairment of LBD

Presence of REM Sleep Behavior disorder (RBD), which was included in the last revision of DLB criteria (McKeith et al. 2005), has demonstrated 52.4% increased 12-year risk of developing DLB (Postuma et al. 2009) and is thought to be associated with presence of synucleinopathy (McKeith et al. 2005). Therefore, presence of RBD and cognitive decline can be a type of MCI due to LBD and may include other cardinal symptoms such as Parkinsonism or visual hallucinations. The cognitive profile of MCI with LBD shows prominent visuoperceptual and/or attention deficits, nonamnestic profile.

Dementia with Lewy Body Disease (DLB)

International diagnostic criteria (McKeith et al. 2005) include:

1. Central feature (for diagnosis of possible or probable DLB): Presence of dementia (i.e., progressive cognitive decline which significantly interferes with daily functioning)
   (a) Prominent or persistent memory impairment may not necessarily occur in the early stages but is usually evident with progression.
   (b) Deficits on tests of attention, executive function, and visuospatial ability may be especially prominent.
2. Core features (Probable DLB: 2 features, Possible DLB: 1 core feature)
   (a) Fluctuating cognition with pronounced variation in attention and alertness
   (b) Recurrent visual hallucinations that are typically well formed and detailed
   (c) Spontaneous features of Parkinsonism
3. Suggestive features (Probable DLB: at least 1 suggestive feature and at least 1 core feature while possible DLB includes: at least 1 suggestive feature in the absence of core features)
   (a) REM sleep behavior disorder
   (b) Severe neuroleptic sensitivity
   (c) Low dopamine transporter uptake in the basal ganglia demonstrated by SPECT or PET imaging
4. Supportive features (commonly present but not proven to have diagnostic specificity)
   (a) Repeated falls and syncope
   (b) Transient, unexplained loss of consciousness
   (c) Severe autonomic dysfunction, e.g., orthostatic hypotension, urinary incontinence
   (d) Hallucinations in other modalities
   (e) Systematized delusions
   (f) Depression
   (g) Relative preservation of medial temporal lobe structures on CT/MRI scan
   (h) Generalized low uptake on SPECT/PET perfusion scan with reduced occipital activity
   (i) Abnormal (low uptake) MIBD myocardial scintigraphy
   (j) Prominent slow wave activity on EEG with temporal lobe transient sharp waves
5. A diagnosis of DLB is less likely:
   (a) With evidence of cerebrovascular disease (focal neurologic signs or on brain imaging)
   (b) In the presence of any other physical illness or brain disorder sufficient to account in part or in total for the clinical picture
   (c) If the parkinsonism only appears for the first time at a stage of severe dementia
6. Temporal sequence of symptoms:
   DLB should be diagnosed when dementia occurs before or concurrently with parkinsonism (if it is present). The term Parkinson disease dementia (PDD) should be used to describe dementia that occurs in the context of well-established Parkinson disease. In a clinical practice setting, the term that is most appropriate to the clinical situation should be used and generic terms such as LB disease are often helpful.

D

Frontotemporal Lobar Degeneration

Frontotemporal lobar degeneration (FTLD) is a heterogeneous collection of diagnoses (Pick's disease or Primary Progressive Aphasia) and syndromes (FTD with motor neuron disease, corticobasal degeneration), and etiologies (e.g., tauopathies versus TDP-43 proteinopathies) (Smith and Bondi 2013; Josephs 2008).

Frontotemporal dementia (FTD) refers to the dementia phase of FTLD. Currently, the three main recognized phenotypes of FTD are: behavioral variant-FTD (bvFTD), semantic dementia (SD), and primary progressive aphasia (PPA). Furthermore, PPA can be subclassified into three variants: logopenic (lvPPA), semantic (svPPA), and agrammatic (agPPA) or nonfluent progressive aphasia (PNFA) (Gorno-Tempini et al. 2011). Pathology for semantic and agrammatic variants of PPA are largely consistent with tauopathies and TDP-43 suggestive of FTLD spectrum disorders, while the lvPPA variant is strongly associated with AD pathology (Josephs 2008). The diagnosis of FTD is challenging due to the complexity and heterogeneity in FTLD. Individuals may be misdiagnosed as psychiatric disorder or AD early in the disease course.

Preclinical stage of FTD involves being a carrier of genetic mutations associated with FTD such as MAPT, GRN, and C9ORF72 genes (Rohrer et al. 2013). The behavioral variant FTD presents with impairments in "social cognition"

including behavioral disinhibition, apathy, loss of empathy, perseverative or compulsive behavior, and hyperorality or dietary changes early in the disease process (Piguet et al. 2011). Other variants of FTD demonstrate predominant language or speech deficits. Core features of semantic PPA include impaired naming and single-word comprehension. Logopenic variant of PPA is characterized by hesitant speech (impaired single-word retrieval in speech) and impaired repetition of complex sentences. Core features of the agrammatic variant of PPA are agrammatism in speech or written output and reduced comprehension (Gorno-Tempini et al. 2011).

Conclusion

Recent advancements in biomarkers in varied scientific fields including molecular genetics, neuroimaging, behavioral neurology, and neuropsychology have accelerated research and shifted nomenclature in neurodegenerative disease. Identification of these biomarkers has led to a clear articulation of the distinction between syndromic phases of neurodegenerative disease across most dementia etiology (preclinical or asymptomatic phase, mild cognitive impairment, and dementia). It is now possible to have biomarkers of neurodegenerative disease without being (and possibly never becoming) symptomatic. These changes will be instrumental in future research focused on prevention, early detection, or delayed progression to dementia (Smith and Bondi 2013).

Cross-References

- Alzheimer's Disease, Advances in Clinical Diagnosis and Treatment
- Delirium
- Frontotemporal Dementia (FTD)
- Geriatric Neuropsychological Assessment
- Lewy Body Disease
- Mild Cognitive Impairment
- Primary Progressive Aphasia
- Vascular and Mixed Dementia
- Young-Onset Dementia, Diagnosis, Course, and Interventions

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Depression and Cognition

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Synonyms

Mood disorder; Neuropsychology

Depression	A psychiatric disorder that includes symptoms of sad mood, hopelessness, poor sleep and appetite, guilt or worthlessness, low energy, and severe suicidal thoughts
Cognition	Thinking skills or abilities that include attention, processing speed, working memory, language, visuospatial skills, language, and executive functioning

Introduction

As an age group, older adults have a smaller prevalence rate for depression compared to middle-aged adults that continues to decrease with advancing age (e.g., Byers et al. 2010). Although the prevalence of depression is lower with older age, the effects of depression in the daily life of patients may be greater with advancing age due to other factors that make older adults a more vulnerable population (e.g., decrease in physical health, cognitive changes due to normal aging, comorbidity with other health/mental health disorders). In particular, cognitive declines due to age-related changes in the brain may compound the effects of depression on the daily functioning of older adults.

A review of the rate of comorbid depression and cognitive impairment in older adults estimated it to double every 5 years after the age of 70, with over 25% of community dwelling 85-year olds living with comorbid MDD and cognitive impairment (Ellen and David 2010). Lee et al. (2007) noted that a high number of depressed older adults present with "mild cognitive impairment," defined in the literature as the stage between normal aging and dementia. Moreover, these cognitive impairments that accompany an acute depressive episode continue long after the remission of depressive symptoms (Ellen and David 2010; Lee et al. 2007). Furthermore, in a recent review and meta-analysis by Diniz et al. (2013), they determined that late-life (geriatric) depression is in fact associated with a higher risk of dementia, including vascular and Alzheimer's disease.

This encyclopedia entry will first review the neurobiological effects of depression in older adults. Then it will describe the effects of

depression on global cognitive functioning as well as specific cognitive domains including attention and working memory, processing speed, spatial skills, language, memory, and executive functioning. Afterward, the special considerations of age moderating the impact of depression on cognition, dementia/pseudodementia, and the effects of antidepressants on cognition will be covered.

Neurobiology of Depression in Older Adults

The impact of depression on cognition in older adults and in other age groups has been hypothesized to be mediated by the neurobiological effects of depression in the brain. In fact, there are several different hypotheses about how this mediation occurs. For instance, there is evidence that older adults with depression have a higher prevalence risk for cardiovascular disease and dementia. There is a "vascular depression hypothesis" (e.g., Sneed and Culang-Reinlieb 2011) that theorizes that heart disease may cause, be a result of, or prolong depression in older adults. Furthermore, this link has also been connected to brain-related changes. For instance, MRI studies have found significant relations between ischemic lesions in the brain and depression severity or diagnosis (Sneed and Culang-Reinlieb 2011). Specifically, for late-life depression, the vascular depression hypothesis is specific regarding the location of deep white matter hyperintensities (DWMH) within frontostriatal circuits that are involved in executive functioning (Sneed and Culang-Reinlieb 2011). In the update by Sneed and Culang-Reinlieb (2011), the authors reported other MRI studies that have found DWMH, reduced volume in frontal and subcortical areas, neuronal abnormalities within the prefrontal cortex, and reduced neuronal density in the dorsolateral and ventromedial areas of the caudate nucleus. Sneed and Culang-Reinlieb concluded that neuronal abnormalities in some LLD are present in the frontal and striatal brain regions, which is consistent with the vascular depression hypothesis.

In a review by Byers and Yaffe (2011), they reported several other neurobiological factors related to how depression can impair cognition through changes in the brain in older adults. These factors include increased levels of cortisol and hippocampal atrophy, increased deposition of β-amyloid plaques, inflammatory changes, and deficits of nerve growth factors. In relation to greater cortisol, higher levels of depression would cause the HPA axis to increase glucocorticoid production that would damage the hippocampus and result in a downregulation of glucocorticoid receptors ultimately resulting in a vicious cycle leading to impairments in cognition. As for beta-amyloid relationships, Byers and Yaffe hypothesized that depression may increase β-amyloid production due to a stress response to depression resulting increase of cortisol. Although the research findings are mixed, they reported some evidence that depression with a high ratio of plasma β-amyloid peptide 40 (Aβ40) to Aβ42 has been associated with memory, visuospatial abilities, and executive function deficits. As for the inflammation hypothesis, Byers and Yaffe stated that depression is associated with increased levels of cytokines that can lead to a decrease in inflammatory and immunosuppressant regulation, resulting in inflammation of the central nervous system that would ultimately result in cognitive impairment and an increase risk of dementia. The increase in cytokines may also interfere with serotonin metabolism that can lead to decrease in synaptic plasticity and hippocampal neurogenesis. Lastly, they mentioned problems with nerve growth factors, specifically, such as brain-derived neurotrophic factor (BDNF). They stated that impairments in BDNF functioning have been found in animal and human models of depression that have been linked to declines in cognitive functioning.

In all, there seems to be multiple pathways of how neurobiological changes due to depression can then impact cognitive functioning and increase the risk of cognitive disorders, including dementia. More research is needed in this area to determine which pathways are most related to cognitive decline in geriatric depression.

Depression on Cognition in Older Adults

Mental Status

Mental status is also commonly referred to as global cognitive functioning as is typically

measured using the mini mental status exam. Older adults with depression have been found to have lower MMSE scores than healthy older adults (Pantzar et al. 2014). But this may also be related to the depressed group being older (healthy control age mean = 72.6 years, mild depression mean = 78.6 years, and moderate-severe depression mean = 75.9 years) and having less years of education (healthy control education mean = 12.1years, mild depression mean = 10.7 years, and moderate-severe depression mean = 10.5 years). However, in a study by Rapp et al (2005), they also found significantly lower MMSE scores in the older adults with recurrent or late-onset depression versus those with no history of or current depression. These diagnostic groups did not significantly differ in age, years of education, nor gender.

In a 13-year longitudinal study, depression at baseline predicted decline in general cognitive functioning using the MMSE even after controlling for covariates that include age, sex, and years of education (van den Kommer et al. 2013). Using the Cognitive Abilities Screening Instrument (CASI) as a measure of global cognition, greater depression severity is related to poorer cognitive performance even after controlling for age and education in elderly Chinese males (Tzang et al. 2015).

Thus, research indicates substantial evidence that global cognitive functioning is impaired in older adults with depression.

Attention and Working Memory

Simple attention can be defined as the limited capacity to passive hold information in the mind such as repeating a list of numbers in the same order spoken as in Digit Span Forward from the Wechsler Adult Intelligence Scale. For this task, no effects of depression on attention were found in American (Pantzar et al. 2014) and Chinese older adult samples with depression (Tzang et al. 2015).

Working memory is related to general attention but includes active (versus passive) manipulation specifically reversing the order of digits, such as in Digit Span Backward. In Digit Span Backward, no effects of depression were found in American (Pantzar et al. 2014) and Chinese older adult samples (Tzang et al. 2015). In another study that used the N-back task as a measure of working memory, the depression group performed worse than healthy older adults (Nebes et al. 2000). This deficit was also seen in older adults whose depression remitted compared to older adults without any history of depression (Nebes et al. 2000).

Processing Speed

Processing speed is broadly defined as the rate at which an individual can process incoming information in order to carry out a task (e.g., Nebes et al. 2000). While normal aging has been known to slow down the speed of information processing for a majority of older adults (Salthouse 1996), this cognitive domain is significantly more impaired in older adults with depression (Dybedal et al. 2013; Ellen and David 2010; Pantzar et al. 2014) compared to healthy older adults. Using the trail-making task, Rapp et al. (2005) found no significant processing speed differences in the easier task of Trail A but did find differences in diagnostic groups on a harder task of Trail B, where older adults with no history or no current depression were faster than older adults with recurrent depression and slowest with older adults with late-onset geriatric major depression (when the age of onset for a first episode of depression occurs is 65 years old or older). Another study also concluded slowed speed of information processing persist even after the clinical symptoms of depression remit in older adults (Thomas and O'Brien 2008). Butters et al. (2004) and Dybedal et al. (2013) also determined that late-life depression is associated with a slower speed of information processing. In fact, Sheline et al. (2006) concluded that processing speed has emerged as the most salient cognitive impairment in older adults diagnosed with depression.

Longitudinal studies have also found associations between depression and slower processing speed in older adults. For instance, a 9-year longitudinal study examined the impact of depression on cognitive functioning in older women (Rosenberg et al. 2010) found that baseline depression ratings were strongly associated with impairments on measures of psychomotor speed. Another longitudinal study examining a large

cohort of older adults found that the level of depression at baseline predicted the rate of decline in speed of information processing, such that more severe depression led to slower speed consistently during the 13-year follow-up period (van den Kommer et al. 2013). These results remained even after controlling for age, sex, and education. Notably, the slower processing speed at baseline also predicted worsening of depression severity over time.

Salthouse has theorized that the effects of declines in cognitive functioning such as memory and executive functioning are mediated by slowed processing speed in older adults (Salthouse 1996). This also appears to be true in older adults with depression. For instance, Nebes and colleagues (2000) conducted hierarchical regression analyses that depression explained a significant amount of neuropsychological variance on global cognition, visuospatial construction, and verbal and visual memory. However, when processing resources (working memory as measured by then-back task and processing speed as measured by digit symbol substitution test) were removed first, depression no longer accounted for a significant amount of neuropsychological performance. Butters et al. (2004) also determined that late-life depression is associated with a slower speed of information processing, which then impacts all other cognitive domains including memory, language, visuospatial skills, and executive functioning. In addition, Sheline et al (2006) found that processing speed mediated the impact of other factors including age, education, race, depression severity, and vascular risk factors on working memory, episodic memory, language processing, and executive functioning (Sheline et al. 2006). However, in a relatively more recent 4-year longitudinal study, Köhler et al. (2010) found that although processing speed partially mediated some of the deficits in their depressed older adult participants, it did not adequately account for the differences between them and the normal control group participants.

Visuospatial Ability

In general, there are only a few studies that examined spatial ability in geriatric depression. Using simple drawings and block design, Butters et al. (2004) found significant differences between older adults with late-life depression and healthy older controls. Nebes and colleagues (2000) found depression group differences (recurrent/current depression, remission from depression, and no history of depression) on a block design task. Notably, when controlling for working memory or processing speed, the effects of depression on the visual-construction task were no longer significant. In a timed, visual pattern-matching task, there was no difference in correct responses between older adults with depression and those without depression, but those with depression had overall slower reaction time compared to the controls (Hofman et al. 2000). Incidentally, when controlled for MMSE scores, the older adults with depression had similar reaction times on this task as those with dementia. In a mental rotation task, no differences were found between older adults with depression and were not on antidepressants compared to healthy older adults (Pantzar et al. 2014).

In sum, these studies indicate limited evidence of the association of depression with impairments in visuospatial and visuo-construction skills.

Language

As in visuospatial ability, relatively less research has been conducted in examining the relation of depression and language, compared to other cognitive domains in older adults. Dybedal and colleagues (2013) found that after controlling for age, there were no differences between the older adults with versus those without depression on animal or letter fluency. Similarly, Butters et al. (2004) found impaired language performance of older adults with late-life depression compared to healthy older adults for a task of verbally naming pictures but no differences on letter or animal fluency. Furthermore (Rapp et al. 2005), no diagnostic group differences were found between older adults with recurrent depression, late-onset depression, remitted depression, and no history of depression. In conclusion, the limited research in this cognitive domain indicates that there is generally little to no relationship between depression and language ability.

Learning and Memory

Memory has been one of the most studied cognitive domains for depression in older adults as well as other age groups. Many studies have focused on verbal memory and most commonly used word lists or stories to measure learning, short- and long-term recall, and recognition. In older adults, many studies have found poorer memory performance in depressed groups versus healthy controls (Butters et al. 2004; Pantzar et al. 2014). For instance, Rapp et al. (2005) used a 10-item list learning task and found that older adults with no history of depression and no current depression performed significantly better on learning, delayed recall, and recognition compared to older adults with recurrent depression and those with late-onset depression.

Studies have also found that poorer verbal memory performance is related to increased severity levels of depression. A relatively recent study (Mesholam-Gately et al. 2012) examined learning and memory performance in older adults with two severity types of depression using the California Verbal Learning Test. The study compared older adults with minor depression (defined as "subsyndromal depression that meets duration criteria but not symptom count criteria for Major Depressive Episode") (Mesholam-Gately et al. 2012, p. 197), to those meeting criteria for major depressive disorder, and healthy control participants. The findings indicated individuals with major depressive disorder performed significantly worse than older individuals with minor depressive symptomatology, who in turn performed comparably to normal control participants. Similarly, a population-based study found that only older adults with moderate to severe levels of depressive symptomatology had verbal memory impairments compared to healthy controls (Pantzar et al. 2014). However, no differences were found between the older adults with mild depression from the healthy controls.

Longitudinal studies have also indicated a predictive relationship between depression and verbal memory. For instance, in a 9-year longitudinal study examining the impact of depression on cognitive functioning in older women, Rosenberg et al. (2010) found that baseline depression ratings were strongly associated with greater verbal memory declines in a list learning task, over time.

However, not all studies have reported significant results. For example, Butters et al. (2004) found no group differences with older adults with late-life depression compared to healthy older adults on verbal memory performance for story and list learning tasks. Consistent with this, Dybedal et al. (2013) conducted a more recent study that also found no verbal memory differences on a list learning task between those with late-life depression and healthy older adults after controlling for age.

In comparison to verbal memory, there are relatively fewer studies that examined the relation between depression and spatial memory in older adults, compared to verbal memory. Burt et al. (2000) found that within a group of patients diagnosed with major depressive disorder, patients older than 60 years showed significantly greater impairments on a delayed memory task of visuospatial construction and organization (Rey complex figure test) compared to younger patients. Additionally, depression severity was significantly associated with poor delayed recognition. In contrast, Dybedal et al. (2013) found no visual memory differences between those with late-life depression and healthy older adults after controlling for age.

In sum, while there are substantial evidences that depression and depression severity impair verbal memory in older adults, the findings are not always consistent. Conflicting findings can be due to differences in sample size, medication, types of memory task, and use of covariates in the data analyses. In visual memory, the research is relatively sparse and indicates further need of more research in this area.

Executive Functioning

Executive functioning is a broad term used to refer to higher-order cognitive skills involved in carrying out goal-directed behavior. The skills involved in executive, goal-directed behavior include, but are not limited to, identifying future goals, developing a plan, reasoning, solving complex problems, choosing among various alternatives, and inhibiting irrelevant responses. Many studies have found executive function to be one of

the most profoundly impacted cognitive domains in depressed older adults (Lockwood et al. 2002; Pantzar et al. 2014; Rapp et al. 2005).

There are many studies that have found significant relationships between depression and poorer executive functioning in older adults. In particular, the performance of older adults with depression on executive measures revealed impairments in response to initiation and inhibition (e.g., Dybedal et al. 2013), active switching (e.g., Butters et al. 2004; Dybedal et al. 2013; Pantzar et al. 2014), and problem solving using error feedback (Lockwood et al. 2002). Longitudinal studies have also shown declines in executive functioning in geriatric depression such as in a 9-year longitudinal study that examined the impact of depression on cognitive functioning in older women. Rosenberg et al. (2010) found that, in terms of subtypes of depression, both early and late onset of depression in the elderly, has also been linked to executive functioning deficits (e.g., Butters et al. 2004). However, the decline in executive functioning has been found to be greater for older adults with late-onset than the early-onset cohort (Herrmann et al. 2007).

Notably, antidepressant treatment and remission studies have also found that executive dysfunction can still occur in older adults. Dybedal et al (2013) found that older adults with late-onset depression were still significantly impaired executive function compared to healthy older adults even after controlling for processing speed. Similarly, Elderkin-Thompson et al. (2007) found that older adults continued to show residual deficits in executive functioning even after successful treatment of depression. Interestingly, even when the depression is in full remission, Thomas and O'Brien (2008) found declines in executive functioning in older adults.

In all, there is substantial evidence that depression impairs executive functioning in older adults and may continue to persist despite the use of antidepressants.

Special Considerations: Pseudodementia Versus Depression

Understanding the cognitive sequelae of geriatric depression is especially challenging because of the age-related changes in the brain that may contribute to cognitive deficits or to the etiology of the depression itself. Moreover, many of the affective, behavioral, and cognitive issues among the elderly are often the result of an interaction between multiple psychiatric, neurological, and medical conditions (Ellen and David 2010).

A critical clinical question is whether cognitive deficits associated with depression resolve following remission of the depressive episode. A growing body of evidence suggests the presence of a syndrome of cognitive impairment that is reversible after the successful treatment of depression in older adults. This syndrome, popularly termed "pseudodementia" or "reversible dementia," can masquerade as dementia and, as such, is an important consideration in the differential diagnosis of dementia in the aging population (Ellen and David 2010). It is estimated that 18–57% older adults with depression present with a reversible syndrome of dementia that resolves upon alleviation of depressive symptoms (Alexopoulos and Meyers 1993). However, it is extremely challenging to reliably differentiate between geriatric depression and reversible or irreversible dementia.

This issue becomes more complicated because cognitive impairments that can result from dementia can manifest with depressive symptoms as well (Kang et al. 2014). Some researchers have suggested that depressive pseudodementia may be a transient state that eventually progresses to dementia. For example, a recent review suggested that late-life depression is a strong predictor for the progression of reversible dementia to an irreversible one (Kang et al. 2014). This is also consistent with the meta-analysis of 23 studies conducted by Diniz and colleagues (2013), which found that geriatric depression was significantly associated with higher risk of all-cause dementia, including vascular and Alzheimer's disease.

Thus, the question of pseudodementia and depression remains unclear. While some researchers have concluded that depression can mimic dementia, others state that it can also be a risk factor for dementia in late life and that depression is likely an early manifestation of dementia

rather than a risk factor for the neurodegenerative disease Panza et al. (2010).

Age Moderating the Impact of Depression on Cognition

In the adult depression literature, researchers have reported greater relationships between depression and cognitive impairment in the older adult groups compared to the younger adult age groups. Sparse research indicates some evidence that this pattern also exists in the old age group. For instance, Pantzar et al. (2014) found that the effect size of depression on cognitive performance in depressed sample was greater for old-old age group (85 years and older) than young-old age group (60–84 years old).

Although there are several hypotheses of how depression causes neurobiological changes that can result on cognitive decline, there is sparse data of how chronicity of the depression affects the brain and cognitive performance in older adults. Perhaps part of that problem is because chronicity is so intimately related to age and age is a significant factor of the relation between depression and cognition, especially in old age.

Effects of Antidepressants on Cognition in Geriatric Depression

In general, typical pharmacological intervention for depression includes the use of tricyclic antidepressants (TCAs) and monoamine oxidase inhibitors (MAOIs). Additionally, newer classes of antidepressant drugs including selective serotonin reuptake inhibitors (SSRIs), serotonin-norepinephrine reuptake inhibitors (SNRIs), and medications acting on noradrenergic and dopaminergic neurotransmission [e.g., bupropion (Wellbutrin)] are increasingly being used for treatment. However, when treating late-life depression, it is important to pay special attention to aging considerations for this patient population. There is evidence to suggest that age-associated changes can alter the pharmacodynamics and pharmacokinetics of drugs and dictate the type of medication and dosage that will be safe and effective for the elderly.

Researchers generally agree that the newer antidepressants including SSRIs and SNRIs have been shown to be relatively safer for older adults (e.g., Culang et al. 2009). Some researchers postulate that the use of antidepressants in elderly patients can improve memory and other cognitive domains through their effects on improving the depressive symptoms and by the pharmacodynamic effects that are mediated by neurophysiological changes in the brain (Bali et al. 2016). For instance, Doraiswamy and colleagues (2003) pooled data from two double-blind 12-week studies that included 444 older adults with depression comparing sertraline, fluoxetine, and nortriptyline. They found that there was an improvement for short-term memory and psychomotor speed for those patients whose depression improved (responders) and had lower anticholinergic side effects. In order of the highest correlations between depression improvement and cognitive improve, it was sertraline, then nortriptyline, and then fluoxetine.

In contrast, other studies have shown that cognitive deficit either persists or still ensues after successful treatment for depression. For instance, Nebes et al. (2003) conducted a randomized double-blind design examining the effects of an SSRI (paroxetine) or a tricyclic antidepressant (nortriptyline) on cognition in older patients with depression. They found that after 12 weeks of treatment, their cognitive functioning did not improve more than the control group, suggesting that the impairment in cognition due to depression still persists despite response to antidepressants. Culang et al. (2009) conducted an 8-week, double-blind, placebo-controlled study that examined the effects of SSRI, specifically, citalopram, on neuropsychological functioning on older adults with late-life depression. They found that those who did not respond to the citalopram (depression symptoms did not improve), declines were found on verbal learning and memory and in psychomotor speed. For those who did respond to the medication, they improved in visuospatial functioning compared to nonresponders but not better than those in the placebo group.

In a recent longitudinal study by Saczynski and colleagues (2015), over 3000 adults from the National Health and Retirement Study (mean age 72) were followed for 6 years on their use of

antidepressants, depression symptoms, and cognition, as measured by a battery of cognitive test that included memory, working memory, and naming. The researchers found that those taking the antidepressants declined on cognitive tasks at the same rate as those who were not on antidepressants after controlling for baseline cognition, age, and duration of antidepressant use.

In sum, there is little evidence that antidepressant usage can improve cognitive functioning even if they improve depression severity in older adults. There is evidence that the cognitive impairments due to depression persist whether or not the older adults respond to the medications. Furthermore, there is evidence that the use of these medications will not decrease the rate of cognitive decline over time in geriatric depression.

Conclusions

Some cognitive domains seem to be not or minimally affected by depression in older age such as attention and working memory, visuospatial skills, and language, while other cognitive domains seem to be consistently and negatively impaired by depression and/or depression severity such as processing speed, memory, and executive functioning. Mediating factor of processing speed (with but sometimes without working memory) seems to be the way depression affects higher-order or more complex cognitive functioning such as memory and executive functioning. In addition, age and medication seem to also moderate the effects of depression on some of the cognitive domains. All these studies point to a relationship between depression and cognitive functioning; however, there are diverging hypotheses about whether depression causes cognitive declines or if the relationship is bidirectional.

Cross-References

- ▶ Cognition
- ▶ Comorbidity
- ▶ Dementia and Neurocognitive Disorders
- ▶ Depression in Later Life
- ▶ Executive Functioning
- ▶ Executive Functions
- ▶ Memory, Episodic
- ▶ Mental Health and Aging
- ▶ Working Memory in Older Age

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Depression in Later Life

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Definition

The term depression can have different meanings. It can be regarded as a "symptom" (low mood), a

"syndrome" (a set of symptoms with various definitions), or as a medically defined diagnosis according to a classification system. Depressive symptoms can be viewed dimensionally, from more or less normal reactions to pathologically severe depressive symptoms. The symptoms occur on a continuum of severity from mild reactions to complete disablement. The classification systems have traditionally viewed depressive symptoms and depression categorically (Baldwin 2014).

There is no defined biomarker for depression; the diagnosis is based on a clinical interview, observation, and supplemental information from relatives and caregivers. A diagnosis of depression is made according to two main classification systems: the American Psychiatric Association's Diagnostic and Statistical Manual, Fifth Edition (DSM-5), or the International Classification of Diseases, Tenth Revision (ICD-10). To fulfill the criteria for a diagnosis of a depressive episode in ICD-10, four depressive symptoms must be present. To fulfill the criteria for a major depressive disorder (MDD) in DSM-5, at least five depressive symptoms must be present. In both systems, the symptoms have to be present for at least 2 weeks, causing clinically important impairment in daily life function, and one (DSM-5) or two (ICD-10) of the symptoms should be among the core symptoms, which are depressed mood, loss of interest or pleasure (DSM-5 and ICD-10), or decreased energy (ICD-10). DSM-5 and ICD-10 comprise similar criteria but may differ in the identification of people fulfilling the criteria for depression (Table 1).

Depression in Later Life, Table 1 Diagnostic criteria of depression according to DSM-5 and ICD-10 (abbreviated)

	DSM-5	ICD-10
Core symptoms	Depressed mood	Depressed mood
	Loss of interest or pleasure	Loss of interest or pleasure
		Decreased energy or increased fatigability
Other symptoms	Weight loss or weight gain, increased or decreased appetite	Decreased or increased appetite with corresponding weight gain
	Insomnia or hypersomnia	Sleep disturbance of any type
	Psychomotor agitation or retardation	Psychomotor agitation or retardation
	Fatigue or loss of energy	Loss of confidence and self-esteem
	Feelings of worthlessness or excessive or inappropriate guilt	Unreasonable feelings of self-reproach or excessive and inappropriate guilt
	Diminished ability to think or concentrate or indecisiveness	Diminished ability to think or concentrate
	Recurrent thoughts of death, suicidal ideation, attempt, or plan	Recurrent thoughts of death or suicide or suicidal behavior

DSM-5 Diagnostic and Statistic Manual, Fifth Edition
ICD-10 International Classification of Diseases, Tenth Revision

A substantial proportion of older persons can have clinically important depressive symptoms but not fulfill the DSM-5 or ICD-10 diagnostic criteria for depression. Only DSM-5 includes specific criteria for depressive episodes with insufficient symptoms, also termed minor depressive disorder or subsyndromal or subthreshold depression. Subthreshold depressive symptoms persisting for more than 2 years may be diagnosed as dysthymia in both classification systems. In DSM-5, persistent depressive disorder also includes persistent MDD. Finally, the DSM-5 and ICD-10 have specific criteria for bipolar depressive disorder, including different kinds of mania as part of the depressive disorder. It is important to keep in mind that DSM-5 and ICD-10 have been developed mainly in younger populations without cognitive impairment or substantial physical disease, and it has been argued that this makes the classification systems less valid in older people, particularly in the presence of cognitive impairment.

Depression in later life (DLL), also termed late-life depression or geriatric depression, is traditionally defined as depression occurring in

persons older than 65 years, but other age cutoffs have been suggested, such as 60 years and even 55 years. Conversely, it has been suggested that the DLL should use a higher age cutoff than 65, because older people now experience better health and everyday function than they did in earlier times. Older persons can have DLL as part of a previously established mood disorder, or the depression can arise for the first time in late life. DLL is sometimes subdivided according to the age of the first lifetime depressive episode. Studies have used different age cutoffs (e.g., 50, 60, or 65 years) to distinguish between depression beginning in early life (early-onset depression [EOD]) and depression with the first manifestation in later life (late-onset depression [LOD]).

There is a complicated interplay between DLL and dementia. Some important issues are summarized in Table 2.

Depression in Later Life, Table 2 Depression and dementia

Depression increases the risk of dementia	There is an association between early-life depression and risk for dementia. It is less clear whether DLL is an independent risk factor for dementia
Depression and dementia share biological pathways	Vascular disease, hippocampal atrophy, pro-inflammatory states, decreased neurotrophic factors are potential biological mechanisms linking depression and dementia
Depression as a prodromal feature of dementia	Patients with depression and substantial cognitive impairment are at an increase risk for developing dementia
People with dementia are at a higher risk of having depression	Almost one in four individuals with dementia experience significant depressive symptoms. Depression is more common in vascular dementia or dementia with Lewy bodies than in Alzheimer's disease
Symptoms of dementia and depression overlap	Diminished interest in activities that were once enjoyed, sleep changes, psychomotor changes, and problems concentrating are common symptoms in both depression and dementia
Symptoms of depression can present different in older adults with versus without dementia	Aphasia in dementia can impede reporting of subjective depressive feelings. Thus, provisional diagnostic criteria for depression in dementia have been suggested, which include observable symptoms such as withdrawal, irritability, and agitation
Treatment of depression with antidepressants is less effective in patients with dementia	The efficacy of antidepressants for treating depression in dementia is uncertain suggesting different biological pathways in depression in patients with dementia

Epidemiology

Depressive disorders are debilitating health problems and important causes of death for adults. Depression among adults across the life span is projected to be the leading cause of disability in middle and higher income countries by 2030. As the population of those aged 65 and over grows, DLL will become a major health problem worldwide. The prevalence estimates of DLL vary according to which diagnostic criteria have been applied, but overall the prevalence rates do not seem to be higher in older persons than they are in younger age groups. However, in subgroups of older persons, the prevalence rates are considerably higher. As in younger age groups, women are more likely to experience depression than men. Compared to the younger group of old adults, depression seems to be more common among the oldest old, often defined as 85+, as most studies find an increasing prevalence of depression with a higher age. However, the association between depression and increasing age seems to disappear when adjusting for physical disease and increased disability in older age. In

community-based samples, the point prevalence of MDD in older people has been reported to be between 1 and 6%, but rates for subthreshold depression seem to be two to three times higher (Meeks et al. 2011). Higher prevalence rates of depression are found among old individuals in institutions, such as residential care or nursing home care facilities. Depression is also more prevalent in individuals with somatic disease, particularly brain disorders. Depression may occur in up to half of those who suffer from Parkinson's disease or in those who have had a stroke. The prevalence estimates of depression in dementia are high but vary widely, reflecting the difficulty in defining and diagnosing depression in the context of dementia. To improve the diagnosis of depression in dementia, provisional diagnostic criteria for depression have been suggested, but their validity remains uncertain. Overall, depressive episodes in later life are more likely to be a recurrence rather than a first-time episode.

Etiology

Several biological, psychological, and social factors can interact and thus contribute to the development of depression. A biopsychosocial model of etiology seems to be particularly appropriate to DLL, highlighting that the causes of DLL are multiple and range across all three domains (Blazer 2003). It is useful to consider both predisposing and precipitating factors when putative causes of depression in an individual are assessed. There is still limited knowledge about why some older adults develop depression and others do not, even though they seem to be affected by the same set of risk factors.

Biological Factors

DLL regularly arises in the context of medical illness. There are several well-established physical risk factors like ischemic heart disease, chronic obstructive pulmonary disease, diabetes, malignancy, chronic pain, and organic brain diseases.

In addition, the use of drugs may play a central role in the development of depression in older adults. The role of alcohol is especially important to consider in the etiology of DLL given that the rates of alcohol consumption have risen among older adults, and it is well established that alcohol use is linked to lower mood and depression. Older individuals also use more medication more often than younger individuals, and it has been suggested that polypharmacy may be associated with the risk of depression. However, empirical evidence is not consistent, and the results are difficult to interpret because the condition for which the medication is taken often confers an increased risk of depression. Finally, substance dependence can also be a factor in the etiology of DLL and can be easily missed if not assessed in an older patient.

Brain Anatomy

Research indicates that certain areas or circuits of the brain are relevant to the etiology of DLL (Naismith et al. 2012). These areas include the dorsolateral prefrontal cortex, orbitofrontal cortex, anterior cingulate cortex, subcortical white matter, basal ganglia (especially striatum), and the hippocampus. Dysfunction in frontal-subcortical neural networks involving these areas seems to be associated with the onset and prognosis of DLL.

Neurotransmitter Dysfunction

The monoamines, namely, serotonin, noradrenaline, and dopamine, are important modulating neurotransmitters for mood and behavior. Dysfunction in serotonergic and noradrenergic neurotransmission and, to a lesser extent, dopaminergic transmission has been demonstrated in DLL (Thomas 2013). An association between abnormalities in these neurotransmitters and depression is also supported by the fact that antidepressant medication targeting serotonin and noradrenaline function improves depressive symptoms. Dysfunction in other neurotransmitters associated with the occurrence of depression includes gamma-aminobutyric acid (GABA) and glutamate. All of these neurotransmitters have widespread projections to the prefrontal cortex. Even though dysfunction of monoaminergic transmission is shown in DLL, it is not completely clear how aging affects the neurotransmitters.

Some evidence suggests, however, that the age-related changes of the neurotransmitters can make older persons more vulnerable to mood disorders.

Genetics

Hereditary factors could predispose some older persons to depression. There has been great interest in genetic susceptibility across the life cycle, but specific genetic markers for DLL have not been identified. Heritability appears to be related to multiple loci of the genetic material (DNA) with small effects rather than few loci with large effects. Genetic factors have been found to have a greater impact in DLL with EOD. Recent genetic research has focused on the serotonin transporter (5HTTLPR) gene, apolipoprotein E (ApoE) gene, brain-derived neurotrophic factor (BDNF) gene, and 5-methylenetetrahydrofolate reductase (MTHFR) gene and has found that these genes may be involved in the development and treatment response of DLL (Naismith et al. 2012).

Immune System

Scientific knowledge regarding the interplay among the nervous, endocrine, and immune system has expanded immensely in recent years. It is suggested that these systems should be regarded as a single network that gives rise to the new discipline of psychoneuroimmunology (Thomas 2013). Research has shown that aging can lead to an increased peripheral immune response, impaired communication between the immune system in the central nervous system (CNS) and peripheral nervous system (PNS), and a shift toward a pro-inflammatory state of the immune system in the CNS. Raised levels of pro-inflammatory cytokines, such as IL-1β, IL-6, and TNF-α, have been reported in studies of DLL. It is probable that aging and comorbid diseases may alter neuroinflammation and predispose individuals to DLL (Alexopoulos and Morimoto 2011).

Dysregulation of the HPA (hypothalamic-pituitary-adrenal) axis has been suggested as a cause of depression in older and younger adults. The associated high glucocorticoid levels may have a toxic effect on the brain, particularly the hippocampus. This has been forwarded as an explanation for the increased risk of dementia in people with depression, although findings linking high glucocorticoid levels with hippocampus atrophy are conflicting.

Vascular Disease

There is a well-established bidirectional association between vascular disease and depression. This includes coronary heart disease as well as cerebrovascular disease (i.e., stroke). The white matter of the brain is composed mainly by myelinated nerve fibers. Lesions to the white matter identified on MRI, or white matter hyperintensities (WMH), have been studied extensively in relation to depression. It is presumed that WMH are caused by chronic hypoperfusion of the white matter and the disruption of the blood–brain barrier. WMH are related to vascular risk factors, the risk of depressive episodes, poorer remission, and cognitive impairment. The strong relationship between cerebrovascular disease and depression has led to the "vascular depression" hypothesis, which postulates that cerebrovascular disease can predispose, precipitate, and perpetuate depressive syndromes in later life by damaging frontal-subcortical circuits (Alexopoulos 2005). However, the concept of a vascular depression has received some criticism and it has proved difficult to reliably identify such a subgroup. Nevertheless, vascular disease is likely to be an important factor in about 50% of people with DLL (Thomas 2013).

Psychosocial Factors and Personality

It is a common view that psychosocial factors are most important in mild to moderate depression, whereas biological factors play a greater role in severe depression. The scientific evidence for this view is rather limited, and the evaluation of possible psychosocial etiological factors should be part of the assessment regardless of the severity of depression.

Several psychological factors are associated with depression. Relatively little research on the association between personality and depression has been done, and the interpretation of the results is difficult. Most studies are cross-sectional or retrospective, and the recall of earlier personality

traits may be influenced by the present situation. It is also difficult to establish what came first, the depressive disease or the presumed personality trait. Furthermore, it is complicated to disentangle the contribution of the personality traits from the social situation of the person as risk factors for depression.

There is some evidence that a high level of neuroticism is linked to DLL. Neuroticism is a personality trait characterized by worry, fear, anxiety, guilt, and moodiness. People with a high level of neuroticism can be sensitive to life stressors and may interpret minor situations as threatening or hopelessly difficult. It has been suggested that older persons with depressive syndromes can display cognitive distortions, where they generally overrate their own mistakes and exaggerate negative outcomes of life events and where loss and defeat are core themes.

High levels of mastery of one's environment and self-efficacy have been shown to provide protection against DLL. A higher sense of control, an internal locus of control, and more active strategies have been found to be associated with fewer depressive symptoms (Bjorklof et al. 2013).

Learned helplessness is the idea that individuals behave according to the expectation that acting in continually stressful situations has no meaning. Older adults frequently encounter circumstances such as chronic physical illness and disability that may lead to learned helplessness, and this notion has been linked to the occurrence of DLL (Aziz and Steffens 2013).

Life Events

Stressful life events can be seen as an integral part of becoming old, but some types of stressful life events, such as divorce or criminality, are less common in old age. It could also be argued that stressful life events are more often expected in late life, making it easier to deal with them.

As a person grows older, he or she will inevitably deal with different types of loss. For example, these losses include loss of position in society, loss of a job, loss of financial and functional independence, and loss of a social network and loved ones. These losses may produce grief that develops into depression.

Social support may act as a buffer to stressful life events, and it is documented that impaired social support is related to DLL. However, it is important to bear in mind that the majority of people who experience significant losses in old age do not develop depression. Hence, the meaning of loss has to be interpreted in the context of the person's mastery style, social situation, and other predisposing factors (Aziz and Steffens 2013).

Clinical Picture

Several studies have shown that clinicians at various levels fail to recognize depression in older persons. There may be a tendency to attribute depressive symptoms to the normal aging process. This may also explain the reluctance of some old people to view their symptoms as signs of depression. It is important to stress the fact that depressive symptoms are not a consequence of normal aging. The most plausible reason for the low detection levels of depression is probably the rather complicated interplay between normal age-related changes, symptoms of somatic disorders and depressive symptoms. This may cause clinicians to miss the diagnosis or also hinder insight by the person with depressive symptoms.

The ICD-10 criteria for depressive episode and DSM-5 criteria for MDD are identical for both younger and older patients (Table 1). The core symptoms of depression are depressed mood, loss of interest or pleasure, and decreased energy (the latter only in ICD-10). Additional symptoms defined in the diagnostic criteria are loss of confidence, an excessive feeling of guilt or worthlessness, difficulty concentrating, change in psychomotor activity, disturbance of sleep, change in appetite with corresponding weight change, and suicidality.

The clinical presentation of depression in old people differs from what is seen in younger age groups. The aging process, cognitive impairment, reduced physical health, polypharmacy, and disability can contribute to a more heterogeneous presentation of a depression syndrome in older individuals. Older adults may be less likely to

describe their suffering in ways that match up to common depressive symptoms. For instance, older persons with frank depression rarely describe experiencing feelings of sadness. This has led to the term "depression without sadness." More recent research, however, has challenged the view that there is a specific phenotype in depression among old people, suggesting that the key symptoms of depression are the same, irrespective of age (Thomas 2013).

However, it seems that some symptoms are more prominent in DLL, with cognitive impairment being the most important. Various expressions have been used to describe cognitive impairment in depression, with pseudodementia being the most common. Pseudodementia refers to depression that is misdiagnosed as dementia due to marked symptoms of cognitive impairment. This term has fallen out of use, however, given the persistent nature of cognitive deficits in depression, even after the depression has been successfully treated and recent evidence suggesting that depression is a risk factor for dementia (Butters et al. 2008). The characteristic pattern of cognitive impairment in depression includes impaired attention and executive and amnestic impairment, whereas apraxia, visuospatial impairment, and aphasia may indicate that the cognitive impairment stems from a comorbid dementia disorder. People with a substantial cognitive impairment as part of their depressive episode should be followed-up closely, even if the cognitive impairment is reversed after the treatment of depression, because the risk of developing dementia in the following year is higher in this group.

Other patterns of the symptom profile in DLL are somatization or hypochondriasis, psychomotor retardation, anxiety, and agitation. It should be noted that some of these symptoms are also common in other diseases that frequently occur in old age, such as chronic obstructive pulmonary disease and coronary heart disease.

Psychotic symptoms seem to be more common in DLL compared to depression in younger adults.

There is evidence that for many patients with dementia, the depression syndrome may differ from the diagnostic criteria in the ICD-10 and the DSM-5. Thus, provisional criteria for depression in patients with Alzheimer's disease have been suggested. These criteria require fewer symptoms for a diagnosis of depression and the symptoms do not have to be present nearly every day. In addition to the depressive symptoms described in ICD-10 and DSM-5, the criteria for depression in Alzheimer's disease also include social withdrawal or isolation and irritability (Olin et al. 2002).

Assessment of Depression

In addition to a thorough disease history that considers biological and psychosocial risk factors, the use of a structured assessment scale for depression is recommended. A few scales have been developed for use in old people, such as the Geriatric Depression Scale (GDS) and the Cornell Scale for Depression in Dementia (CSDD); the latter is also used in people without dementia. Other well-known scales, such as the Montgomery-Åsberg Depression Rating Scale (MADRS), the Hamilton Depression Rating Scale (HAM-D), the Beck Depression Inventory (BDI), the Patient Health Questionnaire (PHQ), and the Hospital Anxiety and Depression Rating Scale (HADS), are frequently used, and the psychometric properties of most of these scales are found to be acceptable in the assessment of DLL. Reporting depressive symptoms may be hampered by cognitive impairment and the assessment may have to include a proxy-based assessment, such as the CSDD. Given the large proportion of people with DLL who experience impaired cognition, a structured assessment of cognition should be included in the diagnostic process, whether or not a dementia disorder is suspected.

Suicidality

The suicide rates in older adults, particularly in men, have risen. Older men have few suicide attempts per completed suicide, i.e., they choose more lethal methods. An assessment of suicidality should be part of all assessments of DLL. As with any patient population, the older patient must be approached sensitively. Nevertheless, an explicit and specific exploration of suicidal thoughts

should be carried out during the assessment. Older men who commit suicide often seek medical help prior to the attempt, but symptoms of depression or suicidal thoughts are rarely mentioned. Practitioners need to be aware of this and have suicidality in mind when older men seek advice about other conditions, particularly issues concerning pain management. Established risk factors for suicide among old people are bereavement, social isolation, earlier attempts, chronic painful illness, disability or the threat of increasing disability, drug or alcohol use, and sleep problems (Manthorpe and Iliffe 2010). Despite the concern about the high rate of suicide among old people, this issue has received little attention, particularly when compared to the attention toward suicidality in younger people. Practice guidance on how to reduce the risk is lacking, and intervention studies are scarce.

LOD and EOD

Some researchers suggest etiological and clinical differences between EOD and LOD. EOD is associated more with a family history of depression, personality dysfunction, and severe disorders. EOD is regarded as a risk factor for the later development of dementia. LOD is associated more with WMH on MRI, prominent cognitive impairment, and it relates more to systemic vascular risk and neurodegenerative disorders. There is a debate as to whether the symptom profile of depressive symptoms defined in the classification systems is different in EOD and LOD patients.

Bipolar Disorders in the Late Life

The number of people seeking care for bipolar disorders is increasing. Bipolar disorders can develop early, i.e., onset before 50 years of age, or can arise with a late onset, i.e., after 50 (different cutoffs between 50 and 65 have been used). Bipolar disorders in late life include both early and late onset. Due to the complexity and heterogeneity in the classification of the disease, prevalence rates vary. Among older patients with bipolar disorder, most have their first episode of mania or depression early in life; in the minority, a bipolar disorder may present itself for the first time in old age. In that case, the diagnostic process may be challenging due to the extensive medical comorbidity. Medical comorbidity in bipolar illness is associated with a more disabling course of the illness and a higher risk of suicide (Sajatovic and Chen 2011). Psychiatric comorbidity, such as anxiety disorders or substance use disorders, is often less common among older people than younger people with bipolar disorder. Patients with a late onset of bipolar disease tend to have less history of mood disorders in their family. About half of all older patients with bipolar disorder have depression as their first mood episode.

Treatment

Before starting treatment, a careful assessment focusing on the biopsychosocial aspects of DLL is needed. The assessment should not be restricted to counting symptoms in order to establish a diagnosis; the meaning or the impact of the depressive symptoms to the individual person needs to be taken into account. Functional limitations and disability, disease history, and the duration of symptoms are key issues to keep in mind when weighing the benefits of treatment against risks. Earlier treatment experiences and preferences of the patient should be taken into account. A careful explanation of the treatment plan involving the patient – and if appropriate a family caregiver – is mandatory for treatment success, as low treatment adherence has been reported among old people.

A stepped care approach, identifying the least restrictive and least costly intervention that will be effective for a person's presenting problems, is recommended (NICE 2010). People with subthreshold depression without a significant impact on everyday life should be offered supportive and psychosocial interventions, but they should normally not be offered medical treatment. In milder forms of depression or persistent subthreshold depression, more intensive psychotherapeutic approaches are advocated. Drug treatment should still not be a first-line treatment option, but should be considered if other alternatives fail to produce

substantial improvement. In moderate or severe depression, drug treatment should be offered, often in combination with intensive psychotherapeutic treatment.

In the treatment of depression, it is important to aim for remission (i.e., patients do not meet the diagnostic criteria for depression or they have no more than minimal depressive symptoms according to a depression assessment scale) and not merely for response (i.e., significant symptom reduction), because residual symptoms after treatment are strongly associated with a risk for relapse. Once in remission, a plan for the continuation of treatment should be established. There is reason to believe that maintenance therapy should be offered more liberally in DLL than in younger age groups, due to a greater risk of relapse.

Psychosocial Interventions

Older patients with minor or mild depression can benefit from participating in various types of social activities to prevent isolation and loneliness, e.g., befriending services and attending day centers and local community events. Physical exercise includes bodily activity that enhances overall health and wellness. There is evidence that structured exercise programs can help older patients with milder depressive syndromes. Different kinds of exercises can be beneficial, but results are most consistent from aerobic exercise. However, there are also studies that have failed to find a positive effect of physical exercise in DLL.

Psychotherapy

Research indicates that psychotherapy can be an effective treatment for DLL even though the quality of studies is relatively low (Wilson et al. 2008). There is a variety of therapies that may be applied, such as supportive therapy, life-review therapy, cognitive-behavioral therapy (CBT), interpersonal therapy (IPT), and problem-solving therapy (PST). Psychotherapy can be offered to individuals (in- or outpatients), couples, families, and as group therapy. Supportive treatment and adding structure to the day can be effective in patients with minor depression syndromes. CBT focuses on here-and-now situations as well as the link between negative thought patterns and mood and behavior and is often structured in sessions and length. CBT is widely studied and applied in DLL with mild to moderate severity. IPT is also based on here-and-now situations, but emphasizes interpersonal relationships. PST is based on CBT principles, but is a more focused treatment approach. PST aims to teach patients to better define their problems and goal, and the strategies to cope with the problems, carry out the strategies, and then evaluate them. PST has shown strong results for depressed patients with executive dysfunctions. As a result, it has been suggested as a key treatment approach in "vascular depression," where it has been implicated that the dysfunction of frontostriatal circuits gives rise to executive impairment (Espinoza et al. 2014).

Psychotherapy, in combination with medical treatment, may be more efficacious than any of the two modalities alone in the treatment of DLL, both in the acute phase and as maintenance therapy.

Medication

A number of issues need consideration when prescribing antidepressive medication to older adults. As noted earlier, polypharmacy is common in older individuals. Medication with negligible side effects in healthy young people may cause serious side effects in older adults who take many prescribed drugs, especially when several of those drugs could have direct effects on the brain. An example is the rather weak anticholinergic effect of a drug like paroxetine; in combination with other drugs with weak anticholinergic effects, it may cause confusion or delirium in susceptible individuals. Pharmacokinetic changes, such as increased distribution volume, reduced hepatic metabolism, and reduced glomerular filtration rates, may lead to higher plasma and brain levels of the drug. However, there is great variation among older adults in these changes. The slogan "start low, go slow" that was often voiced in old-age psychiatry may be appropriate, but should not prevent older patients from being treated with adequate doses. When evaluating dosing regimens, the polymorphisms of key enzymes of the cytochrome P450 system involved in the metabolism of several psychopharmacological

substances should be taken into account. A considerable proportion of individuals can have polymorphisms that may cause great variation in the plasma level of a medication. In cases of unusual side effects at low doses or treatment resistance, an analysis of P450 enzymes may be indicated.

Most studies regarding drug treatments for depression have been done in samples with MDD. Hence, the results cannot readily be extrapolated to people with mild depression or subthreshold depression. The effect of antidepressants in treating DLL is well documented (Nelson et al. 2008). However, there is great variability among studies. The older tricyclic antidepressants (TCAs) have a comparable effect to the new ones but a higher prevalence of side effects – particularly anticholinergic and antiadrenergic effects – that have made them less useful in treating DLL.

Contrary to the positive treatment effect in older adults without substantial cognitive impairment, most of the studies concerning the use of antidepressive treatment in patients with dementia have failed to show an effect (Nelson and Devanand 2011). This may be because of an inability to define homogenous patient groups with depression and dementia. Symptoms of depression and dementia partially overlap and cognitive impairment may prevent any verbalization of the depressive symptoms. Furthermore, people with dementia may be more susceptible to adverse events. Taken together, there is not enough evidence to suggest antidepressive therapy as a first-line treatment in people with dementia except in specific cases, such as very severe depressive symptoms or a history of earlier episodes that have responded to treatment.

There are a large number of antidepressive drugs to choose from, but selective serotonin reuptake inhibitors (SSRIs) are the first choice in most instances, in line with most clinical guidelines. These drugs are generally well tolerated and they have a predictable interaction profile. Nonetheless, recent studies indicate that SSRIs may also be associated with serious adverse outcomes, such as increased QT interval, falls, and hyponatremia. SSRIs may be combined with other antidepressants; combining SSRIs with mirtazapine or mianserin can be particularly useful for patients with sleeping problems and low appetite. Serotonin and noradrenaline reuptake inhibitors (SNRIs) have adverse event profiles similar to SSRIs and are a useful second-line treatment option because of their somewhat broader receptor profile. Because older adults with depression constitute a heterogeneous group, the prescription of antidepressive medication should be individualized based on the side effect profile of the drug, previous medication history, somatic diseases, and the use of other drugs.

Monotherapy is preferred, but in cases of treatment resistance, augmentation therapy with other drugs may be tried. The best evidence is for augmentation with lithium, used for bipolar disorder (Cooper et al. 2011). However, lithium serum levels have a very narrow therapeutic window and require careful observation in order to avoid potentially serious adverse events.

Electroconvulsive and Neuromodulation Therapies

Electroconvulsive therapy (ECT) is well tolerated and efficacious in treating DLL (Riva-Posse et al. 2013). It should be an option in people with severe depression when other treatment alternatives have failed. In many countries, ECT is reserved for severe depression with psychosis, suicide risk, or life-threatening refusal of food or fluids. Concerns about using ECT in DLL have been raised, especially the fear of precipitating delirium or memory impairment. Recent studies demonstrate a faster remission in patients treated with ECT than patients treated with antidepressants, without extra side effects. This suggests that the indication for ECT could be broader. The high relapse rate after ECT is a therapeutic challenge; maintenance therapy may be indicated. Other stimulation therapies, such as transcranial magnetic stimulation, vagal stimulation, or deep brain stimulation, have been tried out in selected patient groups, but these alternatives are not easily accessible and there is limited evidence to date to justify their use in clinical practice.

Prognosis

DLL is associated with a number of negative outcomes, such as disability, cognitive impairment, poorer outcomes of physical disorders, and an increased risk of mortality. Remission rates of DLL after treatment are not different from those in younger age groups; however, relapse rates are higher (Mitchell and Subramaniam 2005). The risk of relapse is highest for the first 6 months. Hence, it is important to continue treatment for at least 6–9 months. Even after the first depressive episode in old age, the relapse rate is high after the treatment has been discontinued. This has led many to recommend lifelong maintenance treatment even if the first depressive episode has a later onset, particularly if it was an episode of great severity. This recommendation has to be weighed against risks associated with polypharmacy, side effects, and other risk factors for relapse, such as cerebrovascular pathology, other physical diseases, and cognitive impairment.

Conclusion

DLL is a disease with vast consequences for affected individuals and society as a whole. The symptoms are well characterized and there is a huge knowledge base around epidemiology, etiology, and treatment. However, a substantial part of the knowledge relates to younger age groups, which has been extrapolated to DLL. There is reason to believe that there are important etiological and clinical issues that apply to DLL, which differ from what we see in younger age groups. Yet, there is probably greater diversity among older versus younger age groups. This calls for a careful assessment and consideration of biological and psychosocial issues common with advancing age. Particular attention should be paid to comorbid physical diseases, cognitive impairment, and distressing life events. The increased vulnerability of some older adults to depression in itself and treatment side effects, the uncertain efficacy of treatment in subgroups, and the high relapse rate in DLL call for close follow-up. Lastly, the high risk of suicide, particularly in older men, warrants special attention among all health workers providing care for older individuals.

Cross-References

- ▶ Anxiety Disorders in Later Life
- ▶ Bipolar Disorder in Later Life
- ▶ Cognitive Behavioural Therapy
- ▶ Comorbidity
- ▶ Dementia and Neurocognitive Disorders
- ▶ Grief and Bereavement: Theoretical Perspectives
- ▶ Mental Health and Aging
- ▶ Mild Cognitive Impairment
- ▶ Problem-Solving Therapy
- ▶ Psychological and Personality Testing
- ▶ Subsyndromal Psychiatric Disorders
- ▶ Suicide in Late Life

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Disability and Ageing

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Synonyms

Age-related disease; Age-related impairment; Chronic disease; Functional change and loss; Impairment

Definition

Disability is a broad term that has multiple definitions. The World Health Organization (World Health Organization 2012) defines disability as encompassing:

1. Impairments: a problem or problems with bodily structure or function
2. Activity limitations: a problem or problems experienced by an individual when attempting to carry out an action or task
3. Participation limitations: a problem or problems in dealing with life situation (e.g., social, vocational)

The term disability, however, is not limited to health conditions. In fact, the International Classification of Functioning, Disability, and Health views *disability* as an umbrella term (World Health Organization 2012). In their definition, disability is the interaction between environmental and personal factors (e.g., stigmatization, access to healthcare, social support) and a health condition (e.g., schizophrenia, cardiovascular disease). This means that experiencing a disability is really the combination of *both* some health

condition and how you are treated and/or limited as a result of it (World Health Organization 2012).

Disability is not an inevitable part of aging, but the odds of experiencing a disability or living with a disability increase with age. As this entry will show, many age-related changes are associated with disability (e.g., age-related eye degeneration resulting in cataracts leading to visual disability) (Hoyer and Roodin 2009).

The Nature and Causes of Disability for Older People

As of 2010, approximately a billion people (around 15% of the world's population) were estimated to live with some form of disability. Among these, 2–4% were estimated to have severe disability that dramatically impaired functioning (e.g., quadriplegia, blindness). Rates of disability (i.e., experiencing difficulty in performing activities), and severe disability (i.e., being prevented from performing activities), increase with age (World Health Organization 2011). We can use the USA as an example here. In the USA, fewer than one in five people aged under 65 report a disability (2010 US Census data) (Brault and United States. Bureau of the Census 2012). This increases to about 50% in adults aged 65 years and over. In this age bracket, one in two will report a disability. Over a third will live with a severe disability. For people in their 80s, rates of disability are close to 75% and severe disability 60% (Brault and United States. Bureau of the Census 2012).

The way that disability is assessed and recorded, however, differs country to country. This means that it is hard to accurately estimate how many people are disabled, let alone how many older people are disabled. According to best estimates, however, approximately 30% of adults aged 60 years and over in higher-income countries have a disability, and approximately 45% of adults over 65 in lower-income countries live with a disability. For example, the rates of disability are substantially higher in African and Southeast Asian nations than they are in the Americas or Europe (World Health Organization 2012). International differences notwithstanding, with an expanding older population, globally, there are more people living with disability today than there have been in the past.

In general, part of the reason that we are living longer is because we are better at preventing and treating communicable disease. Communicable diseases are those that can be spread between people or between people and animals. Vast reductions in infectious and parasitic disease have resulted from effective and available immunization, attempts to manage poverty, and improvements in diets and infrastructure. Thus, at present, in developed or high-income countries, the leading causes of disability (as well as disease and death) are noncommunicable (e.g., arthritis, cancer, mental health disorders). The same is true of middle-income countries – it is only in developing countries that the leading cause of disease and death remains communicable disease (alongside maternal, perinatal, and nutritional conditions). It is estimated that by 2030 this will change – noncommunicable disease will be the leading cause of disability, disease, and death worldwide (World Health Organization 2011).

To some extent, this represents a challenge to the way that disability is traditionally conceptualized. When we think about disability in aging, we often draw on standard stereotypes – imagining someone in a wheelchair or someone who is vision impaired. The reality is that disability is varied, not only in its nature, but also in the extent to which it affects or limits people.

The most common disability-related health conditions in Australia and Canada are arthritis, back problems, and hearing problems (World Health Organization 2011, 2012; Australian Bureau of Statistics 2012). Others include heart disease, hypertension, asthma, diabetes, stroke, depression, dementia, speech disorders, and vision disorders. In the USA, rheumatism and heart problems represent the most common causes

of disability among adults 65 or older (World Health Organization 2011; Centers for Disease Control Prevention 2009). As of 2011, the most common health conditions in developing countries were heart disease, stroke, cancers (breast, prostate, and lung), sensory problems (cataracts and glaucoma), hearing loss, and musculoskeletal impairment (osteoarthritis and osteoporosis) (World Health Organization 2011).

Older people with disability can either enter old age with a preexisting disability or develop a disability in later life (either due to age-related factors or other factors such as communicable disease or accident). As highlighted above, however, rates of disability increase with age, in part due to biological change. For example, as we age, visual deterioration is common. This includes declines in accommodation (the ability of the lens to focus), contrast sensitivity, and sensitivities to glare (Hoyer and Roodin 2009). Changes in the eye give rise to visual pathologies. Approximately 70% of adults aged 80 or over have cataracts, with 20% and 7% of the same age group experiencing age-related maculopathy and glaucoma, respectively (Resnikoff et al. 2004). Similarly, with age comes a predictable breakdown of cells in the inner ear (albeit at different rates for different people). This can result in hearing impairment, with approximately 35% of men and 22% of women aged 70–74 experiencing such impairment, and this rises to 58% of men and 49% of women at 85 years or older (Mathers et al. 2000). Taste, smell, and touch sensitivities also decline. In the case of touch, this can be particularly problematic – as insensitivity to touch and pain can lead to accidents and subsequent disability (Hoyer and Roodin 2009). Finally, loss of bone density and muscle mass, circulation, and respiration are also part of the normal aging process (Deschenes 2004). As this highlights, many factors contribute to disability and all can hinder effective participation in many activities of daily living (Hoyer and Roodin 2009).

Factors Exacerbating Disability

While age increases the risk of developing diseases and disabilities, the cumulative effects of adverse lifestyle or environment can expedite disability in later life (World Health Organization 2011, 2012). One of the most consistent predictors of disability is socioeconomic disadvantage. Poor nutrition, and inability to access healthcare, increases the risk of developing a disability (World Health Organization 2012). At the international level, rates of disability in the USA are high when compared to other developed countries. The reason for this is largely assumed to be ready and equal access to healthcare provided by governing bodies. Similarly, healthcare is often difficult to access in low-income countries. Just as there are higher rates of disability within low-income countries than within high-income countries, so too are there higher rates of disability in people of low socioeconomic status (SES). Poverty has a cumulative effect, and this becomes more evident in later life. Further, poverty is more evident among the elderly (World Health Organization 2011). Those born into poverty are more likely to develop a disability – and if they survive into old age, carry it with them. The prognosis and quality of life for those with a disability who experience poverty are worse than for those with a disability who do not experience poverty. Thus, there appears to be a cycle of disability – where poverty breeds disability and also exacerbates it. Gender also interacts with poverty. As women live longer than men, on average they are more likely to experience poverty in old age (Hoyer and Roodin 2009).

The Impact of Disability on Older Adults

The most obvious impact of disability on older adults is in the realm of self-care. Physical disability in older adults can prevent them from being able to independently move in and out of bed, leave the house, and engage in house maintenance (Brault and United States. Bureau of the Census 2012). In fact, as of 2010, at least one in ten American adults aged 65 or older reported needing assistance in leaving the house, with a similar proportion reporting needing assistance with housework (Brault and United States. Bureau of the Census 2012). When we consider the fact that

many people with disabilities require doctor or hospital visits, as well as pharmacy medication, any disability that prevents them from leaving the house would exacerbate challenges associated with disability management should they not have access to assistance.

Disability can impact on basic activities of self-care or *activities of daily living*. These include the ability to bathe, dress, and toilet independently. *Instrumental activities of daily living* – like paying bills, shopping and food preparation, and taking medications appropriately – require some degree of planning and intellectual engagement (Cavanaugh and Blanchard-Fields 2014). In the USA, of Medicare enrollees 65 years or older, approximately 41% needed some assistance with these activities. Twelve percent of adults aged 65 years or older needed help with instrumental activities only, with the remaining 29% also requiring assistance with at least one activity of daily living (Cavanaugh and Blanchard-Fields 2014). The most common problems include difficulties in walking, bathing, dressing, using the toilet, getting in and out of bed, and eating (Cavanaugh and Blanchard-Fields 2014). Impairments in these areas increase with age. In the case of walking, approximately 15% of adults aged 65–74 years are having difficulty in doing so, compared to almost 50% of adults aged 85 years and older. Around 20% of adults aged 65 or over require either the use of a walking aid (e.g., cane, walker, crutches) or wheelchair for mobility (Centers for Disease Control Prevention 2009). Importantly, when an older person becomes restricted in some capacity, their decline is more rapid and recovery protracted, thus increasing the likelihood of additional disability that further limits their ability to live independently (World Health Organization 2011; Hultsch et al. 1999)

Chronic disabilities are a robust predictor of falls in the elderly (as can be assumed with over 50% of adults 85 and older reporting difficulties walking). Further, in the USA, accidental injury is the fifth leading cause of death in older adults after cardiovascular disease, cancer, stroke, and pulmonary disease (Rubenstein 2006). Falls themselves account for approximately 60% of these deaths. As a consequence, data in the USA reveals that falls are the most common causal factor of restrictions of activities of daily living (Rubenstein 2006). The more chronic health conditions an elderly person reports, the more likely they are to fall and fall recurrently (Tinetti et al. 1986). Thus, we can see that disability in and of itself puts people at the risk of future disability (World Health Organization and Ageing Life Course Unit 2008).

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Participation in Society

Disability in the elderly often puts limits on their ability to live happy, fulfilled lives. This is not just because of problems associated with activities of daily living (e.g., walking, getting dressed); rather, impairments can also impose barriers to social and vocational interactions. For example, difficulties in vision can prevent older adults from driving (Hoyer and Roodin 2009). Mobility difficulties can prevent catching public transport, as can the availability or affordability of public transport (Gilhooly et al. 2002). Thus, disability can lead older adults to withdraw from social activities or cease attending gatherings or going on outings.

Physical and/or cognitive disability may also prevent older adults from engaging in work-related activities. There are a number of reasons for this. Firstly, physical disability may prevent someone from performing a job that they previously held (e.g., problems with walking may prevent a farmer from farming). However, potent misconceptions about the disabled elderly (including those held by the elderly themselves) can also prevent older adults with disabilities who desire employment from seeking and attaining it (World Health Organization 2012). For example, older adults with a disability are often excluded from disability services that aim to provide rights and opportunities to those living with a disability (Jönson and Larsson 2009). In Sweden, for example, a system of long-term support (personal assistance) has been introduced for those living with a disability who are *under the age of 65* (Jönson and Larsson 2009). Researchers argue that ageism affects disability here – whereby many conflate disability and aging (i.e., assume that disability is a normal and natural part of aging) (Jönson and Larsson 2009). Thus, older adults with a disability

often are not able to take advantage of programs designed to support them in pursuing paid work, among other things.

As prefaced above, older adults with disabilities are likely to face discrimination. Some researchers have argued that the recent focus on "positive," "successful," or "healthy" aging has meant that older adults with a disability are stereotyped as people who age "badly" or "unsuccessfully" (Minkler 1990). When looking at specific prejudices, mental disabilities are stigmatized, and physical disabilities are often assumed to extend to cognitive impairment. Those with dementia are sometimes seen as "less than human" and consequently are not afforded time and companionship.

Whether through physical barriers, or social exclusion, isolation can have a severe negative impact on older adults with a disability. A primary predictor of longevity is the strength and quality of our social relationships (often marriages (Tucker et al. 1996)). Older adults with strong social networks thrive – especially when they enjoy close and meaningful relationships (Hoyer and Roodin 2009). When disability limits this, either through preventing socializing or through increased incidents of discrimination, older adults with disability are likely to experience declines in health and quality of life.

Societal Impact and Management of Disability in Aging

Given the rising number of people with a disability, there is a considerable burden experienced globally – both in terms of health and finances. Financial costs are borne by the disabled themselves, governments, and individual carers (and families). One report estimated that, in the period between 2006 and 2015, the financial cost of heart disease, stroke, and diabetes in 23 low- and middle-income countries approached $US100 billion (World Health Organization 2011). In 2009, the cost associated with new cancer cases in the USA was estimated at $US286 billion. The worldwide cost of dementia in 2010 was estimated to exceed $US600 billion (World Health Organization 2011). Note that this figure includes nonprofessional care provided by family members. In fact, the majority of older adults with a disability do not live in aged care facilities. In Australia, one in ten people reports being a carer for a person with a disability (Australian Bureau of Statistics 2012). The majority of carers are female (70% of primary carers), and of carers themselves, approximately a third have a disability. Labor force participation is lower for carers, who often spend more than 40 h a week in their caretaking roles (Australian Bureau of Statistics 2012).

The cost of caring for older adults is not just financial; it is also emotional. Carers are typically overworked and often unpaid. They face substantive stress, especially because their role often involves negotiating and managing the current impairment and the future consequences of the impairment (palliative care and death), which is often not recognized publically (Hoyer and Roodin 2009).

The costs detailed above, both to societies and individuals, highlight the importance of looking at disability in older adulthood at national, and global, levels. When it comes to disability, generally, the World Health Organization recommends that multiple environmental changes should be implemented to improve the lives of those with disabilities (World Health Organization 2011, 2012). For example, it is recommended that policies concerning accessibility of education and healthcare be designed with specific reference to meeting the needs of disabled people. Funding and the provision of services for those with disability need to be increased. At a very basic level, built environments should be designed to be accessible to all. Negative attitudes and poor standards of care need to be combated. In each case, it is recommended that extensive consultation with people with disabilities is undertaken and that any programs instituted are rigorously documented and evaluated (World Health Organization 2012).

When it comes to programs specifically designed to help older adults with disabilities, multiple successful examples can be found. For example, in Japan, free social exercise classes are made readily available to older adults living in large cities (Hoyer and Roodin 2009). Indeed,

exercise programs in the USA have been shown to reduce disability and pain for older adults with knee osteoarthritis (Ettinger et al. 1997). Community-based programs in the USA aimed at preventing disability in older adults, as well as promoting disease self-management, have been shown to reduce functional decline and length of hospital stays (Wagner et al. 1998).

On an individual level, managing disability in older adulthood – much like disability itself – is complex. Several factors have been identified, however, that reliably delay the onset of disability. Most importantly, exercise is a factor that has been shown to increase both physical health and mental health and is effective in delaying the onset of dementia (Cotman and Berchtold 2002) and preventing physical disability (see above). Cognitive stimulation is also important. Older adults who remain in the workforce until later in life display better cognitive integrity than those who retire early, and it is likely that cognitive challenge is protective (World Health Organization 2011). Finally, a strong social network is vital. Older adults do not necessarily benefit from having a large social group. Rather, they are most healthy when they report close, developed, and deep friendships (Hoyer and Roodin 2009). A reduction in smoking, drinking, and drug taking also reduces the chance of disability (Hoyer and Roodin 2009; Cavanaugh and Blanchard-Fields 2014).

Conclusion

Disability is multifaceted. The current statistics on rates of disability vary country to country, in large part due to differences in definition and measurement. Despite this, it is clear that disability increases with age. This is largely due to biological changes that are associated with aging, such as reduction in bone and muscle density. Much of this, at present, is unchangeable – we cannot stave off the process of aging. What is malleable, however, is how we treat and support older adults with a disability, and how we prepare for old age ourselves. At present, the bulk of the caring for older adults with a disability is undertaken by partners and relatives – who are often unpaid and under-resourced. Further, older adults with a disability attract substantive discrimination. They are often treated as if they are childlike or impaired beyond their disability. Increases in support to caregivers, better government and aged care services, as well as improvements in attitudes toward older adults with a disability would lead to improved quality of care and life for older adults with a disability (as well as their caregivers). Finally, while avoiding disability in later life is probably unrealistic, it can be delayed and managed more positively. Specifically, regular exercise and social interactions have both been shown to be protective, as has income equality. Some changes can be made at a personal level, such as adherence to an exercise program. Others will need to be tackled at a societal level. Income inequality, for example, is to a large degree a product of public expenditure, taxation, laws, as well as government provision of healthcare and education. With an aging population, it is clear that changes must be considered, if we are to reduce the global burden of disability.

Cross-References

- ▶ Age Stereotyping and Discrimination
- ▶ Aging, Inequalities, and Health
- ▶ Loneliness and Social embeddedness in Old Age
- ▶ Social Cognition and Aging
- ▶ Social Connectedness and Health
- ▶ Stress and Coping in Caregivers, Theories of

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Distance-to-Death Research in Geropsychology

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Synonyms

Distance-to-death and time-to-death; Terminal changes and time-to-death-related changes; Terminal decline and terminal drop; Time-to-death-related trajectory and time-to-death-related growth curves

Definition

In the broadest sense, distance-to-death research in geropsychology includes all kinds of examinations of associations between facets of psychological functioning and time-to-death. In a narrower sense, however, the term refers to the study of terminal changes in psychological functioning, that is, intraindividual changes that occur time-to-death related at the end of the individual's lifespan. Up to the present, geropsychological distance-to-death research for the most part consists of studies of terminal decline and terminal drop in cognitive functioning and subjective well-being.

Distance-to-Death Research in Geropsychology

Across the past decades, research in geropsychology increasingly considered distance-to-death as indicator of psychological changes that unfold at the end of the human lifespan. That is, this research is based on the rationale that crucial changes in psychological functioning may occur in late life as individuals approach their death, meaning that the occurrence of these changes is closely related to biological processes of deterioration that precede and will finally precipitate the death of the individual. Historically, this approach was initiated in the field of geropsychological research on cognitive functioning early in 1960s by Robert Kleemeier, who presented evidence of an association between late-life declines in intellectual function and mortality, suggesting *the existence of a factor, which might be called terminal drop or decline, which adversely affects intellectual performance and is related to impending death of the aged person* (Kleemeier 1962, p. 293). Such terminal change might unfold late in people's life over some time period before death, hence timely associated with distance-to-death, rather than with calendar age (i.e., distance-to-birth). To put this reasoning simply, if humans are not hit by lethal developments that unfold short termed (such as accidents or severe acute illnesses), crucial changes driven by end-of-life degradations may not occur "normatively" in terms of age related, but "terminally" in terms of time-to-death related.

Thus, in the broadest sense, geropsychological distance-to-death research includes all kinds of empirical studies that examine associations between psychological functioning and time-to-death. In a narrower sense, however, the key objectives of such research refer to terminal *change*. This distance-to-death research was largely driven by the concepts of *terminal decline* and *terminal drop*. The latter term has been used to differentiate time-to-death-related processes that unfold rapidly and accelerated aggravation or loss of functionality prior to death (i.e., "drop") from processes that run in a more steady and less accelerated way (i.e., "decline"). This distinction has been grounded on theoretical considerations regarding the causal processes driving the terminal changes in psychological functioning: *Terminal decline reflects a gradual accumulation of underlying biological and environmental causes, whereas terminal drop implicates a threshold model with an acute precipitating mechanism* (Bäckman and MacDonald 2006, p. 227). However, both terms are often used interchangeably in distance-to-death research, supposedly because clear-cut empirical criteria to distinguish terminal decline from terminal drop in observations of time-to-death-related changes are hard to establish. That is, any notion of changes that occur uniquely time-to-death-related at the end of individuals' lifespans implies some kind of acceleration, in that these changes occur after the onset of the terminal process, adding to ongoing normative age-graded (or otherwise time-graded) changes or stability. Therefore, the remainder of this chapter will not follow a strict distinction between slow-running and fast-running terminal changes, but rather deal with terminal change in general, including both dynamics of terminal decline and terminal drop.

Doing so, this chapter will mainly focus on conceptual and theoretical aspects of distance-to-death research. Thus, only a brief overview on empirical findings on terminal change will be given first. Second, methodological concepts that are key constituents of distance-to-death research will be outlined. Third, the relevance and potentials of distance-to-death research will be considered: Which insight about late-life development *does* – or *could* – distance-to-death research provide to geropsychology?

Empirical Evidence: Terminal Decline in Cognition and Subjective Well-Being

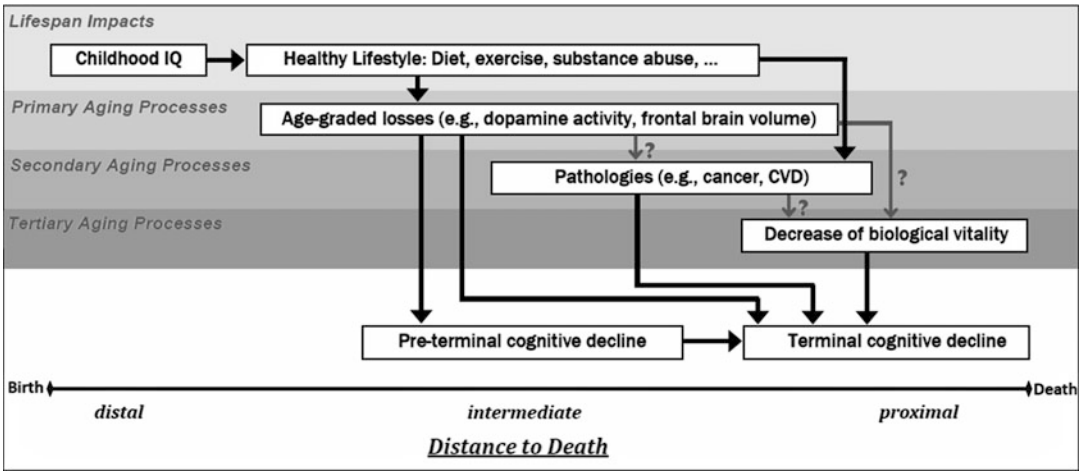
Starting with Kleemeier's investigation, gerontologists' interest in phenomena of terminal decline

has long been focused on cognitive functioning (e.g., Kleemeier 1962; Riegel and Riegel 1972; Siegler 1975; White and Cunningham 1988). This "early" research revealed manifold and strong evidence that (a) levels of cognitive function predicted subsequent survival (for review Small and Bäckman 1999) and (b) intraindividual declines of cognitive performance were associated with distance-to-death (for review, see Bosworth and Siegler 2002). Overall, these findings suggested that terminal change accounts for a substantial portion of the differences in cognitive performance among older individuals, leading to questions on the nature of the phenomenon, particularly concerning the causal processes underlying terminal change and the pervasiveness of such distance-to-death-related changes across different – including also "non-cognitive" – facets of psychological functioning. With the terminal decline paradigm well established in aging research, more recent investigations in the broadest sense dealt with these questions.

The further progress of distance-to-death research up to the present may be summarized with respect to two predominant topics, namely, (a) increasing evidence of distance-to-death-related changes not only in cognitive functioning but also in indicators of subjective well-being (SWB) and (b) the provision of more and in-depth insights about the course and predictors of terminal decline. Up to the present, a large body of research provides massive evidence of terminal decline in cognitive functioning, unfolding in a dedifferentiated manner across various cognitive abilities (Wilson et al. 2012). Moreover, broadening the distance-to-death perspective beyond the focus on cognitive functioning, SWB emerged as important field of terminal changes in recent years. An increasing body of studies provided evidence of changes in SWB associated with time-to-death – showing patterns of terminal decline of cognitive (i.e., life satisfaction) and affective components of SWB (e.g., Gerstorf et al. 2008a, b, 2010; Palgi et al. 2010; Schilling et al. 2013; Vogel et al. 2013; Windsor et al. 2015).

Given its historical "forerun," in particular cognitive distance-to-death research revealed manifold hints toward proximal and distal influences across the lifespan that might impact on the onset and speed of terminal declines (for review, see Bäckman and MacDonald 2006; for more recent findings, see, e.g., Gerstorf and Ram 2013; Muniz-Terrera et al. 2013; Cadar et al. 2015). Overall, terminal decline in cognitive functioning appears as developmental dynamic not fully mediated by specific diseases, but a phenomenon determined by multiple impacts, including some "core" of time-to-death-related change that still could not be attributed to particular causes and might be understood in terms of a "deterioration of global biological vitality" (Bäckman and MacDonald 2006, p. 225). Figure 1 summarizes proximal and distal impacts of terminal cognitive decline, adapted from Bäckman and MacDonald's (2006) respective summary (leaving out predictive pathways from genes and early environment to childhood IQ and also direct links from childhood IQ and normative age-graded influences to death that were part of their figure). The original figure has been modified by adding potential causal pathways among impacts, which Bäckman and MacDonald did not include in their model, but may be considered at least hypothetically.

Thus, keeping this chapter's conceptual focus, empirical distance-to-death research up to the present might briefly be characterized as a process moving from mere evidence of terminal change in psychological functioning toward an understanding of these terminal changes as driven by proximal and distal impacts across the individual's lifespan. This course of the investigation of the phenomenon – from disclosure to causes – seems also implied in Gerstorf and Ram's (2013, see also for more review of empirical findings) suggestion to organize objectives for future research on terminal decline according to five basic rationales (Baltes and Nesselroade 1979), namely, (a) identification and description of terminal changes and (b) the interindividual differences in terminal changes, (c) analysis of interrelationships between terminal change in different attributes or multiple aspects of functioning, and (d) identification of the causes of terminal change and (e) of the interindividual differences in



Distance-to-Death Research in Geropsychology, Fig. 1 Distal and proximal impacts on terminal cognitive decline (*Note.* Modified figure adapted from Bäckman and MacDonald, European Psychologist 2006, 11(3), p. 229. *Black arrows* denote impacts considered originally by Bäckman and MacDonald)

terminal change. It could be expected, hence, that ongoing and future distance-to-death research will increasingly focus on the causes of terminal change in psychological functioning.

However, crucial to such understanding of causes, research on terminal decline in cognitive functioning suggests that psychological changes preceding one's death are driven by impacts which do not all unfold distance-to-death-related, but differentially timed within the individual's life course. The "classic" distinction of primary, secondary, and tertiary aging processes – also added in Fig. 1 to the model adapted from Bäckman and MacDonald (2006) – has been suggested as conceptual framework to disentangle this temporal overlay and interplay of the driving forces of late-life changes (Ram et al. 2010): Primary aging denotes processes that are intrinsic to aging (i.e., unfolding regularly and irreversibly within individuals at certain ages), whereas secondary aging refers to pathological changes that do not occur age-graded and may be preventable or reversible (Busse 1969), and tertiary aging denotes biological degradations that unfold under impending death (Birren and Cunningham 1985). Thus, processes that unfold normatively age-related, or nonnormatively across some limited time period in one's life, or uniquely distance-to-death-related might impact on terminal changes in the psychological functioning observed at the end of the lifespan. However, key to distance-to-death research, this rationale implies that *unique* statistical association of intraindividual change with time-to-death (controlling for age- and pathology-related time metrics, such as time since diagnosis) means strong evidence for the effectivity of tertiary aging processes.

Methodological Concepts of Distance-to-Death Research

Time-to-Death as Predictor of Change in Psychological Functioning

Across the past decades, distance-to-death research gained tremendous inspiration from appearance of longitudinal growth curve methodologies (e.g., Curran et al. 2010). Growth curve modeling of time-to-death-related trajectories – mostly done by means of longitudinal mixed/multilevel models employing time-to-death as within-subject predictor (e.g., Vogel et al. 2013; Sliwinski et al. 2003) – is a suitable and effective tool to analyze the association between intraindividual changes and time-to-death, meaning evidence for terminal *change* in a strict sense. Since the 1990s, studies of terminal change increasingly used longitudinal data

to model time-to-death-related trajectories of the variable under study.

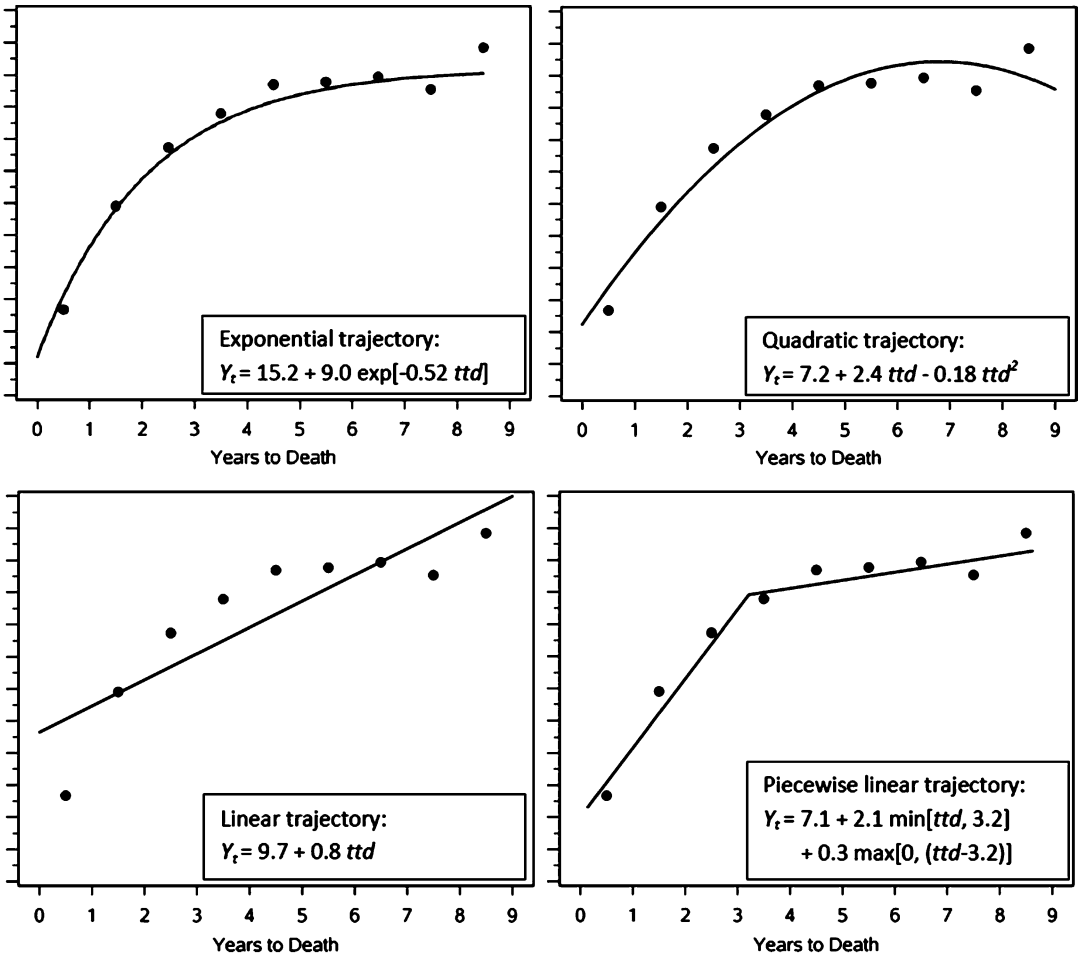
By means of time-to-death-related growth curve modeling, fundamental objectives concerning terminal change can be addressed. For instance, the abovementioned objectives suggested by Gerstorf and Ram (2013) can be linked to model parameters of a time-to-death-related growth curve model (e.g., terminal change may be identified in terms of the fixed level and slope effects and described by the "curvature" of a growth curve model, whereas interindividual differences in terminal change are mirrored statistically in the random level and slope variances) or could easily be operationalized by more elaborate growth curve model specifications (e.g., latent dual growth curve models may be used to analyze interrelationships between terminal declines in different attributes and potential causes of terminal change might be included as predictors of time-to-death-related slopes; McArdle 2009).

By now, the longitudinal growth curve modeling approach has become key to the analysis of terminal change and therefore is essential for geropsychological distance-to-death research. However, a methodological drawback often present in these analyses of terminal change should also be noted: In the typical scenario of distance-to-death research, using data from longitudinal samples to model time-to-death-related trajectories, only those participants can be included that had deceased (and those time of death had been recorded) when the analysis is conducted. Thus, the participants still alive at the last mortality follow-up are excluded from these analyses. This practice could lead to considerable selectivity of the subsample used for analysis, as participants from early birth cohorts that survived to very old age are excluded and/or only these from the younger birth cohorts that died already at rather young ages are included. Concerning, for instance, that the terminal processes of the long-living could differ systematically from those that die at rather early ages, such selectivity could lead to biased evidence of terminal change.

Trajectories of Terminal Change

Piecewise growth curve models (also referred to as multiphase models, change point models, or transition point models, e.g., Cudeck and Harring 2007) have become a particularly relevant tool to model time-to-death-related trajectories: As an implication of the concept of terminal change, trajectories of psychological functioning in late life might typically be shaped such that they mirror some kind of transition from a phase of preterminal change – which might show only minor change or change unfolding normatively age graded – into the phase of increased terminal change prior to death. That is, the typical end-of-life growth curves might appear as compound of two pieces, namely, the preterminal trajectory showing relatively low rates of change and the terminal trajectory showing higher, accelerated change. For illustration, Fig. 2 shows different widely used distance-to-death-related trajectory functions, including a piecewise growth curve, fitted to the hypothetical values observed from one individual at varying temporal distance-to-death. Piecewise growth curve models hence are the statistical "translation" of this implicit characteristic of distance-to-death-related processes and have been used in many studies of terminal changes (e.g., Gerstorf et al. 2008a, b, 2010; Vogel et al. 2013; Wilson et al. 2003; Sliwinski et al. 2006).

An alternative to modeling piecewise trajectories are growth curve models employing a nonlinear growth function that also may reflect the transition from a preterminal phase of moderate changes into a terminal phase of accelerated change – for instance, curvilinear (quadratic) trajectories showing accelerating trends toward the end of life, or exponential growth functions that could follow a pattern of high stability across a period more distant from death, turning into rapid change as death comes close. These nonlinear functions may be more realistic in that they do assume a continuous transition from preterminal to terminal change, instead of a sudden onset of the terminal phase at a single point in time. However, it is this "coarseness" of the piecewise growth curve model that makes it attractive for research on terminal change: Fitting a series of measures obtained at decreasing distance-to-death to a piecewise trajectory with a distinct change point includes an estimation of the onset and



Distance-to-Death Research in Geropsychology, Fig. 2 Illustration of widely used time-to-death-related growth curve functions

duration of the terminal process. That is, even if one does not assume that terminal change will start suddenly within a short temporal range (say, a day or a week), an estimate of the change point in time provides valuable evidence of the timing of the terminal phase, indicating at about which time-to-death the terminal processes began to evoke perceptible and observable changes in the study variable. For example, for the individual depicted in Fig. 2, the onset of the terminal phase would be estimated at about 3.2 years before death.

Moreover, comparing the growth curves depicted in Fig. 2 it should also be evident that the choice of a growth function is relevant with respect to the distinction of terminal drop versus terminal decline. Piecewise or exponential growth functions are better suited than the polynomial functions (linear or quadratic) to fit a terminal drop pattern of sharp and steep decrease within a shorter time period before death.

Psychological Functioning as Predictor of Time to Death

In contrast to the longitudinal approaches that employ time-to-death as predictor of psychological functioning, time-to-death is also a widely used outcome variable mainly in epidemiological

research. These studies apply event history analyses to predict time-to-death (Yamaguchi 1991), for instance, using cognitive abilities measured in a sample as predictor of survival. Thus, this approach could be classified as cross-sectional, in that it models the statistical association between time-to-death and the interindividual differences in the predictor at a given point in time (e.g., White and Cunningham 1988; Smits et al. 1999). Cross-sectional survival analytic findings of time-to-death-related variability in a variable under study might be taken as indirect evidence of time-to-death-related *changes* that could have caused these differences. However, these analyses do not provide clear-cut evidence of terminal changes, leaving it unexplored whether and when intraindividual changes did generate the interindividual differences that are analyzed. For example, interindividual differences in cognitive abilities that predict survival might have persisted stably since early phases of the lifespan (Deary et al. 2004).

While it presents a weakness of the cross-sectional approach to distance-to-death research that survival analytic findings cannot provide evidence of terminal *change* in a strict sense, it should also be noted that this procedure is not affected by the potential selectivity problems due to the exclusion of study survivors (which may affect longitudinal analysis of terminal change, as explained above). In cross-sectional event history analyses, time-to-death can be treated as right-centered variable. That means that participants that have not deceased until the last mortality follow-up are included in these analyses, with their time-to-death considered as unknown but above the maximum value observed in the sample.

Distance-to-Death Research in Geropsychology: What Is It Good For?

Terminal Versus Age-Graded Changes in Late life?

In very general terms, gerontological research deals with changes in biological, psychological, and social functioning that unfold with some regularity as humans approach and traverse the old age period of life. Therefore, analyses of age-related change had always played the important role to provide gerontologists with basic knowledge of such regularity, in terms of normative changes which are to be expected at certain ages, as well as the interindividual variability of such changes, pointing at the plasticity of aging processes. A great deal of research interest in psychological development in late life has focused on the losses and hardships that accumulate in old age, considering in particular how psychological functioning – such as cognitive performance, subjective well-being, etc. – gets affected by fundamental biological degradations that must occur in old age at least among those that prevented acute lethal diseases and other causes of premature death. With regard to this question, the analysis of age-related changes in psychological outcomes could be understood as an application of chronological age as indicator of such accumulation of loss: The older, the worse the physical health and other "objective" living conditions; hence, age may predict decline in psychological functioning. However, distance-from-birth may not be the optimal predictor of old age development driven by the biological degradations and the losses that tend to accumulate toward the end of the human lifespan. Taking into account that the occurrence, onset, and speed of such late-life aggravations are to some extent driven by nonnormative developmental influences, which may or may not affect individuals' development more or less strongly at different times of their life course, a great deal of late-life development may come in old age, but not strictly age-graded (Baltes and Nesselroade 1979). Thus, chronological age might be unreliable indicator of impacts that promote changes in psychological functioning in old age.

In contrast, distance-to-death may do a better job in indexing the accumulation of crucial biological degradations (and other kind of loss) late in an individual's life, considering that this accumulation itself marks the process that will end up in the individual's death. That is, the health status of a 75-year-old who will not survive until age 80 could be expected worse compared to another

75-year-old who will live another 20 years, but might rather resemble the health status of a 90-year-old who will die before age 95. Following this reasoning, a focus on distance-to-death-related changes seems promising to add to the traditional age-related perspective in research on psychological late-life development in threefold respects, namely, (a) enabling the disclosure of non-age-graded developmental late-life dynamics, (b) promoting insights in the nature of processes that drive psychological late-life development, and (c) advancing geropsychological reasoning with paradigms of terminal phase of life and psychological terminality.

Disclosure of Non-age-graded Late-life Developments

The distance-to-death perspective can provide some "instrumental" value for the empirical detection of change dynamics unfolding at the end of the human lifespan. That is, using time-to-death as a metric of time-graded changes in psychological variables under study could reveal changes that occur frequently and with some regularity in late life, which otherwise, grading change to age or calendar time of measurement, would not be detected.

Such added value gained from shifting the focus from an age-related to a distance-to-death-related perspective became apparent in recent years from studies that examined longitudinal changes in subjective well-being (SWB) using both time metrics, chronological age, and time-to-death (Gerstorf et al. 2008a, b, 2010; Palgi et al. 2010; Schilling et al. 2013; Vogel et al. 2013; Windsor et al. 2015). These studies reported changes in SWB associated with time-to-death – showing patterns of terminal decline of life satisfaction and affective components of SWB – but weaker (or no such) associations with age. This evidence of time-to-death-related decline is inconsistent with the notion of a "stability-despite-loss paradox" of SWB in old age (e.g., Kunzmann et al. 2000): Age-graded longitudinal SWB trajectories or cross-sectional age-SWB associations showed no age-related decline – or even some age-related improvement – in many studies, suggesting that SWB in general is maintained largely stable across large parts of the old age period (noticing, however, reports of accelerated age-graded declines in the oldest-old ages; e.g., Pinquart 2001; Schilling 2005). Such apparent stability has been taken as evidence of old people's overall high resilience toward the losses they are confronted with in late life (e.g., Kunzmann et al. 2000; Charles and Carstensen 2009). However, rather than "paradoxical" stability of SWB, the absence of age-related decline might mirror effects of differential survival, in that those who suffer from severe health losses that could aggravate their SWB will soon die or otherwise be prevented from study participation. Evidence of time-to-death-related decline in SWB supports this latter interpretation. Thus, shifting the focus from age-graded to time-to-death-graded changes in SWB was "instrumental" in drawing a more clear-cut picture of SWB development toward the end of the human lifespan, disclosing late-life change dynamics that imply a correction of a widespread notion of stability built on the age-related perspective.

Insights in Processes Driving Psychological Late-life Development

Disentangling time-to-death-related changes from age-graded developments (or other intraindividual changes that unfold neither age- nor time-to-death-graded), could be essential to deepen the insights in the driving forces that impact on late-life psychological functioning. Usually, the time metric used to index changes in developmental studies is not considered a causal variable, but a proxy variable representing a set of processes covarying with the index time, considered causally linked with the change in the developmental variable under study. Interest in distance-to-death-related changes in psychological functioning follows an inherent rationale that these changes are driven by (or might even drive reciprocally) those "fatal" processes that will end in the loss of the individual's biological capability needed to survive. Thus, psychological changes that unfold in association with distance-to-death are usually considered as linked with tertiary aging processes, denoting the biological degradations that unfold under impending death (Ram et al. 2010; Birren and Cunningham 1985).

Disentangling time-to-death-related change in a given psychological study variable from changes that unfold age-related and or related with the duration of some pathological conditions provides insight in the nature of the developmental process, telling the researcher whether the respective psychological changes are driven by terminal degradations or could be considered as consequence of biological aging in a strict sense or of the individual's particular pathological conditions (Ram et al. 2010; Sliwinski et al. 2003).

However, stressing such conceptual relevance of the distance-to-death perspective, some principal limitation of every time metric used to grade developmental changes should also be kept in mind. Regarding the study of age-related change, Wohlwill stated that age *is at best a shorthand for the set of variables acting over time, most typically identified with experiential events or conditions, which are in a direct functional relationship with observed developmental changes in behavior; at worst it is merely a cloak for our ignorance in this regard* (Wohlwill 1970, p. 50). This rather critical view might also apply to the use of time-to-death as time metric in developmental studies. That is, evidence of time-to-death-related psychological changes – such as terminal decline in cognitive performance or affective well-being – points at tertiary aging processes underlying such change, but of course it does not include an identification and confirmation of the particular causal impacts that drive this terminal change. Thus, in the quest for an in-depth understanding of late-life psychological development, evidence of time-to-death-related change does not mark the final destination, but rather a stopover, directing further scientific inquiry toward the specification of and the causal interplay between particular variables involved in the underlying process of tertiary aging.

Moreover, the clear-cut distinction of changes uniquely related with the timing of primary, secondary, and tertiary aging processes – by means of statistical modeling with given longitudinal data – might be an ideal hardly met. In particular, primary, secondary, and tertiary aging processes may not only co-occur and overlap but also interact in determining the course of individual developmental trajectories. Obviously, mortality and pathology risks increase with age, and "secondary" pathology processes might also increase mortality risks. That is, the onset of tertiary aging processes cannot be considered independently from the onset and course of secondary aging processes, and both might depend on the course of the primary aging (considering plasticity of aging in terms of interindividual differences in the severity of age-graded changes). Thus, an interplay, rather than mere co-occurrence, of primary, secondary, and tertiary aging processes should be considered (for illustration see again Fig. 1).

Concerning statistical analyses that employ specific time metrics as proxy variables representing the impacts of these different processes, this consideration should take into account the "uniqueness" of the separation of time-to-death, age, or other time metrics' effects on the developmental outcome variable studied: Most of the findings on terminal decline in cognitive performance or SWB published over the past decades rested upon some kind of longitudinal analysis of intraindividual differences in the outcome predicted by time-to-death and/or chronological age (commonly done by running multilevel or latent growth models). Typically these studies focused on evidence of unique time-to-death-related change that may not be accounted for by normative age-graded development, by either comparing separate models of age- versus time-to-death-related change in terms of model fit or intraindividual variance accounted for (e.g., Gerstorf et al. 2008a, b, 2010; Windsor et al. 2015), or by employing both time metrics simultaneously in one model in order to estimate their "unique" effects mutually controlled for the other time metric (e.g., Vogel et al. 2013; Sliwinski et al. 2003). If primary, secondary, and tertiary aging processes *interact* to some degree in causing the interindividual changes in the psychological outcomes studied, the estimates of time-to-death-related variability obtained with these statistical designs would not be perfectly "freed" from primary age-graded or secondary pathological processes. The potential interplay between such differentially time-graded processes might

be modeled statistically by inclusion of respective interaction effects between different time metrics in growth models (see, e.g., the statistical strategy proposed by Ram et al. 2010).

However, regarding the conceptual meaning of the statistical effects, the crucial point is that time-to-death-effects found in empirical data do not strictly correspond with tertiary aging processes and hence do not strictly discriminate the impacts of terminal degradations on late-life development from those of normative aging and "nonterminal" pathology. Thus, again, evidence of time-to-death-related psychological changes marks an important stopover on the pathway to an in-depth understanding of end-of-life development, pointing at terminal degradations of the human system that affect psychological functioning, but proceeding further on this pathway will need a specification and confirmation of the processes "proxied" by the time-to-death metric.

Considering Paradigms of Terminal Phase and Psychological Terminality

In view of the so far massive evidence of intense changes in many domains of human functioning that co-occur and accelerate over individuals' final years of life, the distance-to-death perspective in the study of late-life development may be driven further theoretically, considering psychological terminality and the terminal phase of the human lifespan as theoretical paradigms that might inspire and enrich future research on late-life development.

As a basic conclusion drawn from the large body of distance-to-death research, individuals approaching their end of life frequently undergo changes in psychological functioning along with physical health degradations, which did not unfold in some continuous manner across the adult lifespan, but occur specifically over some limited time period preceding the end, at whatever age it occurs. Therefore, the aging person's "final years" might be considered distinct from previous life phases: An individual might pass on to the terminal phase of life when the accumulation of losses caused by primary and secondary aging processes sum up to a critical mass, triggering dynamics of physical and psychological change specifically related with impending death. The co-occurrence and interaction of these particular dynamics might activate causal linkages which are not effective at "earlier" stages of the human life course, but particularly involved in the degradation of the human system in the approach of death.

For instance, research on nutritional health effects in very old subpopulations indicated a "risk factor paradox," in that mortality risks implied by the nutritional status in the general adult and young-old population were reversed (e.g., obesity seems protective against mortality and decline of physical function; Kaiser et al. 2010), also adding to other findings of so-called reverse epidemiology (Kalantar-Zadeh et al. 2005). Though "nonpsychological" and not taken from distance-to-death research, this denotes an exemplary case of specific causalities – different from those found in the healthy general population – that emerge under conditions of aggravated physical health and biological degradations. Similarly, the severe physical and functional loss conditions typically met in the terminal phase of life might interact in triggering consequences that will reveal causal dynamics not only quantitatively more intense, but qualitatively different from those driving preterminal development. Therefore, the terminal phase of life might be viewed conceptually as a period of unique meaning, to be distinguished from age-graded segmentations of the lifespan such as the "third" and "fourth" ages.

Furthermore, a crucial aspect which could hold particular importance for psychological functioning in this terminal phase is the individual's subjective perception of distance-to-death-related accumulations and accelerations of degradative changes. These might generate a sense of impending death, provoking behavioral and affective responses which could be understood in terms of psychological terminality. The self-regulatory reactions of individuals who "feel it coming" may at least to some extent be directed toward the impending death, serving to facilitate the unavoidable process of dying. Thus, criteria of successful preterminal adaptation – such as maintenance or restoration of goal achievement and primary

control capacities (Heckhausen et al. 2010), protection or optimization of positive SWB outcomes, and so forth – might no longer be sufficient to understand end-of-life self-regulation. Reasoning in such a way about psychological terminality could inspire research on late-life development, at least by creating "paradoxical" views of adaptive changes, conflicting with the motivational constructs assumed as driving forces of adaptation across the lifespan. For instance, it might be asked whether terminal declines of hedonic well-being could be adaptive in supporting the self-regulation of impending death, in that individuals may easier disengage from life when it has become less hedonically rewarding. Similarly, one might even consider some cognitive declines adaptive in the terminal phase of life: For instance, reduced memory function might help to prevent too intense cognitive processing of the loss of life, which otherwise might cause feelings of regret and despair.

The arguments for such uniqueness of the terminal phase of life and psychological terminality unfolding within are quite speculative at this stage of distance-to-death research, as empirical research findings relevant to the particular matter of such uniqueness are barely present in the gerontological publication arena. Thus, these theoretical propositions should be understood as prospective paradigms for the further proceeding of distance-to-death research (noticing also theoretical work that provided at least implicitly some ideas of psychological terminality, such as Joan Erikson's addition of a ninth stage of development to the Eriksonian psychosocial theory of lifespan development, (Erikson 1997); and the thanatopsychological premise that knowing about their death impacts on human's attitudes and behavior, (Kastenbaum 2000)). That is, with substantial evidence of time-to-death-related changes in key domains of psychological functioning established, future research might move toward distance-to-death-related changes of structural relationships and dynamic interactions, involving these psychological domains. For instance, key questions that are still hard to answer include: How do people cope with health experiences signaling impending death – do they adapt to the health aggravations in the terminal phase of life differently than to health problems experienced earlier in a "nonterminal" life situation? Which role do fears of death and dying play for such adaptation in the approach of life's end?

Conclusions

Up to the present, distance-to-death research in geropsychology has developed over a period of more than five decades, revealing a body of solid evidence on terminal decline of cognitive functioning and, more recently, in SWB. Altogether, this research suggests that the end of life typically comes with intense and accelerated intraindividual changes of psychological functioning, which reflect the degradation of the biological and psychological systems that drive these changes. In such a way, the terminal phase of life appears as some kind of mirror image of the initial phase of life, in that rapid changes unfold at both ends of the lifespan, driven by causal mechanisms related with the respective endpoint – maturational processes unfolding after birth and terminal processes promoting the degradation of the organism.

However, distance-to-death research at present also appears as a still emerging field of geropsychological inquiry, far from any state of completion. The manifold findings of terminal changes reported so far inspire further questions concerning the interrelationships between time-to-death-related changes in different psychological domains and on the nature and specification of the underlying processes. Also, the generality of the terminal change phenomenon has yet to be explored (Gerstorf et al. 2013): Which other domains of psychological functioning – in addition to cognitive abilities and well-being – undergo time-to-death-related changes? Finally, in view of the co-occurrence and interplay of terminal changes in different psychological attributes, considering the terminal phase of life as a distinctive developmental segment of the human lifespan might be a paradigm advancing research on late-life development. In the terminal phase, the accumulation and acceleration of biological degradations preceding an individual's death might

make special adaptive demands, not faced so far in previous developmental phases. Distance-to-death-related changes of psychological functioning might then be better understood in regard of their terminality, driven by these demands.

Cross-References

► Life Span Developmental Psychology

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Dual Sensory Loss

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Synonyms

Combined sensory loss; Deafblind; Dual sensory impaired; Dual sensory impairment; Vision and hearing loss

Definition

Dual sensory loss (DSL) is the acquired loss, in various degrees of severity of both vision and hearing acuity, associated with aging and prevalent in older adults.

Dual sensory loss (DSL) is the acquired, combined loss of vision and hearing prevalent in older adults. As adults get older, they often also experience changes in their sensory acuity as well. In particular, significant eye and ear changes occur. Commonly, the sclera of the eye changes in color, the number of mucous cells in the conjunctiva may decrease, the retro-orbital fat atrophies causing the eye socket to recede, eyelid tissue becomes lax and the levator muscle weakens causing the eyelid to droop, deposits of calcium and cholesterol salts often appear, retinal changes take place, and the pupil weakens and changes size (Nigam and Knight 2008). Visual conditions such as cataract, diabetic retinopathy, retinitis pigmentosa, glaucoma, and macular degeneration are common in older adults often leading to devastating visual difficulties (including low vision or legal blindness). The degree of impairment arising from the visual difficulty varies. Early cataract changes often only affect glare, while in more advanced stages, cataracts cause blurred vision and impaired contrast sensitivity and in severe cases blindness may occur, although cataract surgery

often restores functional vision. Diabetic retinopathy usually affects both eyes and results in blurred, distorted vision of the central visual field although laser surgery is sometimes successful in restoring functional vision. Glaucoma results in loss of the visual field and if controlled, sight loss may be minimal. If uncontrolled, impaired vision or blindness often results. Finally, age-related macular degeneration is the progressive loss of reading vision and sharp distance vision. This retinal disorder usually occurs bilaterally and affects the central part of the visual field frequently leaving peripheral vision unaffected. According to the WHO ICD-10 (World Health Organization), the severity of visual impairment ranges from moderate visual impairment (distance visual acuity worse than 6/18 and equal or better than 6/60), to severe visual impairment (distance visual acuity worse than 6/60 and equal or better than 3/60), to blindness (distance visual acuity worse than 3/60 to no light perception).

Age-related visual loss frequently results in light sensitivity and reduced tolerance for glare. Central or peripheral field losses cause a multitude of problems ranging from intolerance to variations in luminance to dependence on high levels of luminance, reduced contrast sensitivity, the inability to see fine detail of large low contrast objects, difficulty visualizing distant objects, discriminating detail, adapting to darkness, and distinguishing between colors. Additional visual difficulties include the reading of print even when using visual aids (e.g., reading legal documents, notices, magazines, or recipe books) and restricted mobility which frequently interferes with a person's ability to move around safely in the environment. These difficulties are disabling having severe psychosocial ramifications, such as decreased ability to participate in activities of daily living (ADLs) and independent activities of daily living (IADLs) independently, depression, and decreased social interaction.

Likewise, ear changes associated with the aging process occur and include: changes to the external pinna (such as enlargement), loss of elasticity of the external auditory canal, thinning and stiffening of the eardrum, calcification of the ossicles, atrophy of the muscles of the middle ear, atrophy and diminishing cochlea hair cells, and vestibular and neural changes (Nigam and Knight 2008). Ear conditions that are prevalent in older people are cerumen (earwax) accumulation, conductive hearing loss (e.g., due to middle ear ossification), sensorineural hearing loss (e.g., due to presbycusis, noise-induced hearing loss, or multiple sclerosis). Central auditory processing disorder (CAPD) may occur due to neural changes in the central auditory nervous system. Hearing loss is usually defined according to the corresponding decibel loss consisting of mild, moderate, moderate-severe, severe, or profound hearing loss categories. These acquired hearing disorders are often slow to deteriorate and difficult to identify early due to the subtle changes that develop gradually. The hearing loss is typically more severe in the high frequencies affecting the perception of sounds (such as f, th, sh, and s speech sounds) and speech reception or understanding (particularly in poor listening situations or when there is high background noise or reverberation), difficulty with speech discrimination and the processing of auditory information.

Any combination of vision and hearing loss (even when a mild loss occurs in both vision and hearing) is termed DSL. The impact of DSL is devastating for older people, having significant implications for their health care. These prevalent conditions (vision and hearing loss) need to be recognized and considered by clinicians, researchers, and policy makers, particularly since the prevalence of these conditions is expected to rise in future years.

Prevalence

In line with global population aging, there will be an increased number of older adults with vision and hearing loss. According to the WHO (2012a), amongst the 285 million people worldwide who are visually impaired, in the 50 year and over age group, 65% are visually impaired and 82% are blind. Similarly, of the 328 million adults with disabling hearing loss worldwide, approximately one-third is aged 65 years and over (World Health Organization 2012b).

Since the prevalence of vision loss and hearing loss is high in the older adult population, it is a rightful assumption that the prevalence of the combined sensory loss (DSL) would be high in this segment of the population and worthy of further investigation and discussion. Research in the prevalence of DSL, however, reflects a relatively small body of work in comparison to other chronic conditions affecting older adults such as diabetes or dementia. Estimates of the prevalence of DSL vary greatly in the literature. This is primarily due to the different methodological approaches used to investigate DSL and the specific population investigated in studies of DSL. The following are two examples of studies that illustrate the disparity in prevalence estimates: Caban et al. (2005) found that the prevalence of DSL in their sample of 1110 community residing people in the USA was 7.3% in those participants aged 69–79 years and 16.6% for those aged 80 years and over. Schneider et al. (2012), however, obtained considerably different results in their longitudinal study of 2015 adults living in the Blue Mountains in Australia. Participants were aged 55 years and older at baseline. Results suggested that the prevalence of DSL (termed DSI in this study) was 6% at baseline, increasing from 0% for ages <60 years to 26.8% for participants aged 80 years and over.

The type of cohort included in studies of DSL also produces different prevalence rates. While Smith et al. (2008) concluded that DSL ranged from 5.0% to 7.4% in the older adult veteran cohort, increasing to 20% in veterans aged 85 years and over, Cacchione et al. (2003) found that 52.6% of their sample of older adults living in rural long-term care facilities were visually impaired; 44.1% were hearing impaired; 24.6% were dually impaired (had DSL); and 23.4% had no sensory impairment.

Gender is also an important factor that influences the prevalence of DSL. The literature shows that moderate and severe vision impairment and blindness have a higher prevalence rate in women than men (West et al. 1997). However, men are at a higher risk for developing hearing loss due to their increased participation in the military (noise exposure) or having worked in noisy occupations during their lifetime. Physically, hearing loss is associated with different phenomena in men compared to women. In men, hearing loss is associated with high triglyceride levels, high resting heart rate, and a history of smoking, while hearing loss in women is associated with high body mass index, high resting heart rate, fast pulse wave velocity, and low Ankle-Arm Index (Helzner et al. 2011).

A variety of vision and hearing measurement methods (such as self-report, standardized measures or observation) have been used to investigate DSL (Heine and Browning 2015). This use of different measures as well as the above-mentioned factors has made the comparison of studies difficult.

Consequences of DSL

DSL affects older adult's everyday lives, functioning and participation in activities, and has implications for their health and psychosocial well-being. In particular, difficulty with communication is frequently observed (Heine and Browning 2002). Many older adults with severe visual loss cannot see their communication partner's face clearly and therefore cannot lip-read or perceive cues such as gesture, facial expression, and body posture and thus need to rely heavily on the auditory modality for adequate speech reception (Heine et al. 2002). For people with DSL, auditory acuity is reduced and even if hearing loss is mild, the auditory modality cannot compensate for diminished visual acuity. Communication difficulties such as reduced conversational fluency, adequate reception of a verbal message, and difficulty with identification of verbal and nonverbal cues result in communication misunderstandings or breakdowns (Heine and Browning 2002). In turn, conversational difficulty interferes with performance and confidence in social-communication situations often resulting in diminished psychosocial functioning.

Many older adults with DSL are at risk for developing a multitude of difficulties, including depression and decreased well-being. Kiely et al. (2013) investigated the association between DSL and mental health in 1611 adults aged

65–103 years. They found an association exists between depressive symptoms and DSL that was attributed to adults with DSL experiencing difficulty with completing ADLs and having limited social engagement. In line with these findings, Crews and Campbell (2004) also found that older adults with DSL had difficulty with everyday competence, experienced poorer health, and had decreased social roles.

The consequences of DSL are extensive as was evident in a study by Wallhagen and colleagues (2001) who investigated the relationship between DSL and several comorbidities in 2442 adults aged 50–102 years. These authors concluded that DSL had a strong impact on physical and social functional status.

Clinical Assessment

To date, there is no consensus regarding the identification and assessment of DSL for either research or clinical purposes. While self-report, questionnaires, and tests such as the Snellen eye test (for vision) and pure-tone air audiometry for hearing have been commonly used in research studies (Heine and Browning 2015), little literature exists regarding the identification of both disorders in one clinical setting. Service providers almost always identify vision and hearing disorders within separate contexts. That is, the vision specialists (such as the optometrist and ophthalmologist) assess vision and evaluate the client's perceptions about their visual loss, while audiologists assess hearing and appraise the client's perceptions about their hearing loss. The identification of DSL clinically is therefore reliant on a collaborative approach between professionals, which assumes that additional education concerning DSL has been provided to all team members working with older adults. For example, the audiologist needs to take into account a client's visual difficulties (such as "blurry" vision) and conducts the audiological consultation by considering the necessary accommodations that are required. These might include: reducing the distance between conversationalists, accounting for mobility needs, and adjusting the room lighting (e.g., by reducing the glare). Likewise, it is essential for the vision specialist to accommodate a client's hearing difficulties by reducing the distance between conversationalists and using effective communication strategies such as speaking slower, clearer, and louder and repeating or expanding utterances for clarification purposes. The lack of DSL clinical guidelines and professional education programs educating visual specialists about hearing loss and audiologists about vision loss are significant barriers to the early identification of DSL and is thus an area for further investigation.

Management of DSL

This interdisciplinary area of practice requires the collaboration of a number of medical and allied health professionals including general physicians, ophthalmologists, otolaryngologists, vision specialists (such as optometrists), audiologists and speech-language pathologists (SLPs). Following diagnosis, the vision specialist and audiologist counsel and advise clients about their sensory acuity and provide rehabilitation or management strategies, especially fitting the necessary devices (such as magnifiers to enlarge print for visual enhancement and hearing aids and assistive listening devices for amplification). The SLP is often included as a team member in the rehabilitation program and is in an ideal professional position to provide clients with DSL strategies and practice to improve their communication. Heine et al. (2002) conducted a cross-sectional study at a day center for visually impaired people investigating the communication, situational difficulties and conversational needs of older adults with sensory loss, and their communication partners. Results suggested that older adults with DSL experienced a range of functional vision and hearing and communication difficulties and would benefit from specifically devised training programs.

The management plan for those with DSL needs to take into account the client's unique sensory status, competencies, and barriers. Again, the vision specialist, audiologist, and SLP need to be educated in the area of DSL and its

management in order to delineate and implement an adequate rehabilitation program. For example, as part of the rehabilitation program, when fitting a hearing aid for a client who has DSL, the audiologist needs to consider the client's visual difficulties and possible inability to manipulate small objects such as the battery of a hearing aid or read written instructions. In these instances, visual accommodations such as the use of a magnifier or enlarged font size may be warranted and from an audiological viewpoint a magnetic-tipped device for battery removal may be useful. From a technological perspective, it is imperative that the audiologist considers that a client who has DSL has different audiological needs to a client who has a hearing loss. Simon and Levitt (2007) discussed numerous audiological issues in relationship to DSL including specific recommendations for amplification fittings. These authors proposed that hearing aid fittings should be adjusted for people with DSL (e.g., consider the use of directional microphones) to improve their sound localization and binaural processing which are of primary importance for speech perception and spatial orientation.

Visual and hearing devices are one aspect of the rehabilitation process. Other relevant clinical target areas include speech perception training, communication programs for clients and carers (Heine et al. 2002), and the provision of informational counseling and psychosocial support (Brennan and Bally 2007). Tye-Murray (2009) has been instrumental in researching audiovisual speech perception in people with vision and/or hearing loss. Outcomes of their research suggest that audiovisual speech perception is related to auditory and visual word neighborhoods (context) and multisensory integration. Thus for people with DSL, practice drills including these concepts should be included in the intervention program.

Over the past two decades, communication training has gained popularity as a valuable rehabilitation method for people with DSL. In accordance with a client-centered approach, the person with DSL and if possible, their frequent communication partner/s (family or carer) participate in a communication training program to address the environmental, situational, and conversational needs of the person with DSL (Heine et al. 2002). In these training programs, the clients and carers practice effective listening skills, situational management (such as being proactive and preselecting a quiet listening environment for a conversation; reducing glare or background noise), and the use and implementation of communication strategies (such as identification of conversational breakdown and the use of communication repair strategies). Communication training programs can enhance conversations, minimize communication breakdown, and increase social confidence thereby improving the social interaction, quality of life, and well-being of older adults with DSL.

A complimentary unique model of intervention is the biopsychosocial model discussed by Brennan and Bally (2007). This model focuses on the coping and adaptation strategies that older adults with DSL can use to improve their functioning, independence, and well-being. Counseling and assertiveness training are beneficial especially since many older adults with DSL often feel vulnerable and have decreased self-esteem and confidence. This emotional reaction in turn often leads to social isolation, depression, and decreased feelings of well-being.

Although DSL is a new field of research and clinical practice, the diverse management programs show promising progress.

Future Directions

Between 2000 and 2050, the number of people aged 80 years and over in major areas worldwide will more than quadruple (United Nations, Department of Economic and Social Affairs. Population Division 2004). With increasing longevity, and the increase in the size of the older adult population, the prevalence of DSL will increase dramatically.

While DSL in this segment of the population is still under-researched, gains have been made in the recognition of DSL as a clinical entity that is currently being researched more widely. Prevalence studies are more common although they

still reflect disparate findings. Interventions such as enhancing signal processing by modifying the dimensions of hearing aids, (Simon and Levitt 2007) multisensory integration (Tye-Murray 2009), communication strategy usage, and psychosocial adaptations continue to be researched and discussed in the literature. Further research is, however, necessary to increase professional and community awareness, knowledge and understanding of this group's communication and psychosocial needs. In order to achieve an exceptional professional service for older adults with DSL, including methods of early identification of DSL and the use of effective visual and auditory rehabilitation strategies, a richer discussion among researchers and professionals is required. The adoption of an interdisciplinary perspective is imperative, and the development of clinical guidelines to support this collaboration is an essential step in informing clinical practice.

For older adults with DSL, the future looks promising as research continues. Promoting the topic of DSL provides this area with the important recognition it deserves, especially since outcomes of research and clinical studies can contribute to these adults' well-being and improved quality of life.

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Dynamic Analyses to Optimise Ageing (DYNOPTA)

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Definition

The Dynamic Analyses to Optimise Ageing (DYNOPTA) project has harmonized and pooled nine epidemiological studies of human aging to examine pathways to compressing morbidity and optimizing healthy aging in the Australian population. Research using the DYNOPTA dataset has focused on four main outcomes that contribute to disease and disability burden among older adults: cognitive function, sensory function, mental health, and mobility or activity limitations.

Project Background and Aims

DYNOPTA is a cross-institutional and multidisciplinary project that has harmonized and pooled nine independently designed longitudinal studies of aging, creating a large nationally representative dataset of older adults in Australia. Aggregating data from a number of cohort studies has the advantages of enhancing population coverage (reducing coverage error), increasing sample size of underrepresented groups (such as the oldest old or those with rare medical conditions), facilitating instantaneous replication across studies, allowing cross-population comparisons, and investigating the impact of study idiosyncrasies on research findings. The broad aims of the DYNOPTA project are to identify effective pathways to compress morbidity and optimize aging (Anstey et al. 2010a, 2011a).

The DYNOPTA dataset is rich including the theme areas of cognitive functioning, sensory-motor functioning, mental health, mobility, and functional independence. The pooled dataset also includes background variables that cover sociodemographics, health, lifestyle, medical conditions, carers, and mortality. Within the DYNOPTA dataset, there are over 400 harmonized variables, which, when combined with individual observations, results in excess of 18 million data points. DYNOPTA therefore provides some of the most comprehensive available evidence on the health and well-being of older Australians between the years 1990 and 2006.

Contributing Studies and Sample Composition

The target population for the pooled DYNOPTA dataset is defined as all Australians born prior to December 1955, resulting in a baseline age range of 45–103. However, the target populations for the individual studies vary by geography and demography. There are study design differences in sample frame, random sampling procedures (simple, stratified, and clustered), data collection procedures (clinical interview, postal, telephone and self-completion questionnaire), baseline year (ranging from 1990 to 2001), age range, sample size, time intervals, and the number of follow-up waves. It is therefore important that analyses account for study design effects either through weighting and/or

Dynamic Analyses to Optimise Ageing (DYNOPTA), Table 1 Nine studies contributing to the DYNOPTA dataset

Study	Location	N	Baseline age range	Waves	Period
Australian Diabetes, Obesity and Lifestyle (AusDiab) study	National	7,296	45–95	2	1999–2005
Australian Longitudinal Study on Women's Health (ALSWH)					
Middle-aged cohort	National	13,706	45–51	4	1996–2005
Older-aged cohort	National	12,431	68–76	4	1996–2005
Household, Income and Labour Dynamics in Australia survey (HILDA)	National	6,164	45–90+	5	2001–2006
Australian Longitudinal Study of Ageing (ALSA)	Adelaide	2,087	65–103	7	1992–2004
Blue Mountains Eye Study (BMES)	Blue Mountains	3,654	45–100	3	1992–2004
Canberra Longitudinal Study (CLS)	Canberra, Queanbeyan	1,134	70–103	4	1990–2002
Melbourne Longitudinal Study on Healthy Ageing (MELSHA)	Melbourne	1,000	65–94	11	1994–2006
Personality and Total Health (PATH) Through Life	Canberra, Queanbeyan	2,550	60–66	2	2001–2006
Sydney Older Persons Study (SOPS)	Sydney	630	75–97	5	1991–2003

modeling adjustments. The contributing studies include three nationally representative panel surveys and six regional studies that were representative of the local community (see Table 1). The national surveys contribute 65% of participants.

The full DYNOPTA dataset is large and complex, consisting of 50,652 participants who were followed longitudinally, on up to 11 measurement occasions over a 15-year period. Over all studies, there was an average of 4.4 measurement occasions over a period of 9.4 years (SD = 2.9) and on average sample members participated in 3.1 measurement occasions. The mean age at baseline was 61.7 years (SD = 12.4, range = 45–103), and 77% of the sample were women, reflecting the inclusion of the all-female cohorts from the ALSWH and women's greater longevity (excluding participants from the ALSWH, 53% were woman). Study participants were generally community living, but five studies did include adults who resided in institutions such as nursing homes. Further information about each of the contributing studies, full description of the sample, and project background can be found in a cohort profile published in the International Journal of Epidemiology (Anstey et al. 2010a) and a summary of policy-relevant findings in the Australasian Journal on Ageing (Anstey et al. 2011a).

Variable Harmonization

Variable harmonization is the rescaling of functionally equivalent measurement instruments onto a common metric. While this process may result in coarse-grained data (information loss), it has the advantage of providing a framework for the direct comparison of data obtained from independently sampled populations. Within DNOPTA, variables were primarily harmonized retrospectively using the *by fiat* method, which involves identifying common variables and, when necessary, recoding response categories onto the same scale where the possibility of disputing the recode is deemed trivial by a panel of experts. Modeling-based approaches were used to standardize mental health variables such as depression symptoms. Table 2 shows a selection of variables harmonized in each of the content domains. Where possible, variables were harmonized to conform to Australian national standards. Some measures were not collected by all contributing studies, resulting in

Dynamic Analyses to Optimise Ageing (DYNOPTA), Table 2 Selection of harmonized variables for each domain

Domain	Example measures
Cognitive function	Mini-Mental State Examination (MMSE)
Sensory function	Visual acuity, pure-tone audiometry, self-reported vision and hearing difficulties, hearing aid use, dual sensory loss
Mental health	SF-36, probable depression, psychological distress
Mobility and disability	Activities of daily living, driving
Mortality	Date of death
General health and medical conditions	Diabetes, hypertension, cardiovascular disease, stroke, arthritis, BMI, self-rated health
Sociodemographics	Age, sex, partner status, education, career occupation, labor force status, domicile
Health behaviors	Alcohol consumption, smoking status, physical activity

study censoring. For example, the Mini-Mental State Examination (MMSE) was only collected by the six regional studies. Mortality data were obtained by linkage with the Australian National Death Index. Weights have been calculated to account for design differences in sampling, selection, and response rates. Weighted estimates are intended to reflect the Australian estimated resident population in 1996.

Profiling the General Health and Population Norms for Older Adults

One of the main contributions of the DYNOPTA project has been the estimation of population prevalence, national trends in behavioral patterns, and calculation of normative data for older adults. Population estimates derived from single studies are often restricted to reporting norms for broad age ranges (e.g., 75+) or may even exclude adults aged older than 85 due to lack of recruitment (Anstey et al. 2010b; Burns et al. 2013a). Of particular importance, data pooling in DYNOPTA has increased the number of participants aged over 80, allowing for more robust and reliable norms of those in older age. For example, population prevalence has been estimated for chronic disease and medical conditions (Bielak et al. 2012), probable dementia (Anstey et al. 2010b), hearing loss (Kiely et al. 2012a), depressive symptoms (Burns et al. 2012a), self-rated health (Anstey et al. 2007), and driving trends (Ross et al. 2009) in older Australians. Normative data that is representative of the older adult population has been generated for common neuropsychological tests and assessment scales including the National Adult Reading Test (NART) (Kiely et al. 2011) and SF-36 (Bartsch et al. 2011). Population level norms have also been published for health behaviors such as smoking and alcohol consumption (Burns et al. 2013a) and engagement in physical activity (Sims et al. 2014). Burns and colleagues (Burns et al. 2013a) examined period effects by comparing patterns of alcohol and smoking consumption during the years between 1990–1994 and 1996–2002. They reported a decline in the proportion of adults who consumed alcohol at high risk levels or currently smoked tobacco over this period.

By mapping the demographic profile of a number of longitudinal cohort studies, DYNOPTA researchers have been able to reveal public health knowledge gaps by identifying subpopulations with low participation rates. In particular, they have documented the poor representation of Indigenous Australians in longitudinal studies of aging (Anstey et al. 2011b).

Cognitive Function

There are no existing prevalence data for dementia based on clinical diagnoses in Australia, and other national surveys have limited numbers of participants in ages 75 years and older. To address this, DYNOPTA has provided the most recent national prevalence estimates of cognitive impairment (probable dementia) (Anstey et al. 2010b). Cognitive impairment was defined by an MMSE score of 23 or less. A cut point of 23 on the MMSE was reported to have a specificity of 0.96 and 0.91 and sensitivity of 0.75 and 0.60 for dementia diagnoses in the Canberra Longitudinal Study (CLS) and

Sydney Older Persons Study (SOPS), respectively. Cognitive impairment was estimated to occur in 15.8% (95% CI: 14.0–17.7) of adults aged 75 years and older, increasing to 41.4% (95% CI: 31.3–50.8) of adults aged 90 years and older. These estimates were highly consistent with results from meta-analyses of European studies. There were no significant sex differences in the prevalence of cognitive impairment, although higher education was associated with higher MMSE scores.

Healthy life expectancy research on cognition had previously focused on social inequalities in cognitive impairment-free life expectancies, by demonstrating differentials in years lived with cognitive impairment by level of educational attainment. DYNOPTA data has been used to extend this literature by investigating the effects of modifiable risk factors for dementia. Multistate models were used to estimate the impact of obesity, smoking, and sedentary behavior on cognitive impairment-free life expectancies (Anstey et al. 2014). Smoking was associated with the largest reductions in total life expectancy and years lived without cognitive impairment for men and women, regardless of their education level. However, with the exception of obesity in men, all risk factors were also associated with fewer years lived with cognitive impairment. The key conclusion from this analysis was that although healthy lifestyle behaviors delayed the onset of cognitive impairment, they did not necessarily prevent it. Crucially, as age is the strongest risk factor for dementia, and dementia risk reduction also increases longevity, risk reduction strategies may result in more years lived with cognitive impairment at a population level. This finding has important implications for statistical modeling of the impacts of dementia risk reduction and projections of future dementia prevalence.

Sensory Function

The DYNOPTA dataset includes measures of self-rated vision and hearing loss as well as clinically assessed measures of visual acuity and pure-tone audiometry. To date, most DYNOPTA studies of sensory function have focused on age-related hearing loss. Notably, over 70% of adults aged 80 years and older were estimated to have at least a mild degree of hearing impairment as defined by a pure-tone average of speech frequencies (0.5–4 kHz) greater than 25 dB in the better ear (Kiely et al. 2012a). An evaluation of the utility of self-reported hearing loss in comparison to hearing loss defined by pure-tone audiometry demonstrated that the prevalence of hearing loss based on self-report data was likely to be overestimated for adults younger than 75, but underestimated for older age cohorts (Kiely et al. 2012a).

Another significant study modeled longitudinal trajectories of audiometric hearing thresholds in 3,526 adults. Importantly, these analyses examined an extensive range of risk factors for hearing loss not elsewhere investigated, including sociodemographics, noise exposure, medical conditions, and cognitive impairment. It was found that age, cognitive impairment, and hypertension were associated with faster rates of decline in hearing thresholds. However, many other factors commonly associated with differences in hearing levels did not predict rates of decline in hearing thresholds (Kiely et al. 2012b). A currently active stream of research on sensory functioning involves calculating sensory impairment-free life expectancies. These analyses demonstrate that in addition to being highly prevalent, hearing and vision impairment can affect older adults for substantial periods of their remaining life.

Mental Health

The burden of psychological distress of older adults, particularly those living in the community, is unclear; some findings purport an increase in depression risk with increasing age; others suggest a decline in depression risk. However, many of these findings are confounded by increasing heterogeneity with age and small sample sizes. This is particularly the case for older men with some suggestion that men are at greater risk of reporting depression in late life. Analyses with DYNOPTA indicate a pattern of increasing

depression risk in men although this failed to reach a level of statistical significance (Burns et al. 2012a, 2013b). More robust evidence was found for increasing levels of depressive symptomatology among older men (Burns et al. 2013b); this appears to mirror rates of suicide in older Australian men. Gender differences in mental health in late life have also been reported when examining terminal mental health decline. That is, in both men and women, there is evidence that depressive symptoms increase substantially in the years preceding death. Findings from DYNOPTA indicate this association is more strongly pronounced in men and that most of the effect in women can be accounted for by comorbid physical health states (Burns et al. 2013c).

With increasing interest in dimensions of positive mental health and well-being, DYNOPTA has provided substantial evidence of the need to examine dimensions of psychological health and well-being that are not necessarily captured in clinically relevant dimensions of psychological distress. For example, in contrast to measures of psychological distress, vitality – a sense of vigor, energy, and engagement – has been implicated as a stronger predictor of self-rated health (Burns et al. 2014a), falls (Burns et al. 2012b), and mortality (Burns et al. 2014b, c).

Another study examined age differences in high- and low-arousal positive and negative affect. Lower levels of negative affect and higher levels of low-arousal positive affect were reported by older adults relative to those in midlife (Windsor et al. 2013). Interestingly, physical function suppressed the association of older age with reduced high-arousal positive affect and lower negative affect. In other words, age differences in affect were amplified after additionally accounting for covariation with physical function. These findings were interpreted as being consistent with the notion that older adults tend to restrain high-arousal emotions in order to avoid uncomfortable levels of physiological arousal and provided further evidence that age differences in the expression of negative and positive affect are underpinned by lower levels of physical functioning among older adults.

Driving

DYNOPTA has been used to provide national trends in driving rates and predictors of self-reported driving and to investigate the proportion of older drivers with low levels of cognitive and/or sensory functioning (Ross et al. 2009). In this study it was reported that 46% of adults over the age of 65 were nondrivers. The proportion of nondrivers was greater for women and for older age groups, such that for those aged 85 years and older, 37% of men and 5% of women reported that they were current drivers. Discontinued driving was more likely to be reported by participants who were women, older, not married, had careers in lower-skilled occupations, were living with impaired levels of visual acuity, and had poorer health. Although people with suspected cognitive impairment (MMSE < 23), and visual impairment (visual acuity > 0.3 logMAR) did generally reduce or cease driving, there remained substantial numbers of men who continued driving with cognitive or visual impairments.

By combining data from a number of state jurisdictions across Australia, Ross and colleagues were able to evaluate the implications of differing licensure policies for older adult driving rates (Ross et al. 2011). They compared differentials in driving rates between state jurisdictions with and without mandatory age-based license testing. It was reported that mandatory age-based testing for renewal of driving licenses was associated with lower rates of driving, but was not effective in reducing the proportion of older drivers who had either a visual or cognitive impairment.

In summary, the pooling of existing datasets to create DYNOPTA has produced the largest dataset on aging in Australia. This resource has enabled both population-based research of a descriptive nature and developmental research on trajectories, trends, and patterns of characteristics at ages for which Australia previously lacked large datasets. The process of developing the pooled dataset has demonstrated the feasibility and utility of this approach.

Cross-References

- ▶ Australian Longitudinal Study of Aging (ALSA)
- ▶ Mental Health and Aging

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