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A Lifespan Perspective on the Interconnections between Personality, Health, and Optimal Aging

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Abstract

The seeds of Healthy Aging are sown much earlier in life, through the health choices we make in younger adulthood and midlife. Cumulative effects of good health behaviors throughout one's life often pay off in older adulthood, in the form of healthy aging (optimal physical, cognitive, and psychological functioning), whereas poorer choices earlier in life can lead to suboptimal or even pathological aging. Personality characteristics are important predictors of health behaviors and thus influence the course of healthy aging. Personality also impacts other precursors to healthy aging, such as educational attainment, work and career choices, financial success, relationship and family outcomes, and attitudes toward engagement and involvement in later life. Through multiple pathways, not only health and health behavior, personality predicts positive aging-related outcomes.

There is renewed interest in the concept of Healthy Aging, which had known previous incarnations with such labels as Successful Aging (Baltes & Baltes, 1990; Rowe & Kahn, 1997) and Optimal Aging (Aldwin, Spiro & Park, 2006). Whether it is called healthy, successful, or optimal, the concept refers to aging that is not pathological (e.g., early aging) and at the same time is better than normal aging. We invoke images of a physically active 90 year old or a cognitively intact 80 year old to illustrate successful or healthy, as opposed to normal or even pathological aging. Perhaps the key question in the study of healthy aging is how to promote it or bring it about to some extent. Of course, genetics and social structural variables (e.g., family SES, living in a rich country in the developed world, living in an environment that facilitates healthy habits versus one that promotes obesity) influence the ability of individuals to age in a healthful manner. Yet other individual factors such as personality traits also play a role in whether a person is a successful ager or not. The seeds of Healthy Aging are sown early in life, through the health choices we make in younger adulthood and midlife. Cumulative effects of good health behaviors throughout one's life often pay off in older adulthood, in the form of healthy aging, whereas poorer choices earlier in life can lead to suboptimal or worse, pathological aging (Mokdad, Marks, Stroup, & Gerberding, 2004; Peel, McClure, & Bartlett, 2005). Personality shapes those choices (Friedman, 2000; Smith, 2006; Turiano, Chapman, Gruenewald, & Mroczek, 2015).

This chapter focuses on personality characteristics and healthy aging. First, we define healthy aging. Next, we suggest that personality traits throughout the lifespan play a central role in setting the stage for healthy aging. We discuss long-term explanatory chains that explicate how personality can have its effect on healthy aging not only in later life but also in earlier periods within the lifespan including childhood. Then, we describe more complex bidirectional

and multidirectional associations between personality and health across the lifespan. Finally, we posit that understanding the complex associations between personality and health can help create more effective interventions to promote healthy aging that are targeted and tailored based upon people's personalities.

The Phenomenon of Healthy Aging

There are multiple definitions of healthy aging. These definitions typically include functional status, or the extent to which a person can function independently, take care of one's self, and contribute to society (WHO, 2017). The World Health Organization (WHO) definition of functional status incorporates intrinsic capacity, which recognizes the multitude of mental and physical capacities that one can draw upon, as well as environments that support one's functional ability. In addition to functional status, many definitions of healthy aging also encompass the capacity to thrive and flourish in older adulthood (Baltes & Baltes, 1990). For example, remaining a productive older worker or travelling the world in later life would constitute successful or healthy aging. These definitions illustrate that healthy aging is a multi-faceted phenomenon that encompasses the holistic health and well-being of older adults.

David Barker and Long-Term Explanatory Chains: From Cradle to Grave

Personality characteristics impact many aspects of healthy aging in both distal and proximal ways. The causes can be found in factors that are nearer (in time) to older age and in factors that predate older adulthood by many decades. This idea rests upon the central tenets of lifespan development theory which maintains that developmental variables (like personality, physical functioning, cognitive functioning, emotions, etc.) are interconnected over long periods of time, even across the full life course (Baltes, 1987). The Barker fetal origins hypothesis that we discuss below is such an example. Other lifespan development principles are also highly

relevant for the topic of personality and healthy aging in this chapter. For example, the tenet of multidirectionality in development (Baltes, 1987) bears directly up the bidirectionality and feedback loop ideas we discuss later. The concept of development encompassing both gains and losses is quite relevant for the idea that the effects of a different personality dimension on health may sometimes comprise a gain (a net positive for health) but at other times frame a loss. The lifespan developmental concept of plasticity also frames the issue of personality and health, as that association may be altered by changes in personality or health, or both. Personality characteristics and health variables are each plastic, or malleable across the lifespan. Further, when one changes, how does that effect the other? Does the association between personality and health become stronger under such circumstances, or does it become weaker? The association between the two variables is itself defined by the lifespan developmental principle of plasticity.

There are other tenets of lifespan developmental theory (e.g., historical embeddedness, contextualism) but multidirectionality, plasticity, development as including both gains and losses, are the ones most relevant to how we conceptualize healthy aging in his chapter. A final tenet from Baltes (1987) is ontogenetic development which we described earlier. It simply means development is a lifelong processes and events early in life are often interconnected in important ways to events that occur in later life. Sometimes this is framed in terms of proximal vs. distal associations. In this section, we describe these proximal and distal processes using a well-known example.

Distal associations can be defined by long-term explanatory chains that weave throughout the lifespan. The best example of a distal chain is the Fetal Origins of Late-Life Heart Disease, informally known as the Barker Hypothesis (Barker, 2004). It is one of the most well-known and well-documented of all long-term explanatory chains that describe a human lifespan biological or

psychological process. The Barker Hypothesis states that very early life events, especially conditions in the womb, set the stage for late-life disease. It is astonishing to think that events that occur before birth, when we were in the fetal stage, could influence disease events that are as distal as heart disease in older adulthood. Decades separate the predictor and the outcome. Yet, the current thought on this topic posits the existence of many mediators or intervening variables that come between pre-birth womb conditions and heart failure that happens perhaps 5 or 6 decades post-birth. The hypothesis states that slower fetal growth rates lead to a “thrifty phenotype.” In the short term, this leads to lower birth weight, but also a lifelong slowdown of metabolic activity. This thrifty phenotype, with its metabolic limitations, does not necessarily lead to heart disease as long as a healthy, low-lipid diet is maintained. This thrifty phenotype is much less able to cope with the modern unhealthy diet, rich in fats and sugars, though. Such a diet, which is common in Western countries, leads to greater cardiometabolic problems in thrifty phenotype individuals.

Thus we can visualize how a lifespan chain might operate with respect to the thrifty phenotype. It has its origins in the environment of the womb, perhaps due to nutrient deprivation of the mother due to poverty, wartime circumstances, or economic depression. It then manifests itself in low birth weight at the time of birth, and continues to manifest via slower metabolic processes in childhood, which if paired with a lipid-heavy diet would have obesigenic outcomes (after the 1930s and 40s, caloric intake improved in many countries but perhaps not the quality of those calories). This in turn would lead to cardiometabolic problems in adulthood, eventually terminating in heart disease and earlier mortality in midlife and older adulthood. The Barker fetal origins model is a truly lifespan model of health, acknowledging that events from early life can influence healthy aging or other health outcomes that are years or decades distant.

The Barker Hypothesis is also a nice example of a lifespan chain model in which variables that are present early in life influence health much later in life through a multi-step chain that unfolds across the lifespan. It is also an example of Baltes' lifespan principle of ontogenetic development, or development as a cradle-to-grave, lifelong process. Abstracting this idea, we can conceive of how levels of a variable at the outset lead to an event or change in some other variable at a second time point, which in turn lead to an event or variable change at a third point, and on and on, until some endpoint (often much later in the lifespan) is reached. As an example, see Figure 1: lower conscientiousness in young adulthood leads to the formation of sedentary habits, which leads to lack of physical fitness, which leads to further sedentary behavior, which leads to muscle atrophy and weight gain, which leads to joint problems, which leads to lack of mobility. Mobility issues are a central part of functional impairment, a key component of the WHO definition of non-healthy aging. The effects of personality may be very indirect, as this example illustrates. Here, personality initiates a very long-term developmental chain that unfolds over many decades, leading to an outcome relevant for healthy aging.

[Figure 1 here]

Yet there are examples of personality operating in a less distal, more proximal manner. For example, lower conscientiousness in late midlife (age 55-65) leads to many missed doctor appointments where the issue of sedentary lifestyle may have been addressed, leading to worse functional ability in late life. In other instances, proximal links between personality and health may be a single step in a longer chain. For example, weight gain in early and mid-adulthood puts strain on one's joints, leading to faster deterioration of cartilage in one's knees that requires knee

replacement surgery. Low conscientiousness in older adulthood leads to putting off the knee surgery, increasing the number of years with lower functional ability, as well as worse surgical complications when the surgery is finally done (if it is undertaken at all). In this example, conscientiousness had a proximal effect on functional ability, but the chain of events leading to lower functional ability began with weight gain in early adulthood. As these examples illustrate, personality is involved in both distal and proximal processes which lead to healthy or unhealthy aging outcomes (Friedman, Kern, Hampson, & Duckworth, 2014; Hampson, Edmonds, Goldberg, Dubanoski, & Hiller, 2015; Shanahan, Hill, Roberts, Eccles & Friedman, 2014).

Feedback Loops and Bidirectionality

Having described these hypothetical linear chains of causality, it is worth noting that more complex non-linear chains may be even more common. Undoubtedly, some causal chains may run neatly from A to B to C to D. This may hold when the elements of the chain (A, B, C, etc.) are comprised at least partially of events that have some kind of discrete ending. For example, a childhood disease (a discrete event) leads to a cognitive deficit (not an event but more akin to a trait or dimension) which in turn leads to less completed schooling and lower SES, which leads to poorer health, and ultimately to a heart attack (a discrete event). Yet other chains may not contain discrete events. Often, the elements in a chain are not events but rather are trait-like dimensions or perhaps chronic conditions that continue to exist even after they have exerted some effect on the next element in the chain. In such a circumstance, the leading element in the chain (A) exerts an effect on the second element (B), but A is still there even after it influences B. In turn, B may be a trait-like element itself that can exert an effect back on A, in addition to any effects B has on downstream elements (C, D, E). In such a feedback loop, B, which was changed

by A, exerts an effect on A changing it further, which in turn feeds back and has an additional effect on B.

This undoubtedly sounds confusing. Yet if we think in a longitudinal and lifespan manner, it becomes much clearer. Think of the various elements in terms of time points or time segments. At time one, A exerts an effect on B, thereby changing B as it moves forward into time two. At time two, A and B are both extant, albeit B is changed from the previous time point. At time two, B exerts an effect on A. Now it is A's turn to change as it moves forward into time three. Of course, B is also extant at time three and is changed by A (which had been changed by B at time two, which had been changed by A at time one). This process of two or more variables influencing each other in a cyclical way as they move forward through time is often described by the term bidirectionality. In such cases, two or more variables influence each other in a cyclical way as they move forward through time.

An example of bidirectionality that has relevance for healthy aging research is the well-known association between wealth and health (Pollack et al., 2007). Wealth and health feed off each other and promote one another in a bidirectional manner, reaching a desirable terminus in late adulthood in the form of healthy aging and morbidity compression (i.e., a shorter period of poor health at the end of the lifespan). Higher earnings and greater net worth tend to lead to better health through various mediators, such as better health education and more skills to navigate the often byzantine world of health care and health insurance. In youth, when health issues tend to be at a minimum, higher family-of-origin SES often leads to higher levels of education. More education tends to be associated with greater knowledge about what causes health problems and what kinds of health behaviors can promote a healthier life. Education also tends to boost skill levels with respect to solving complex problems and processing complicated

information. Such skills can help people master the often byzantine world of health care and health insurance. So greater wealth, as operationalized by higher family SES, can have a positive effect on health, through various mediators. Additionally, better health can feed back and boost one's wealth. One needs to be healthy to do complex and high-paying jobs, and to work long hours. Thus, the better health that was brought about by greater wealth in turn creates even greater wealth. Eventually, the twin action of wealth and health over a lifespan promote successful or healthy aging. Wealth and health feed off each other and promote one another in a bidirectional manner, reaching a desirable terminus in late adulthood in the form of healthy aging and morbidity compression (i.e., a shorter period of poor health at the end of the lifespan).

Bidirectionality in Personality and Healthy Aging

Turning to the bidirectional phenomenon in the context of personality and healthy aging, a handful of studies have demonstrated transactional or bidirectional relationships between personality traits and many other kinds of variables. Ironically, many of these other variables are regularly considered outcomes or endpoints. Yet in bidirectionality studies they are often shown not to be endpoints at all but rather part of a back-and-forth flux with a personality dimension that can go on for years, and presumably over the course of an entire lifespan.

Jackson (2011), for example, discovered that conscientiousness and academic engagement reinforced each other in a bidirectional manner over a 4-year period with annual measurements. Increases in conscientiousness from year one to year two led to increases in academic behaviors (studying, going to class) from year two to year three, which in turn led to further increases in conscientiousness from year three to year four, which led to even further increases in academic behavior at the terminus of the study at year 4. Essentially, a bidirectional or transactional relationship was in operation between these the two series of variables, making it

much less clear which variable was changing the other. Was personality change the leading indicator? Perhaps academic behavior changes in the years before the study had promoted increase in conscientiousness. Yet, perhaps self-control or perseverance increases that date to middle or late childhood preceded those high school academic changes.

The previous example raises the question: which variable was the predictor and which was the outcome ? The picture is fuzzy, calling into question our traditional beliefs of what is causal and what is caused. As we have previously noted, there are obviously some outcomes that are true endpoints, such as divorce, disease onset, and mortality (Mroczek & Spiro, 2007; Mroczek, Graham, Turiano, & Oro-Lambo, in press). However, many variables related to personality are not clear points but rather variables that continually change over the lifespan, such as health status and cognitive functioning, which are two variables that form the basis of healthy aging. These variables are likely candidates for entry into bidirectional feedback loops (or multi-directional in the case of three or more variables) with personality, thereby making the predictor-outcome association opaque.

Since Jackson (2011), other studies have provided evidence consistent with personality-related bidirectionality in the areas of physical activity (Allen, Magee, Vella, & Laborde, 2017), work and job experiences (Le, Donnellan, & Conger, 2014), living arrangements (Jonkman, Thoemmes, Lüdtke, & Trautwein, 2014), relationships (Mund & Neyer, 2014; Robins, Caspi, & Moffitt, 2002) and life events (Kandler, Bleidorn, Riemann, Angleitner, & Spinath, 2012). Further work has shown that changes among traits can be subject to bidirectional processes as well. That is, change in one trait can lead to changes in other traits, feeding back upon the first trait (Klimstra, Bleidorn, Asendorpf, van Aken, & Denissen, 2013). These correlated change or coupled change models (Allemand, Zimprich & Martin, 2008; Bollen & Curran, 2004), can help

establish evidence of bidirectionality, as well as evidence for other relevant concepts such as the “corresponsive principle” (Roberts & Wood, 2006) which holds that people’s personality traits lead them to seek out experiences that reinforce those very traits. What this means for healthy aging is that one’s state in older adulthood may be the product of longstanding bidirectional processes that in many cases were set in motion decades earlier. The process is continual and mutually reinforcing, and if this promotes optimal or healthy aging, then this is a positive feature of a person’s overall health in late life. However, one can envision problematic bidirectional processes, such as high neuroticism setting off frequent fight-or-flight activation that leads to high blood pressure which in turn creates anxiety that maintains or even increases neuroticism. In these cases, it will not necessarily lead to healthy aging.

As we argued elsewhere (Mroczek et al., in press), these ideas and bodies of evidence may require us to give up traditional notions of causality. These traditional notions may not depict reality very well. Older ideas (such as those in behaviorism) posited simple stimulus-effect relationships. In contrast, both bidirectionality and “corresponsiveness” (Roberts & Wood, 2006) imply ongoing feedback loops in which cause and effect are difficult or even impossible to disentangle. That said, most bidirectional processes probably have some vague sort of beginning, and thus a distal “cause.” Yet once a process is set into motion, feedback loops take over and traditional stimulus-effect causality is very difficult to determine. This is likely how many developmental processes work, including those that define healthy aging. It is also very far away from the simplistic independent variable-dependent variable mindset that still dominates in much of psychology. That said, there may be some processes in healthy aging that look like the lifespan mediation chains that Barker described (long-term chains of linear causality). However, it is likely more processes in healthy aging are defined by feedback loops in which causality is

hard to determine. Healthy aging researchers need to consider these processes, even if they are difficult to model or pin down, because health in late life for any person by definition is something that emerges closer to the end than the beginning of an individual lifespan. Therefore, healthy aging is likely the cumulative product of myriad feedback loops, linear chains, ontogenetic processes, increases or decreases in plasticity, and other developmental phenomena that have brought a person to health and vitality in older age, versus something less than that (in the worst case scenarios, pathological aging).

Differential lifespan effects of the same trait

In addition to possible feedback loops operating across the lifespan, there is another possibility that may complicate the association between personality and healthy aging. Even if a trait level remains stable, it is possible that the association between that trait and a health variable (and by extension, to healthy aging) varies over different phases in the life course. This interesting possibility was proposed by Shanahan et al. (2014), who argued that that the manner in which a given trait influences a health outcome can operate differentially across the lifespan (see also Mroczek, 2014). That is, the mechanisms by which a trait influences health may differ across the lifespan and/or the importance of a trait for health may differ across the lifespan.

For example, Shanahan et al. (2014) suggest that the mechanisms by which self-control influences health differ across the lifespan. In childhood and adolescence, high self-control may be related to the formation of good health habits (choosing better foods, getting exercise). In adolescence and adulthood, high self-control may have its greatest effect through avoidance of risky behaviors such as drug use, risky sex, drunk driving, and thrill-seeking endeavors that can lead to injury, permanent damage to health, or even death. In adulthood, the action mechanism of self-control may center more on medication adherence and engagement with the health system to

manage emerging chronic conditions, although diet and exercise likely still play a role here too. Shanahan et al. (2014) also argue that the importance of self-control for health may differ across the lifespan. Specifically, in late adulthood, the effects of self-control may “dissipate” due to biological aging. Such biological processes may overwhelm even the most self-controlled people and halt any effect of self-control on health in this last phase of life. In essence, differential action mechanisms and differential degrees of influence add layers of complexity to any effects of linear chains or of bidirectional feedback loops.

Using Long-Term Linear Chains and Bidirectional Loops to “Target and Tailor”

Regardless of whether a particular personality process leading to healthy (or unhealthy) aging takes the form of a bidirectional feedback loop or a simpler long-term linear chain, personality traits may form the basis for targeting or tailoring efforts that can promote healthy aging as well as other health outcomes (Haggar-Johnson & Whiteman, 2008). The idea behind targeting and tailoring is to use information about individual differences to make interventions more powerful and efficient. It is a form of psychological precision medicine. Targeted and tailored personality interventions are still largely hypothetical. However, we can imagine examples in which they have the potential to be more effective than “one size fits all” approaches. Take our earlier example of neuroticism leading to high blood pressure which reinforces the extant neuroticism. Certain negative affect- and anxiety-reducing interventions, such as meditation or expressive writing, may lower neuroticism and in turn, lower blood pressure. However, these interventions may not work well with people who are extremely high in neuroticism (say, above the 90th percentile). More comprehensive techniques, such as cognitive behavioral therapy, may work better for these individuals. If so, people who are moderately high in neuroticism may benefit from meditation and expressive writing, whereas people who are

extremely high in neuroticism may be better served with a targeted intervention that is based on CBT. In this example, the treatments have been targeted to be more precise and in theory are more powerful because they take into account individual differences.

A successful intervention for these two neuroticism groups – “very high” and “high” – could potentially break the bidirectional coupling of this trait with a physiological variable such as blood pressure. Often, targeted personality-based interventions (which remain largely hypothetical) are seen as valuable because they have the ability to break a linear personality-outcome association. However, their effect may be more far reaching and powerful because they may disrupt a bidirectional feedback loop that has the potential to cycle on indefinitely.

We’ve just described “targeting” which means a segment of people based on an individual differences variable is designated for a particular intervention. A slice of the trait spectrum is targeted. However, “tailoring” often refers to much more intense and specific individual-level intervention. In this sense, tailoring is akin to molecular precision medicine approaches in which a drug or treatment is designed specifically for one individual. Imagine a personality-based tailoring effort whereby an intervention is created for a single person. This is obviously expensive but almost certainly much more powerful than a targeting effort, and probably many times more powerful than the typical “one size fits all” interventions that are common in psychology, medicine, and education.

Personality-based interventions throughout the lifespan have the potential to promote successful aging. Personality-based interventions early in the lifespan can disrupt long-term explanatory chains or bidirectional feedback loops. In older adulthood, personality-based interventions can lead to better proximal outcomes such as higher medication adherence, more physical activity, and greater social engagement. Interventions that are targeted and tailored

based on people's individual traits have the potential to be even more impactful than "one size fits all" approaches. However, more research is needed to develop and test such personality-based precision medicine before it can enter the health care toolbox in the coming years.

Conclusion

We've discussed how personality influences healthy aging through distal (long-term) as well as proximal (short-term) predictive chains. Yet we also argued that such linear chains may be too simplistic, and that dynamic processes likely play a major role as well. Specifically, transactional or bidirectional processes in the form of longitudinal feedback loops may describe the manner in which a person ages healthfully or not. We also considered differential predictive power of personality over different life course periods (Mroczek, 2014; Shanahan et al., 2014). Lastly, we discussed a relatively new idea, personality-based interventions, and how they could be part of either targeting or tailoring efforts that promote healthy aging as well as other health outcomes (at any age) by increasing the precision and power of otherwise clumsy, sledgehammer-like interventions (Hagger-Johnson & Whiteman, 2008). Through personality-based precision medicine, lifespan perspectives on personality science could play a key role in promoting the healthful aging of the world's growing population of older adults

Keeping as much of the world's older adult population as healthy as possible will be a major challenge in the coming decades. Many developed countries that have seen declines in birth rates paired with increases in life expectancy (such as Japan and Switzerland) are already seeing a large increase in the percentage of their populations that are over 60 years of age. Yet the developing world has seen increases in life expectancy as well. In China and India alone, there will be several hundred million new people over the age of 60 by mid-century. Multi-pronged and multi-disciplinary efforts will be needed to ensure that as many of these individuals

as possible grow older in a healthy manner, retaining functional status and cognitive capacity and maintaining some degree of independence. Insights from personality science could play a key role in those efforts.

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Figure 1. An illustrative example of the varying effects of trait conscientiousness on health throughout the lifespan. Conscientiousness in young adulthood influences a trajectory of health behaviors; in this case, low conscientiousness leads to sedentary habits, which in turn influence a chain of worsening health and worsening health habits. Adulthood conscientiousness further influences this cycle, through missed doctor appoints and avoidance of procedures which may alter the health trajectory.