Intelligence 63 (2017) 56–65

Contents lists available at ScienceDirect

Intelligence

journal homepage: www.elsevier.com/locate/intell

Inter-connected trends in cognitive aging and depression: Evidence from the health and retirement study

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A B S T R A C T

The cohort process of cognitive aging is a contested topic in population research. The literature is largely in disagreement over how and why inter-cohort trends in cognitive aging occur in the United States. This paper examines significant trends in the rate of cognitive decline and conceptualizes the role of the depression trajectory as a late life course process that accelerates cognitive aging at the individual and population level. To this end, I draw my study sample from the Health and Retirement Study (N = 24,678) and use aging-vector models as an extension of parallel-process latent growth modeling to analyze repeated measures of cognition and depression. Findings show the acceleration of cognitive decline ("negative" Flynn Effect) and worsening of depression risk for recent cohorts. The upward trends in depression account for significant acceleration in cognitive decline among later cohorts, thus providing a new insight into socio-genic population dynamics of cognitive aging.

1. Introduction

The cohort process of cognitive aging is a contested topic in population research and disciplinarians are in disagreement over the concept of cognitive aging itself. One perspective commonly held by biological specialists is that cognitive decline is inevitable and ontological, and that it is one manifestation of the normative aging process (Alwin & Hofer, 2011; Baltes, Cornelius, & Nesselroade, 1979). Social scientists challenge the narrowly ontological definition of cognitive aging on the basis that cognitive aging is malleable to external influences (Elder, 1974, 1998). The cohort process is a seminal concept in this framework, which posits cognitive development is differentiated across successive cohorts "by the changing content of formal education, by peer-group socialization, and by idiosyncratic historical experiences" (Ryder, 1965, p. 843). In fact, recent evidence suggests that cognitive development follows different patterns from one generation to the next (Finkel, Reynolds, McArdle, & Pedersen, 2007; Gerstorf & Ram, 2011).

While the role of the cohort process in cognitive development is widely acknowledged, the literature has yet to reach a consensus on its exact mechanisms. In the 1980s, James Flynn (1987) discovered that in almost all developed countries, IQ test scores are increasing for more recent birth cohorts. This phenomenon is known as the Flynn Effect (Flynn, 1987; Williams, 2013). Although the Flynn Effect was initially accepted as a globally ubiquitous phenomenon, recent evidence from the United States challenges this claim (Alwin & Hofer, 2011). Some suggest that the Flynn Effect may not have occurred in the United States at all, citing lower verbal skills of recent cohorts due to TV-watching habits and the over-simplification of vocabulary at school (Glenn, 1994; Hayes, Wolfer, & Wolfe, 1996). Dubbed as the "negative" Flynn Effect, some report that older adults in recent years actually score lower in terms of short-term memory and vocabulary (Alwin, 2008).

The current literature remains divided over the negative Flynn Effect however, with some studies finding no supporting evidence (Skirbekk, Stonawski, Bonsang, & Staudinger, 2013; Williams, 2013). Even if the negative Flynn Effect is to be believed, it remains perplexing to many that cognitive performance is becoming worse among older adults, despite their higher levels of educational attainment compared to their predecessors (Alwin, 2008). Toward a more comprehensive theory of cohort differences in cognitive aging, Alwin and Hofer (2011) call attention to life course processes that concurrently evolve with cognitive aging. It could be that independently of demonstrated cognitive abilities in adulthood (i.e., higher educational attainment), cognitive aging is differentiated across cohorts through their changing experiences in mid/late life stages. Using this perspective as a theoretical compass, the present study identifies individual depression trajectories as one such life course process.

For clarity, I define cognitive aging as a measurable aging process by standard cognitive tests, rather than based on diagnoses of neurological disorders. Similarly, I refer to depression strictly in terms of self-reported symptoms, rather than a formal diagnosis. Justifications of focusing on depression are several fold. First, depression is known as a

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http://dx.doi.org/10.1016/j.intell.2017.05.004
Received 4 November 2016; Received in revised form 25 April 2017; Accepted 19 May 2017
Available online 05 June 2017

cognitive risk factor (Ganguili, 2009; Ganguili, Du, Dodge, Ratcliff, & Chang, 2006). Second, significant variability in depression risk trajectories is expected particularly around the age of 60 and onwards, when many experience the onset of cognitive decline (Kim, 2008; Mirowsky, 2013). Third, depression trends are distinct across birth cohorts (Yang, 2007; Zivin, Pirraglia, McCammon, Langa, & Vijan, 2013). When taken together, the existing evidence makes a compelling argument that changing patterns of cognitive aging can be attributed to differing experiences with depression in mid/late life. The goal of the present study is to test this thesis.

2. Cohort trends in cognitive aging

The body of research on cognitive aging is vast and has diverse branches, yet attention to the cohort process of cognitive aging is relatively new. Considered as a fundamental structural force in the life course, the concept of the cohort process represents historical, social, and cultural contexts that uniquely represent each cohort's life experiences (Elder, 1974, 1998; Ryder, 1965). Life course theorists argue that human development is a socially conditioned phenomenon, and in doing so, they challenge the narrow ontological notion of cognitive aging (Elder, 1998). Under this framework, the Flynn Effect represents crucial evidence on the cohort process of cognitive development. (Flynn, 1987; Skirbekk et al., 2013).

Stipulating inter-cohort trends in cognitive aging from the Flynn Effect requires careful consideration, because the Flynn Effect mainly refers to improvement of cognitive functioning among children, and says little about its impact on cognitive aging. The proponent of the negative Flynn Effect, Duane Alwin (2008), argues that the Flynn Effect was never the case for children in the United States, citing the evidence of lower verbal scores among recent cohorts through media exposure (Glenn, 1994) and the progressive simplification of vocabulary used in textbooks (Hayes et al., 1996). It seems intuitive to assert that recent cohorts perform poorer than their predecessor in old age, particularly regarding vocabulary (Alwin, 1991). This negative Flynn Effect hypothesis, however, is yet to reconcile with the fact that educational attainment is improving for later cohorts, and is also challenged by contradicting reports that show improvement in cognitive performance among recent cohorts of older adults (Bowles, Grimm, & McArdle, 2005; Gerstorf & Ram, 2011; Schaie, Willis, & Pennak, 2005; Zelinski & Kennionson, 2007).

3. Re-defining the negative Flynn effect

The current disagreement over the Flynn Effect on cognitive aging is based on the implicit assumption that patterns of cognitive aging are directly linked to cognitive development in adulthood. Although the link is indisputable, cognitive performance is known to retain considerable malleability to external influences over the life course (Oi & Alwin, 2017). The negative Flynn Effect may be better understood as the changing patterns of cognitive aging in response to mid/late life experiences that are unique for recent cohorts. In this way, the negative Flynn Effect refers to how quickly recent cohorts lose their cognitive abilities and knowledge that they once possessed in relation to their predecessors, rather than what they know less about or perform worse on. I therefore redefine the negative Flynn Effect as the acceleration of cognitive decline driven by some concurrent life course processes.

There are two other competing scenarios where significant inter-cohort trends could occur yet disagree with the negative Flynn Effect hypothesis. The first scenario is that inter-cohort gaps narrow with age, so that regardless of cohort-specific influences earlier in life, cognitive trajectories eventually converge in late life (i.e., leveled). The second scenario is that cognitive deficits for recent cohorts directly translate to their lower levels of cognitive functioning, such that cohort-differences in cognitive performance remain parallel with age. The pre-existing studies have yet to confirm the negative Flynn Effect in relation to these two alternatives. The present study aims to fill the gap.

4. Linkage between depression and cognitive aging

Despite the persistent malleability of cognitive development over the life course, little to no attention is given to life course factors that continuously evolve in late life and shape their pattern of cognitive decline in the process. Although this oversight has being cogently pointed out (Alwin & Hofer, 2011; Mirowsky, 2011), no existing studies to date rectify it. In the face of population aging, the interlocking mechanisms of the aging process continue to garner academic interests in the United States (Mirowsky, 2011). A better understanding of multiple processes that involve cognitive aging can thus contribute to this large research program on aging. The careful synthesis of the literature led me to identify depression as a life course process that plays a seminal role in cognitive aging.

Depression in late life, acute or chronic, can affect one's cognitive abilities directly by hampering one's information-processing speed and working memory (Nebes, Butters, Mulsant, Pollock, & Reynolds, 2000). One theory posits that depressed individuals focus on negative thoughts about themselves, and allocate less processing resources for other tasks (Hartlage, Alloy, Vazquez, & Dykman, 1993; Nebes et al., 2000). Another theory suggests neuropathology and physiological damage caused by depression, which in turn induces dopaminergic dysfunctions that interfere with cognitive processing (de Paula et al., 2016). Through these pathways, depression can interfere with cognitive processing without triggering dementia or severe cognitive impairment (Campbell, Marriott, Nahmias, & MacQueen, 2004; Dantzer, O'Connor, Freund, Johnson, & Kelly, 2008; Rubinow, Post, Savard, & Gold, 1984).

In addition, the chance of developing depression symptoms rises with age (Mirowsky, 2013), posing increasing cognitive risks in late life. Like cognitive performance, this age-graded pattern of depression symptomatology exhibits considerable variability across birth cohorts. According to results from two different data sources that jointly cover several decades in the past, recent cohorts of older adults are more likely to report depression symptoms (Kim, 2008; Mirowsky, 2013; Yang, 2007). I acknowledge that some studies offer alternative views on inter-cohort trends in depression (Roberts, Eun Sul, & Catherine, 1991; Srole & Fischer, 1980; Zivin et al., 2013). The relevance of these studies is nevertheless limited, because these studies draw results from cross-sectional analysis or clinical diagnosis.

5. The present study

In short, individual cognitive performance and mental well-being dramatically change as individuals transition into late life; their cognitive functioning declines and the tendency to develop depression symptoms rises. Depression affects cognitive performance through various pathways. Taken together, the synthesis of existing evidence suggests that the malleability of cognitive development is systematically driven by the rising risk of depression among recent cohorts, which in turn contributes to the acceleration of cognitive decline among recent cohorts (i.e., the negative Flynn Effect). With the aim to test this thesis, the analytic goal of the present study is to examine the extent to which cohort-differences in depression risk account for accelerated cognitive decline among later cohorts, given the negative Flynn Effect is observed.

This thesis in question rests on the premise that the dynamic link between depression and cognition in late life takes place in mid/late life, independently of earlier-life determinants of cognitive aging. To ascertain this premise, the present study takes into account an array of proxies for so-called cognitive reserve in adulthood. Cognitive reserve is a theoretical construct that indicates individual resources to cope with potential pathological damage and implement strategies to undertake cognitive tasks even in old age (Jefferson, Gibbons, Rentz, Carvalho, & Jones, 2011).

The most broad and widely used proxy of cognitive reserve is socioeconomic status including educational attainment, occupation, and