

Attributing heart attack and stroke to “Old Age”: Implications for subsequent health outcomes among older adults

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Abstract

This study assessed the extent to which older adults attribute a recent heart attack/stroke to “old age,” and examined consequences for subsequent lifestyle behavior and health-care service utilization. Community-dwelling adults ($N = 57$, ages 73–98 years) were interviewed about their heart attack/stroke, and an objective health registry provided data on health-care utilization over a 3-year period. Endorsement of “old age” as a cause of heart attack/stroke negatively predicted lifestyle behavior change, and positively predicted frequency of physician visits and likelihood of hospitalization over the subsequent 3 years. Findings suggest the importance of considering “old age” attributions in the context of cardiovascular health events.

Keywords

aging, health-care utilization, heart attack/stroke, illness attributions, self-directed age stereotypes

The experience of a heart attack or stroke prompts a search for an underlying causal explanation. Perceptions of the causes of heart attack/stroke are varied (Bermejo et al., 2012; French et al., 2001); however, there is a strong implicit assumption regarding the role of age: It is generally agreed that heart attack/stroke can be due simply to old age. In this research we suggest that although chronological age is indeed related to the occurrence of heart attack/stroke, attributing heart attack/stroke to “old age” may be maladaptive from a health-motivation standpoint. We assess the extent to which older adults endorse “old age” as a cause of their own heart attack/stroke and examine the consequences for three health outcomes: Self-report lifestyle behavior change, objective frequency of physician visits in a 3-year

follow-up period, and the likelihood of hospitalization in the same 3-year period.

The relationship between age and heart attack/stroke

There is little debate that chronological age is related to cardiovascular decline over the lifespan. Aging is accompanied by the process of atherosclerosis (narrowing of the arteries due to

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formation of plaque) and, by extension, the related complications of hypertension, heart attack, and stroke (Stout, 1990; Strehler, 1977). However, cardiovascular decline is not entirely an age-determined process; rather, it is an *age-related* process that is heavily influenced by external, modifiable factors (Fries, 1980; Stout, 1990). Indeed, health research has revealed that much of the cardiovascular decline previously attributed to aging is more appropriately attributed to the exacerbating role of three modifiable factors: poor diet, lack of physical activity, and tobacco use (Manson et al., 1992; World Health Organization, 2005). What this suggests is that although cardiovascular decline in old age is not completely within individual control, it is more controllable than once-believed.

Despite increased awareness of the modifiable aspects of cardiovascular decline, the erroneous belief that age causes such illness endures (Goodwin et al., 1999; Stewart et al., 2012). It is possible that this belief derives from a set of well-accepted stereotypes about older adults and the aging process. Among these stereotypes are the deep-seated assumptions that “all old people are ill,” and that “illness is a natural part of aging” (Ory et al., 2003; Palmore, 1999; Rowe and Kahn, 1998). The association between age and illness is learned in childhood and reinforced over the lifetime with repeated exposure to the pairing of older adults and illness through books, television, and other media sources (Hummert, 2011; Levy, 2009). Furthermore, the perspective that illness is caused by age is often inadvertently reinforced by health-care professionals and other caregivers who, in attempting to help older patients come to terms with disability or illness, may overstate the role of aging as a causal factor (e.g. Palmore, 2004).

Once individuals accept stereotypes about age and illness, there is potential for such stereotypes to become internalized and self-directed. In her theory of age-related stereotype embodiment, Levy (2009) suggests that as we get older, generalized stereotypes about aging can become specific expectations about *one's own* aging. In this way, older adults can begin to embody the stereotype of the frail, ill,

disabled elderly adult (Levy, 2009). Empirical work supports this “embodiment” hypothesis by demonstrating that pre-existing age stereotypes do indeed predict subsequent self-perceptions of one's own aging (Kotter-Gruhn and Hess, 2012; Rothermund and Brandtstadter, 2003). Furthermore, self-perceptions of aging go on to predict health behavior and outcomes: The more one's self-view aligns with negative age stereotypes, the more one seems to embody those stereotypes in terms of becoming frail, disabled, and ill over time (Hess, 2006; Levy et al., 2009, 2012). In this study, we suggest that one indication of the acceptance (embodiment) of the stereotype that “all old people are ill” is the attribution of one's own illness to old age.

Attributions and health

According to theories of causal attribution, humans are naturally motivated to seek out the causes of important outcomes, thereby facilitating mastery of one's environment (Abramson et al., 1980; Jones et al., 1972; Weiner, 1985, 2012). All causal factors (i.e. attributions) are classifiable along several underlying dimensions: *Locus of causality* distinguishes between causes that originate within the self (internal) and those outside the self (external); *stability* differentiates between causes that are subject to change over time (unstable) and those that are not (stable); and *controllability* discriminates between causes that can be volitionally controlled by the self/others (controllable) and those that cannot (uncontrollable). The underlying dimensions of any given attribution provide information about the plausibility of success/failure in future settings, and therefore have considerable influence on subsequent motivation and behavior. In the context of health, this means that the attributions used to explain illness have a notable impact on health behavior, adjustment to illness, and recovery (Roesch and Weiner, 2001; Turnquist et al., 1988).

As a cause of poor health, “old age” is considered internal (i.e. *I am old*), stable (not apt to change), and uncontrollable (Banziger and

Drevenstedt, 1984; Townend et al., 2006). These dimensional properties mean that age attributions embody a dangerous logic: If old age causes poor health—and aging is uncontrollable—then health is also uncontrollable. Theoretically, the perception that poor health is due to old age should produce a reduced sense of responsibility for one's health, coupled with behavioral inhibition as individuals negate the importance of lifestyle habits and medical treatment (Bennett and Elliot, 2005; Gjorup et al., 1987; Leventhal and Prohaska, 1986).

Indeed, research demonstrates that when older adults attribute relatively minor health symptoms (e.g. breathlessness) and functional declines (e.g. forgetfulness) to old age, they are less likely to take action in terms of seeking medical treatment or otherwise addressing the possibility of an underlying condition (Ettinger et al., 1994; Goodwin et al., 1999; Kart, 1981; Sarkisian et al., 2001; Stoller, 1993). In this way, the continued attribution of symptoms to old age can culminate in untreated disease, excess disability, and even reduced longevity (Levy et al., 2011; Rakowski and Hickey, 1992; Williamson and Fried, 1996).

Attributing heart attack/stroke to old age

Although the consequences of attributing relatively minor symptoms to old age are well documented, very little is known about the consequences of attributing a major health event such as heart attack/stroke to old age. One reason for this involves the measurement procedures that have been used to assess attributions for heart attack/stroke in past research. Specifically, most past studies have used open-ended measures as a basis for assessing participants' attributions (Affleck et al., 1987a, 1987b; Croog and Richards, 1977; De Valle and Norman, 1992; Koslowsky et al., 1978; Runions et al., 2006; Thompson, 1991; Weinman et al., 2000). Open-ended measures fail to adequately assess attributional complexity and often result in the under-representation of implicit or unconscious beliefs such as "aging causes illness"

(French et al., 2002; Goodwin et al., 1999; Hall et al., 2003).

The current study

The current study employs a close-ended (vs open-ended) measure to assess old age attributions, and will examine the prediction that a greater tendency to attribute heart attack/stroke to "old age" will be associated with negative health outcomes (operationalized as self-report lifestyle behavior change, objective frequency of physician visits, and objective likelihood of hospitalization). This research builds on and extends past studies that have used an open-ended approach to assessing attributions (a strategy that generally fails to elicit "old age" attributions); studies that have considered men only; and studies that have been limited to samples of middle-aged adults < 65 years old (Affleck et al., 1987a, 1987b; Koslowsky et al., 1978; Thompson, 1991; Weinman et al., 2000).

Method

Participants and procedure

Aging in Manitoba (AIM) is one of the longest running population-based studies of aging in Canada, beginning in 1971 with a random, province-wide stratified sample of older adults aged 60+ years (for details, see Chipperfield et al., 1997). The $N = 57$ participants for this study (ages 73–98 years, 54% male) were selected from the 1996 wave of AIM to participate in a small subsidiary study known as the *Successful Aging Study* (SAS) that specifically targeted AIM participants who met the following criteria: (a) residing in one of the province's major cities, (b) living in the community (vs a personal care home), (c) having a satisfactory level of comprehension, and (d) responding to the interview in English (Chipperfield et al., 2004). In addition, to be included in the current analysis, participants had to have suffered a heart attack ($n = 44$) or stroke ($n = 13$). Participants were initially contacted by telephone and were subsequently interviewed in their own homes.

Measures

Covariates. We covaried for participants' chronological age (*range* = 73–98 years, $M = 80.40$, standard deviation (SD) = 6.07), gender (1 = male, $n = 31$; 2 = female, $n = 26$), and their severity of chronic conditions. Severity of chronic conditions was assessed with a procedure adapted from the Seriousness of Illness Rating Scale–Revised (SIRS-R; Rosenberg et al., 1987; Wyler et al., 1968). Based on past research (Chipperfield et al., 2007; Stewart et al., 2012), a list of 22 health conditions was presented and participants indicated (yes/no) whether they suffered from each condition (e.g. arthritis, cancer, diabetes, etc.). Each health condition was assigned a severity rank based on correspondence with the SIRS-R. A weighted severity of chronic conditions score was computed for each participant by summing across the rank values associated with each reported health condition. Thus, higher scores indicate greater severity of chronic conditions (i.e. poorer health; *range* = 1–171, $M = 61.40$, $SD = 34.02$).¹

Age attribution. Participants were asked “how strongly do you agree that your heart attack or stroke was due to old age?” (1 = *strongly disagree*, 6 = *strongly agree*; *range* = 1–6, $M = 2.44$, $SD = 1.62$). Past research demonstrates concurrent/discriminant validity of this measure to the extent that it correlates in the expected direction with various other illness attributions (Stewart et al., 2012) and a range of well-established health belief measures (e.g. health locus of control; Wallston et al., 1978).

Health outcomes. Three dependent health outcomes were examined, the first being a self-report indicator of *lifestyle behavior change*. Participants were asked “To what extent have you changed your lifestyle to try to avoid another heart attack/stroke?” (0 = *not at all*, 1 = *a little*, 2 = *quite a bit*, 3 = *a great deal*; *range* = 0–3, $M = 1.53$, $SD = 1.02$). The remaining two dependent variables were obtained from a provincial health registry that documents contact with the health-care system for all residents

covered through the universal Canadian health insurance. This registry provided a frequency count of *physician visits* over a 3-year period following the in-home interview (*range* = 19–295, $M = 112.77$, $SD = 64.93$), and an index of *hospitalization* over the 3-year period (0 = *no days spent in hospital*, $n = 18$; 1 = *one or more days spent in hospital*, $n = 32$).

Results

Regression was used to examine the relationship between attributing heart attack/stroke to “old age” and the three dependent variables. Separate linear regression models were used for the two continuous dependent variables (lifestyle behavior change and physician visits), and logistic regression was used for the dichotomous hospitalization variable (Pampel, 2000). All models included age, gender, and severity of chronic conditions as covariates. One-tailed critical t values were adopted given the directional hypothesis that old age attributions would be associated with negative health outcomes (Jones, 1952).

Lifestyle behavior change

As hypothesized, attributing heart attack/stroke to “old age” was associated with self-report lifestyle behavior change ($b = -.160$, confidence interval (CI) = $-.317$ to $-.002$, $\beta = -.25$, $t(55) = 2.03$, $p < .05$): The more the participants endorsed “old age” as the cause of heart attack/stroke, the less likely they were to make lifestyle changes following the event. In terms of the covariates, neither gender ($\beta = .16$, $t(55) = 1.33$, *ns*) nor severity of chronic conditions ($\beta = .17$, $t(55) = 1.39$, *ns*) was associated with lifestyle behavior change; however, chronological age had an inverse relationship with lifestyle behavior change ($b = -.074$, CI = $-.115$ to $-.032$, $\beta = -.43$, $t(55) = 3.52$, $p < .05$): The older the participants were, the less likely they were to change their lifestyle behavior following heart attack/stroke. Although a covariate-only model explained a significant amount of variance in lifestyle behavior change ($R^2 = .17$,

$F(3, 52) = 4.74, p < .05$), the addition of the “old age” attribution significantly improved the variance explained ($\Delta R^2 = .06, \Delta F(1, 51) = 4.13, p < .05$).

Physician visits

Attribution to “old age” also predicted physician visits ($b = 10.03, CI = -.79 \text{ to } 20.87, \beta = .25, t(48) = 1.86, p < .05$). The more the participants attributed heart attack/stroke to “old age,” the greater the number of physician visits they had during the 3-year follow-up period. In terms of the covariates, chronological age predicted frequency of physician visits ($b = -4.41, CI = -7.433 \text{ to } -1.40, \beta = -.40, t(48) = 2.95, p < .05$): Unexpectedly, older participants had fewer physician visits than did younger participants. Although speculative, one interpretation is that younger participants were more proactive about their health care, or potentially more mobile in getting to and from appointments. The severity of chronic conditions covariate also predicted physician visits ($b = .49, CI = -.01 \text{ to } .98, \beta = .27, t(48) = 2.05, p < .05$): As expected, greater illness severity predicted more physician visits. Gender was not a significant predictor of physician visits ($\beta = -.03, t(48) = 0.22, ns$). Again, a covariate-only model explained a significant proportion of variance in physician visits ($R^2 = .13, F(3, 45) = 3.55, p < .05$); the addition of the “old age” attribution marginally improved the variance explained ($\Delta R^2 = .06, \Delta F(1, 44) = 3.48, p = .06$).

Hospitalization

Consistent with the emerging pattern, attributions to “old age” also predicted the likelihood of being hospitalized over the 3-year follow-up period ($\beta = .40, Wald = 6.26, odds\ ratio = 2.01, p < .05$). A one increment increase in attributions to “old age” corresponded to a 101-percent increase in the odds of hospitalization (Pampel, 2000). To illustrate this finding, Figure 1 presents the probability of hospitalization (Y axis) by endorsement (yes/no) of the

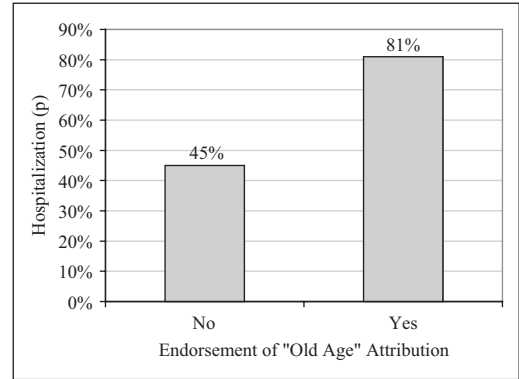


Figure 1. Predicted probability of hospitalization during 3-year follow-up period by endorsement of “old age” attribution.

“old age” attribution (X axis). Endorsement of the “old age” attribution was defined as responses between “2–6” on the 6-point scale ($n = 30$); whereas no endorsement was defined as a response of “1” ($n = 27$).¹ The probability of being hospitalized was 45 percent among older adults who did not endorse “old age” attributions, compared to 81 percent among older adults who did endorse “old age” attributions. In this model, none of the covariates (age, gender, and severity of chronic conditions) were related to hospitalization ($\beta = .03, odds\ ratio = 1.01; \beta = .09, odds\ ratio = 1.75; \beta = .13, odds\ ratio = 1.01$, respectively).²

Discussion

This study assessed the extent to which older adults attributed a recent heart attack/stroke to “old age,” and examined the consequences of this belief for subsequent health outcomes over a 3-year period. Endorsement of the belief that “old age” caused one’s heart attack/stroke was associated with detrimental health outcomes involving self-report lifestyle behavior as well as objective indicators of health-care service utilization. Findings underscore the need for further consideration of the implicit assumption that “age causes illness,” and suggest a potential new avenue for causality-focused health education interventions.

Age causes illness: an implicit belief with potential health consequences

Despite growing recognition of the modifiable causes of heart attack/stroke, the imprecise belief that age causes cardiovascular illness endures (Goodwin et al., 1999; Stewart et al., 2012). In this study, the more an older adult attributed their heart attack/stroke to old age, the less they reported changing their lifestyle behaviors following the event. To the extent that “old age” is perceived as an uncontrollable cause of illness, this finding fits with a broader literature concerning the relationship between perceived control and health (Lachman et al., 2011; Wallston, 2004). This literature repeatedly demonstrates that health-related motivation is compromised when individuals believe that they have little control over illness (Roesch and Weiner, 2001; Turnquist et al., 1988). The low-control mindset that likely results when individuals attribute heart attack/stroke to old age could lead to a reduced sense of responsibility for one’s health, and lowered motivation with regard to lifestyle behavior changes. In effect, believing that one’s heart attack/stroke was caused by old age amounts to the bleak outlook that “it doesn’t matter what I do—I am going to be sick now that I am old.”

In addition to its association with self-report lifestyle behavior, old age attributions predicted an objective indicator of frequency of physician visits. It was expected that participants would have a relatively large number of physician visits: They had all experienced a heart attack/stroke and were therefore likely to be receiving follow-up care. That said, the more an older adult attributed heart attack/stroke to old age, the more often they saw their doctor during a 3-year follow-up period. This finding could imply that older adults who attribute their heart attack/stroke to old age have poorer subsequent health status, slower recovery, and require more follow-up procedures. Conversely, this finding could also imply that older adults who attribute heart attack/stroke to old age are more proactive about their health care—that is, they are more likely to visit their physician when they need to.

However, given that these older adults reported being less proactive in changing their lifestyle behavior following the heart attack/stroke (see above), the former explanation may be more plausible.

Along with frequency of physician visits, endorsement of “old age” as a cause of heart attack/stroke predicted the likelihood of hospitalization during the 3-year follow-up period. In fact, the probability of being hospitalized was nearly double for participants who endorsed the “old age” attribution: 81 percent were hospitalized at some point over the subsequent 3 years, compared to 45 percent of those participants who did not endorse “old age” as a cause of heart attack/stroke.

Taken together, our findings suggest that the propensity to attribute heart attack/stroke to old age may contribute to poor subsequent health and recovery. Throughout, we have suggested that attributions to old age may represent the internalization of underlying stereotypes about older adults and the aging process. Our findings are consistent with the notion of stereotype embodiment: The belief that “age causes heart attack/stroke” may serve as a self-fulfilling prophecy whereby older adults begin to engage in a pattern of health behavior that brings about further poor health outcomes (Levy, 2009).

Implications

The “costs” of heart attack/stroke take several forms. Economically, the annual cost of heart disease and stroke is estimated at over US\$400b: In 2010, US\$1 out of every US\$6 spent on US health care was devoted to heart disease and stroke (Centers for Disease Control and Prevention, 2011). Socially, heart attack and stroke exact a major cost on population health, contributing to high rates of premature death and to caregiver burden at both the family and community level. At the individual level, the experience of heart attack/stroke reduces both quantity and quality of life, and recovery can be among the most challenging life events an individual will face. The findings of this study suggest that the way in which an individual explains

heart attack/stroke is associated with subsequent health and recovery and imply the potential value of causality-focused interventions in the movement to reduce the economic, social, and individual costs of heart attack/stroke.

Causality-focused interventions are those that are designed to enhance patient understanding of the underlying causes of cardiovascular illness. To date, most causality-focused interventions are “attribution prescriptive”: The aim is to prescribe a specific attribution for heart attack/stroke (usually lifestyle behavior). There is not, however, a counterbalanced emphasis on interventions designed to *discourage* competing attributions (such as aging). Without this counter-strategy, it is possible that the well-intended efforts of interventions designed to prescribe lifestyle attributions may be undermined by the implicit lay assumption that aging is the “real” or “most important” cause of illness.

Thus, it seems there is a need for causality-focused interventions designed to disentangle the relationship between age and cardiovascular illness. Because a valid relationship exists between age and cardiovascular illness, the purpose of such an intervention would not be to refute age as a contributor to cardiovascular illness. Rather, the purpose would be to modify patient perception about the underlying nature of aging: Aging is not a fixed trajectory of inevitable physical declines; but rather, the physical declines associated with aging are mutable and heavily influenced by one’s own volitional behavior. This amounts to the distinction between biological aging and chronological aging articulated by Fries (1980; see also Roizen, 1999). The underlying message that physiological decline (biological aging) is not the same thing as “getting older” (chronological aging) may be a useful addition to health education interventions designed to promote a healthier aging population.

Limitations and unresolved issues

The results of this study should be interpreted with the following limitations in mind. First,

analyses are based on a relatively small sample size that could limit the generalizability of findings. Nonetheless it is worth noting that participants were initially selected using a randomized stratified sampling technique (Chipperfield et al., 1997). A second limitation involves the single-item indicator of lifestyle behavior change, which did not permit an estimation of measurement reliability. However, results involving this dependent measure were consistent with those for the objective health outcomes obtained from a reliable health registry (i.e. frequency of physician visits and hospitalization).

An unresolved issue in this study involves the explication of mediating processes regarding the direct relationship between “old age” attributions and health outcomes (physician visits/hospitalization). A supplemental mediation analysis was performed to test lifestyle behavior change as a potential mediator. Although this analysis approached statistical significance, we could not confirm that the relationship between age attributions and physician visits/hospitalization was mediated by lifestyle behavior change. It is possible, however, that a true mediation effect exists and might be detected with increased power and/or a more nuanced measure of lifestyle behavior change.

Conclusion

Although there may be a kernel of truth to the stereotype that age causes cardiovascular illness (Jussim et al., 2009), focusing on age as the cause of cardiovascular illness is not adaptive from a health-motivation perspective. The belief that cardiovascular events such as heart attack/stroke are an inevitable consequence of aging is demotivating, and has the capacity to undermine health promotion behavior as individuals move from early-mid adulthood to old age. If we aim to secure a healthier aging population, we must begin to counter negative beliefs about the relationship between age and illness.

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Declaration of conflicting interests

The results and conclusions presented in this article are those of the authors. No official endorsement by Manitoba Health is intended or should be inferred. All procedures were evaluated and approved by the author's institutional research board of ethics.

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Notes

1. When asked how much their heart attack/stroke was due to old age, 47 percent of participants ($n = 27$) indicated "1" (*strongly disagree*). This finding was not unexpected, and may help explain why old age attributions have been under-researched. Nonetheless, disagreement by 47 percent does not negate the fact that 53 percent of the sample ($n = 30$) agreed that old age was, at least in part, a viable cause of their heart attack/stroke. This suggests that if our sample size was $N = 1000$, 530 participants would endorse old age attributions. However, because nearly half the sample replied "1," the attribution data were somewhat skewed = .64, standard error (SE) = .316. A square-root transformation reduced the skewness to .51, and analyses were re-run with the transformed variable. Because the pattern of results remained identical, and because the originally scaled variable is easier to interpret, we present results obtained using the non-transformed variable.
2. The analysis for lifestyle behavior change was based on $n = 56$ because one participant did not respond. The sample size for frequency of physician visits ($n = 49$) and hospitalization ($n = 50$) was reduced because seven participants passed away during the 3-year follow-up, and

one outlier was removed from the physician visits distribution.

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