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# Aging and Cumulative Inequality: How Does Inequality Get Under the Skin?

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***Purpose:*** This article draws from cumulative disadvantage and life course theories to develop a new theory for the social scientific study of aging. ***Design and Methods:*** Five axioms of *cumulative inequality (CI) theory* are articulated to identify how life course trajectories are influenced by early and accumulated inequalities but can be modified by available resources, perceived trajectories, and human agency. ***Results:*** Although the concept of CI has attracted considerable attention among social scientists, it holds promise for integrating additional disciplinary approaches to the study of aging including, but not limited to, biology, epidemiology, and immunology. The applicability of CI theory to gerontology is illustrated in research on the early origins of adult health. ***Implications:*** Primary contributions of the theory to gerontology include greater attention to family lineage as a source of inequality; genes, gestation, and childhood as critical to early and enduring inequalities; the onset, duration, and magnitude of exposures to risk and opportunity; and constraints on generalizations arising from cohort-centric studies.

*Key Words: Theory, Cumulative disadvantage, Life course, Stress, Psychosomatic processes*

The concept of accumulation has long interested gerontologists and, it appears, this interest has grown in recent years. Many biologists are studying how the aging process is altered by the accumulation of senescent cells, DNA damage, or oxidative damage. At the same time, social and behavioral scientists are examining topics such as cumulative disadvantage, financial accumulation, and stress accumulation. Although the concept of accumulation—gradually collecting or amassing something—is useful for many fields of study, gerontologists appear to be elevating it from a latent, almost silent, process into one which needs explicit attention to better understand the aging process. Indeed, a recent title search of PubMed for the words "accumulation" and "aging" identified 150 publications during the past 40 years. Half of them, however, were published in the past decade.

In the social sciences, much interest has been shown in Dannefer's (1987, 2003) prescient articulation of cumulative advantage/disadvantage (CAD) theory. O'Rand's (1996, 2003) exemplary writings are parallel in many respects, but she prefers the phrase cumulative advantage theory. Many scholars summarize CAD via the maxim *advantage accumulates*, but Dannefer (2003) defines CAD as the "systematic tendency for interindividual divergence in a given characteristic (e.g., money, health, status) with the passage of time" (p. 327). Viewing the life course perspective as too focused on microlevel processes—what Hagestad and Dannefer (2001) call "microfication"—Dannefer sought to privilege "a set of social dynamics that operate on a population, not individuals" (Douthit & Dannefer, 2007, p. 224).

Dannefer's aim to apply CAD theory to population and cohort processes is excellent. We need more attention to age stratification and how macrostructural forces influence aging. Nevertheless, it is also valid to apply some of these concepts to the analysis of micro- and mesolevel processes that influence the process of growing older (without becoming lost in microfied analyses). Perhaps studying the accumulation of inequality will help us understand multiple levels of relationships between individuals and their environments. Indeed,

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when Ferraro (2007) incorporated a tenet for cumulative disadvantage into the gerontological imagination, it spanned both individual and cohort phenomena: "disadvantage accumulates over the life course, thereby differentiating a cohort over time" (p. 336).

Encapsulated in that tenet is a recognition that cumulative disadvantage can be usefully linked with the life course perspective. Furthermore, another tenet of the gerontological imagination focuses on life course analyses: "aging is a life-long process, and using a life course perspective helps advance the scientific study of aging" (Ferraro, 2007, p. 335). Studying the *accumulation* of inequality implies more than studying older people only; it is quite consistent with the life course or life span approach (Elder, 1994).

The purpose of this article was, therefore, to integrate core concepts and principles from cumulative disadvantage and life course theories—as well as from other theories—into a new theory for studying aging and the accumulation of inequality. *Cumulative inequality (CI) theory* is specified as a middle-range theory that incorporates elements of macro- and microsociological content in an attempt to bridge both levels of analyses (Merton, 1968b).

Cumulative inequality theory specifies that social systems generate inequality, which is manifested over the life course via demographic and developmental processes, and that personal trajectories are shaped by the accumulation of risk, available resources, perceived trajectories, and human agency. Recognizing that CI may lead to premature mortality, the theory also emphasizes the use of cohort-inclusive and longitudinal studies to discern how selection processes shape inequality.

Ferraro, Shippee, and Schafer (in press) articulated five axioms of CI theory as they sought to apply it to the research on aging and the life course. In the process, they also enumerated how CI is distinct from CAD theory and provided a rationale for preferring the term "cumulative inequality theory" for this emergent perspective. The present article is designed to extend the development of the theory by focusing on its application to gerontology. The primary aims are twofold.

First, we seek to explicate this new theory for the *social scientific study of aging*, largely by integrating several current theories, especially CAD and life course theory. Second, recognizing the multidisciplinary nature of gerontology, we seek to develop meaningful links between CI theory and selected findings and models of biologic processes associated with aging. Social forces in everyday life can lead to psychological and biologic changes in the body. Unlike the idiomatic use of "under the skin," we hope to illustrate how CI leads to biologic changes in humans that are commonly associated with the process of growing older. In this sense, inequality may well lead to the accumulation of biologic materials under the skin that are markers of aging and predictive of senescence.

## Axioms of CI Theory

In this section, we summarize the five axioms of CI theory and identify their utility for gerontology (Ferraro et al., in press). Subsequently, we show how they can be meaningfully linked to biomedical and behavioral processes for the study of aging.

> Axiom 1: Social systems generate inequality, which is manifested over the life course through demographic and developmental processes.

Cumulative inequality theory draws on the sociological dictum that social structures shape human behavior and interpersonal relations. Heeding the criticism of Hagestad and Dannefer (2001), CI theory identifies social systems as central to the generation of inequality. Inequality is not primarily the result of individual choices and actions but is structurally generated. Even before an individual is born, social forces shape conception, fetal development, and birth. And evolutionary biologists have long noted that adaptation and survival have a social component (Kirkwood, 1977); natural selection is, after all, a selection process (i.e., organisms mature to reproduce, thereby propagating traits favorable to survival).

What CI theory adds to the focus on the social antecedents of inequality, however, is a recognition that inequality accumulates over the life course. Whether two infants (even twins) start with the same genes and environment or vastly different ones, differentiation will occur. Thus, age is an index of life changes and the accumulation of inequality. This is helpful for studying individual lives as well as for studying cohort differentiation. It also points to the importance of reproduction and genetics in shaping cohorts and the life course.

Cumulative inequality theory holds that childhood conditions are important for explaining adult

functioning and well-being; thus, gerontology benefits by systematically considering how *early-life* events and experiences shape *later life* outcomes. If childhood conditions are important for understanding the accumulation of inequality, then it stands to reason that family lineage is critical as well, both for genetic transmission and for shared living environments (and these topics have received little attention in CAD theory). Whereas families are nested within communities, CI theory also prioritizes a multilevel approach for understanding how social systems influence inequality over the life course.

Axiom 2: Disadvantage increases exposure to risk, but advantage increases exposure to opportunity.

Many people think of advantage and disadvantage as opposites, but this may limit our understanding of how inequality accumulates over the life course. Consider the difference between income that is $50,000 and –$50,000. The person with negative income does not just have less of the same units held by the person with positive income; rather, the person with negative income will encounter different social processes to deal with this situation. Instead of thinking of positive and negative income as simply the polar opposites of advantage and disadvantage, it may be useful to consider disadvantage as an exposure to risk (e.g., debt, bankruptcy) and advantage as an exposure to opportunity (e.g., investment, retirement security). Advantage and disadvantage refer to social positions in a hierarchy, but one should not simply assume that advantage is the opposite of disadvantage (see also Willson, Shuey, & Elder, 2007). In analytic terms, gerontologists need to consider nonlinear relationships in how events and processes imprint the life course.

Cumulative inequality theory also holds that there are multiple axes upon which inequality develops. Whereas inequality may diffuse across outcomes, it is useful for gerontological research to monitor multiple domains (Kelley-Moore & Ferraro, 2005). To best capture how these domains interact—and how risk factors accumulate—we need greater attention to conceptualizing and measuring the *magnitude*, *onset*, and *duration* of exposures to advantage and disadvantage. Magnitude refers to the extent or dose of an advantage or disadvantage. Onset refers to when the exposure began, and duration is the length of time that an individual experiences the condition (either risk or opportunity).

Axiom 3: Life course trajectories are shaped by the accumulation of risk, available resources, and human agency.

Although CI theory views social structure as antecedent in the development of inequality, there is no expectation that adverse experiences or events have inexorable effects over the life course. The accumulation of risks and opportunities is central to CI theory, but this does not mean that the individual's life is determined by early exposure to adverse or favorable experiences. Rather, we view trajectories as subject to change, a position consistent with a large body of gerontological research regarding the modifiability of the aging process (Maddox, 1987). How, then, are trajectories modified?

Inequality accumulates over the life course, but resource mobilization and human agency play critical roles in how trajectories are shaped. Resources can blunt the potential impact of earlier disadvantage, enabling the person to better function with or overcome the adversity (Ferraro & Kelley-Moore, 2003). Psychosocial resources are important, and human agency plays a critical role in shaping how likely it is that social adversity can get under the skin. For example, being treated unfairly can provoke a number of psychosomatic processes that can be harmful to health, but some persons seem better able to deflect or adapt to such unfair treatment without it damaging their health (Thoits, 2006). Others seem highly vulnerable to such treatment, even when they have resources that appear adequate given the magnitude of the unfair treatment.

The life course perspective gives priority to timing in one's life, and it may be useful to think of turning points as times during the life course when major change occurs in how the person responds to a risk or an opportunity (Elder & Shanahan, 2006). The point is that people *respond* to these stimuli, and CI theory draws on the richness of life course theory to explicitly incorporate human agency into the process of trajectory modification.

Axiom 4: The perception of life trajectories influences subsequent trajectories.

Another source of trajectory modification is outlined in Axiom 4, thereby highlighting how CI theory is distinctive from previous theories regarding the accumulation of inequality. People have a sense of how they are doing, and this sense influences their subsequent actions (Carstensen, 2006). Drawing from symbolic interaction and social

comparison theories, CI recognizes that subjective views of positions and resources may be more important than the actual positions and resources in shaping subsequent trajectories. Following the Thomas theorem, actors seek meaning for their positions (Merton, 1995), and the interconnectedness of human lives may predispose them to view their positions in the context of their associates (Elder & Shanahan, 2006). People view and evaluate their trajectories in comparison to significant others and reference groups.

The evaluation of trajectories is also part of the process whereby people select activities to optimize. According to the theory of selective optimization with compensation, a person first becomes aware of a deficit in functioning; then he or she selects an activity to optimize (Baltes, Staudinger, & Lindenberger, 1999). Compensation follows the evaluation of a performance concern and a decision to select an activity by which to restore a sense of optimal functioning.

Gerontologists have long been interested in how normative and nonnormative events influence adaptation as well as whether the person feels he or she is on or off time when transitioning to new roles. Cumulative inequality theory holds that favorable sentiments about being ahead of one's peers likely lead to self-efficacy, and self-efficacy may alter the psychosomatic processes initiated by adversity (Pearlin, Nguyen, Schieman, & Milkie, 2007). By contrast, unfavorable sentiments about a life transition, such as involuntary retirement due to corporate downsizing, would challenge self-efficacy and subsequent trajectories.

> Axiom 5: Cumulative inequality may lead to premature mortality; therefore, nonrandom selection may give the appearance of decreasing inequality in later life.

As noted earlier, selection processes are central to the study of how inequality accumulates over the life course. The practical significance is that if CI operates on mortality, the composition of a *population* will change, perhaps resulting in what some refer to as "cohort inversion." Removing persons with the most health problems from a population will make the cohort that was initially disadvantaged appear better off than before the population truncation. The likely result is that mean scores may rise, giving the appearance of improving health and decreasing inequality.

This problem may be exacerbated for *samples* because continued participation in most longitudinal studies is contingent on the ability to respond (independent of availability in the population). As such, gerontologists should give explicit attention to nonrandom selection in their studies as shown recently by Willson and associates (2007). Moreover, the utility of cohort-centric studies for assessing CI merits sober consideration. If the aim is to show how inequality *accumulates*, it should be clear that studying limited age ranges (e.g., 70+) will result in describing changes after considerable population truncation has occurred. Stated plainly, should gerontologists study older people only? If the aim is to identify mechanisms for the accumulation of risk factors and inequality, the limitations of cohort-centric designs should be apparent.

To supplement this brief overview of the theory, Table 1 lists the five axioms as well as propositions for each as specified by Ferraro and associates (in press). The table is intended to formalize the theory for developing hypotheses for future research, and it may clarify some of the ways in which CI differs from CAD and life course theories. For instance, there is little attention to the role of family lineage (Proposition 1c) in CAD theory. In addition, neither CAD nor life course theory specifies propositions that focus on how perceived life trajectories shape self-efficacy and psychosomatic processes (4b and 4c). For the remainder of the article, the table also permits concise reference to the theory as we articulate links to other disciplines and to the field of gerontology more generally.

## Interdisciplinary Linkages for Studying Aging and Senescence

Although CI theory has strong connections to sociology, meaningful links may also be developed to biologic and psychological concepts and theories of aging and senescence. Others have remarked on such potential connections, but we are unaware of any systematic endeavors to explicate the links. For instance, Alkema and Alley (2006, p. 577) astutely asserted that accumulation processes are "closely related to biological concepts such as stress theories of aging," allostatic load, and physiological dysregulation. Thus, in this section, we articulate some of these interdisciplinary links and more directly address the question of how inequality gets under the skin. Two topics are considered to illuminate how CI theory can be utilized, at least in part, to enhance our understanding of the multidimensional phenomenon known as aging.

**Table 1. Axioms and Propositions of Cumulative Inequality Theory**

| Axioms | Propositions |
|---|---|
| 1. Social systems generate inequality, which is manifested over the life course through demographic and developmental processes. | a) Childhood conditions are important to adulthood, especially when differences in experience or status emerge early.<br>b) Reproduction is a fulcrum for defining life course trajectories and population aging.<br>c) Influenced by genes and environment, family lineage is critical to status differentiation early in the life course.<br>d) Cohorts provide the context for development, structuring risks, and opportunities.<br>e) Consider inter- and intra-individual processes and use analytical techniques that explain variability on multiple levels or in multiple domains. |
| 2. Disadvantage increases exposure to risk, but advantage increases exposure to opportunity. | a) Consequences of advantage may not be the inverse of disadvantage.<br>b) Inequality may diffuse across life domains (e.g., health and wealth).<br>c) Trajectories are affected by the onset, duration, and magnitude of exposures. |
| 3. Life-course trajectories are shaped by the accumulation of risk, available resources, and human agency. | a) Human agency and resource mobilization may modify trajectories.<br>b) Turning points in the life course may alter the anticipated consequences of a chain of risk.<br>c) The dialectic of human agency and social structure is essential to cumulative inequality.<br>d) Unfavorable trajectories can be mitigated by the magnitude, onset, and duration of resources; resources can also accelerate favorable trajectories. |
| 4. The perception of life trajectories influences subsequent trajectories. | a) Social comparisons shape trajectories.<br>b) Favorable life review linked to self-efficacy.<br>c) Perceived life course timing influences psychosomatic processes. |
| 5. Cumulative inequality may lead to premature mortality; therefore, nonrandom selection may give the appearance of decreasing inequality in later life. | a) Cumulative inequality creates compositional change in a population.<br>b) Population truncation may give the appearance of decreasing inequality.<br>c) Test for selection effects.<br>d) Interpret results in light of event censoring and cohort inclusiveness. |

*Note*: Adapted from Ferraro, Shippee, & Schafer (2009).

### *Early Origins of Adult Health*

As noted earlier, CI theory gives explicit attention to the role of reproduction, family lineage, genes, and environmental context in shaping the life course (i.e., Propositions 1a to 1c and 2c). Whereas CI focuses on the compounding of status hierarchies, it is important to understand the *early* origins of health. Are there inequalities in how people start the race of life? Gerontologists frequently think of aging as a life course process—from birth to death—but gestation may have a more vital role in gerontology than many of us would have previously imagined. From conception to birth, evidence is mounting that the circumstances of fetal growth may have long-term consequences on health, both during childhood and adulthood.

The work of Barker (2003) has been both enlightening and controversial for providing insight into how fetal health may be related to adult health outcomes. Beyond the obvious consequences that risky maternal behaviors such as smoking may have on fetal health, Godfrey and Baker (2000) have shown that poor fetal nutrition raises the risk of low birth weight as well as *adult* health problems, including hypertension, coronary heart disease, and stroke. The scientific innovation in this genre of research is to track the individuals from conception to later life, even death, and to systematically document the associations. The outcomes

considered are diverse, extending also to diabetes and sarcopenia.

As might be expected, this line of inquiry has raised some controversy. Singhal and Lucas (2004) argue that it is not fetal programming per se but the rate of growth *after* birth (accelerated development) that is the true causal agent. Severely malnourished fetuses may not survive, but most that survive will experience rapid growth. Singhal and Lucas contend that it is the rapid growth during infancy that raises the risk of obesity and insulin resistance. Moreover, both the programming and accelerated growth theses imply metabolic discontinuity. It may be that the "metabolic whiplash" from poor nutrition during gestation to rich nutrition during accelerated infant growth requires major homeostatic recalibration; perhaps this recalibration (or reprogramming) is the pathway to a series of metabolic and health problems. In adulthood, there is evidence that weight cycling creates metabolic problems often leading to weight gain and insulin resistance. Perhaps this whole sequence starts much earlier in the life of the organism (Montani, Viecelli, Prévot, & Dulloo, 2006).

The point is that gestation, infancy, and early childhood are critical periods in the life course. Gerontologists obviously consider later life as a period of profound change and adaptation, but the seeds of many of these changes may have been planted decades earlier. Although it may seem antithetical to gerontology as a field of study, we assert that gerontologists interested in health issues should be educated about and seek to integrate what has been learned in recent decades on the early origins of health. A beginning point is the observation by Forrest and Riley (2004, p. 162) that "childhood is an incubation period for many disorders that affect the health of the whole population."

Controversy has also arisen regarding whether early origins have inexorable effects on adult health. Some investigators hold that the effects of selected early events are indelible—a scarring effect due to the early insult. Indeed, this viewpoint led Barker (2003) to propose a new approach to thinking about disease causation. He is critical of the widely held view that chronic diseases are the result of health destructive actions on the part of adults, seeking rather to improve adult health through better maternal and child health. Cumulative inequality theory concurs in some respects by identifying family lineage, genes, maternal health, and childhood as critical to how health inequalities develop over the life course (Propositions 1a to 1c). Nevertheless, we disagree that the early influences are necessarily inexorable in their effects. Many early imprints on the organism can be neutralized or reversed, and a growing number of investigators are discovering that resources may moderate the effects of adversities (Propositions 3a and 3d). The work of gerontology, therefore, should include identifying how to modify what may appear to be inexorable influences on health. This can occur by prospective tracking of persons over long periods of time or by interventional research seeking to interrupt a presumed chain of events. As Maddox (1987, p. 563) sagaciously concluded decades ago:

> We do not know the limits of modifiability of aging processes and the experience of aging. We do know that if we want to understand aging processes and the experience of aging, we can learn by trying to change them when the observed outcomes are unacceptable.

Indeed, if the events and processes that compromise quality of life cannot be modified, what is the aim of biomedical research? Cumulative inequality theory holds that the imprint of early life is substantial but that there are many ways to modify the deleterious chain of events due to early inequalities.

It may be reasonable to accept the premise that gestation and childhood shape health outcomes in adulthood, but how and why does this occur? Some scholars feel that the fetal programming and accelerated growth perspectives rely on an oversimplified view of the link between early origins and health in later life. The two stages of life may be linked, but why?

First, some scholars argue that there may be a genetic basis for the link, a premise that is plausible but insufficient for explaining the major social changes in disease incidence and prevalence.

Second, there may be complex etiological links between early and later life, which we are only beginning to understand. Although the links are complex, CI theory is designed to help explicate the accumulation processes that alter one's chances to age optimally. Some social scientists have viewed cumulative disadvantage primarily as the growing gap in an outcome between status groups—early disadvantage amplifies inequality over time—but we think this misses a main point. Early inequalities lead to differences in how individuals and groups are *exposed* to risk factors that typically compromise health (Axiom 2; Propositions 2a and

2c). In short, CI is a life course *process*, not just the difference in outcomes due to early adversity.

A research program that provides great insight into how inequality accumulates from childhood to adulthood is led by Felitti (2002). Using data from managed care enrollees, Felitti and colleagues (1998) found that childhood adversities (ranging from parental divorce to sexual abuse) are more common than widely reported and consequential to adult health 50 years later. In related articles, not cited herein, they also showed that a count of adverse childhood experiences increased the likelihood of engaging in behaviors that compromise health (smoking, obesity, and attempted suicide). Thus, early adversities may have both direct and indirect effects on physical health, with the indirect effects often manifested through high-risk lifestyles.

Children who are abused, for instance, may suffer immediate bodily harm as well as impaired psychosocial development and an inclination toward risk taking—and each is a mechanism by which early adversity may lead to poor health (Irving & Ferraro, 2006). This illustration also clarifies that disadvantage in one domain (e.g., family life) may spill over to other domains (e.g., mental and physical health) as specified in Proposition 2b. Moreover, early adversity increases the likelihood of exposure to additional risks, which may lead to additional adversity. Such a pernicious cycle is difficult to overcome, especially when it is situated in an environment and a family rife with problems (Propositions 1a to 1d).

### *Stress Accumulation*

The first axiom of CI theory focuses on how social arrangements shape inequality over the life course. The primary antecedents of inequality are socially patterned—even genetic material is socially transmitted—and one of the major mechanisms by which social forces get under the skin is through stress processes. We have, of course, alluded to these mechanisms earlier, but it may be useful to focus more closely on biologic processes associated with adversity. Social and environmental stressors often precipitate biologic processes that shape the survival and functioning of the organism (Hayflick, 1998). Cumulative inequality theory may be helpful for identifying how these stressors accumulate, modify cohort inequality, and diffuse across life domains (Propositions 1d and 2b).

When people sense stress, common physiological responses include activation of adrenal hormones, such as cortisol and catecholamines, and autonomic nervous system reactivity (Lundberg, 2005). Occasional arousal of these responses is normal and probably conducive to healthy functioning and adaptation. A growing body of research, however, shows that chronic activation of these responses may have adverse effects on the human body. The biologic arousal due to stressors is increasingly seen as the intersection of many processes ranging from social and behavioral determinants of stress exposure and resource mobilization to the endocrine responses that influence immune function.

Recent findings from stress research document how the accumulation of these stress responses can accelerate the aging process, even among people who are asymptomatic of disease. For instance, a recent article by Epel and associates (2006) shows that stress arousal is related to low leukocyte telomerase, a precursor of telomere shortening. Telomeres are DNA–protein complexes found at the ends of chromosomes, which act as caps to keep the sticky ends from fusing together. Telomeres are essential to the stability of genetic information, and it is widely known that telomeres are shorter in older organisms. Moreover, the cellular enzyme, telomerase, helps telomere stability and length, thereby aiding genetic stability and cell function.

Epel and associates (2006) administered a social stress test to a sample of 62 women and found that stress exposure was related to less telomerase and shorter telomeres. Interestingly, they also found that telomerase was positively correlated with education. In essence, they uncovered that social factors and stressful exposures were related to biochemical responses that influence genetic stability and cell function. This finding suggests the utility of telomerase and telomere length as biomarkers of the accumulated effects of stress over the life course.

Drawing from CI theory, people who had early-onset and severe adversity (e.g., child abuse) would be more likely than their peers without such adversity to be telomerase deficient, resulting in shorter telomeres (Propositions 1a and 1c). This hypothesized difference should exist long before the development of heart disease or other diseases commonly associated with stress processes (Proposition 2c). In addition to this hypothesis, it would also be fascinating to determine whether telomerase levels rise with adequate resource mobilization (Proposition 3d). Whatever the case, biogerontologists have

expressed considerable interest in the process of telomere shortening, and Epel and associates (2006) provide an important insight into how accumulated stress may lead to biologic changes that accelerate aging and senescence.

A second illustration of how accumulation processes are critical to understanding growing inequality in populations and cohorts is through *chronic inflammation*. A review by Finch and Crimmins (2004) reveals that infections during the early years of life increase the likelihood that the person will develop diseases as an adult, including cancer, diabetes, and cardiovascular disease (see also Blackwell, Hayward, & Crimmins, 2001). From CI theory, we see that early disadvantage increases the *risk* for additional disadvantage: Early infections increase the risk for chronic adult morbidity, creating a lifetime of chronic inflammation. Advantaged children, who have less exposure to infections, are less likely to become infected during childhood, resulting in a lower risk for chronic disease during adulthood. They would still have bouts of inflammation, but these would be less chronic over the life course, allowing greater time for recovery and optimal functioning. Disadvantaged children, however, such as those with an alcoholic parent, may be more susceptible to infections due to poorer living conditions and the chronic stress of potential abuse (Propositions 1a to 1d).

Several biomarkers can be used to identify chronic inflammation, including interleukin-6, fibrinogen, and tumor necrosis factor-α, but the most widely used is C-reactive protein (CRP). C-reactive protein is an acute-phase inflammatory protein that signals a response to an exogenous or endogenous insult. For instance, CRP will be elevated from external trauma or from internal tissue malfunction. C-reactive protein is functional for short-term adaptation to inflammation but can be maladaptive if chronically elevated. Monitoring CRP and other biomarkers in the population may help identify how chronic inflammation shapes aging and senescence. According to CI theory, we anticipate that accumulated disadvantage will generally be associated with chronic inflammation but that the characteristics of the exposure and the mobilization of resources will also influence the degree of chronic inflammation (Propositions 2c and 3d).

## Implications of CI for Gerontology

Cumulative inequality is proposed here as a new social science theory of aging to help us explain the ways in which accumulated inequality can get under the skin. In this concluding section, we reflect on CI theory's utility for gerontology and offer several observations regarding what might accelerate theory development in social gerontology and, more generally, paradigm development in gerontology.

It should be noted that many elements of our theory have been discussed in previous theories of aging, but the integration of these elements is novel. For instance, we merge concepts and expectations from the following theories: CAD, life course, symbolic interactionism, stress process, and chronic inflammation. The result is a new way of thinking about how inequality accumulates over the life course. In doing so, we also draw several conceptual distinctions such as differentiating between disadvantage and risk (Axiom 2) and noting that disadvantage is not the inverse of advantage (Proposition 2a). We also give priority to family transmission of inequality (both genetic and environmental) and the role that actors play in interpreting their life trajectories. Details of how we constructed the theory and how it is distinct from other theories are presented elsewhere (Ferraro et al., in press).

In this article, we drew from research on the early origins of health and stress processes to provide examples of how CI can build some meaningful links to other fields of study and theories of aging. The attempt was to build bridges. If successful, these are surely footbridges. Much work needs to be done to more formally articulate how CI theory can be integrated with more biomedical findings and biologically based frameworks for understanding aging and senescence.

Several conclusions surfaced from this endeavor. First, gerontology needs greater and more systematic attention to *accumulation processes*. For instance, epidemiologists often refer to the accumulation of risk factors, but what does it mean? There are many ways to accumulate items—continually, intermittently, in large or small quantities. And accumulation may infer dumping (e.g., catastrophe theory) or systematic adaptation to the accumulation of experiences. We need both empirical research and theoretical explication about how accumulation processes shape the aging process. By analogy, we should not aim to study just the contents of the accumulation process but the processes by which the content was accumulated (Propositions 1d, 2c, 5a, and 5c).

Gerontologists would also be wise to study both the positive and negative content of accumulation processes. There is an understandable interest in studying the accumulation of negative content such as health deficits (Kulminski et al., 2007) and damage (Finkelstein, 2007). At the same time, we applaud the growing interest in how positive attributes or characteristics influence aging; examples include compensatory skill (Baltes et al., 1999), cognitive reserve (Richards & Sacker, 2003), and intellectual strategies (Roring & Charness, 2007).

Cumulative inequality theory provides a useful framework for studying accumulation processes, especially in the context of social stratification (Merton, 1968a). As noted earlier, biologic processes are related to patterns of social interaction. Very telling in this regard is research by Cohen, Doyle, and Baum (2006) revealing that socioeconomic status (SES) is inversely associated with stress hormones such as cortisol and catecholamines (e.g., epinephrine and norepinephrine). This is an important finding for stress researchers who want to better understand how accumulated stress influences biologic functioning. At the same time, it is important for social gerontology. The inverse relationship between SES and stress hormones means that there are observable biomarkers of accumulated stress that can be used to examine how people respond to long-term disadvantage. Moreover, it points to SES as an enduring exposure to risk or opportunity (Propositions 2a and 2c)—and one that has been shown to have long-term effects. Growing older entails accumulation. Cumulative inequality theory provides a frame of reference to consider the accumulation processes and the mechanisms that either exacerbate or reduce the risk for chronic stressors (Propositions 2c and 3d, respectively).

Second, it should be clear from this essay that CI theory is not an ontogenetic stage theory of aging, and life stages need not be tightly linked to chronological age. Keeping with another tenet of the gerontological imagination, we need a healthy skepticism for aging as a causal variable (Ferraro, 2007). Cumulative inequality theory uses basic life stages as a frame for understanding accumulation processes involving structural disadvantage, exposure to risk, and mobilization of resources.

Cumulative inequality theory gives special attention to family lineage and, concomitantly, reproduction, gestation, and childhood. The interest in fetal and childhood origins of health is strong, and we believe that gerontology will benefit by studying accumulation processes during these early periods (Propositions 1a, 1c, and 1d). We agree with Finch and Crimmins (2004, p. 1739) that "a new theory of human health in life history could emerge from a fuller accounting of inflammatory exposures from *gestation* to old age" (emphasis added). Although the promise of these early origins is profound, it could be argued that we are in a scientific phase of health studies that might be described as the *black box of middle age*. Cumulative inequality theory holds that early origins are important but not just because "indelible" events directly accelerate aging and senescence. Rather, we view the early origins as precipitating different exposures for those who were disadvantaged early. In this sense, we also need to integrate adult adversity in spheres such as work and family into the study of CI. Our aim was to uncover the pathways by which early disadvantage leads to health problems and early senescence. Cumulative inequality theory, therefore, examines the intertwined phases of the life course for the development of inequality. We do not assume that certain periods of life initiate changes that are universally experienced by a population. Instead, our interest is in studying how people experience events and processes at different times—in both biographical and historical time—and how some people are able to reduce their exposure to noxious events and experiences.

Third, Axiom 5 provides a lens for interpreting the processes of accumulation. This axiom recognizes how mortality changes the composition of a population. In this sense, we must realize that people at advanced ages have survived the insults of everyday life and some have overcome incredible odds. The intellectual consequences for gerontology are twofold: (a) we need greater appreciation for older people as survivors and (b) we need to recognize the limitations of cohort centrism and population truncation for studies of accumulation processes. We also need more long-term life course studies of aging, especially if we intend to test how inequality accumulates (Propositions 5b to 5d).

Finally, gerontology as a field of study has long praised interdisciplinary collaboration, and it remains one of the most exemplary fields of study to build bridges across disciplines. At times, however, the pace of gerontology's transformation from a multidisciplinary to an interdisciplinary field of study almost seems dilatory (Achenbaum, 1995; Bass & Ferraro, 2000). Many gerontologists espouse the virtues of interdisciplinary research, but we suspect that relatively few of those who

claim to be gerontologists actually engage in such research.

This slow pace of interdisciplinary integration, moreover, is changing due to the intense scientific interest in biomarkers in population-based studies. Although collecting biomarkers from clinical samples has been done for centuries, we are now seeing more systematic collection of biologic specimens in population-based surveys. With greater collection of biologic specimens in populations, we are on the verge of exciting scientific developments for studies not only of disease development but also for normal aging. In addition, if biomarkers can be integrated into intervention studies or policy research, we will have a direct window on how social arrangements, whether planned or unintentional, accelerate or retard senescence.

Interdisciplinary collaboration in gerontology, moreover, is now growing at a much faster pace because of the availability of social survey data that are coupled with biomarker data. Indeed, research questions about telomere shortening and chronic inflammation are now being formulated by social scientists. Collaboration with more biologic scholars is surely advisable, but the point is that few sociologists or psychologists a decade ago even considered the prospect that they might have a survey that also included telomerase or CRP as variables. This is a major shift in the science of aging and one that points to our need for interdisciplinary studies of accumulation processes over the life course. Cumulative inequality theory is offered to aid such interdisciplinary collaborations and guide empirical tests of how inequality gets under the skin.


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