Abstract

This chapter reviews a specific line of development of theory in research on aging and inequality that reflects the intersection of the influence of social and biological factors on life course inequality. This research has elevated the importance of health for defining the aging process, albeit as it develops within the sequentially unequal social contexts of the life course. Social inequality and health are mutually influential across the life span: social inequality creates original conditions for health inequalities while health comes to perpetuate and exacerbate social inequality with age. Empirical observations of this process have generated multiple theories of aging and health. In addition, the integration of genomic, physiological and clinical data with social survey data has made this line of investigation central to aging theory. This development has coincided with new concerns in theory development about correlational versus causal explanations of aging.

Keywords: inequality and health, health disparities, correlational, generative and causal theories
Long, Broad and Deep: New Research Directions in Aging and Inequality

The development of theories of inequality over the life courses of aging cohorts is arguably the core theoretical enterprise driving most of aging research over the last two decades. The origins and interdependent trajectories of inequality with age across life domains including race/ethnicity, gender, family, education, socioeconomic status and health are the predominant foci represented in research journals, books and book chapters. Life chances from birth to death and life styles related to social perceptions and behaviors and their life course consequences pervade this literature. The problematic long-term cumulative dynamics among race/ethnicity, class of origin (especially poverty in childhood), education, and health with age have assumed a central position in this research and unleashed a robust conversation about age and inequality, and particularly about how inequality “gets under the skin”--putatively to accelerate the aging process, to constrain the quality of life and to yield wide variations in (healthy) life expectancy.

The focus on health has been generated, in part, by the integration of biological with social data that has introduced new, and potentially transformative, sources for theoretical elaboration. Genomic, physiological biomarker, and clinical and other administrative data collected as repeated observations over the life course are raising questions about gene-environment interactions and about how social exposures to stressors such as sustained poverty, to economic shocks such as unemployment, and to social losses such as late-life loneliness affect the aging process. Research on aging and inequality before the mid-1990s largely could not get under the skin per se, although it identified important surface manifestations of environmental conditions and subjective responses that influenced aging through processes of inequality. Also,
longitudinal data were only beginning to capture mid- to late-life processes of aging that could link earlier to later life conditions and establish associations (and possible causal linkages) of inequality across the life course.

This chapter will focus specifically on this ascendant research area, although the field of aging and inequality is a broader one. In my judgment this research area provides an opportunity to examine theory development and how it proceeds as a product of the interaction of pre-existing theories, new data, and innovative methods for their analysis. Specifically, with the integration of maturing longitudinal panel data including social and biological measures over longer periods of the life course with time-sensitive methods of data analysis, aging is now observable as a continuous lifelong process, not as disjunctive and strictly age dependent.

Institutional factors matter, even those that stratify populations by age, but the dynamics of inequality are continuous, cumulative and pervasive leading to inequalities and disparities whose mechanisms are problematic and motivate new theories.

Long, Broad and Deep: What Aging Is and Why and How It Occurs

The lifelong manifold process of aging implicates biological, psychological, social, and environmental factors that interact over time and across place in complex ways to direct and temporally organize the shapes and boundaries of lives (O’Rand, 2003, 2009). As such, aging is a long, broad and deep process: long, because it occurs continuously across the life span (probably starting before birth); broad, because it continuously integrates diverse factors from across levels of observation (from the molecular to the social to the global); deep, because it is never fully and directly observable as an ongoing generative process. The patterns by which these interactions occur lead to variations in the pace of aging that are only partially evident in
patterns of inequality that include widening socioeconomic stratification and the differential onsets and trajectories of health decline and mortality rates observable within aging cohorts. Some but not all major empirical generalizations that have emerged as components of the phenomena of aging include: lifelong cumulative patterns of sequential contingency (or selection) in which earlier outcomes constrain later ones producing divergent trajectories of well-being; formative (and sometimes fateful) conditions, experiences or traits in early life which have enduring direct or indirect constraints on later ones; mutually influential processes whose causal relationships are confounded (and confounding) over time (especially the relationship between education and health; deflections or redirections from earlier trajectories as a result of encounters with major life course redirections stemming from exogenous or agentic interventions; underlying (latent) processes that may become manifest long after their origination—and that are often observed only through smaller surface manifestations of the deeper generative process.

These are inherently stratification processes that lead to the differentiation over time of cohort members whose fortunes in wealth and health diverge as a result of their successive encounters with stratified and stratifying institutions, including families, schools, health care systems, workplaces, neighborhoods, communities, and others, and with life course risks that disrupt or derail lives, such as illness, job loss, family dissolution, and similar life events. The differentiation process is traceable to observations of the status attainment models of the 1970s that examined the intergenerational transmission of social status and the critical period of adolescence to young adulthood (the transition to adulthood differentiated principally by educational attainment) that had lifelong consequences for socioeconomic status attainment. Status attainment models were refined by the 1980s—in part due to the maturing of longitudinal
databases initiated in the 1960s (e.g., the Panel Study of Income Dynamics; panels of the National Longitudinal Studies; the Wisconsin Longitudinal Study)—to address the significance of variability in the timing and sequencing of life transitions for well-being and their importance in differentiating later outcomes. At the same time, new approaches to incorporate the structural effects of race, gender, class and state and market institutions extended the prevailing stratification model. Some of these surveys and newer ones (the National Survey of Families & Households; the Health and Retirement Study) continue and have been enriched by linkages to administrative records (such as Medicare and Social Security), geographic information systems, and clinical and genomic data collections. The large scale shift to add prospective data on health to these surveys and to initiate new studies with fine-grained attention to health indicators as focal concerns has been a primary thrust over the last three decades that has provided the empirical bases for studying the societal-health nexus in aging, which is now a core concern.

The continuousness of the manifold aging process raises many challenges for theory. These include the causal priority of elements of the process (e.g., genes, environments and individual agency); selection-causation conundra among mutually influential co-occurring processes over time (e.g., SES↔health); the complex multifactorial etiologies of health conditions; seemingly uninterpretable N-way interactions among multiple factors; comparability of data sources across populations (e.g., sampling, harmonization of measures) to afford rigorous comparisons for theory-building; and unmeasured (unobserved) heterogeneity that biases observations, among others. These challenges deserve separate treatment in their own right. For purposes of this general review I define them briefly here:

*Causal priority: what comes first or before the others. Genes? Environments? Individual propensities and behaviors? Since theory is about causation or contingency, causal
priority over time is a fundamental challenge. Genes, environments, and individual agency interact over time in ways that we are only beginning to understand. How do we disentangle cause and effect?

*Selection-causation: related to the earlier challenge is the question of whether prior factors related to social inequality and/or health select individuals into subsequent circumstances and with how much relative effect. How do subsequent conditions mediate or moderate these earlier conditions and why?

*Etiologies of disease refer to the complex (and usually deep) bases of diseases and health conditions and the extent to which they are vulnerable to variations in social environments: what are the relative influences of genes and environments on the onsets and progressions of illnesses?

*Uninterpretable n-way interactions refer to how much sense can be made of the interactions among many factors to produce specific outcomes.

*Data comparability confronts the problem of how across different samples and data sources the same “issue” or “problem” is treated equivalently or similarly enough to be a basis of comparison across studies. Is disease or social inequality measured equivalently across studies to contribute to an empirical generalization?

*Unmeasured heterogeneity is the incapacity to have all the data necessary in a single study to capture all the information we probably need to specify the process of interest. What is “out of sight” in the study and how can we ameliorate this with linked data from other sources or with statistical controls that manage the errors in our observations?

Observation of these general empirical components of aging necessarily require repeated measurements drawn over extended periods and, ideally, from several sources, that may include
social surveys, administrative records from institutional sources, clinical data collections (biomarker and genomic data), geographic information systems, experiments, and on-the-ground direct observational methods. They also require analytical methods that capture the temporal features of aging (i.e. timing, sequence, duration, and trajectories of continuity, accentuation, and deflection) and that estimate potential causal pathways along the life course.

The normal science of aging—and generally of most sociologically interesting phenomena—pursues explanations by following what Goldthorpe (2001) refers to as *causation as robust dependence*. This is a correlational approach to examine multiple covariate effects on an outcome of interest using regression-based techniques. In this approach, efforts to move from association to causation (i.e. that X is a genuine cause of Y) are guided by statistical criteria that the dependence of Y on X is robust, or cannot be eliminated through other variables (some unobserved) or sampling biases (e.g. non-representative samples or selective attrition) by detecting and eliminating spurious causal significance. Causation is defined by the predictive power or explained variation in Y. However, aging researchers are calling for more than statistical predictability. Predictability must be guided by theory, some examples of which follow. And, predictability is not equivalent to causality which requires what Goldthorpe (2001) refers to as *consequential manipulation*, which will be considered at the end of this section.

Three theoretical projects focused on life course (aging) inequality are reviewed below. They are successively more encompassing, beginning with the education-health project followed by the fundamental causes of health inequalities theory and ending with cumulative inequality theory. The large share of publications associated with these theories overwhelmingly follow the causation by robust dependence heuristics. However, some research departs from this in ways that will be briefly summarized.
Education, Health and Aging

The robust dependence of health on educational attainment is now well established across hundreds of publications over 20 years (e.g. Ross & Wu, 1995; Mirowsky & Ross, 2003; Ross & Mirowsky, 2010). Educational attainment decreases age-specific rates of morbidity, disability and mortality and increases aspects of mental and physical health functioning and self-assessed health in adult populations, after controlling for numerous earlier and subsequent life course conditions. The theoretical challenge has been to explain the robust correlation that persists across controls for mediating and moderating factors by situating it in a more generalized theory of action. Human capital theory has been adopted to serve this purpose by leading medical sociologists in this area. The argument is that educational attainment represents accumulated knowledge, skills, values and behaviors learned at school (Mirowsky & Ross, 2005). These specifically include literacy, numeracy, problem-solving, analytical flexibility, observation and experimentation, among other forms of cognitive capital. Importantly, these forms of (cognitive) capital are purported to spill over into health-related behavior by promoting health literacy to benefit of health over the life course. Hence, the mechanism of social action and rational choice within the boundaries of knowledge.

In this research program, the human capital framework has spawned spin-off hypotheses that elaborate the general theory. For example, the gender gap in physical impairment that places women at greater risk for chronic disabilities provides an opportunity to examine gender differences in health and the role of education in their emergence. Since women are in disadvantaged positions relative to men--with fewer opportunities for economic gain, workplace authority, and wider community social status—education assumes a distinctive importance for women in the absence of alternative resources. Lower levels of education will result in higher
levels of impairment among women than among men. Mirowsky and Ross (2010) label this hypothesis the resource substitution hypothesis; they label the competing hypothesis, the reinforcement of advantage hypothesis. Their results support a resource substitution process in which education matters more on average for women’s physical impairment than men’s, although the gender gap disappears among men and women with college degrees.

Non-cognitive factors receive less attention in this work so far, although these variables are receiving more and more attention in the developmental psychology and economics literatures. In developmental psychology a long tradition of interest has focused on the long-term effects of persistent personality traits and levels of social competence and effectiveness such as self-control, ambition and self-efficacy and pointed to a broad domain of non-cognitive factors that probably operate jointly with cognitive skills acquired in-school and out-of-school. Researchers with explicit aging interests have linked such non-cognitive factors as self-control (Moffitt et al., 2011) and conscientiousness (Israel et al., 2014) in childhood to later outcomes in health, wealth and well-being in adulthood. The persistent and self-amplifying features of such personality traits into adulthood are expressed in the conduct of adult roles and health behaviors and hence result in socioeconomic and health inequalities.

Economists, who are most closely identified with cognitively-centered human capital approaches, have turned to non-cognitive factors not only to strengthen the prediction of educational achievement and later related outcomes such as adult earnings, but to establish causal effects of these factors (a point to which I will return later in this essay). Heckman and Kautz (2012) link cognitive and personality factors to predict educational attainment. Cognition is measured as fluid and crystallized intelligence; the former is measured as novel problem-solving that implicates both inductive and deductive reasoning and the latter is the measured use
of general knowledge and accumulated skills developed over a lifetime. Non-cognitive factors are composed of the “big five” personality traits (and related factors) identified by personality theorists: 1-conscientiousness (the tendency to be organized, responsible and hardworking); 2-openness to experience (the tendency to be open to new aesthetic, cultural or intellectual experiences); 3-extraversion (sociability and the orientation towards the outer world of people and things rather than the inner world of subjective experience); 4-agreeableness (the tendency to be cooperative and unselfish); and 5-emotional stability/neuroticism (consistency in emotional reactions, frequency of mood changes, proneness to psychological distress).

The researchers draw data from longitudinal surveys and review experimental studies to establish that personality is a cause of achievement after controlling for cognitive abilities. One study compares high school dropouts, GED completers, and high school graduates. The key finding is that dropouts and GED completers are distinguishable from high school graduates as a result of personality differences more than cognitive differences. They also review random assignment experiments in preschool and kindergarten (e.g., the Perry Preschool Study and Project STAR) settings where children were assigned to treatment groups purposely to change personality traits or to provide classroom settings (smaller class sizes) in which more attention could be directed to individual student behaviors. Treatment effects were demonstrated to be causal of later higher performance levels.

In short, educational achievement and the cognitive and non-cognitive factors that propel it at the individual level appear to constitute a pervasive component of the aging process and life course inequality. Adult health and wealth inequalities appear to be robustly dependent on education. Ross and Mirowsky (2010) refer to it as a “fundamental cause” of aging, following the theory that Link and Phelan (1995) initially proposed regarding the robust dependence of
health on the pervasiveness and underlying generative force of a broader conception of socioeconomic inequality than educational attainment. The latter is a more ambitious theoretical effort to which I will turn now.

Fundamental Causes of Health Inequalities

The persistent associations between socioeconomic status (SES), however measured, and mortality, multiple diseases and overall health, respectively, motivated Link and Phelan (1995) to propose the theory of fundamental causes of health inequalities. That argued that SES was not a simple gradient easily represented by stratified access to a fixed set of resources (knowledge, money, power, prestige, social ties, etc.) that serve as mechanisms through which it operated. Rather, SES was a “basic cause” (following Lieberson, 1985) of health and mortality because of complex and dynamic shifts in the mechanisms linking the two via multiple pathways and their “flexible use” to protect against or to ameliorate poor health. This persistent association could not be explained away by conventional methods associated with robust dependence in which surface indicators of SES could be mediated or moderated. The underlying and pervasive effects of SES were reproduced over time because the mechanisms of their reproduction themselves varied and changed.

A test of the falsifiability of the basic cause is the situation in which causes and cures of fatal diseases are not known (Phelan, Link & Tehranifar, 2010). Under these circumstances, the resources can be deployed are not clear, hence SES should then differentiate outcomes much more weakly. Flexible resources can be deployed to highly preventable diseases (e.g. lung cancer) but less so to unpreventable diseases (e.g. brain cancer). As new knowledge is gained regarding formerly unpreventable diseases, new mechanisms for their prevention or amelioration become new resources that advantage those with access to them.
The theory is built on a narrative of social action similar to rational choice, but more specifically tied to identifying specific changing pathways to health outcomes. How does health come about as a function of SES? It comes about with the flexible deployment of unequally distributed resources that are specifically and temporally suited to diverse health outcomes. This implicates both individual and contextual resources, but deals more explicitly with individuals. Resources “must come from somewhere” (Phelan, Link & Tehranifar, 2010: S30). Meso- and macro-contexts include families, neighborhoods, workplaces, community networks, but any single indicator or set of indicators of context(s) can only partially represent complex and pervasive SES.

Luftey and Freese (2005) apply fundamental cause theory in an ethnographic study of two routine endocrinology clinics for diabetics to capture the dynamics of resource use and context. The two clinics treated patients from different socioeconomic backgrounds (Park Clinic—upper middle class; County Clinic—lower middle and lower class); they also differed organizationally in the access to treatment by physicians versus residents, continuity of care by the same practitioner, and resources for in-clinic diabetes education (all privileging Park Clinic patients). The analytical framework links these three in-clinic characteristics with the resources patients bring with them from the outside (financial limitations; occupational constraints; and social support networks) and patients’ dispositions in two areas: apparent motivations (the cost of compliance and the magnitude of lifestyle adjustment) and apparent cognitive abilities (interactional differences and capacities as practical achievements).

They observed the multiple ways that SES operates in these contexts through “regimen design” and “enacted regimen” that emerge in the interactions between patients and clinics. The control of long-term glucose level through patient self-management is the objective of diabetes
treatment. Yet, this regimen emerges quite differently on a day-to-day basis as organizational resources and practices interact with patient resources and dispositions. The investigators discerned mutual attribution processes in which patients and practitioners appeared to make judgments regarding each other’s capacities, intentions and behaviors. These interactions produced biases among practitioners and resistance among patients to regimen design, especially in the Counter Clinic context. The ultimate consequence was an SES-driven pattern of lower self-management success among poorer patients at both clinics.

The rich complexity portrayed in this project makes a strong case for the fundamental cause argument that SES is so pervasive across levels that its manifestations, taken singly, weakly represent and may obscure its action. SES is a generative process. Following Goldthorpe (2001), SES as a phenomenon exists at a deeper observational level than immediate data afford; it generates the surface causal effects observed, that by themselves under-represent the deeper phenomenon.

Cumulative Advantage/Disadvantage and Inequality

Life course theory throughout the 1970s and early 1980s was driven by an emphasis on inter-cohort variation and within-cohort homogeneity until within-cohort heterogeneity and variation across the life span became more and more evident, especially as longitudinal databases initiated in the 1960s and maturing over two decades were revealing more heterogeneity and inequality within cohorts with age. Indeed, the aging research community had conceptualized cohort aging instead as a process of convergence, as shared experiences of retirement and health decline leveled the playing field at older ages. By the 1990s the appearance of convergence was in part attributable to processes of selective mortality. As average life expectancy has continued
to increase and the mechanisms for its continuation are now better understood, cohorts can be differentiated by schedules of healthy life expectancy and degrees of chronic frailty.

Dale Dannefer (1987) aptly characterized the actual phenomenon as divergent following Robert K. Merton’s “Matthew Effect,” as a cumulative process of inter-individual divergence and increased inequality that resulted from the successive encounters of individual lives with stratifying institutional processes that set them on, and constrained them within, different life paths. The mechanisms driving the divergence were cumulative advantage and cumulative disadvantage (CAD) as path dependent trajectories in which earlier status and achievement has persistent influence on later status and achievement, not just as a result of individual motivation and capacity but as the outcome of institutional processes bearing on individual lives. Hence, for example, educational institutions select and sort students in ways that have lifelong consequences on average within a cohort that lead to inequalities in later years based on factors associated with the educational system which may go unobserved. The experimental studies reported by Heckman and Kautz (2012) summarized earlier reveal that school contexts (e.g. class sizes, curricular directed at learning skills) can differentiate students above and beyond their individual motivations and capacities.

The CAD model has fit the data well. The simple, falsifiable, generative features of CAD have motivated considerable research. The simplicity is reflected in its parsimonious prediction that the impact of prior events increases over time in stratified systems. Falsifiability stems from clear prediction of growing inequality with time that increasingly advantages early higher status, which invites competing hypotheses of reversals, cross-overs or compensatory mechanisms. The theory has been formalized to accommodate rigorous analyses of competing hypotheses (DiPrete
Generativity refers to CAD’s capacity to motivate new questions that extend or revise its boundaries.

Theory-building based on CAD is now focused on deciphering the tempo and critical phases of divergence and on competing hypotheses. Educational achievement and its timing is a robust determinant of later inequality, as we have reviewed. It is identified in the educational-health literature as causal and as a critical phase of the life course with enduring effects. However, childhood conditions are significant precursors to schooling experiences and achievements and apparently exert persistent direct effects, in their own right, on educational attainment and some later life outcomes, particularly in health. The literature on adverse socioeconomic and health conditions in childhood and their enduring effects is vast and cannot be reviewed here. Both prospective and retrospective data have been examined—and while they face specific limitations across datasets, including shorter views of the life course in prospective studies starting in childhood or infancy and errors in memory in retrospective designs—they arrive generally at similar results regarding the robust dependence of some adult health conditions and risks for poverty on adversity in childhood.

The (probably multiple) mechanisms by which these occur are not firmly established. Some are rooted in bio(neuro)developmental factors influenced by in utero and postnatal environments in the family and broader community (e.g. Barker, 1992; Gruenewald, 2013) and some in diverse psychosocial and behavioral responses to these stress-inducing conditions that condition later reactions to stress (e.g. Moffitt et al., 2011). The questions about the tempo of aging now focus on whether the effects of early life conditions reflect “sensitive” or “critical” periods that imprint the bio-psycho-social developmental process in ways that cannot be mediated or reversed with later more enriching conditions or whether these early life conditions
establish an initial disadvantage when facing “chains of risk” that sets path dependent course of disadvantage which may even amplify or accentuate the effects of early conditions with time and under new challenging conditions (e.g. dramatic health decline; unemployment; family dissolution). The patterns of cumulative disadvantage and cumulative advantage are also not symmetrical: the former are probably more path dependent and the latter more stochastic (O’Rand, 2009). By this is meant that initial disadvantage has a significant gravitational force on later life outcomes, while initial advantage does not guarantee the successful navigation through later life shocks and turbulence but affords the resources to confront them although less predictably.

Studies with competing hypotheses are adding to the development of the CAD model in several ways. An exemplary study in the theory-building process uses the MIDUS study to identify psychosocial compensatory mechanisms that off-set the long term effects of early adversity (Schafer, Ferraro & Mustillo 2011). The question is whether “human agency” in the form of optimism or “buoyant expectations” can overcome early adversity’s effects on overall reflections by individuals on their past and future lives. Their results suggest that the experiences of the past constrain expectations for the future, but that human agency is fruitful ground for continuing to test CAD propositions.

Ferraro has moved further on this agenda with a recent effort at theory-building in aging by linking the CAD model to other processes associated with aging, including human agency. He is also linking CAD to the stress process model widely studied in medical sociology. Ferraro proposes an axiomatic theory of cumulative inequality (CI) to explain how inequality gets under the skin (Ferraro & Shippee, 2009). The linkage of the stress process to the CAD stratification process directly situates health as formative in aging. The axiomatic framework draws from the
extensive literature associated with CAD and the empirical generalizations that have emerged from it and links it to the stress model is medical sociology. The five axioms seek to establish the macro-, meso, and micro-level foundations for 19 macro-, meso- and micro-level propositions (Ferraro & Shippee, 2009: 337), which are probably not intended to be exhaustive but illustrative. The complement of axioms and propositions are founded on an even deeper level model of the process: social change can flow in two directions. The micro-meso-macro dynamics of aging encompass demographic processes associated with the aggregation of the day-to-day actions of individuals facing the macro- and mezzo-level conditions of their lives—aggregate individual actions stemming from human agency may produce macro-level changes.

Cumulative inequality (CI) is a program to integrate the long, broad and deep components of the aging process at all levels of observation. It draws on empirical generalizations from the cumulative advantage/disadvantage framework that has developed for two decades to establish fundamental axioms of aging inequality. It then generates propositions from this axiomatic framework with relevance to aging beyond health or medical sociology concerns. It is not possible here to provide specific comments on the propositional derivations of the axioms, especially as this theory is probably still in development as this essay is being composed. However, a brief look at the axioms reflects the wide reach of the CI theory. The five axioms are listed below.

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

Axiom II. Disadvantage increases exposure to risk, but advantage increases exposure to opportunity.
Axiom III. Life course trajectories are shaped by the accumulation of risk, available resources, and human agency.

Axiom IV. The perception of life trajectories influences subsequent trajectories.

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

These axioms apply to aging inequality across the spectrum of the human life course, and are not relevant only to the role of health per se. Family development (including intergenerational processes), work career trajectories and labor market processes, patterns of identity development, and patterns of attitude formation and change, among other sociologically interesting processes, can be investigated using this foundation for question generation. However, it is nevertheless clear that questions of health over the entire life course have colonized aging theory, especially those associated with inequality.

Integrating Biological and Social Factors in Aging

Perhaps the most rapidly developing area of aging research is the linkage between the biological and the social. The patterns of inequality in health and mortality traceable to the “health career” and its SES origins have contributed to the emergent importance of monitoring the “pace of aging,” or the rate at which underlying and manifest health conditions shorten the length of life, diminish healthy life expectancies, and constrain the quality of life with age. Indeed, it can be argued that aging research has changed most dramatically in the direction of health because of the growing sophistication of biological data collection and analysis.
Arguably, the decades of research on stress and health, which focused increasingly on how deleterious social experiences like those associated with lower social status “get under the skin” (Ferraro & Shippee, 2009), ushered in this line of thinking. The stress process tradition became interested in the role of chronic or acute social stress on “allostatic load” (or multiple dysregulation measured by steroid hormones) or “wear and tear” and the effect of allostatic load, in turn, on health and aging. The idea took fire by the 2000s when more biomarker data were being collected. Although reviews of the literature in this area suggest that the biosocial link is still not well established (Juster et al., 2010; Weinstein et al., 2013), the now vast collection of these data promises continued research with perhaps improved methods of analysis to establish causal relationships.

More recently, biosocial research is getting “deeper under the skin” with social genomics as the newest area. This program investigates how social experience regulates gene activity by studying gene transcription or the rate at which DNA is transcribed into messenger RNA which in turn produces proteins that are integral to biological processes like those associated with stress (Gruenevald, 2013) and inflammatory responses (Finch & Crimmins, 2006; see Shanahan, 2013 for a good summary of this area). Genetic transcription is an adaptation to changing circumstances in the organism. The theory proposes that levels of transcription differentially accumulate from early childhood throughout the life course leading to adult health disparities and differential rates of mortality. Early adversity can establish a durable program of the stress response system creating a “defensive phenotype.”

The genetics of health and aging is not a new interest. However, the new social genomics has moved beyond twin-sibling studies and target genes to genome-wide studies (GWAS) of DNA sequence variations (mutations) called SNPs (single nucleotide polymorphisms) that can
provide genetic fingerprints to detect disease susceptibility (e.g. the apolipoprotein E gene (APOE) mutation is associated with a higher risk for Alzheimer’s disease) and behavioral vulnerabilities. The collection, data storage and computational demands of GWAS research are considerable. The logistics of GWAS data collection in national databases are daunting. These are accentuated by the requirements of longitudinal designs, especially those motivated by life course theories like CAD and the childhood adversity hypothesis of long-term phenotypic vulnerability. Shanahan (2013) outlines challenges of these data for theory-building on this question, especially in adjudicating between the “sensitive period” and the “critical period” models of the childhood origins of adult health outcomes. The “sensitive period” hypothesis argues that biological systems are subject to change at specific points of development, while the “critical period” hypothesis argues that it is the only time biological systems are subject to change. Clearly, fine-grained longitudinal study of childhood with multiple follow-ups into adulthood are a major challenge.

Traversing the biological-social divide is the most ambitious agenda for aging research no matter what the hypothesis of interest is. Longitudinal design, accurate repeated measurements of relevant social and biological variables, and the estimation of cause-and-effect relationships are three major tasks, each requiring interdisciplinary collaboration and considerable resources over time. The interaction of research and theory over two decades, however, has brought aging inequality research to this point.

Cause and Effect in Aging Research

Goldthorpe (2001) has identified a third approach to causation in the social sciences besides robust dependence and generative processes. He refers to it as consequential manipulation (CM), identified primarily with experimental and quasi-experimental designs but
also with econometric modeling that is designed to unmask spurious correlations and identify true causal effects, using techniques that are not yet widespread in the robust dependence literature. Often “causal” variables in aging research, like education, are themselves endogenous (i.e. dependent on prior variables that might alter the observed effects of education on an outcome such as health if they are included) or endogenously interdependent (education↔health or SES↔health). Also, sample selection decisions can bias the estimates in an analysis, as in the case of using samples restricted to older populations (say above age 65) that select out younger individuals who have not survived to the age of 65 or select only workers to study wage differences and thus miss non-workers whose characteristics may be relevant to wage differences but are missed in the analysis. Finally, advocates of consequential manipulation propose that besides the major problems of endogeneity and sample selectivity in non-experimental analyses, the real issue is the definition of causation itself. In this vein, causes should be treated as “treatments” that must be manipulated. CM in this sense requires a more counterfactual approach: what would happen to Y if exposed to treatment X or if not exposed to treatment X? This approach to causation requires random assignment to treatment or control groups in a manner not affected by the assignment process and with the assumption that all the theoretically relevant variables are included in the analysis.

CM is considered by some social scientists as the gold standard of causation (e.g. Angrist & Pischke, 2009). Econometric approaches to deal with these problems above have spread to sociology and demography with the application of such techniques as sample selection models and instrumental variables (exogenous variables with unique effects on endogenous variables) to the kinds of survey databases that predominate in aging research (see Bollen, 2012; Elwert & Winship, 2014). These applications have introduced new rigor to statistical analyses that seek to
establish causal relationships in the aging process, although they face challenges in secondary analyses that limit the identification of strong instrumental variables or lead to the overuse of instrumental variables leading to over-identification.

However, experimental designs *per se* have not spread as widely outside of medical and epidemiological research and economics of aging for practical and ethical reasons (Goldthorpe, 2001). The practical concerns about experimental designs are numerous and include such limitations as (1) their potential unrepresentedness, especially when clinical populations or volunteer samples are the source of data; (2) the artificial environments that can be associated with the design; and (3) the potentially unobserved effects of the “treatments” on other variables. Another nontrivial concern about the CM approach to causality is its explicit rejection of intrinsic variables or attributes as treatments because they cannot be manipulated: no causation without manipulation (Goldthorpe, 2001). This means that such variables as sex and race cannot be causes. Sociologists especially express concern about this requirement, although they are quick to admit that these variables always require careful theoretical treatment and serve usually as moderating factors between X and Y, whose effects are themselves embedded in social contexts that imbue them with meaning.

However, CM can reveal sociologically causes that can be treatments. Here I refer you back to the Heckman and Kautz (2012) article on soft skills summarized earlier. A key argument in this study was that school environments matter. In the education and health literature, which is dominant in aging inequality research, school characteristics and related curricular and environmental variables are usually absent as potentially causal variables. Theory-building in this area will benefit going forward by creative approaches to linking individual characteristics
with critical and formative social environments using diverse methods that might include experiments.

Caveat: Global Risks and Aging Inequality

Missing from this review so far is a consideration of theory-building at higher levels of analysis, such as globalization, nation-state, community institutions and family. The macrosocial dynamics of aging inequality implicate demography, economy and policy at a global level. I have addressed some of these issues at some length in another essay composed nearly simultaneously with this one (O’Rand and Bostic, 2015). This essay points to the asynchronies among population aging, migration, global market processes (especially labor market restructuring and financialization), and the retrenchment of national welfare and labor policies that are contributing to growing inequalities between and within aging cohorts across countries. These asynchronies are unraveling what was putatively a more age-related and coherent life course formed by institutional arrangements that characterized the 20th century and they are introducing pervasive new uncertainties and social stresses across age groups and across societies.

A compelling argument has been made that we have entered a post-national era (Beckfield et al., 2013) in which most institutional arrangements that bear critically on the life course well-being of families and individuals no longer fall within the boundaries of the nation-state. And if they do fall within these boundaries, as in the case of aging and health-care policies, they serve to exacerbate social inequalities rather than to ameliorate them as a consequence of global processes. The ascendance of global institutions, especially the restructuring and financialization of markets, has superceded national policies and introduced new risks to labor security, health, retirement and the private accumulation of resources to protect against life
course risks. This occurs through stratified systems of education, income-maintenance, health-care services, and more.

The relevance of these macro-trends to the meso- and micro-level agenda of the long-broad-deep program reviewed here is that the forces of inequality are pervasive. If inequality gets under the skin, then the prospect for healthy future populations is problematic. Hence, understanding the processes by which inequality gets under the skin is appropriate a core element of aging inequality.

Conclusions

Theories develop as they are applied to data and the methods used to collect and analyze them. Methods develop as well, both in response to theoretical demands and in their own right, and thereby serve to develop theory further. Theory development of aging inequality has followed this pattern for over a half century. Aging research demands longitudinal data and statistical methods suited to these data to estimate causal relationships over time. Data and methods, in turn, make demands of theory to specify causal relationships in ways that they can be falsified and can generate new questions.

Over the last two decades theory-building in aging inequality has focused on defining the role of health in the aging process. Arguably, health is now the core metric of aging; the diverse and complex patterns of disease, disability and mortality with age have become the central problem for aging researchers, especially those concerned with social inequality and its pervasive and enduring effects. Major theoretical programs have emerged over two decades to address the seemingly inextricable relationships among health, aging and social inequality across the lifespan. Four of these programs were considered here: the education-health research program focused on the explanation of the robust relationship between years of schooling and
later health; the fundamental cause program that argues that SES is an underlying, pervasive phenomenon that propels the aging process via multiple mechanisms over time that cannot be fully captured by surface indicators alone; the cumulative advantage-cumulative disadvantage (CAD) theory that elevated “time” itself through processes of accumulation that generate inequality; and cumulative inequality (CI) that is attempting to expand the CAD project in a broader theory of aging inequality that incorporates health (using the stress process model) and human agency.

The most recent arrival to this active research area is biosocial research (including social genomics) that has enriched the measurement of health and is raising new questions about causal relationships that are forming a new agenda for the future. One example of this is the potential contribution of biosocial research to the understanding of the origins of health inequalities in early life and the subsequent trajectories of health (and the pace of aging) as individuals encounter successive but unequal social risks and resources over their lives. The challenges to this agenda are theoretical and practical, and causal relationships are not well established, but the promise of this research has made it perhaps the most rapidly developing subarea of aging inequality.

Causal relationships defined by theory are the objectives of aging science. And, as a developing research program, aging inequality has followed three different approaches to causation. Robust dependence is the predominant approach concerned with predictive power, the elimination of spurious relationships and the partialling of focal relationships. The education-health, CAD, and CI literatures overwhelmingly apply diverse statistical methods that fall into the robust dependence category. The fundamental cause project employs robust dependence but conceptualizes the basic underlying causal impact of SES as a multifaceted
generative process that is dynamic over time and difficult to capture with a single variable or small set of repeated variables. This is a strong sociological argument that is moving beyond its epidemiological origins to more structural interests. Finally, consequential manipulation is the experimental approach focused on counterfactual approaches to causality that make more rigorous demands on design that simple regression-based statistics dominant in robust dependence. While practical and ethical concerns constrain the application of experimental designs for some aging inequality research question, efforts in this direction will also contribute to theory development.

The causal processes that concern these theoretical programs do not address the broader macro-level processes that also drive the independent variable(s) of interest, inequality. The aging process and patterns of inequality are being shaped by global forces that affect day-to-day lives. These forces are introducing new risks for individuals and families that bear upon health across the life course through the reorganization or retrenchment of social institutions that once had greater equalizing effects for aging cohorts. The 21st century poses new risks in a post-industrial, post-national era of institution-building driven by a market ideology and a financial logic.

References


