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Integrating Theory, Methods, and Evidence

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By

Kandauda A.S. Wickrama,
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Cambridge Scholars Publishing

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PREFACE

A substantial body of research consistently documents the lasting link between early socioeconomic adversity (ESA)—experienced during childhood or early adolescence and increased physical health risks in later adolescence (ages 13–19), the transition to adulthood (ages 19–25), and young adulthood (ages 25–40) (Costello et al., 2003; Danese, & McEwen, 2012; Wickrama et al., 2017, 2022). This connection significantly contributes to maintaining health disparities throughout the lifespan (e.g., Packard, et al., 2011; Nurius et al., 2015).

The relationship between ESA and physical health has been studied across various research fields and theoretical viewpoints (e.g.; Brody et al., 2015; McEwen & Gianaros, 2010; Miller & Cole, 2012; Raposa et al., 2014; Conger et al., 2010). Although these studies have provided valuable insights, the lack of interdisciplinary integration has limited our understanding of how ESA influences health-risk trajectories and health outcomes throughout the first half of life.

This book presents an integrative theoretical framework that delineates multiple concurrent mediating pathways linking early socioeconomic adversity (ESA) to physical health across the first half of the life course. The framework encompasses neurobiological, physiological, psychological, and behavioral resource pathways over this period, along with potential moderators of these associations, acknowledging relationships among these pathways. In particular, this book focuses on neurobiological development and the mediating role of the brain in the association between adversity and health.

It draws on empirical and theoretical evidence, primarily from life course and developmental perspectives, to support the proposed integrative model. Emphasizing a long-term perspective, the framework highlights continuity in developmental processes and identifies adolescence as a particularly vulnerable period for the impacts of ESA. Such integrated life course approaches offer powerful tools for understanding how health risk trajectories form and evolve, ultimately contributing to health inequalities

from childhood through early adulthood (Moody-Ayers et al., 2007; Stowe & Cooney, 2015).

The development of an integrative framework to inform research on the health and well-being impacts of FEA is timely given that longitudinal datasets with dyadic data are increasingly available capturing a wide array of multi-dimensional constructs (e.g., structural and environmental adversities, neurobiological development, mental and physical health outcomes, biomarkers, and genes).

This integrative framework offers a foundation for developing comprehensive, testable longitudinal models to investigate the pathways from ESA to health outcomes in adolescence and young adulthood. The book also outlines specific directions for future research and discusses appropriate analytical and methodological approaches.

Consequently, to advance this research, this book provides:

- A brief conceptualization with figures for each pathway connecting ESA and subsequent physical health outcomes.
- Illustrative empirical examples from existing studies (particularly research from the Adolescent Health Study (Addhealth) and Iowa Transition Project as described in Appendix 1).
- Demonstrations of results' interpretation for both statistical and theoretical concepts.

Intended Audience

This book is designed to serve as a supplementary textbook, complementing other available texts on social and family epidemiology, for graduate-level courses or self-study purposes among graduate students, instructors, and researchers across various disciplines. This book is suited for a variety of courses and fields of study interested in how contextual factors, particularly family structural and environmental factors, contribute to young adults' physical health outcomes, such as social epidemiology, adolescent development, social determinants of health, public health, family and health, and medical sociology or family counselling, and longitudinal data analysis. Students who have completed basic statistics courses have the knowledge and skills necessary to use this book.

Content

This book is divided into three parts to describe a single, integrative framework that summarizes the various mechanisms responsible for the association between family economic adversity (FEA) and young adults' physical health, acknowledging the interdependence between these mechanisms.

Part One comprises Chapters 1 and 2, which introduce the theoretical perspectives that support the proposed integrative framework connecting FEA to the physical health of young adults. Chapter 2 summarizes the conceptualization and measurement of ESA and physical health outcomes.

Part two comprises Chapters 3 through 9, which collectively discuss each pathway connecting ESAA to young adults' physical health, considering both mediating and moderating processes. These pathways include:

- Neurobiological development (Chapter 3),
- a physiological response pathway (Chapter 4),
- a psychological response pathway (Chapter 5),
- a behavioral response pathway (Chapter 6),
- a psychosocial resource pathway (Chapter 7),
- Moderation by psychosocial resources (8)
- Moderation by individual characteristics (Chapter 9) and
- Chapter 10, which illustrates methodological and analytical advances that can be used for quantitative research in this area.

Acknowledgements

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The studies cited in this book have extensively used data from Add Health, a project directed by Kathleen Mullan Harris and designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University North Carolina at Chapel Hill, and funded by grant P01- HD31921 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development, with cooperative funding from 23 other federal agencies and foundations. Information on how to obtain the Add Health data files is available on the Add Health website (http://www.cpc.unc.edu/ addhealth).

The studies cited in this book have also used data from the Iowa Transition Project. Over the years of this project, support for this research has come from multiple sources, including the National Institute of Mental Health (MH00567, MH19734, MH43270, MH48165, MH51361), the National Institute on Drug Abuse (DA05347), the Bureau of Maternal and Child Health (MCJ-109572), the National Institute on Aging (R01AG043599-01A1), he MacArthur Foundation Research Network on Successful Adolescent Development among Youth in High-Risk Settings, the Iowa Agriculture and Home Economics Experiment Station (Project No. 3320), and the Spencer Foundation.

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FOREWORD

I count myself fortunate to have entered the field of prevention science and family studies at a time when the foundational work synthesized in this volume was beginning to reshape how we think about health disparities. As a graduate student in the late 1990s, I was assigned to read papers from the Iowa Youth and Families Project initiated and lead by Dr. Rand Conger. Those papers changed how I understood the connections between family economic hardship, family processes, and youth development. They provided both theoretical clarity and empirical rigor that continues to shape my research program today. This book represents the culmination of nearly three decades of that scholarship—work that has fundamentally transformed our understanding of how socioeconomic adversity becomes embedded in biology and behavior across the life course.

The Iowa studies began during the farm crisis of the 1980s, when agricultural communities across the Midwest faced devastating economic collapse. Dr. Kandauda Wickrama and his colleagues, documented how macroeconomic forces affected family systems to shape individual trajectories across decades. The family stress model that emerged demonstrated how economic pressures create subjective strain, disrupt parents' emotional well-being, increase marital conflict, and undermine effective parenting. Crucially, the team showed that these effects are not inevitable—they are mediated through family relationships and parenting practices, suggesting that strengthening family processes could buffer children from poverty's toxic effects. These insights shaped an entire generation of prevention research, including my own.

What distinguishes this volume is how it synthesizes and extends those foundational insights through a comprehensive new framework. The Early Socioeconomic Adversity and Young Adult Health across the Life Course (ESAYHLT) model traces multiple interconnected pathways through which early adversity shapes health across the first half of life: neurobiological, physiological, psychological, behavioral, and resource-based. The framework distinguishes between structural adversity and environmental adversity, addresses sensitive developmental periods, and provides methodological tools for testing these complex processes empirically. No other framework in the literature achieves this level of integration across

biological, psychological, and social levels of analysis while maintaining coherence with life course principles.

The book's arguments gain force from their grounding in decades of empirical work. These are not abstract propositions but pathways documented and tested across multiple samples, time periods, and contexts. Drawing primarily on the Iowa Youth and Families Project and the National Longitudinal Study of Adolescent to Adult Health (Add Health), the authors demonstrate how early family economic pressure predicts trajectories of adolescent stress exposure, how these stress trajectories relate to patterns of psychological adjustment and physiological dysregulation, how risk pathways interact and compound across adolescence, and how they ultimately crystallize into disparities in chronic disease risk by young adulthood. The empirical grounding lends credibility that purely conceptual models cannot achieve.

Equally important are the book's contributions to understanding health equity. The authors examine how race and ethnicity, gender, and community context moderate pathways from adversity to health, documenting both vulnerability and resilience. Their finding that Black youth demonstrate greater resilience to some stressors during adolescence but face intensified impacts in young adulthood challenges simplistic narratives about racial health disparities. Of particular interest, their documentation of "skin-deep resilience"—where educational and socioeconomic success among marginalized youth comes at physiological cost—raises profound questions about the hidden tolls of striving against structural barriers. These findings carry significant implications for both research priorities and policy interventions aimed at reducing health inequalities.

The methodological chapters present cutting-edge techniques—not as technical exercises, but as analytic strategies that embody the theoretical commitment to understanding how individual lives evolve through sequential, cumulative, and interactive processes across time.

The clear exposition, coupled with empirical demonstrations using real data, will make these advanced techniques accessible to new researchers. I plan to use these chapters in my own graduate seminars, as I have long relied on Wickrama's earlier methodological writings in teaching longitudinal analysis.

Looking back at the trajectory of this research program—from studies of farm families in crisis to comprehensive models of neurobiological embedding—I am struck by the consistency of vision. Wickrama and his colleagues remained focused on a fundamental question: How do social and economic circumstances shape human development and health across the life course? They progressively deepened our understanding of mechanisms, first attending to family processes, then to developmental timing and trajectories, then to physiological processes and biomarkers, and now to neurobiological development and brain function. Each phase built upon prior insights while maintaining theoretical coherence. This cumulative, integrative approach exemplifies the most productive form of programmatic scholarship, and it reflects not just Wickrama's vision but decades of collaboration with students and colleagues who have contributed to this research program. Having worked with him over the years, I can attest that his scholarly contributions are matched by his generosity as a mentor.

This volume should be required reading for graduate students and faculty in human development, family science, sociology, public health, developmental psychology, and social epidemiology. It provides both a comprehensive theoretical framework and practical analytical tools for investigating how health disparities develop. In an era when such disparities threaten the fabric of society, the book arrives at a critical moment. By demonstrating how socioeconomic adversity becomes biologically embedded across development, it makes clear that addressing health inequalities requires comprehensive efforts to support families, strengthen communities, and reduce childhood poverty. The science presented here demands a public health and social policy response commensurate with the scale of the problem. I commend this volume to all who are concerned with adolescent health, family well-being, and health equity. It represents the best of what our field can offer.

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CHAPTER 1

INTRODUCTION

Previous research extensively documents the link between early socioeconomic adversity (ESA) and a wide array of physical and mental health outcomes across a person's life. These outcomes include depression, anxiety, substance abuse, cardiovascular disease, cancer, diabetes, respiratory conditions, chronic pain, gastrointestinal and metabolic disorders, neurological and musculoskeletal issues, and premature death (Conger et al., 2010; Danese & McEwen, 2012; Danese et al., 2009; Bellis et al., 2014; Lee et al., 2018; McLaughlin et al., 2014; Plakard et al., 2011). Studies also indicate that childhood socioeconomic adversity is linked to various biomarkers associated with cardiometabolic diseases, cancer, thyroid problems, arthritis, and psychobehavioral issues in adults (Alley et al., 2025). Long-term associations tend to be especially strong—often more consistent than those seen at the same time—particularly for specific physical health issues like obesity in early adulthood (McLarn, 2007). However, the mechanisms underlying these links remain poorly understood. The primary goal of this book is to present a comprehensive life course developmental framework that explains how early socioeconomic adversity influences physical health during adolescence and early adulthood. This framework combines life course and developmental perspectives, supported by several mid-level theories. It includes concepts such as multiple socioeconomic risks, cumulative risk processes, lifelong influences, age-specific stress exposure, sensitive periods, neurological development, related risk trajectories and continuities, and cumulative physiological dysregulation over time.

This integrated framework offers a comprehensive understanding of how early socioeconomic adversity (ESA—also called early socioeconomic risk) influences physical health outcomes in the first half of life. It also explores the different pathways through which this relationship may develop. The framework aims to lay a foundation for future research that develops and tests long-term models examining the connections between ESA, adolescent

stressors, health risks, and physical health across adolescence and young adulthood.

Instead of providing a detailed review of all studies related to each mechanistic pathway, this work offers a broad synthesis of key findings, selectively citing empirical research to support the proposed pathways from ESA to physical health outcomes in young adulthood. Therefore, this work should be viewed as a focused and illustrative overview that underpins the proposed integrative framework, rather than a systematic review.

Selection of Studies

The empirical studies included in this work mainly rely on two major panel studies: the Iowa Youth and Families Project / Iowa Transition Project (YTP) (Principal Investigator: Glen H. Conger; 1986-2008, N=610) and the National Longitudinal Study of Adolescent to Adult Health (Add Health) (Principal Investigator: Kathleen Mullan Harris; 1995–2016; N=20,000). Both YTP and Add Health provide extensive longitudinal data tracking adolescents into adulthood over several decades. These datasets offer rich, prospective, and longitudinal information, including biopsychosocial and behavioral data from youth and their parents (see Appendix 1 for additional details). Collectively, these studies have generated over 8,000 peer-reviewed journal articles.

Findings from YTP and Add Health are supported by evidence from other relevant empirical studies, reinforcing the proposed pathways and associations. These additional articles were identified through thorough searches of key databases such as APA PsycNet and Google Scholar, using important terms related to the proposed pathways. Search terms included: “early socioeconomic adversity,” “socioeconomic risk,” “family economic hardship,” “parental practices,” “family socialization,” “community adversity,” “brain development,” “puberty,” “adolescent maladjustment,” “precocious development,” “delinquency,” “adolescent stressors,” “adolescent transition,” “mental health,” “physical health,” “polygenic scores,” “biomarkers,” and “allostatic load.” The focus was on articles published after 2000; however, foundational and highly cited studies, including theoretical works published before 2000, were also included when relevant. To ensure cultural and contextual consistency, only studies conducted in European and North American settings were considered, as the nature of the associations being examined may vary across different cultural, geographical, and historical contexts.

Theoretical Principles Integrated in The Organizing Framework

Life course perspective and midlevel theories

The proposed framework integrates life course and developmental perspectives to explain how early socioeconomic adversity (ESA) continues to affect physical health throughout the first half of life. The life course perspective highlights the cumulative impacts driven by early socioeconomic risks, interacting with individual traits (human agency), and their influence on health risk patterns over time (Elder et al., 2003; Elder & Giele, 2009). Meanwhile, the developmental perspective examines how structural socioeconomic factors, early life environments (such as family), and stressors specific to different life stages—particularly during sensitive periods—collectively shape ongoing health risks (Greyson & Holmbeck, 2002). To further support the framework, several mid-level theories are incorporated, including:

- Cumulative advantage/disadvantage theory (Dannefer, 2003),
- The stress process theory (Pearlin et al., 2005),
- Family stress models (Conger et al., 1994),
- Delinquency theory (Gottfredson & Hirschi, 1990),
- Attachment theory.

The principles of the life course perspective, as they relate to health development in the first half of life, are illustrated in Figure 1.1. The figure demonstrates how early exposure to multiple sources of socioeconomic adversity—such as low socioeconomic status (SES) and negative family or community environments—interacts with individual traits and larger structural factors to influence stress patterns, health risk pathways (or continuities), and long-term health outcomes. Additionally, the life course perspective argues that experiences in later life stages (e.g., early adulthood) are shaped by earlier life experiences (e.g., childhood and adolescence). The term "life course" refers to a lifelong developmental process. This means that health development does not occur in just one life stage; instead, it begins in infancy and progresses over time, leading to diversity—and, in some cases, inequality—in physical health outcomes in adulthood. For example, early socioeconomic adversity, usually rooted in childhood, influences physical and mental health paths that continue into adulthood (Wickrama et al., 2013). Therefore, a long-term, process-focused approach is vital for understanding adult health. As a result, life is viewed as a series

of experiences or circumstances that begin with early challenges at individual, relational, and contextual levels. These chains create pathways, often called intraindividual trajectories or autoregressive continuities over time (Elder & Giele, 2009).

Intraindividual trajectories illustrate how a person's experiences—such as economic hardship, exposure to stress, or health risks—change over time. These encompass biological, psychological, and socio-behavioral health risk processes, also known as stress trajectories and biopsychosocial health risk trajectories. For instance, depressive symptoms during adolescence may increase, decrease, or remain stable over time (Wickrama et al., 2016).

Autoregressive continuities, also known as rank-order continuities, demonstrate the persistence of individuals' relative positions over time in terms of risk exposure or health outcomes. In this context, prior exposure to ESA may result in consistent differences among individuals in risk levels, even if intraindividual change occurs. Depending on the research focus, these pathways linking ESA and adult health can be seen either as intraindividual trajectories or autoregressive continuities.

These ideas highlight that cross-sectional data only offer a snapshot of long-term trajectories. Gaining a more accurate understanding of development and health requires a dynamic view that considers how stress and health patterns evolve.

The interconnectedness of stress and risk over time is illustrated through concepts such as triggering, clustering of socioeconomic risks, and risk proliferation. These ideas suggest that early socioeconomic stressors or risks can increase the likelihood of facing more difficulties later in life, possibly through mechanisms such as social selection. For example, early economic hardships or depressive symptoms might raise the chances of experiencing more severe adversities or mental health issues over time. Structural socioeconomic factors play a key role by enabling or limiting access to resources and opportunities, thereby reinforcing or reducing disadvantages. This process aligns with the cumulative advantage/disadvantage theory (Dannefer, 2003; DiPrete & Eirich, 2006), which proposes that the influence of early socioeconomic conditions on health and well-being tends to grow over time, widening disparities between individuals. This theory extends the life course perspective by explaining how health inequalities develop and deepen throughout life, similar to the saying "the rich get richer and the poor get poorer." In this framework, individuals from resource-rich backgrounds tend to enjoy increasingly

better health outcomes, while those from disadvantaged origins face growing challenges. For instance, early depressive symptoms can predict more severe or longer-lasting depression in adulthood, showing lagged effects or continuity. These trajectories often follow an exponential pattern, resulting from the interaction between risk exposure and time, thereby heightening health disparities. As a result, risk trajectories may take a "fan-shaped" pattern—though some individuals may experience recovery trajectories, with symptoms decreasing more quickly after episodes, emphasizing resilience and the potential for positive change despite early adversity. Autoregressive continuities involve consistent rank-order risks over time, indicating that a person's relative standing regarding health risk remains stable even if their risk levels fluctuate. Such patterns, known as risk continuities, suggest that past socioeconomic adversity can have a lasting influence on health risks, thereby maintaining differences among individuals throughout their lives. Depending on the research focus, biopsychosocial pathways linking early socioeconomic adversity (ESA) to adult health can be viewed as either intra-individual trajectories (reflecting change within a person over time) or autoregressive continuities (highlighting stability in individual rankings). Both perspectives recognize that life stages are interconnected, and change is part of a lifelong, ongoing process rather than isolated, disconnected phases.

As shown in Figure 1.1, the life course perspective suggests that early exposure to multiple socioeconomic risks—both structural (such as socioeconomic status [SES]) and contextual/environmental (such as family environment)—can influence the development of various health risks, including stressful life events, psychological challenges, socio-behavioral problems, and physiological dysregulation. Prolonged exposure to the same socioeconomic risks over time (regarding duration or chronicity) may lead to the accumulation of physiological stress responses, a process called weathering (Geronimus et al., 2006). This physiological wear and tear can present as disorganization, often indicated by higher biomarker levels.

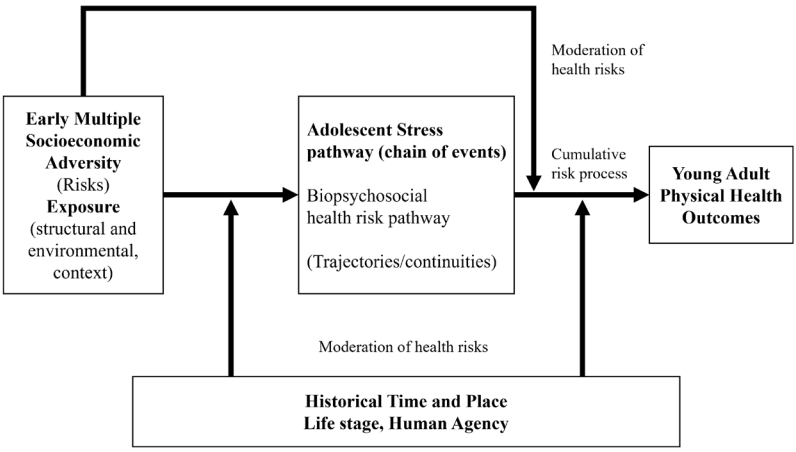

Figure 1.1. Life Course Theoretical Perspective for Youth Health Development

Aligned with the idea of cumulative advantage and disadvantage, health risks—similar to socioeconomic risks—can build up over a person's lifetime through a compounding process, eventually causing disparities in physical health outcomes in adulthood. Several mechanisms may contribute to this cumulative health-risk process.

a. Multiplication of Health Risks: Early health risks can trigger and multiply with additional risks. For example, high BMI might lead to type 2 diabetes and a sedentary lifestyle, with each risk independently and cumulatively worsening physical health outcomes (Wickrama et al., 2017).
b. Inter-domain Health-risk Proliferation: Risks in one area can influence the development of risks in other areas. For example, stressful life events during youth may predict trajectories of depressive symptoms and BMI, creating interconnected risk pathways (Wickrama et al., 2021). This shows how early stress can become embedded, impacting the development of later physical health risks.
c. Parallel Trajectories of Health Risks: Health risks can develop concurrently over the course of life. Studies indicate that trajectories of depressive symptoms are often connected with trajectories of BMI and educational failure, showing linked developmental paths (Wickrama et al., 2014; 2022). The idea of parallel trajectories

explains this simultaneous development of risk areas within individuals (Wickrama et al., 2016).

Another key aspect of the life course perspective is the concept of linked lives (Elder et al., 2009), which emphasizes the interdependence of people in close relationships, such as between parents and children or spouses. These interconnected risk trajectories can reinforce each other, leading to cumulative health risks across generations. Just as daily routines (like meals, homework, and chores) are shared within families, so are their life and risk pathways.

Shared experiences of socioeconomic adversity (ESA) within families can jointly influence the health trajectories of both parents and children. For example, research has shown that adolescents' depressive symptoms are linked to their parents' depressive symptoms, mainly because of shared family environments, which may serve as a broader family construct (Lee et al., 2024; Papp, 2012). These connections are not limited to psychological outcomes; similar patterns are also seen in physical health risks, such as unhealthy behaviors and lifestyles that are shared between parents and children (Wickrama et al., 1999). Such mutual influences often stem from the presence—or absence—of social, emotional, and economic resources within the family.

The life course framework also shows that people's lives are shaped by the historical time and place in which they live. These factors influence health over time through opportunities and barriers created by larger social and historical contexts. Differences between generations in their experiences of major events—such as the Great Depression, 9/11, the farm crisis of the 1980s, the 1990s recession, or the COVID-19 pandemic—can lead to distinct life and health paths. For example, teenagers living in rural Midwest during the late 1980s faced the farm crisis, which significantly impacted family resources and well-being. Those who experienced this event may follow different social and behavioral paths than peers from unaffected areas or other periods (Conger et al., 1994; Lorenz et al., 2000), potentially leading to varied health outcomes in adulthood.

Another key principle of the life course perspective is human agency—the ability of people to make choices and influence their own lives. While structural constraints shape life paths, individuals are not passive; they actively influence their trajectories. Personal traits—such as self-esteem, mastery, self-regulation, or, conversely, neuroticism and hostility—can promote resilience or increase vulnerability to adversity. These traits affect

decisions like educational achievement, employment, and relationships, which can impact health trajectories over time (Lee et al., 2018; Settersten, 20037).

Another key concept of the life course perspective, especially regarding youth, is that of turning points. Turning points are positive or negative life events that can potentially change current developmental paths (Wheaton & Gotlib, 1997). Positive events, such as finishing education, securing stable employment, and marriage, can shift youths' developmental or health risk trajectories toward a more health-promoting direction. Conversely, adverse events like early pregnancy or dropping out of school can reinforce existing adverse developmental paths, leading to negative outcomes in young adulthood.

The life course framework also emphasizes the ideas of latent effects and biological embedding. This explains how early life experiences impact biological development, leading to lasting effects on adult health (Nist, 2016). These latent effects of early adversity occur through biological embedding, where early physiological damage appears later in life (McGowan, 2012; Shonkoff et al., 2009). Research has shown that epigenetics functions as a mechanism that underpins early biological embedding.

Stress process theory

The stress process theory (Pearlin et al., 2005) complements the life course approach by illustrating how stressors persist and influence individuals over time (see Figure 1.2). Both theories emphasize the significance of social and economic contexts—such as income, social class, and employment—that shape access to resources and limitations, ultimately affecting health outcomes throughout life. For instance, family economic hardship can limit children's access to healthcare and insurance (Hsu & Wickrama, 2017). The stress process theory identifies primary stressors—such as economic hardship or low SES—that can lead to secondary stressors, thereby increasing adversity. Many primary stressors (including race/ethnicity, immigration status, and family SES) are assigned at birth but have enduring effects across the lifespan. The family stress model (Conger et al., 1994) illustrates this by demonstrating how parental job instability, income, and debt lead to economic hardship, which in turn influences marital conflict and parenting quality, ultimately shaping children's development.

Additionally, this theory highlights how the duration and intensity of stress exposure can affect whether psychosocial resources are weakened or strengthened, thereby influencing future health risks—even beyond the effects of ongoing life events (Moody-Ayers et al., 2007; Wickrama et al., 2013). Importantly, consistent with the life course focus on human agency, stress process theory underscores the protective role of psychosocial resources during youth. These resources can either buffer or exacerbate the effects of stress on health. Furthermore, gene-by-environment (G × E) interactions suggest that genetic predispositions can shape these effects, influencing an individual's susceptibility or resilience to adversities and risks.

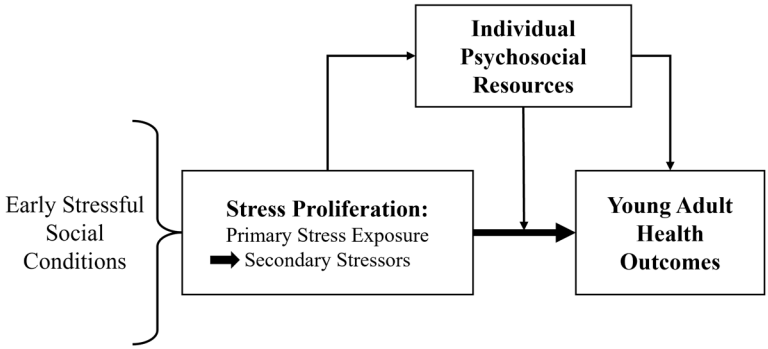

Figure 1.2. Stress Process Theory for Youth Health Development

Stress Appraisal Theory

The stress appraisal theory (Lazarus & Folkman, 1984) explains the coping process as involving a series of psychological and cognitive responses. According to this theory, individuals interpret stressors subjectively based on their cognitive resources, vulnerabilities, and past experiences. In other words, it is not the stressor itself, but how a person perceives and interprets the situation that affects the resulting psychophysiological responses.

The Developmental Perspective and Mid-Level Theories

The developmental perspective emphasizes how socially patterned exposures to risk factors during sensitive life stages influence developmental and health trajectories (Greyson & Holmbeck, 2002;

Steinberg, 1987). It points out that health risks and outcomes throughout a person's life are shaped by stage-specific stressors, especially during key developmental periods such as infancy, childhood, and adolescence (see Figure 2.1). Central to this idea is the concept of timing or critical periods, which suggests that the developmental stage during which stress occurs significantly impacts stress sensitivity and long-term results. For instance, the negative effects of socioeconomic adversity (ESA) can differ depending on whether the exposure happens in childhood, adolescence, or adulthood. A common example is the varied socioeconomic outcomes of World War II veterans, which depended on their age at the time they entered military service (Elder et al., 1994) and their predispositions.

Delinquency theory and attachment theory

Mid-level theories like delinquency and attachment theory expand on the developmental perspective by explaining how stress affects social and behavioral outcomes—such as delinquency and relationship stability—during adolescence and early adulthood. According to the delinquency theory of social control (Gottfredson & Hirschi, 1990), delinquency occurs when a person's bonds to society are weak or broken. Adolescents may engage in deviant behavior when social control mechanisms—such as attachment, commitment, involvement, and belief—are underdeveloped, especially in challenging family environments. Similarly, attachment theory (Mikulincer & Shaver, 2007) proposes that patterns of emotional regulation and relationship behavior in adulthood are rooted in early attachment experiences. Insecure attachment—often shaped by early family adversity—can lead to relationship instability, emotional withdrawal, and a heightened fear of abandonment in romantic and social settings relationships.

Neurological Development

Neurobiological research supports the developmental perspective by showing how early and ongoing stressors influence brain development. Since the brain is the main organ involved in experiencing and adapting to stress—especially during childhood and adolescence, times marked by high plasticity and reorganization—it demonstrates how early stress can cause lasting changes in neurodevelopment, affecting brain structure, function, and neural connections (McEwen, 2000; Sturman & Moghaddam, 2011). In childhood, key milestones include the development of the temporal lobe and hippocampus (linked to language and memory), growth of the limbic system

(related to emotional regulation), and maturation of the prefrontal cortex (which oversees self-regulation and executive functions). During adolescence, brain development features significant changes in the limbic system (which becomes more sensitive to emotions and rewards), the prefrontal cortex (which enhances decision-making and executive functions), and the corpus callosum along with white matter (improving communication between brain hemispheres and neural integration) (Sheridan et al., 2014; Shonkoff et al., 2009; Sturman & Moghaddam, 2011). As shown in Figure 1.3, neurodevelopment is closely tied to health risk pathways (Manuck & Hariri, 2008). Deficits in neurodevelopment can lead to maladaptive emotional, social-behavioral, or physiological responses. Therefore, early and ongoing stress—particularly within the context of ESA—plays a crucial role in shaping long-term physical and health outcomes. Importantly, as indicated in Figure 1.3, this relationship is bidirectional: new evidence suggests that health risk pathways and outcomes can also influence brain development, emphasizing both the brain's plasticity and vulnerability (McEwen, 2000).

The Need for Theoretical Integration: Life Course Developmental Perspective

Both the developmental and life course perspectives complement each other and work together to provide a comprehensive understanding of health development throughout life. Combining these perspectives enhances our understanding of health, especially in the first half of life. As Jones et al. (2009) note, both the developmental life course perspective and the structural life course perspective offer valuable insights. The structural perspective highlights how social stratification results in an unequal distribution of risks and resources, ultimately affecting individual health trajectories (Jones et al., 2019). In contrast, the developmental perspective focuses on how social patterns and sensitive developmental stages, along with timing and life-stage-specific exposures, shape health outcomes. The structural view helps to understand health in adulthood and later life, as it considers changes in socioeconomic environments, such as family dynamics, work conditions, and retirement. Meanwhile, the developmental perspective is more relevant in early life, as it highlights the rapid biological, psychological, and social changes that occur during infancy, childhood, and adolescence. Merging these approaches provides a more complete understanding of how health risks and outcomes develop early in life, enabling analysis of both structural factors and developmental processes that contribute to health disparities. Additional enriching theories in this

integration include the stress process theory (Pearlin et al., 2005), the stress appraisal theory (Lazarus & Folkman, 1984), and the cumulative advantage/disadvantage theory (DiPrete et al., 2006).

This book builds on this comprehensive approach by incorporating findings from biopsychosocial and neurodevelopmental research. Together, these frameworks support a multidisciplinary, unified life course perspective on physical health, well-being, and health disparities—especially in early adulthood and early life. These combined life course strategies are widely adopted by social epidemiologists and researchers in developmental, neurological, social, and behavioral sciences to better understand how health risk trajectories and disparities form during childhood, adolescence, and adulthood (Moody-Ayers et al., 2007; Settersten, 2003; Stowe & Cooney, 2015; Wickrama et al., 2017).

Introducing the Integrative Framework

The proposed integrative framework (ESAYHLT, Figure 1.3) offers a comprehensive, long-term perspective on health development during the first half of life. The ESAYHLT framework begins with early socioeconomic adversity (ESA) (shown in the far-left box) and ends with young adult physical health (HLT) outcomes (depicted in the far-right box). This framework explains how ESA, along with ongoing adolescent stressors, has a lasting impact on health risk trajectories throughout childhood and adolescence. These trajectories act as mediating mechanisms or pathways that lead to physical health and disease outcomes in early adulthood (Wickrama et al., 2022). These pathways may involve both baseline levels and changes over time (i.e., trajectories).

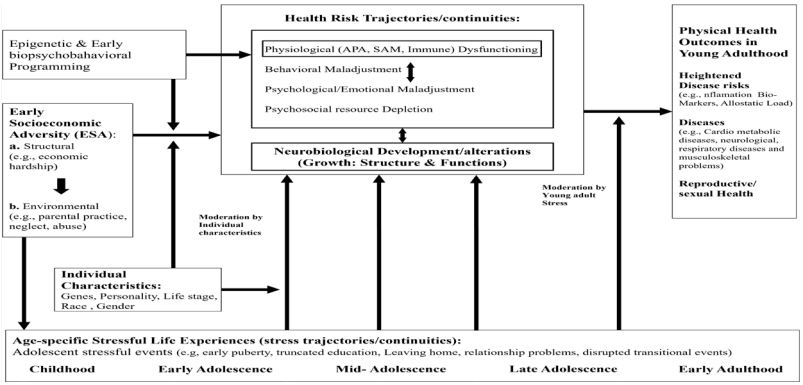

Figure 1.3. The proposed integrative framework (ESAYHLT) for Early Socioeconomic Adversity and Young Adult Physical Health Outcomes.

As shown in Figure 1.3, the impact of stress on risk trajectories depends on the intensity, duration, modality, and chronicity of the stressor exposure, as well as the sensitive period during which the exposure occurs.

The framework highlights six key risk domains that serve as interconnected pathways linking ESA to physical health in young adulthood.

1. Physiological functioning, including dysregulation of the SAM and HPA axes.
2. Psychological adjustment, such as symptoms of depression and anxiety
3. Behavioral adjustment, including problems with delinquency and relationship issues,
4. Psychosocial resource depletion,
5. Neurological development, including brain structure, function, connectivity, and ongoing adolescent stressors.

The vertical double-headed arrows in the figure depict dynamic, bidirectional relationships across these pathways. These connections between pathways are primarily cross-domain and reciprocal, highlighting the complexity of developmental processes. Importantly, as shown in Figure 1.4, neurological development plays a crucial and widespread mediating role among ESA, ongoing stressors, risk trajectories, and eventual health outcomes (McEwen, 2000; Sturman & Moghaddam, 2011).

Each domain has its own specific mechanisms. For example:

In the behavioral domain, mechanisms include delinquency, peer relationships, aggression, substance use, sleep issues, dietary habits, exercise, and challenges in forming romantic relationships.

Stressors beginning with ESA can cluster and multiply—both within and across different areas—leading to pathways of stress and health risks. ESA acts as a primary stressor that often triggers secondary stressors, such as stressful life events (SLEs), which are associated with a wide range of health risk trajectories.

Cumulative and Reciprocal Influences

Risks across and within domains can act independently or together, creating cumulative threats to physical health. For example, psychological and behavioral risk patterns can influence health outcomes both additively and multiplicatively. The impact of early or ongoing ESA may increase during adolescence and early adulthood because of interactions with other stressors and the passage of time. These interactions can lead to escalating or recovering risk trajectories, fueling adverse cumulative health effects. For instance, depressive symptom patterns often worsen during adolescence as the impacts of ESA and early family adversity increase (Bae et al., 2014). Such rising health risks significantly contribute to health disparities observed in adulthood.

Physical health outcomes in this model consist of:

- Allostatic load
- Chronic diseases, such as cardiovascular and metabolic conditions
- Morbidity and Chronic Pain
- Physical impairments

Self-reported health (SRH) serves as a physiological indicator, reflecting overall inflammation and health perception, often beyond standard objective measures—even in individuals who are generally healthy (Christian et al., 2011). Stressors also affect physical health complaints, including limitations that shape subjective perceptions of health (Phyo et al., 2022).

Although not fully shown in Figure 1.3, reverse causal effects are acknowledged. For instance, health-risk behaviors like smoking and alcohol