CHAPTER 4

LIFE COURSE PERSPECTIVES IN HEALTH PSYCHOLOGY

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Health psychology has long been concerned with how psychosocial processes can influence health risks and disease outcomes. Over past decades, a large literature has convincingly linked psychosocial factors, such as psychological stress (e.g., Booth et al., 2015) and personality traits (e.g., Graham et al., 2017), to risks for a number of chronic diseases and mortality. Some of the best observational studies on these topics assessed psychosocial characteristics at baseline, and then followed participants for years to document the onset or progression of disease (e.g., Kittleson et al., 2006) or have investigated how longitudinal changes in psychosocial constructs predict disease risks over time (e.g., Slopen et al., 2012). These types of studies highlight that many chronic conditions have long latency periods with pathogenic processes unfolding and becoming established over decades. Nonetheless, many traditional health psychology models neglect how psychosocial processes vary by developmental stages as well as the implications of such temporal variations for health. Yet, incorporating developmental perspectives is important as there is increasing evidence for phase-specific effects, where the magnitude or direction of an effect may vary over different segments of the life course (Shanahan et al., 2014). For example, some psychosocial effects on health may dissipate in late life due to the overwhelming force of biological aging, which becomes the greatest driver of health in the final years.

The present chapter takes a life course perspective on health psychology by focusing on one seminal health psychology model and discussing it from a life course perspective. Specifically, we use the stress-buffering model (Cohen & Wills, 1985) to demonstrate how the inclusion of a developmental perspective can refine theories by illustrating that the types and characteristics of social relationships that serve stress-buffering effects may vary across the life course. Such consideration of how effects vary across developmental stages allows for more nuanced approaches in intervention efforts, potentially maximizing their effectiveness.

In this chapter, we first provide a brief overview of both the stress–health and stress-buffering models. Then, we discuss, specifically for childhood, adolescence, and adulthood, the (a) the stress–health link, (b) the health-relevant social relationships during that life stage, and (c) the empirical evidence for buffering effects. As the stress–health literature is a very large one, this chapter focuses largely on stressors related to adverse life exposures that can span across the life course, namely socioeconomic and racial/ethnic (hereafter “racial”) disadvantages.

HEALTH PSYCHOLOGY: STRESS-HEALTH AND STRESS-BUFFERING MODELS

Numerous conceptual models have proposed how stress can get under the skin to impact biological systems and health (e.g., McEwen, 1998; Miller, Chen, & Parker, 2011). A typical stress–health model postulates that stressors give rise to psychological responses, such as heightened emotion reactivity, which in turn can elicit biological and behavioral responses. Biologically, stress triggers physiological responses in multiple body systems, such as the hypothalamic–pituitary–adrenal (HPA) axis and the immune system. For example, standardized laboratory stressors have reliable short-term associations with increased secretion of cortisol, a hormone released by the adrenal glands (Dickerson & Kemeny, 2004), as well as increased circulating levels of pro-inflammatory cytokines, proteins primarily secreted by immune cells (Steptoe et al., 2007). Over time, repeated or prolonged activation of these systems can lead to longer-lasting physiological alterations. One such alteration has been termed *allostatic load*, referring to the long-term wear-and-tear of physiological systems, which in turn is predictive of disease outcomes (McEwen, 1998). In addition, exposure to stressors can also confer health risks through behavioral pathways. Stress and the efforts to manage stress have been linked to more health-compromising behaviors such as substance use, and the effects of these behaviors can accumulate over time to confer risks for diseases (e.g., Schulte & Hser, 2013).

Although robust, the stress–health link is not deterministic. Indeed, various psychosocial factors may attenuate it, and one of the most widely discussed factors is social support. Specifically, the stress-buffering theory posits that social support experienced during times of stress can mitigate the typical psychological responses, and thus also mitigate the biological and behavioral sequelae that typically ensue (Cohen & Wills, 1985). For example, instrumental and emotional support may attenuate the perceived level of threat posed by a stressor and increase one’s perceived ability to manage it, thus also attenuating the aforementioned biological or behavioral responses. Ample empirical evidence supports this stress-buffering model. For instance, social support has been found to buffer the negative impact of stress in observational studies of real-world stressors such as discrimination, in laboratory studies that induced psychosocial stressors, and in intervention studies that manipulated social support (Cohen & Wills, 1985; Miller et al., 2014). In sum, the stress-buffering model is well-established conceptually and empirically.

LIFE COURSE STRESS-BUFFERING MODEL

One component that is missing, however, from the traditional stress-buffering model is the incorporation of a developmental perspective. This perspective is important because developmental stage can shape the social support needs and experiences of an individual, and thus define what is beneficial or detrimental for health. Notably, this approach could help shift the stress-buffering literature from an assumption that "social support is good, conflict is bad" to one that allows the protective value of social relationships to vary as a function of developmental stage and the social needs that individuals have at different stages (Chen et al., 2017). Indeed, a recent overview proposed that there are temporal variations in developmental needs relating to family relationships across childhood and adolescence, and whether family relationships can provide a buffering role depends on their fit with specific developmental needs (Chen et al., 2017). The current chapter will further this model by covering childhood, adolescence, and adulthood and by including a wider range of relationships from family to peer relationships. Specifically, we will demonstrate how the social needs and experiences individuals have as well as the types and characteristics of social relationships vary across the life course. Then, we will discuss how relationships that best serve the social needs of that life stage may provide the most optimal chance for buffering the negative impacts of social disadvantage on health.

As the disadvantage-health and disadvantage-buffering literatures are broad, this chapter reviews a wide range of health-related outcomes. While some studies utilized clinical measures (e.g., self-reported disease control and mortality) that are directly interpretable as health measures, most studies utilized biomarkers that reflect processes in different body systems including the autonomic, endocrine, and immune systems. These biomarkers are sometimes referred to as biological mediators that reflect underlying processes that contribute over the long term to a number of chronic diseases. To orient readers, Table 4.1 summarizes the biomarkers mentioned in this chapter and their corresponding conventional interpretations.

TABLE 4.1

Biomarkers Mentioned in This Chapter and Their Conventional Interpretations

Measure	Conventional interpretation
	Endocrine biomarkers
Cortisol reactivity to laboratory stressor or naturalistic/daily stressors	Thought to reflect acute activation of the hypothalamic–pituitary–adrenal (HPA) axis; over time, repeated experiences of conditions that elicit HPA activation would be theorized to be linked to adverse health outcomes (Dickerson & Kemeny, 2004).
Cortisol awakening response (CAR)	Cortisol levels typically increase about 50%–60% in the first 30–45 minutes after awakening, but there are individual differences in the size of this response. Interpretation is under debate—one the one hand, CAR is thought to be adaptive to help mobilize resources to meet the anticipated demands (such that attenuated CAR is seen as maladaptive); on the other hand, chronically high CAR responses may have long-term health costs (Adam et al., 2006).
Diurnal cortisol slope	Cortisol levels typically decline rapidly (post CAR) over the first few hours after waking and then decline more slowly across the day. Morning-to-evening slopes are typically estimated to gauge individual differences. Flatter diurnal cortisol slopes are thought to indicate disruption of cortisol's circadian pattern have been linked to worse physical health outcomes (Slatcher & Robles, 2012).
	Immune biomarkers
Circulating levels of cytokines or proteins (e.g., IL-6, CRP)	Thought to reflect nonresolving, low-grade inflammation, and has prospective links with increased risks for diabetes, heart attack, stroke, as well as mortality (Nathan & Ding, 2010).
Stimulated production of cytokines	Typically assessed by culturing cells with a microbial stimulus and then quantifying the ensuing production of pro-inflammatory cytokines. Greater production of cytokines indicates a more aggressive inflammatory responding (Miller, Chen, & Parker, 2011).
Sensitivity to inhibition signals	Typically assessed by culturing cells with a microbial stimulus and a compound that exerts anti-inflammatory effects; lower production of cytokines indicates a greater cellular sensitivity to inhibitory signals, which is considered adaptive for shutting down inflammatory responses (Miller, Chen, & Parker, 2011).
	Genetic expression biomarkers
Inflammatory and anti-inflammatory gene expression	Assesses the differential expression of messenger ribonucleic acid for different targets, such that over expression of inflammatory targets and reduced expression of anti-inflammatory targets are thought to promote sustained low-grade inflammation (Miller, Chen, & Parker, 2011).
	Metabolic biomarkers
Body mass index	Thought to reflect overall adiposity and has prospective links with increased risks for cardiovascular diseases and mortality (Czernichow et al., 2011; Steptoe et al., 2019).
Resting and ambulatory blood pressure	Higher blood pressure is associated with increased risks for cardiovascular diseases (Fuchs & Whelton, 2020).

Metabolic syndrome components	Metabolic syndrome represents a clustering of different cardiovascular risk factors. Components of metabolic syndrome typically include waist circumference, high-density lipoprotein, triglycerides, blood pressure, and fasting glucose. There are defined cutoffs for each component and the total number of components that meet the cutoffs are summed such that typically a score of three or more meets the criterion for having metabolic syndrome. Both metabolic syndrome status and component counts have been linked to increase risks for cardiovascular disease (Dekker et al., 2005).

Note. IL-6 = interleukin-6; CRP = C-reactive protein.

CHILDHOOD

Social Disadvantages and Health in Childhood

Exposure to poverty and racial disadvantage during childhood are associated with physical health problems throughout the life course. Black children and children from poorer families are more likely to have asthma, obesity, and poorer health behaviors during childhood and adolescence (e.g., Braveman & Barclay, 2009; Hanson & Chen, 2007), and are more likely to have cardiovascular, respiratory, and autoimmune diseases during adulthood (Cohen et al., 2010). These findings spurred numerous conceptual models attempting to explain how disadvantage during childhood can contribute to such a wide range of health problems across the life course. Many of these models are based on the idea that childhood is a sensitive period of development characterized by heightened plasticity of bodily systems and postulate that stress during childhood can impact health via altered biological functioning. One example is the biological embedding model, which posits that stress experienced during sensitive periods of development can "calibrate" immune cells to mount more exaggerated cytokine responses to microbial challenges and become more insensitive to hormonal signals that normally terminate inflammation (Miller, Chen, & Parker, 2011). The biological embedding model further posits that exposure to childhood stressors may also give rise to psychosocial characteristics, such as heightened threat vigilance and poor health behaviors, that further exacerbate the pro-inflammatory state instigated by immune cells. Over time, both biological and psychosocial factors may result in the body sustaining a chronic, low-grade inflammatory profile (Miller, Chen, & Parker, 2011). Indeed, social disadvantage has been linked to higher levels of low-grade inflammation markers assessed in childhood, adolescence, and adulthood (Lam et al., 2021). In turn, low-grade inflammation is associated with the same chronic diseases to which social disadvantages have been linked (e.g., Danesh et al., 2000).

Given the far-reaching impacts of childhood social disadvantage on health throughout the life course, identifying protective factors in childhood may be particularly efficacious in curtailing the subsequent health risks that typically follow. As family plays an instrumental role in childhood and has known implications for children's health, it can be a viable context for identifying buffering factors.

Family Relationships in Childhood

Family relationships are typically children's first social encounters and are major shapers of their social, emotional, and cognitive development (Harden, 2004). During childhood, the family is responsible for providing a safe environment and critical social experiences that foster a sense of physical safety and emotional security in children. These family experiences are linked to a number of outcomes, including secure attachment with parents/caregivers (hereafter "parents"), more prosocial skills, and better academic achievement (Harden, 2004). Notably, these family experiences have implications across the life course because they also form the foundation upon which many adult experiences and relationships are built. For example, adults who grew up with more negative (vs. positive) family relationships tended to have increased negative reactivity to stressors and difficulties in maintaining close relationships (Repetti et al., 2002). As such, family relationships during childhood are thought to have far-reaching impacts on an individuals' emotional, social, and behavioral functioning decades later.

But which of these family-related needs and experiences are relevant for health during childhood? We posit that there are four: (1) parental monitoring, (2) attachment, (3) parental warmth, and (4) family routines and parenting consistency. First, especially earlier in childhood, children's physical safety relies on parents monitoring and tracking children's activities. In addition to the physical well-being it ensures, parental monitoring has been linked to children's lower levels of circulating inflammatory markers and secretory immunoglobulin A, a proxy for immune activation (Byrne et al., 2017). Conversely, children with parents who engaged in less monitoring displayed flatter morning-to-evening cortisol slopes, which is a profile thought to indicate disruption of cortisol's circadian pattern and signaling that has been linked to poorer health outcomes (Martin et al., 2014).

Second, attachment and evolutionary theories suggest that the parent–child relationship is a special bond, one that is necessary for optimal human development (Tottenham, 2012). This has been demonstrated through studies of children who have been institutionalized, as these children typically receive adequate nutrition, safety, and medical care, but may have fewer and lower-quality caregiver-child interactions, thus resulting in lower levels of attachment. Indeed, children with (vs. without) a history of institutional care are at higher risk for developmental delays physically (e.g., smaller in size), cognitively (e.g., language delays), and emotionally (e.g., difficulties in identifying emotional expressions; Tottenham, 2012). In addition to developmental delays, the lack of secure attachment to parental figures poses challenges to physical health as well, including higher levels of a salivary inflammation marker, C-reactive protein (CRP; Measelle et al., 2017), and attenuated cortisol awakening responses, a pattern that has been linked to chronic stress in adults (Oskis et al., 2011). By contrast, among children with asthma, perceptions of parents as a secure base (a characteristic of secure attachment) have been associated with lower production of type 2 T-helper cell (Th2) cytokines, which are thought to promote airway inflammation (Ehrlich et al., 2018).

In addition to these qualities, family relationships can also vary in their valence and structure. In terms of valence, parent–child relationships can be generally characterized as warm and supportive or as harsh and conflictual. Because children typically have not mastered emotion regulation skills, they largely rely on parents managing their children's emotional experiences during times of distress, rendering high levels of

parental warmth and low levels of family conflict important family characteristics during childhood. In turn, both have been associated with physiological profiles in children. For example, among children with asthma, greater parental warmth was linked with higher sensitivity to anti-inflammatory signals in immune cells, lower circulating levels of eosinophil cationic protein, a marker of airway inflammation (Miller et al., 2009), as well as greater anti-inflammatory gene expression (Stanton et al., 2017). On the other hand, children who experienced greater conflicts at home had lower cortisol upon waking and a flatter diurnal cortisol slope throughout the day, a profile that is associated with worse physical health outcomes (Slatcher & Robles, 2012).

Parent–child relationships also vary in their structure. For example, in more hierarchical parent–child relationships, the parent is in a position of power and makes decisions for the child, whereas in less hierarchical relationships, there are greater amounts of joint parent–child decision making and mutuality. Earlier in childhood, parent–child relationships are typically more hierarchical as rules and boundaries help children become socialized and acculturated to norms. Notably, these hierarchical relationships mean that consistency is important, particularly earlier in childhood, as consistency in terms of family routines (establishing day-to-day regularity in family practices) and parenting behaviors (responding to children's behaviors with rewards and punishments that are predictable across time and situations) helps children learn social norms related to attitudes, values, and actions. Family environments that are less able to provide predictable routines may interfere with children's development of the belief that they are effective agents of their surroundings and have been linked to learned helplessness in children (Evans & Stecker, 2004). Consistency also appears to have implications for child health with greater disorganization within the household associated with poorer control of symptoms in children with asthma (Weinstein et al., 2019), greater odds of obesity (Anderson & Whitaker, 2010), and worse sleep (Boles et al., 2017).

In brief, family relationships play a foundational role in childhood development and are thought to have far-reaching impacts on psychosocial adjustments and well-being even decades later. During childhood, children look to parents to fulfill many of their social needs: parental monitoring ensures the physical safety of children, an attachment figure functions as a safe haven and secure base, parental warmth and support serve as sources of comfort during distress, and hierarchical and consistent parenting behaviors allow optimal socialization. In turn, these developmentally needed parent characteristics have been linked to more positive physiological health profiles in children.

The Buffering Effects of Family Relationships in Childhood

In addition to the main effects on health, there is also burgeoning evidence suggesting that the aforementioned family characteristics may buffer the negative impact of chronic disadvantages on health specifically during childhood. This literature has largely focused on parental warmth as a protective factor. For example, maternal warmth has been found to buffer the negative impact of community crime exposure, a stressor common in the context of low socioeconomic status (SES), on childhood adiposity (Gartstein et al., 2018). Specifically, the body mass index (BMI) of children living in neighborhoods with high crime rates but who experienced high levels of maternal warmth were no different from those who live in neighborhoods with low crime rates (Gartstein et al., 2018). In addition, maternal warmth was found to protect against the negative impact of chronic maternal intimate partner violence on children's risks of developing asthma (Suglia et al., 2009) and children's cortisol reactivity to laboratory stressors (Hibel et al., 2011). Buffering effects have also been found in the context of racial discrimination. For example, parental warmth weakened the link between racial discrimination and poorer health behaviors in a sample of Black children (Zapolski et al., 2016).

Given the associations of childhood social disadvantage with adulthood health, studies have also examined whether childhood experience of parental warmth would buffer such associations. Indeed, adults who grew up in low SES households and experienced high levels of maternal warmth as children were protected from the pro-inflammatory phenotype (Chen et al., 2011) and the number of metabolic syndrome components that met clinical cutoffs typically exhibited by those who grew up in low SES households (Miller, Lachman, et al., 2011). Maternal warmth has also been found to attenuate the association between childhood abuse and multisystem health risks, assessed with a composite of various biological markers of HPA, Sympathetic Nervous System (SNS), immune system, and metabolic syndrome (Carroll et al., 2013). Moreover, such buffering effects have also been examined using the viral-challenge paradigm where participants were exposed to a virus that causes a common cold and then monitored for signs of infection. Adults with low childhood SES were more likely to develop a cold, but this increased risk was attenuated among those who experienced parental warmth during childhood (Cohen et al., 2020). Many of the above studies utilized retrospective measures of chronic childhood disadvantage and parental characteristics, precluding causal inferences. However, a family-oriented psychosocial intervention that targeted Black children with low SES was shown to reduce inflammation levels assessed during young adulthood. Notably, these effects were mediated by increases in nurturant-involved parenting (including items assessing parental warmth) from baseline to postintervention (Miller et al., 2014). Although causal inference cannot be ascertained, these findings support the proposition that parental warmth can attenuate the link between childhood social disadvantage and a variety of physiological health outcomes, both during childhood and into adulthood.

The buffering role that secure attachment plays in the link between childhood disadvantages and health risks has also been documented. For example, one study examined the role of infants' attachment to parents in the context of maternal depression, a stressor common in the context of low SES. Maternal depression was only associated with higher levels of inflammation among infants with insecure attachment, but not among infants with secure attachment (Measelle & Ablow, 2018). Another study assessed family income, secure attachment behaviors, and cortisol responses to a naturally occurring stressor, medical exam and inoculations, in a sample of young children. Lower family income was associated with greater cortisol response to the stressor only among children without a secure attachment to their parents, but not among those who were securely attached (Johnson et al., 2018). Similarly, uncontrollable life events (e.g., the home being broken into), which are common among families facing social disadvantages, were associated with children's heightened cortisol reactivity to a standardized laboratory stressor. However, this pattern was not found among children with higher (vs. lower) secure attachment beliefs (Bendezú et al., 2019).

Although relatively less extensively, the other childhood family characteristics—family routines, parenting consistency, and parental monitoring—have also been examined as buffering factors. For example, a longitudinal study assessed consistency in parental behaviors and

family routines during childhood and then followed these children to late adolescence/early adulthood to assess cortisol responses to a laboratory stressor. Results suggested that adolescents/young adults who received more consistent and structured parenting styles during childhood exhibited lower cortisol responses when experiencing a laboratory stressor (Ellenbogen & Hodgins, 2009). Similarly, high levels of parental monitoring weakened the longitudinal link between exposure to violence and the initiation of substance use among a sample of older children (Sullivan et al., 2004).

To conclude, childhood is a sensitive period of development during which environmental inputs, such as social disadvantage, have high potentials for shaping the operating tendencies of biological systems in enduring ways throughout the life course (Miller, Chen, & Parker, 2011). Given such far-reaching impacts, it is important to identify protective factors during childhood; we propose four family characteristics that may be particularly fitting for children's social needs during this developmental stage. Specifically, parental monitoring fulfills the need of physical safety; having a secure attachment with parents fulfills the need to have a secure base; parental warmth fulfills the need for comfort during distress; and family routines and consistency in parenting behaviors allow optimal socialization. Each of these developmentally fitting family needs has been found to attenuate the negative impact of social disadvantage, or its associated stressors, on physical health outcomes, highlighting their potential as protective factors during childhood.

ADOLESCENCE

More conceptual frameworks highlight adolescence as a second crucial period of development with implications for health across the life course. During adolescence, biological, psychosocial, and behavioral systems undergo major changes. Biologically, puberty introduces rapid and drastic changes to multiple body systems, primarily the hypothalamic–pituitary–gonadal axis, and the adrenal system, that lead to physical growth and reproductive maturation (Patton & Viner, 2007). There are also major changes to brain development that have implications for executive functioning (prefrontal cortex), emotion processing (amygdala), responses to rewards (ventral striatum), and connectivity among these areas (Tottenham & Galván, 2016). Psychosocially, adolescents progress towards acquiring autonomy and independence as well as resuming more roles and responsibilities. In addition, peer relationships become more salient in adolescence, often with less supervision by adults, and adolescents put more emphasis on the expectations and opinions of their peers (Brown & Larson, 2009). Behaviorally, many health behaviors emerge and become consolidated during adolescence. For example, epidemiology studies have documented the onset of cigarette, alcohol, and cannabis use in adolescence, and longitudinal studies have shown the continuity of these behaviors into adulthood (Paavola et al., 2004). In brief, adolescence is marked by rapid and pervasive changes across biological, psychosocial, and behavioral systems.

Social Disadvantages and Health in Adolescence

Despite the many marked changes evident in adolescence, stressors from the broader social context—that is, social disadvantages—show similar associations with health outcomes as they do in childhood. For example, social disadvantages during adolescence have been linked to higher levels of circulating inflammation markers (Liu et al., 2017), flatter diurnal cortisol slopes throughout the day (Deer et al., 2018), and poorer health behaviors, including cigarette smoking, poorer diets, and less physical activity (Hanson & Chen, 2007). Notably, consistent with the idea that health behaviors become established during adolescence, many of these behaviors, and their associations with disadvantages, persisted into adulthood (Melchior et al., 2007). As adolescence lies between childhood and adulthood, it is also viewed as a period where putative mechanisms connecting childhood exposures to adulthood health outcomes may accumulate and become established. Supporting this notion, a longitudinal study that followed children from birth to age 32 found low *childhood* SES to be associated with *adolescent* tobacco smoking, which in turn was associated with *adulthood* tobacco dependence (Melchior et al., 2007).

As such, identifying protective factors during adolescence is important for curtailing the impact of current or previous disadvantages on adolescents' health. Given the increasing prominence of peer relationships during adolescence, we review both the family relationship characteristics that remain important for health during adolescence, as well as the peer relationship characteristics that also play a role in health during this period.

Family Relationships in Adolescence

A large body of conceptual and empirical work points to the continued importance of family relationships to adolescents, as they continue to shape adolescents' social and emotional development (Collins & Laursen, 2004). But which parental characteristics and experiences would be beneficial for an adolescent? While some characteristics that are relevant during childhood extend to adolescence, others may no longer fulfill adolescents' changing needs (Chen et al., 2017). We propose that (a) attachment, (b) parental warmth, and (c) parental monitoring continue to be beneficial for health during adolescence, but that family routines and consistency decline in importance for health during this period.

First, we hypothesize that attachment to parental figures remains as important during adolescence as it was in childhood, with similar implications for health. Parents, particularly mothers, continued to be identified as the primary attachment figures over friends and romantic partners during adolescence and even early adulthood (Rosenthal & Kobak, 2010). Such attachment to parents continues to have ramifications for adolescent's health—adolescents with anxious (vs. secure) attachment style to parents had higher cortisol levels at awakening and attenuated cortisol awakening responses (Oskis et al., 2011). Second, parental warmth continues to be critical in serving as a stable source of comfort for adolescents, and higher levels of parental warmth have been linked to reduced inflammatory profiles (Manczak et al., 2018) and less alcohol and drug use during adolescence (Klevens & Hall, 2014). Third, parental monitoring continues to be important in adolescence, but its function slightly changes. Rather than being important for protecting the physical safety of young children, parental tracking of adolescents' activities becomes important for preventing the onset of a number of risky health behaviors that typically emerge in adolescence. Indeed, longitudinal

studies have found that high levels of parental monitoring predicted a later onset of alcohol use and lower levels of subsequent alcohol use (Yap et al., 2017).

Therefore, as in childhood, we propose that secure attachment, parental warmth, and parental monitoring will remain beneficial for adolescents' health. However, as adolescents shift toward increased autonomy, there are corresponding shifts in the structure of parent–adolescent relationships from more hierarchical to more egalitarian with greater amounts of joint decision-making (Collins & Laursen, 2004). Such shared power relationships mean that strict family routines and more consistent parenting behaviors are no longer as critical to adolescents' family needs, rendering them less relevant to adolescent health.

The Buffering Effects of Family Relationships in Adolescence

Nascent findings support the idea that attachment, parental warmth, and parental monitoring may buffer the negative impact of disadvantages, or their associated stressors, on adolescents' health. Of these characteristics, parental warmth has been examined more extensively as a buffering factor during adolescence. For example, parental warmth has been found to attenuate the link between racial discrimination and substance use (Gibbons et al., 2010), between daily stressors and flatter evening cortisol slopes (Lippold et al., 2016), and between negative responses to stressors and poorer trait cortisol profiles (Stroud et al., 2020) among adolescents. In addition, findings from longitudinal studies suggest that the protective effects of parental warmth may be enduring. For example, parental warmth during adolescence buffered the link between racial discrimination during adolescence and allostatic load (Brody et al., 2014) as well as cellular aging (Brody et al., 2016) during young adulthood.

Consistent with the hypothesis that parental monitoring may help curtail health behavioral risks, higher levels of parental monitoring buffered the longitudinal link between witnessing violence and initiation of substance use (Sullivan et al., 2004). Intervention studies can help delineate causality, and one intervention study targeted parents of Black adolescents living in low-income urban areas to promote parental monitoring in the context of adversity. Relative to control group, adolescents in the intervention group had less risky sexual behaviors six months after the intervention (Stanton et al., 2000). Similarly, in a family-centered intervention that promoted parental monitoring (along with other resources for parents such as strategies for dealing with discrimination), rural Black adolescents in the intervention group were less likely to use alcohol, cigarettes, or marijuana relative to the control group (Brody et al., 2012). These findings suggesting that parental monitoring may be effective in curtailing behavioral health risks associated with social disadvantage.

Although less examined, attachment to parents has also been found to buffer adolescent boys from the negative impact of life stress on cortisol awakening response (Miller et al., 2017). Similarly, studies that utilized laboratory stressors to examine physiological responses found that adolescents with secure (vs. insecure) attachment to parents had attenuated cortisol responses to a frustration stressor task (Cameron et al., 2017).

In brief, family relationships remain important to adolescents, and although less well-examined relative to the childhood period, there is some evidence to suggest that parental warmth, parental monitoring, and attachment continue to serve as protective factors against the negative effects of social disadvantages and their associated stressors on health behaviors and physical or physiological health outcomes during the adolescent period.

Peer Relationships in Adolescence

Despite the importance of parents, as children progress into adolescence, they typically spend less time with parents and more time with peers (Brown & Larson, 2009). Longitudinal studies suggest normative changes throughout the adolescence period such that the influence of friends and closeness with friends increase from early to middle or late adolescence, and that both parents and friends become equally important in late adolescence (De Goede et al., 2009). These peer relationships have implications for physical health, and this literature largely focuses on quality measures of peer relationships such as acceptance, support, and conflict. For example, negative peer relationships, such as ones marked by rejection and conflicts, have been linked to higher levels of circulating inflammation markers, such as CRP and interleukin-6 (IL-6; Murphy et al., 2013), increased levels of inflammatory gene expression (Murphy et al., 2013), and the initiation of tobacco and marijuana use (Chassin et al., 1986; Fite et al., 2008). As such, peer relationships appear to also play a role in adolescent health.

The Buffering Effects of Peer Relationships in Adolescence

Although examined less extensively as moderators of the disadvantage–health link, nascent evidence supports the idea that positive peer relationships can have protective effects in the relationship between social disadvantage and adolescent health. For example, in a sample of Black adolescents, the link between uncontrollable life events in the past year (e.g., family member being robbed, a parent losing a job) and sexual risk-taking behaviors 1 year later was attenuated by having supportive friendships (Brady et al., 2009). Similarly, chronic peer conflict was linked to higher levels of risk-taking behaviors only among those who had less (vs. more) accepting and supportive peer relationships (Telzer et al., 2015). Supportive peer relationships have also been found to buffer the link between cyber harassment and self-reported health among adolescent boys (Fridh et al., 2015). Furthermore, positive peer relationships have also been found to buffer the longitudinal link between adolescent racial discrimination and allostatic load in young adulthood (Brody et al., 2014), suggesting the potential for longer-term protective effects of positive peer relationships.

To conclude, adolescence is marked by heightened changes across biological, psychological, and behavioral domains in ways that have implications for health. In particular, the links between social disadvantages and the onset of substance use and other risky health behaviors emerge during adolescence and persist into adulthood. Family relationships remain important for adolescents with parental warmth, attachment, and parental monitoring remaining linked with positive health outcomes during adolescence. Peer relationships increase in importance, and ones characterized by acceptance and support are beneficial for health. The literature testing moderating effects among adolescents is small, but these specific family and peer characteristics have been found to serve buffering roles in the context of disadvantage.

ADULTHOOD

Adulthood is marked by a number of normative life events (i.e., events that are highly probable for most), such as first job entry, long-term romantic partnership, and parenthood, which promote changes in social relationships. Biologically, adults experience gradual physical and cognitive declines from middle to late adulthood. Although plasticity is heightened during early decades of life and decrease across the life course, recent findings suggest that some degree of plasticity may be retained in adulthood for some body systems, such as certain regions of the brain (Davidson, Jackson, & Kalin, 2000) and certain components of the immune system (Stout et al., 2005). As such, exposures as well as protective factors during adulthood still impact health.

Social Disadvantages and Health in Adulthood

In adulthood, socioeconomic and racial disadvantages continue to be associated with intermediate risk markers, such as inflammation, with emerging evidence suggesting that these disparities are wider in adulthood than in childhood and adolescence (Lam et al., 2021). This is consistent with the cumulative disadvantage theory (Ferraro & Shippee, 2009), which posits that differences in advantage and disadvantage present earlier in life accumulate with the passage of time, thereby leading to divergence across people in varied types of life course health trajectories (Lee et al., 2019; Morton et al., 2018). Specifically, cumulative benefits accrue to those who possess social advantages, whereas cumulative losses accompany those who are disadvantaged, creating “fanning” effects in which individual health trajectories diverge from one another. Unique to adulthood is that numerous aging-related chronic diseases clinically manifest during this period, among which are some of the leading causes of death, including cardiovascular diseases, respiratory diseases, and some cancers. Morbidity of these diseases and the severity of symptoms are not uniform across SES and race groups—Black (vs. White) individuals and individuals with low (vs. high) SES are at greater risks for developing and dying from cardiovascular diseases and some cancers (Mozaffarian et al., 2016; Ward et al., 2004), and these effects are independent of each other (Ward et al., 2004).

Despite its robustness, there are factors that can moderate the disadvantage–health association in adulthood, including social relationships. One feature of social relationships in adulthood that is different from aspects covered in the childhood and adolescence sections is that both receiving social support from and giving social support to close others become important components of social relationships in adulthood. Therefore, this section reviews the health implications of various aspects of adult social relationships, positing three dimensions relevant for adult health: (1) social network size, (2) receiving social support from others, and (3) giving social support to others.

Social Relationships in Adulthood

As mentioned previously, a number of normative life events during adulthood influence social relationships and networks (Wrzus et al., 2013). For example, first job entry, which typically occurs during early adulthood, expands social networks as coworkers and new friends from the workplace emerge; marriage may increase social networks to include in-laws and the partners friends; the transition to parenthood typically reduces social networks as parents focus more on their relationships with their romantic partners and children (Wrzus et al., 2013). In addition, social goals and relationship priorities can shift from early adulthood to older adulthood. Specifically, the socioemotional selectivity theory posits that when one s remaining lifetime is perceived to be relatively unlimited (e.g., during early adulthood), goals focused on acquiring knowledge and information are more salient, and in turn propel individuals to develop diverse relationships from various sources to maximize the chances of gathering information. By contrast, when one s remaining lifetime begins to seem increasingly limited (e.g., during older adulthood), goals focused on emotion regulation become more salient, and in turn propel individuals to seek close relationships that are expected to be pleasant and thus help individuals to manage their emotions (Carstensen et al., 2003). As such, because of both normative life events and the changing perceptions of time remaining in life and corresponding social goals, the size of social networks typically increases during young adulthood but decreases during middle and older adulthood (Wrzus et al., 2013).

How then do these age-normative variations in social network size relate to health? There is some evidence that among younger adults, larger network size was associated with better physiological outcomes, such as higher antibody responses to an influenza vaccine (Pressman et al., 2005), whereas among older adults, investment in social ties, but *not* network size, was linked with better physiological outcomes, such as diurnal cortisol profiles (Lai et al., 2012). However, findings are mixed, perhaps because there may be a lower-bound threshold, under which a small social network size becomes detrimental to health even for older adults. For example, studies have consistently found that social isolation and loneliness are associated with worse physiological profiles across younger, middle-aged, and older adults, including higher levels of circulating inflammation markers (Smith et al., 2020). Similarly, during middle and older adulthood when clinical diseases manifest, social isolation has been linked to cardiovascular events (Valtorta et al., 2016). Together, these findings suggest that despite normative decreases in the overall size of social networks, middle-aged and older adults may still need to sustain a certain level of social integration and derive satisfaction from these relationships to avoid negative health consequences.

While social network size normatively decreases, close relationships, such as with family, remain important throughout all of adulthood, and the size of close relationships appears to be stable all through adulthood (Wrzus et al., 2013). One reason why close relationships are important is because close others provide adults with support during times of distress, with this feature also remaining stable across adulthood (Huxhold et al., 2013). Receiving social support from others has implications for physical health. Perceived social support was linked with lower level of inflammation (Uchino et al., 2018) and lower relative risks of developing and dying from heart disease (Barth et al., 2010).

In addition to receiving social support, providing social support also becomes more prominent during adulthood (relative to childhood and adolescence). One feature of adulthood is an increased sense of generativity—a need to provide care for offspring in ways that contribute to the next generation. Generativity increases from young to late adulthood, peaking at middle adulthood, and generativity in adulthood has been linked to providing social support to others (Keyes & Ryff, 1998). Such developmentally normative increases in generativity behaviors may be important as some models suggest that providing social support is necessary for survival and reproductive success, and that support-giving

behaviors may be beneficial for giver's health (Inagaki & Orehek, 2017). Indeed, independent of receiving social support, providing social support was associated with lower ambulatory blood pressure and lower heart rate (Piferi & Lawler, 2006) as well as lower mortality rates (e.g., Qu et al., 2020).

The Buffering Effects of Social Relationships in Adulthood

Although social network size has been linked to physical health, studies have yet to examine it as a moderator of the disadvantage–health link. However, numerous studies have examined the protective effects of receiving social support in the context of disadvantage and its associated stressors in adulthood. In a sample of Black adults, contextual stress, including racial discrimination and financial strain, was associated with greater inflammation only among those who perceived less (vs. more) social support (Beach et al., 2019). Similar findings have been documented in a sample of sexual minorities, in which discrimination was linked with higher levels of CRP only among those who had lower (vs. higher) levels of perceived social support (Wood & Cook, 2019). Similarly in the context of socioeconomic disadvantage, perceived social support attenuated the impact of low SES, and its associated stressors, on blood pressure (Coulon & Wilson, 2015), inflammation markers (Mezuk et al., 2010), and mortality (Krause, 2006). Notably, empirical evidence for buffering effects comes from samples of younger, middle-aged, and older adults, suggesting that receiving social support may buffer the negative impact of disadvantage on physical health across adulthood.

In addition to buffering concurrent stressors, some studies suggest social support during adulthood can buffer the negative impact of stress exposures that happened earlier in life. In one study, adults exposed to childhood adversity had higher levels of inflammation if they perceived lower (vs. higher) levels of social support as adults (Runsten et al., 2014). Extending these findings to a clinically relevant outcome, childhood abuse was linked with increased mortality among those with low, but not high, adulthood support received (Chiang et al., 2018). These initial findings suggest that, despite decreased plasticity in adulthood, social support that occurs decades after stress exposures may still serve buffering functions.

Aside from receiving social support, the buffering value of giving social support has also been examined in a few studies. For instance, the link between financial strain and mortality was apparent only among those who provided less (vs. more) social support, and these results were independent of receiving support (Krause, 2006). Similarly, stressful life events, such as financial difficulties and job loss, were only associated with increased mortality risk among adults who did not provide help to others (Poulin et al., 2013). By nature of assessing mortality as the outcome, these studies used samples of older adults, but similar buffering effects have been found in middle adulthood, where a related construct, volunteering, buffered cortisol reactivity to daily stressors (Han et al., 2018). As such, providing social support may serve protective functions against the impact of social disadvantage on health across adulthood.

However, there are likely boundaries to the positive effects of giving social support, such that too much giving may become detrimental to health. For example, studies on caregiving have consistently found that those who provide long-term care for a family member with a chronic illness are more likely to have elevated levels of inflammation (Roth et al., 2019). Thus, it has been proposed that giving may only be beneficial for health when the individual chooses to give support and perceives the support they give as effective, both of which do not apply to the context of chronic caregiving (Inagaki & Orehek, 2017). There may also be other adult contexts beyond caregiving, such as chronic or lifetime social disadvantage, in which individuals similarly have a lower sense of agency, and in which the regular giving of social support may not be able to provide the same degree of protection with respect to health problems.

Furthermore, giving and receiving social support are not done in isolation; rather, adults typically engage in both. Thus, it may be important to consider the amount of giving that one does relative to receiving, as some individuals may give much more support than they receive, whereas others may receive more support than they give. Such a balance of support giving to support receiving may have implications for health, with disproportionate giving or disproportionate receiving potentially being detrimental. Nascent studies have begun to answer this question by considering giving relative to receiving (and vice versa). One study found that lower SES was associated with higher levels of inflammation only among adults who disproportionately gave support to others (gave more emotional support than they received; Austin et al., 2021). Similarly, a longitudinal study found that chronic stressful life events, such as the death of someone close, were associated with worse self-rated health over a 5-year period among adults who disproportionately gave support to others (gave more social support than they received; Moskowitz et al., 2013). Of note, in the second study, the magnitude of the buffering effect when only considering support received was stronger than that when considering net support received relative to given, suggesting that the beneficial value of receiving support may decrease with increasing levels of support given. These findings highlight the importance of considering the relative amount of giving to receiving. When giving becomes too great relative to receiving, not only can it override the buffering effect, but it can become detrimental to health, particularly for socially disadvantaged individuals.

To conclude, adulthood is marked by clinical manifestations of chronic diseases, for which morbidity and mortality are patterned by SES and race. Although social network size decreases after young adulthood, close and supportive relationships remain prominent throughout adulthood. Both support receiving and giving are important features of adult relationships. Social network size, receiving, and giving support all have been linked as main effects to physical health outcomes in adulthood. With respect to buffering, empirical evidence suggests that both receiving and giving social support can protect against the negative impacts of disadvantage on health in adulthood. In addition, initial findings suggest that the balance between giving and receiving support may also determine whether support can serve a buffering effect in the context of social disadvantage during adulthood.

CONCLUSIONS AND FUTURE DIRECTIONS

Disadvantage is linked with worse health outcomes across the life course, but social relationships can attenuate these links. As social needs and experiences vary from childhood to adolescence to adulthood, the specific aspects of social relationships that best serve buffering roles also vary

by developmental stage. In childhood, families characterized by high parental monitoring, children's secure attachment with parents, high parental warmth, and consistent family routines and parenting behaviors have been found to attenuate the disadvantage-health link. During adolescence, family relationships remain important with parental warmth, attachment, and parental monitoring continuing to serve protective roles. Peer relationships grow in importance with accepting and supportive ones serving buffering effects. In adulthood, social network size decreases after young adulthood, but close relationships remain prominent throughout adulthood, with both having main effect associations with adult health. Adults receive and give social support in these relationships, and both receiving and giving support have independent protective effects with respect to health. However, initial findings suggest that if support giving becomes disproportionately more than support receiving, it can backfire and have negative health consequences for socially disadvantaged adults.

Although much work on stress-buffering effects across the life course has been conducted, there are notable gaps in the literature that represent important future directions. First, in childhood, more studies are needed that examine consistent family routines and parenting behaviors as moderators in the context of real-world disadvantage stressors that predict physiological processes and clinical health outcomes. Second, substantial empirical research has documented how negative aspects of peer relationships (e.g., targeted rejection, conflicts) can be detrimental to adolescents' development and health, but much less research has been conducted on the health consequences and moderating effects of positive aspects of peer relationships (e.g., peer acceptance). Examining these effects is important as they may provide another social target for prevention and intervention efforts, particularly if family support is not a viable option. In addition, relative to childhood, there are more mixed findings on whether parental support buffers negative impacts of adolescents' stress. Future research is necessary to elucidate the adolescence-sensitive factors (e.g., type of support, support delivery approach) that may render changes in the buffering magnitude of parental support. Third, adults normatively engage in both social support receiving and social support giving, more research is needed on the balance between receiving and giving. Imbalanced social relationships might be particularly detrimental for individuals experiencing high levels of social disadvantage, but more research is needed to clarify the boundaries of when social relationships are beneficial and when they might be detrimental to health among adults experiencing social disadvantage. Fourth, there is little research that examines whether the timing of social relationship exposures relative to the timing of disadvantage exposures matters for buffering effects to occur. The majority of extant research examined how social relationships may buffer *concurrent* stressors, whereas less research has examined how social relationships may buffer *previous* stressors (e.g., whether *adulthood* perceived support would buffer *childhood* poverty) or *future* stressors (e.g., whether *childhood* parental warmth would buffer *adolescence* discrimination events). Yet, these are important questions to examine as they can reveal whether there is a sensitive period during which a social buffering factor needs to occur to reap the protective effects. Finally, although there are known cultural differences in social relationships as well as developmental goals (Posada & Jacobs, 2001), few studies examine how stress-buffering effects may vary as a function of one's cultural background. Overall, addressing these gaps in the literature will help contribute to a more holistic understanding of how characteristics of social relationships that serve buffering effects for physical health may vary across the lifespan.

Of note, although this chapter focuses on the moderating role social factors play in the disadvantage-health association, numerous aspects of social lives are patterned by disadvantage, and this raises the possibility that aspects of social relationships may also mediate associations between disadvantage and health. For example, families from low SES typically lack discretionary income to cover household responsibilities (e.g., regular paid household labor or childcare), leading to more chaos and less routine in youth's family lives (Roy et al., 2004). Economic hardship has also been linked to more conflicts, angry exchanges, and neglectful relationships between parents and between parents and youth (Conger et al., 2010). In adult relationships, poverty has been associated with strained romantic and family relationships (Conger et al., 2010; Hardie & Lucas, 2010). In turn, adverse social relationships have been linked to poorer health outcomes across the life course—in youth, harsher family relationships have associations with greater risks for obesity and worse asthma symptoms (Chen et al., 2007; Hemmingsson, 2018); in adults, strained social relationships or social isolation have been linked to increased risks for cardiovascular diseases and mortality (Holt-Lunstad et al., 2015; Valtorta et al., 2016). Most previous research has tested social relationships as either a moderator or mediator in the disadvantage–health link, and hence future research would benefit from more direct comparisons within the same study to examine whether social relationships better fit a moderator versus a mediator model.

In sum, this chapter focuses on the stress-buffering model to illustrate the advantages of taking a developmental perspective. However, such an approach could in theory be applied to other potential psychological buffers. For example, coping, emotional adjustment, and personality are all psychological factors that have robust associations with health and potentially change across development in ways that might affect their potential buffering influence. Understanding the developmental variation in any of these factors might help reveal whether it would be beneficial or detrimental for health depending on its fit with specific developmental needs. To conclude, the incorporation of a developmental approach across a variety of health psychology models has the potential to allow for more nuanced theories and identify specific targets for intervention and prevention efforts in an age-sensitive manner.

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