Part I

Psychology Health and Illness

Section 1

Psychological Aspects of Health and Illness

Theme 1: Lifecourse and Health

In *As You Like It*, William Shakespeare waxes poetic in describing the seven stages of man:

> *All the world's a stage,*
> *And all the men and women merely players;*
> *They have their exits and their entrances,*
> *And one man in his time plays many parts,*
> *His acts being seven ages.*

Shakespeare had observed an important part of health psychology: that life stage and the normative developmental transitions within each stage set the backdrop for our understanding of all psychological phenomena, including the determinants of health.

The three chapters in this section may be less poetic, but review important areas within lifespan development. The determinants of health across the entire lifespan are influenced by early life experience, and in Chapter 1 (*Developmental Influences on Health)*, Turner-Cobb departs from the book's focus on adult health and behavior that makes up most of this handbook to explore how both internal (biological) processes and external (environmental) influences affect health as well as how children understand matters of health and illness. Chapter 2 (*Lifecourse Perspective and Health)* by Siegler and Ogle provides the long view of how health changes over the normative life cycle. To do this they draw on the University of North Carolina Aging & Heart Study, which was originally designed to examine personality in young adulthood as a predictor of coronary heart disease later in life, but also contributes to our understanding of how the developmental timing of exposure to stressors impacts health. Chapter 3 (*Disability and Health*) by Elliot and Armstrong addresses the fact that physical and mental health disabilities affect people of all ages. Describing medical, social and minority models of understanding disability, they synthesize the different definitions and research to suggest clinical practice and policy implications.

1 Developmental Influences on Health

Julie M. Turner-Cobb
Research Centre for Behaviour Change, Department of Psychology, Faculty of Science & Technology, Bournemouth University

Tara J. Cheetham
Faculty of Health Sciences, University of Southampton

The determinants of health across the entire lifespan are influenced by early life experience. Complex interactions of the physical and psychosocial environment with biological and genetic factors in early life determine health outcomes not just during childhood and adolescence but into adulthood and old age. Developmental influences on health are vital to understanding, predicting, treating and preventing poorer health outcomes and improving health across the lifespan. Such is a life-course perspective; it provides an important stance on understanding health trajectories from the cradle to the grave.

Due to developmental changes occurring during childhood and adolescence, early life experience can have a substantial effect on determining the trajectory of health throughout life, representing a pattern of logarithmic growth.

These changes centre around biological maturation, including the development of the immune and endocrine systems; cognitive understanding and language development; and attachment and social learning. Influences on these developmental processes, whether biological, psychological or social, and their interactions, can determine the degree to which vulnerability or resilience to ill health emerge. Biological influences include genetic predisposition and susceptibility; psychological factors include stress and coping responses, cognitions and emotions, and perception of social support; and social factors include social networks, socioeconomic status (SES), demographics, culture and lifestyle.

Biological Influences

Critical periods during childhood and adolescence are particularly influential in setting the subsequent health trajectory. The complex interplay between environmental context and biological characteristics has been referred to as biological sensitivity to context, biological embedding, differential susceptibility or developmental programming (Belsky *et al.*, 2007; Hertzman, 1999; Lupien *et al.*, 2001, 2009) and the experience of stress or early life adversity is central. The 'orchid gene hypothesis' (Ellis & Boyce, 2008) depicts two metaphoric descriptions, the 'orchid child' and the 'dandelion child', to describe the differential ways that underlying genetic variability interacts with the environment. The orchid child shows a heightened stress reactivity or sensitivity to their context or environment; under conditions of adversity this is likely to result in negative health effects, but with a sufficiently supportive context can result in positive health outcomes. Dandelion children, however, are naturally resilient, able to thrive and flourish in any environment.

From a biological perspective, the stress response, evolved in order to survive physical threats and challenges, can be activated through psychological threats, challenges and experience of loss or harm (see 'Stress'). Both the sympathetic nervous (SAM) system (producing adrenalin and noradrenaline) and the hypothalamic–pituitary–adrenal (HPA) axis (the end product of which is the glucocorticoid, cortisol) become activated under stressful conditions (see chapters 'Stress' and 'Stress and Coping Assessment'). While stress responses are normal and necessary to cope with events, prolonged or repeated stress exposure can have health repercussions. Learning how to cope with stress therefore has implications not just at a psychological level but also at a biological one, with consequences for long-term health. Alterations in stress hormone levels can increase the likelihood of developing chronic conditions later in life as well as have health implications during childhood. The mechanism by which stress and other psychosocial factors can interact with developing biological systems to influence health is explained well by the concept of stability through change, known as allostasis (McEwen, 1998; Sterling & Eyer, 1988). While allostatic systems, such as the SAM system and HPA axis, promote change and enable adaptation, allostatic load occurs when there is an imbalance or overload in allostatic systems.

During childhood development, the HPA axis shows relative plasticity in the natural circadian rhythm until the age of approximately 3–4 years, indicating this as a critical period for the impact of life adversity. Evidence suggests that while infants show a cortisol increase in response to minor stressors (e.g. vaccination, bathing), there is increasing support for existence of a hyporesponsive period to stress between the ages of one year and the onset of puberty (Gunnar & Quevedo, 2007; Jansen *et al.*, 2010). Similarly, in laboratory social stress testing, cortisol results are mixed with respect to effects in children prior to puberty. However, there is evidence that a response is elicited, albeit at a lower magnitude to that seen in adolescent or adult stress testing and with no sex differences observed (Yim *et al.*, 2010).

Psychosocial Influences: Stress and the Psychosocial Environment

The stress and coping paradigm (Lazarus & Folkman, 1984) describes stress as a transactional process that occurs between the person and their environment (see 'Stress'). The experience of stress is best seen as a 'mosaic' of simultaneously and dynamically overlapping stressors emanating from a number of sources at any one time (Michaud *et al.*, 2008:190). At its most generic, stress can be classified based upon duration and severity into acute (short term, mild to severe) and chronic (long term or enduring).

Childhood

With respect to stress experienced during childhood, the Center on the Developing Child, Harvard University (n.d.) classifies stressful events by level of severity, in order to distinguish between stressors. At the mildest end of the spectrum they classify events that produce momentary

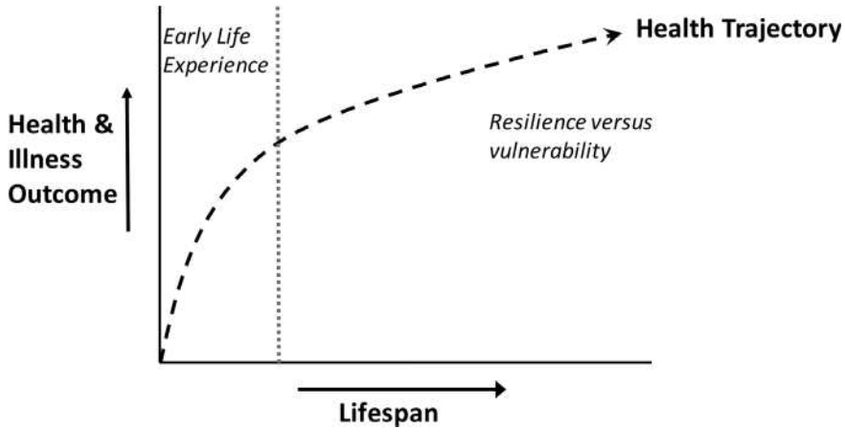

Figure 1.1 Illustration of health trajectory across the lifespan driven by early life experience.

alteration in physiological activity, as 'positive' (e.g. receiving an immunization injection or attending a new daycare). By contrast, 'tolerable' stress from events such as loss or injury is of greater severity but able to be adapted to with sufficient support. At the most severe end of the spectrum are 'toxic' stressors (e.g. poverty, neglect, abuse or violence) which pose the strongest risk for long-term negative mental and physical health outcomes. Such a distinction is important in delineating between stressors, acknowledging that not all stress is associated with negative outcomes particularly if countered by an enriched environment, while highlighting the negative consequences of more severe stressors or stress in the absence of a supportive environment.

Yet the health effects of stress are not necessarily linear with respect to severity, and reactivity to stress is influenced by a myriad of individual difference factors. What has been labelled as the 'shift and persist' strategy is a good demonstration of one way that individuals from disadvantaged backgrounds have been found to adapt to the stressful experiences they encounter and improve their likelihood of a positive outcome (Chen *et al.*, 2012). The 'shift' part of the strategy is focused around the use of secondary control; making cognitive adaptions to cope with an unchangeable stressor rather than using primary control where the situation can itself be changed (Chen *et al.*, 2012). This enables the individual to 'persist' in the stressful situation while looking to the future with hopeful expectation. Children raised in low socioeconomic status (SES) conditions who as adults show this coping technique of shift and persist have been found to have a more adaptive outcome with a lower allostatic load than those who did not develop this strategy; for those without early deprivation there was no additional benefit to using this technique (Chen *et al.*, 2012).

The experience of childcare and transition to school have both been used as vehicles for observing stress reactivity and examining the impact of stress on developing physiological functioning. In these contexts the quality of care and the temperament of the child have been highlighted as important interacting factors influencing endocrine and immune parameters (Dettling *et al.*, 2000; Vermeer *et al.*, 2012). An increase in salivary cortisol levels has been identified in four-year-olds transitioning to school as well as raised cortisol levels up to six months in anticipation of starting school (Turner-Cobb *et al.*, 2008). This response links to vulnerability for subsequently developing an upper respiratory tract infection (common cold) (Turner-Cobb *et al.*, 2011). The experience of mild and moderate stressors is important in learning to cope with stress both at psychological and physiological levels, and the ability to mount a stress response may be protective in the short term. However, repeated stress from different sources, being unable to adapt in the absence of protective environments or the experience of more severe stressors, may have long-term damaging effects on health.

Adolescence

Adolescence is seen as a stress-sensitive period in which the combined effects of accumulated childhood stress and current state of stress reactivity have the potential to influence health. The effect of stress during adolescence represents another critical period, with an increase in basal levels of cortisol and steeper stress reactivity following pubertal development, and effects of early life adversity often emerging during adolescence. Lupien *et al.* (2009) highlight the vulnerability of the frontal cortex of the developing brain during these teenage years as an explanation for this being a critical developmental period for subsequent health.

Prenatal Psychobiological Influences

The effect of psychosocial influences on developmental and health outcomes begin not at birth but in the prenatal environment, representing another critical period. Some studies extend this to the pre-conception environment in respect to parent health practices and endocrine/immune environment around conception or between pregnancies (Guardino *et al.*, 2016). The natural state of pregnancy represents for the mother a period of constant relative high levels of cortisol (Mastorakos & Ilias, 2000) with the development of the HPA–placental axis. There is considerable evidence to suggest that although the foetus is to some extent protected from maternal stress effects during pregnancy, if the mother experiences high levels of stress at certain points in the pregnancy, most notably during the second trimester, this can create a heightened stress hormone environment for the foetus and possibly result in pre-term delivery and low birth weight, indicators for subsequent poorer health outcome.

Prenatal stress also can set the HPA axis response, nervous system and immune parameters to function at an altered level of alert. Such biological rewiring, known as prenatal programming or foetal imprinting (Merlot *et al.*, 2008) is thought to be enabled via a combination of stress-induced reduction of the enzyme 11-beta-hydroxysteroid dehydrogenase type 2 (11beta-HSD2), required to convert cortisol to its inactive form, and abnormal uterine blood flow (O'Donnell *et al.*, 2009; Van den Bergh *et al.*, 2005). The effects of maternal prenatal stress on child postnatal outcomes have included: increased cortisol reactivity, poorer mental and motor development, and behavioural/temperament difficulties in infants; increased

acute stress reactivity, neurodevelopmental and cognitive endangerment, and learning and memory problems in children 2–9 years old; and alterations in diurnal cortisol profile in 10–15-year-olds (for review see Turner-Cobb, 2014).

Early Childhood Adversity

Interest in the health effects of early adversity that reach into adulthood and across the lifespan has grown in recent years.

Maternal Stress

Children exposed to mothers with postnatal depression have been found to have raised or more variable morning cortisol at 13 years, indicating a possible route for intergenerational transmission of depression (Halligan *et al.*, 2004). Shirtcliff *et al.* (2009) have termed this type of delayed effect a 'lingering influence', referring to evidence for raised levels of latent antibodies to herpesimplex virus (HSV) type 1 in 9–14 year-olds who experienced physical abuse or institutionalization shortly after birth and were subsequently adopted.

Literature linking early life stress and physical health outcomes has frequently focused on the impact of family environment and exposure to maternal stress and depression, across a range of ages throughout childhood and adolescence. Among young children (3–4.5 years), higher cortisol has been found in those whose mothers had lower job satisfaction or higher work-related emotional exhaustion (Chryssanthopoulou *et al.*, 2005). Exposure to maternal depression during the first two years of life has been reported as a strong predictor of baseline cortisol at age seven years (Ashman *et al.*, 2002). The combination of ongoing chronic family stress and acute stress events induced asthma symptoms and a Th2 inflammatory response (IL-4/IL-5) in children aged 9–18 years (Marin *et al.*, 2009). Similarly, in 8–18-year-olds with asthma, the perceived stress of their parents predicted greater production of the cell-mediated T-helper 2 (Th2) inflammatory cytokine interleukin 4 (IL-4) than in healthy control children over a period of six months (Wolf *et al.*, 2008).

Early Childhood Stress

Literature on post-traumatic stress in children suggests a higher level of physiological reactivity, which has the potential to develop into the paradoxical pattern of hyporeactivity (low reactivity/inability to respond at usual levels), has been observed in adults with PTSD (see Heim *et al.*, 2000).

In a review of the effects of child abuse in adult life, Wegman and Stetler (2009) concluded that the increased risk for poorer physical health outcomes is comparable to that of psychological health outcomes. For example, childhood abuse has been linked with an increased adult risk for developing cardiovascular or coronary heart disease (Roy *et al.*, 2010) and multiple sclerosis (Spitzer *et al.*, 2012) in younger adults. Similarly, childhood trauma has been linked with chronic pain in adulthood (fibromyalgia, osteoarthritis) and greater diurnal salivary cortisol in middle-aged women (Nicolson *et al.*, 2010). C reactive protein (CRP), an inflammatory marker associated with atherosclerosis and heart disease, was found to be significantly higher in middle-aged men and women who reported having poorer emotional functioning at age seven years (Appleton *et al.*, 2011).

Accelerated ageing associated with psychological stress exposure early in life has been assessed through measurement of chromosomal telomere length (an indicator of the degree of deterioration of the telomeres or DNA protective caps at the end of chromosomes which shorten with age). For example, in a sample of Alzheimer's caregivers and controls, the experience of multiple adversities during childhood was associated with shorter telomeres, predicted to reduce lifespan by 7–15 years (Kiecolt-Glaser *et al.*, 2011). However, with the exception of the study of emotional functioning in childhood and adult CRP (Appleton *et al.*, 2011), all of these studies have relied on retrospective self reports of early life adversity and prospective designs are needed to address these relationships more robustly (Wegman & Stetler, 2009).

Two further domains are important in describing the impact of psychological factors that contribute to developmental influences on health. These are the level of a child's cognitive development and a consideration of the prenatal influences.

Cognitive Development: Children's Understanding of Health

A child's developmental understanding of health and illness also influences their ability to engage in health-related behaviours or to cope with the stress of illness itself. While discrete stages of development in cognitive understanding of illness have been proposed (Bibace *et al.*, 1994), others have suggested a more complex approach accounting for individual differences. There is also evidence that children have some innate understanding of illness that allows them to comprehend more about illness, and from an earlier age, than previously thought (Hergenrather & Rabinowitz, 1991; Normandeau *et al.*, 1998). Knowledge and experience of illness have also been found to affect children's understanding (Crisp *et al.*, 1996). In one study, children as young as six years are able to comprehend a link between stress and illness (Cheetham *et al.*, 2016).

Social Influences: Demographic, Socioeconomic and Lifestyle Factors

Social influences on development that impact on health outcomes include a range of factors from health behaviours including diet, exercise, exposure to tobacco smoke, alcohol consumption or drug use, to lower SES, particularly the early life experience of poverty. These factors may occur through prenatal exposure to parental health behaviours or those experienced in early childhood.

Evidence suggests that low SES is associated with higher basal cortisol levels at least up to the pre-teen years (Lupien *et al.*, 2001). Miller *et al.* (2007) found low SES at age 2–3 years to influence pro-inflammatory markers of gene expression (including glucocorticoid receptors) in adolescence, which may provide a mechanism through which stress has an influence on respiratory infection and cardiovascular disease (Miller *et al.*, 2007). The interaction of lower SES and race is illustrated in a study of middle-aged African-Americans who experienced early life poorer living and working conditions, suggesting that lower SES and racism in childhood have synergistic effects (Slopen *et al.*, 2010).

The longitudinal social patterning of disease is demonstrated well by Evans and Kim (2012), who examined the amount of time spent in poverty during the first nine years of life, in combination with psychological (family turmoil, separation from family, exposure to violence) and physical (noise, crowding, housing problems). They found a relationship between poverty during early life and allostatic load at age 17 years mediated by cumulative risk at the interim age of 13 years. Interestingly, allostatic outcome at age 17 years was unrelated to cumulative risk at this age, showing the importance of social embedding during childhood and early life on illness vulnerability in adulthood.

Summary

There is an increasing amount of evidence that biopsychosocial factors and perhaps more importantly their interactions are key factors in considering developmental influences on health. Experience during early life and particularly during critical periods in pregnancy, childhood and adolescence are powerful drivers of vulnerability and resilience for future adult health. Increased understanding of these biopsychosocial influences in operation during childhood and of the timeline of influence over the health trajectory provides evidence for the development of interventions to improve health outcomes during childhood and across the lifespan at all stages of adulthood.

REFERENCES

Appleton, A. A., Buka, S. L., McCormick, *et al.* (2011). Emotional functioning at age 7 years is associated with C-reactive protein in middle adulthood. *Psychosomatic Medicine*, **73**, 295–303.

Ashman, S. B., Dawson, G., Panagiotides, H., *et al.* (2002). Stress hormone levels of children of depressed mothers. *Development and Psychopathology*, **14**, 333–349.

Belsky, J. Bakermanns-Kranenburg, M. J. & van Ijzendiirn, M. H. (2007). For better *and* worse: differential susceptibility to environmental influences. *Current Directions in Psychological Science*, **16**, 300–304.

Bibace, R., Schmidt, L. R. & Walsh, M. E. (1994). Children's perceptions of illness. In G. N. Penny, P. Bennett & M. Herbert (eds), *Health Psychology: A Lifespan Perspective* (pp. 13–30). London: Harwood.

Center on the Developing Child, Harvard University (n.d.). Toxic stress. Retrieved from http://developingchild.harvard.edu/science/key-concepts/toxic-stress.

Cheetham, T. J., Turner-Cobb, J. M. & Gamble, T. (2016). Children's implicit understanding of the stress-illness link: testing development of health cognitions. *British Journal of Health Psychology*, **21**, 781–795.

Chen, E., Miller, G. E., Lachman, M. E., Gruenewald, T. L. & Seeman, T. E. (2012). Protective factors for adults from low-childhood socioeconomic circumstances: the benefits of shift-and-persist for allostatic load. *Psychosomatic Medicine*, **74**, 178–186.

Chryssanthopoulou, C. C., Turner-Cobb, J. M., Lucas, A. *et al.* (2005). Childcare as a stabilizing influence on HPA axis functioning: a reevaluation of maternal occupational patterns and familial relations. *Developmental Psychobiology*, **47**, 354–368.

Crisp, J., Ungerer, J. A. & Goodnow, J. J. (1996). The impact of experience on children's understanding of illness. *Journal of Pediatric Psychology*, **21**(1), 57–72.

Dettling, A. C., Parker, S. W., Lane, S. *et al.* (2000). Quality of care and temperament determine changes in cortisol concentrations over the day for young children in childcare. *Psychoneuroendocrinology*, **25**, 819–836.

Ellis, B. & Boyce, W. (2008). Biological sensitivity to context. *Current Directions in Psychological Science*, **17**, 183–187.

Evans, G. W. & Kim, P. (2012). Childhood poverty and young adults' allostatic load: the mediating role of childhood cumulative risk exposure. *Psychological Science*, **23**, 979–983.

Guardino, C. M., Dunkel Shetter, C., Saxby, D. E. *et al.* (2016). Diurnal salivary cortisol patterns prior to pregnancy predict infant birth weight. *Health Psychology*, **35**, 625–633.

Gunnar, M. & Quevedo, K. (2007). The neurobiology of stress and development. *Annual Review of Psychology*, **58**, 145–173.

Halligan, S. L., Herbert, J., Goodyer, I. M., *et al.* (2004). Exposure to postnatal depression predicts elevated cortisol in adolescent offspring. *Biological Psychiatry*, **55**, 376–381.

Heim, C., Ehlert, U. & Hellhammer, D. H. (2000). The potential role of hypocortisolism in the pathophysiology of stress-related bodily disorders. *Psychoneuroendocrinology*, **25**, 1–35.

Hergenrather, J. R. & Rabinowitz, M. (1991). Age-related differences in the organization of children's knowledge of illness. *Developmental Psychology*, **27**, 952–959.

Hertzman, C. (1999). The biological embedding of early experience and its effects on health in adulthood. *Annals of the New York Academy of Sciences*, **896**, 85–95.

Jansen, J., Beijers, R., Riksen-Walraven, M., *et al.* (2010). Cortisol reactivity in young infants. *Psychoneuroendocrinology*, **35**, 329–338.

Kiecolt-Glaser, J. K., Gouin, J. P., Weng, N. P., *et al.* (2011). Childhood adversity heightens the impact of later-life caregiving stress on telomere length and inflammation. *Psychosomatic Medicine*, **73**, 16–22.

Lazarus, R. S. & Folkman, S. (1984). *Stress, Appraisal and Coping.* New York: Springer.

Lupien, S., King, S., Meaney, M. J., *et al.* (2001). Can poverty get under your skin? Basal cortisol levels and cognitive function in children from low and high socioeconomic status. *Development and Psychopathology*, **13**, 653–676.

Lupien, S. J., McEwen, B. S., Gunnar, M. R., *et al.* (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews Neuroscience*, **10**, 434–445.

Marin, T. J., Chen, E., Munch, J. A. & Miller, G. E. (2009). Double-exposure to acute stress and chronic family stress is associated with immune changes in children with asthma. *Psychosomatic Medicine*, **71**, 378–384.

Mastorakos, G. & Ilias, I. (2000). Maternal hypothalamic–pituitary–adrenal axis in pregnancy and the postpartum period: postpartum-related disorders. *Annals of the New York Academy of Sciences*, **900**, 95–106.

McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *New England Journal of Medicine*, **338**, 171–179.

Merlot, E., Couret, D. & Otten, W. (2008). Prenatal stress, fetal imprinting and immunity. *Brain Behavior & Immunity*, **22**, 42–51.

Michaud, K., Matheson, K., Kelly, O. & Anisman, H. (2008). Impact of stressors in a natural context on release of cortisol in healthy adult humans: a meta-analysis. *Stress*, **11**, 177–197.

Miller, G. E., Chen, E. & Zhou, E. S. (2007). If it goes up, must it come down? Chronic stress and the hypothalamic–pituitary–adrenocortical axis in humans. *Psychological Bulletin*, **133**, 25–45.

Nicolson, N. A., Davis, M. C., Kruszewski, D. & Zautra, A. J. (2010). Childhood maltreatment and diurnal cortisol patterns in women with chronic pain. *Psychosomatic Medicine*, **72**, 471–480.

Normandeau, S., Wins, I., Jutras, S., *et al.*, (1998). A description of 5- to 12-year old children's conception of health within the context of their daily life. *Psychology & Health*, **13**, 883–896.

O'Donnell, K., O'Connor, T. G. & Glover, V. (2009). Prenatal stress and neurodevelopment of the child: focus on the HPA axis and role of the placenta. *Developmental Neuroscience*, **31**, 285–292.

Roy, A., Janal, M. N. & Roy, M. (2010). Childhood trauma and prevalence of cardiovascular disease in patients with type 1 diabetes. *Psychosomatic Medicine*, **72**, 833–838.

Shirtcliff, E. A., Coe, C. L. & Pollak, S. D. (2009). Early childhood stress is associated with elevated antibody levels to herpes simplex virus type 1. *Proceedings of the National Academy of Sciences of the United States Of America*, **106**, 2963–2967.

Slopen, N., Lewis, T. T., Gruenewald, T. L., et al. (2010). Early life adversity and inflammation in African Americans and whites in the midlife in the United States survey. *Psychosomatic Medicine*, **72**, 694–701.

Spitzer, C., Bouchain, M., Winkler, L. Y., *et al.* (2012). Childhood trauma in multiple sclerosis: a case-control study. *Psychosomatic Medicine*, **74**, 312–318.

Sterling, P. & Eyer, J. (1988). Allostasis: a new paradigm to explain arousal pathology. In S. Fisher & J. Reason (eds), *Handbook of Life Stress, Cognition and Health* (pp. 629–649). New York: Wiley.

Turner-Cobb, J. M. (2014). *Child Health Psychology*. London: Sage.

Turner-Cobb, J. M., Rixon, L. & Jessop, D. S. (2008). A prospective study of diurnal cortisol responses to the social experience of school transition in four-year-old children: anticipation, exposure, and adaptation. *Developmental Psychobiology*, **50**, 377–389.

Turner-Cobb, J. M., Rixon, L. & Jessop, D. S. (2011). Hypothalamic–pituitary–adrenal axis activity and upper respiratory tract infection in young children transitioning to primary school. *Psychopharmacology (Berl)*, **214**, 309–317.

Van den Bergh, B. R., Mulder E. J., Mennes M., & Glover V. (2005). Antenatal maternal anxiety and stress and the neurobehavioural development of the fetus and child: links and possible mechanisms. A review. *Neuroscience and Biobehavioral Reviews*, **29**, 237–258.

Vermeer, H. J., van Ijzendoorn, M. H., Groeneveld, M. G., *et al.* (2012). Downregulation of the immune system in low-quality child care: the case of secretory immunoglobulin A (SIgA) in toddlers. *Physiology and Behavior*, **105**, 161–167.

Wegman, H. L. & Stetler, C. (2009). A meta-analytic review of the effects of childhood abuse on medical outcomes in adulthood. *Psychosomatic Medicine*, **71**, 805–812.

Wolf, J. M., Miller, G. E. & Chen, E. (2008). Parent psychological states predict changes in inflammatory markers in children with asthma and healthy children. *Brain, Behavior & Immunity*, **22**, 433–441.

Yim, I. S., Quas, J. A., Cahill, L., *et al.* (2010). Children's and adults' salivary cortisol responses to an identical psychosocial laboratory stressor. *Psychoneuroendocrinology*, **35**, 241–248.

2 Lifecourse Perspective and Health

Ilene C. Siegler
Department of Psychiatry and Behavioral Sciences, Duke University School of Medicine

Christin M. Ogle
Department of Psychology and Neuroscience, Duke University

Introduction

A lifecourse perspective provides important insights into maintaining health in later life. This has become more complex as the lifespan has reached 122 years of age (so far). Data needed to understand this generally come from cohort studies. A cohort is any defined group of people, often by year of birth or a location or some combination of factors. That is, studies often look at one cohort over time or multiple cohorts at one time to make conclusions. While different studies have different enrollment criteria, the studies themselves are also aging: some have data collected for up to 75 years. Thus, they provide us with important information about what the lifecourse is for the persons studied over their lifespan. This is conditioned by the location of the individuals within their social classes and the geographic location of the persons. The psychological literature had adopted Schaie's (1965) developmental model, which focused on age, cohort and time of measurement. This model organizes the complexity of the information found in deceptively simple descriptions of how particular individuals age. The important point that Schaie makes is that these are not independent components. For example, a person who is 70 in 2017 was born in 1947. For people born in 2017, it is unlikely that their experience of 70 years of life will be the same in the future as it has been in the past. Birth cohort is also a useful way to think about the age when major historical events happened and to study the impact that they have on psychological development.

Power *et al.* (2013) provide a very useful framework that recognizes the levels of the environment (national, neighborhood and household levels) combined with family generational influences (grandparent, parent and offspring generations) that move through time together. Trying to understand what in the lifespan is only due to aging and what is due to other factors is a complex problem. Useful interventions to improve health of persons wherever they are in the lifecourse depend on understanding these interacting components accurately.

In this chapter, we cite some published reviews of the findings from lifespan research sources for data across the lifecourse. We then review a case study of our own work that illustrates the usefulness of a lifecourse perspective and conclude with some observations about the status of this enterprise. We use the terms lifespan and lifecourse together to include the range of work in the behavioral, social science disciplines that contribute to our knowledge and the work in epidemiology representing the biomedical and public health disciplines.

We next turn to a case study reflecting how a lifecourse perspective on health can contribute to our understanding of one disorder, post-traumatic stress disorder (PTSD). This case study illustrates how knowledge concerning how the developmental timing of exposure to stressors impacts lifespan development, by adding new outcome measures to an ongoing study population.

PTSD in the UNC Alumni Heart Study (UNCAHS): A Case Study

The UNCAHS was originally designed to examine personality as a predictor of coronary heart disease in a cohort of college attendees who completed the MMPI during college registration (Siegler, 2016; Siegler *et al.*, 1992;). Based on a growing body of evidence concerning the link between coronary heart disease and PTSD (e.g. Boscarino, 2008; Kubzansky *et al.*, 2007), the scope of the UNCAHS was expanded in 2008 (wave 12) to investigate the relation between these two important health conditions in a non-clinical sample. An inventory of traumatic life events and a measure of PTSD symptom severity were included in the 12th wave of the UNCAHS, followed

by a second measure of PTSD symptoms in the 13th wave. Measures of several individual difference factors shown to be important to the development and persistence of PTSD symptoms were also added at wave 13, including event centrality, measures of the phenomenological properties of trauma memories and adult attachment. With 13 waves of data collection spanning five decades of the lifespan now complete, the UNCAHS contains an extensive archive of data concerning lifecourse personality, psychological health and disease outcomes.

Through a series of papers utilizing the data on post-traumatic stress symptoms (PTSS)[1] in the UNCAHS cohort, we have advanced knowledge of the impact of stressful and traumatic events on psychological health and wellbeing in older adults. It has become critically important to understand the factors that contribute to poor psychological health and wellbeing during older adulthood following recent demographic changes that have resulted in older adults comprising an increasingly larger proportion of the population in industrialized countries, and the implications of these changes for economic and health care policy. Moreover, given that community-dwelling older adults are typically underrepresented in epidemiological research on the frequency of traumatic life events and PTSD symptom prevalence, our first goal was to provide base rate information on lifetime exposure to stressful and traumatic events in community-dwelling older adults with exposure to a broad range of negative and traumatic events across the lifespan. Our initial findings revealed when particular types of traumatic events are likely to occur during the lifecourse, as well as which types of negative and traumatic life events are most likely to cause psychological distress, including PTSS during older adulthood (Ogle *et al.*, 2013b). In our subsequent work, we further investigated the differential effects of exposure to traumatic life events during developmental periods throughout the life cycle (Ogle *et al.*, 2013a). Specifically, we compared traumas experienced during childhood, adolescence, midlife and the young-old period of older adulthood to determine the developmental period during which individuals are most vulnerable to long-term negative post-traumatic outcomes. An extensive body of research has shown that each of these developmental periods is characterized by age-related changes in cognitive, emotional and social processes that may influence the likelihood of negative outcomes following trauma exposure, including the onset of autobiographical memory during early childhood, development of the sense of self during adolescence, increases in social support from romantic partners and coworkers during young adulthood, and declines in social connections coupled with the increase in risk factors for chronic disease during middle and older adulthood. In addition to PTSS, we also examined the influence of the developmental timing of trauma exposure on several psychosocial measures that have been linked to psychological adjustment following trauma exposure, including perceptions of the quality of support individuals receive from others, the ability to cope with stress and subjective happiness. Our results showed that older adults who experienced traumatic events in childhood – 40 or more years prior to our investigation – reported more severe PTSS and lower psychosocial functioning compared to individuals who experienced relatively recent traumas in adulthood. The differential effects of early life compared to adulthood trauma exposure emerged across a wide range of indices, including current PTSS and measures of psychosocial functioning. Furthermore, our results showed that the differential effects of childhood compared to adulthood trauma exposure were not fully explained by differences in the objective and subjective characteristics of the traumatic events. Overall, these results illustrate the enduring nature of traumatic events experienced early in the lifecourse, and suggest that individuals exposed to early life trauma are at greater risk of a wide range of adverse outcomes in older adulthood compared to individuals exposed to traumatic events after the transition to adulthood.

Our subsequent work advanced research on post-traumatic outcomes in older adults by demonstrating that the cumulative burden of exposure to multiple traumatic events across the lifespan persists into older adulthood among individuals from the Baby Boomer generation (Ogle *et al.*, 2014a). Due to their education and cohort membership, the Baby Boomer generation may have greater access to resources that protect against the detrimental consequences of negative and stressful life events compared to other generations. Despite these potential advantages, our results indicated that compared to other known predictors of PTSD, including event severity, personality traits, social support and subjective appraisals of trauma, cumulative trauma exposure predicted greater PTSS in a manner consistent with an accumulation of risk model, whereas the severity of individuals' single most distressing life event did not explain unique variance in PTSS. Comparisons of distinct categories of traumatic events revealed that greater cumulative exposure to childhood violence was the strongest predictor of PTSS, followed by adulthood physical assaults. Analyses concerning individual difference factors further showed that the extent to which older adults construed their most distressing trauma as central to their identity accounted for the largest percentage of explained variance in PTSD symptom severity. This finding is commensurate with a core principle of Life Course Theory (Elder, 1998), which holds that the degree to which cumulative disadvantages negatively impact an individual's developmental trajectory depends on the individual's perceptions of their personal circumstances and their ability to mobilize resources. Perceptions of the centrality of a traumatic event may be especially consequential during older adulthood, given that the process of evaluating and accepting the events of one's life is the primary developmental task of older adulthood (Erikson, 1982). This finding concerning the role of event centrality in post-traumatic outcomes suggests that treatment efforts aimed at reducing the centrality of traumas as they are evaluated during the life review process (Butler, 2002) may be especially beneficial for older adult trauma survivors. Overall our results underscore the importance of examining the broader context of lifetime exposure to traumatic events, in particular cumulative exposure to assaultive violence throughout the lifespan, when seeking to identify factors contributing to current distress in older adults.

Our examination of PTSS in the UNCAHS has also focused on advancing knowledge concerning mechanisms that promote the development and maintenance of post-traumatic stress. The rich archive of data in the UNCAHS concerning lifecourse personality, psychological health and disease outcomes provided the opportunity to test the most extensive set of PTSD risk factors measured in a single participant sample to date (Ogle *et al.*, 2016a). Furthermore, our analysis of prospective data and comparison of factors assessed before and after traumatic event exposure allowed us to elucidate the temporal order of PTSD symptoms and their correlates. Results indicated that individual difference measures assessed after the traumatic event exposure, including insecure attachment and characteristics of the current trauma memory, better accounted for symptom severity than factors measured before the traumatic event occurred. These findings support a model of post-traumatic stress in which characteristics of the individual that represent stable personality factors and phenomenological properties of the current trauma memory combine to account for severity of PTSS. This work also provides empirical evidence of factors that are most likely to be important in treating PTSD. In particular, our findings suggest that intervention and treatment programs targeted at altering particular properties of individuals' trauma memories, such as diminishing the physiological experience of the trauma memory and the centrality of the memory to one's personal identity, may be effective at reducing PTSS.

In addition to advancing our understanding of post-traumatic outcomes in older adults, our research on the UNCAHS cohort has contributed to the