CHAPTER 27

Biological Models of Behavior and the Life Course

MICHAEL J. SHANAHAN
SCOTT M. HOFER
LILLY SHANAHAN

Many topics of interest to life course sociology are linked in significant ways to biological processes. These topics include, for example, trajectories of physical and mental health, the stress process, patterns of aggression and deviance, sexual behavior, fertility, parenting, and manifold dimensions of aging and mortality. Many other topics are also likely to be linked to biological processes, albeit less conspicuously, including educational and occupational careers, patterns of close interpersonal relationships both within and beyond the family, and one's involvement and status in organizations. With few notable exceptions, relatively little interest has been expressed in these possibilities to date. Yet as George (*this volume*) notes, the future of the life course will hopefully be characterized by its intellectual exchanges with other subfields. Given their many plausible links to the life course, biological models of behavior are excellent candidates for interdisciplinary research.

In this chapter we explore connections between life course sociology and contemporary biological approaches to human behavior. We focus on three such approaches: life-history theory, behavioral genetics, and behavioral endocrinology. Because all three subfields are vibrant, large areas of inquiry, our scope is necessarily limited: for each area, we provide a brief introduction

MICHAEL J. SHANAHAN • Department of Sociology, University of North Carolina, Chapel Hill, North Carolina 27599-3210

SCOTT M. HOFER AND LILLY SHANAHAN • Pennsylvania State University, University Park, Pennsylvania 16802-6504

Handbook of the Life Course, edited by Jeylan T. Mortimer and Michael J. Shanahan. Kluwer Academic/Plenum Publishers, New York, 2003.

to the biological principles that inform the perspective, an overview of characteristic themes and analytic strategies, and points of connection with the life course paradigm. That is, we do not consider specific areas of investigation in detail but rather explore possible links between these biological models and the life course at a thematic level. Moreover, because of the paucity of prior exchanges between biology and life course studies, our overview is largely conceptual in nature. Given space limitations, we focus on micro dimensions of the life course, with special attention to how social and biological forces jointly shape transitions between roles, and patterns of continuity and discontinuity that extend across the phases of life. We begin with several observations about both the appropriateness and the hazards of linking biology and sociology.

BIOLOGICAL AND SOCIAL FORCES: CAUTIONARY TALES AND PROMISE

The links between biology and sociology have not always been harmonious. The cases of Herbert Spencer (Peel, 1971) and his American champion Lester Ward (Scott, 1976) are instructive. A key proposition of Spencer's Synthetic Philosophy was that society and its institutions would become progressively complex, reflecting a universal, evolutionary process toward greater differentiation. This proposition predated the publication of *The origin of species* (1859) and was unfortunately at odds with Darwin's emphasis on natural selection as the primary mechanism of evolution. According to Darwin and his early followers, natural selection meant that organisms adapt to their ecological settings and this adaptation may or may not lead to a more complex organism. That is, evolution by natural selection was not viewed as inherently "progressive," but rather as adaptive to the prevailing conditions. Spencer was thus forced to reject natural selection as the predominant force of evolution in order to salvage his belief in the ever-increasing differentiation of society.

Lester Ward extolled the virtues of Spencer's vision and promoted sociology as a science that could hasten this progressive differentiation through social interventions. For Ward, the efficacy of these interventions hinged on Lamarckian inheritance, which held that characteristics acquired by one generation during their lifetime could be transmitted to their offspring. Thus, successful interventions in one generation would have lasting effects down the familial lineage. With the re-discovery and widespread appreciation of Mendel's Laws—which described basic rules of the heredity of innate, not acquired, characteristics—Ward believed that either Mendel was wrong or sociology and its interventions would hardly be worth the effort. In the final analysis, he chose his brand of sociology over his understanding of Mendel.

These case studies would be interesting footnotes to the history of the behavioral sciences except that many sociologists continue to resist the integration of biology with sociology, fearing that biological models run contrary to cherished values about the malleability of people and the importance of progress (not unlike both Spencer's and Ward's suspicions about natural selection) or that an appreciation of biology could lead to a simple-minded reductionism that views biological forces as necessary and sufficient conditions for behavior (not unlike Ward's fear of Mendelian genetics). Yet such reactions are both unwarranted and counter-productive. Contemporary biological theories of human behavior are not inherently laden with social values nor are they reductionistic. Rather, biological models of behavior have

undergone nothing short of a paradigm shift in the last few decades, leading to the widespread assumption that "nature and nurture" interact in complex ways and indeed are often inseparable.

Such a view is consistent with propositions of systems theory (Ford & Lerner, 1992), several of which are especially relevant to a discussion of biology and the life course. First, human behavior is the product of multiple levels of analysis, including, for example, levels characteristically associated with sociology, psychology, biology, and anthropology. By extension, there is no *a priori* reason to believe that any one level will have special explanatory value. For example, genes do not simply cause behavior (Gottlieb, 1996) and, at the same time, behavior is not purely the result of social forces. Second, all levels of analysis are characterized by plasticity, which refers to the range of behavioral possibilities (Lerner, 1984). Thus, each person's behaviors represent one set of possibilities from among a finite range of possibilities; similarly, every social order represents one form of organization out of a range of possible social orders. Third, although each level is likely to operate according to its own laws, the levels interact to produce behavior (Cairns, McGuire et al., 1993). That is, systems theory assumes that many factors at multiple levels interact to form sets of "correlated constraints" that include the behaviors of interest and their covariates. These behaviors and their covariates represent organized systems, and ongoing reciprocal interactions among their levels explain continuity and provide a map of opportunities for change.

When viewed jointly, these principles define a central theme of life history theory, behavioral genetics, and behavioral endocrinology, and consequently of our chapter: *Social and biological forces interact in complex and dynamic ways to define ranges of likely behaviors*. By itself, this theme acknowledges the importance of context and its interplay with biology. Yet our review suggests a second overarching theme that links biological models of behavior to the life course more directly: *Behavior reflects a lifetime of reciprocal exchanges between person (including biological make-up) and context*. This theme acknowledges that behavior cannot be fully understood without reference to prior experience and indeed each of the three biological models that we consider promotes concepts consistent with this view.

Although biological models of behavior and life course sociology are thus thematically well-suited for interdisciplinary collaborations, care must nevertheless be exercised in avoiding the "the twin dangers of destructive cynicism and gullible expectations" (Rutter, 2002, p. 1). Gullible expectations are understandable given the excitement that currently surrounds developments in the biological sciences. In truth, however, once behavior is viewed as a product of long-standing interactions between biology and social context, the causal field becomes exceedingly complex. Quick progress is thus highly unlikely, as has already been demonstrated in the study of psychopathology (Rutter, 2002). Indeed, some scientists familiar with newly emerging insights from biology have tended toward a skeptical view that emphasizes the intractable contingency that characterizes how biological and social forces jointly produce behaviors. Whether such skepticism is warranted, however, can only be resolved through empirical study that accurately and dynamically assesses both biological and social features of the developing person and changing context. This phase of empirical research is only just beginning, and the purpose of our chapter is to consider, in very broad terms, the ways in which life course sociologists can contribute to these research efforts, and the ways in which biological models of behavior might inform life course research.

LIFE HISTORY THEORY AND THE LIFE COURSE

Although several Darwinian approaches to behavior are often recognized,* life-history theory is most closely connected with life course sociology because of their mutual interest in transitions and connections among the phases of life (Charnov, 1993; Hill & Kaplan, 1999; Stearns, 1992). Life history theory applies principles of evolution to biological features of the life course that have demographic consequences (Stearns, 1992). For example, in his path-setting research, Cole (1954) observed that patterns of sexual maturation and reproduction in the lives of people have, when viewed in the aggregate, consequences for the size of the population. An understanding of the dynamics of a population thus depends on an understanding of how factors that relate to reproduction and survival actually occur in the lives of individual people.

These factors, or life-history traits, include, for example, patterns of growth, age of maturity, number and spacing of offspring, parental investments, mortality schedules, and length of life (Hill & Kaplan, 1999). Clearly, many of these concerns are linked to topics of interest to life course sociologists, including the pubertal transition, features of the family cycle (e.g., the transition to parenthood), and morbidity and mortality. Although life-history theory draws on evolutionary principles broadly, it characteristically emphasizes how natural selection favors specific constellations of these traits as they optimize survival and reproduction in a given time and place. We briefly review relevant principles of Darwinian evolution and then apply these principles to life history theory and research.

The Life History Perspective

Evolution by natural selection holds that there is variation in one or more characteristics in every generation, that heritable similarities exist between parents and their offspring, that some organisms are better suited to survive and reproduce in a particular environment than others, and that the proportion of these better-adapted individuals in the population will increase through time.† These central ideas are actually part of five theories that comprise the evolutionary synthesis of "variational evolution" (Mayr, 1982). One of these central ideas, natural selection, has special relevance for life-history theory.

The theory of natural selection provides a mechanism by which evolution occurs. Selection refers to differential survival and reproduction based on traits. Many biologists believe that selection works directly on the phenotype, which refers to the totality of all

*Evolutionary psychology tends to focus on naturally selected psychological mechanisms that ultimately promote survival and reproduction; these mechanisms include, most prominently, aspects of emotions, cognition, and motivation. Sociobiology focuses on fitness-enhancing mechanisms that are associated with social psychology, family and kin relations, and group behavior (Nielsen, 1994). Life history theory represents a subfield of behavioral ecology, an anthropological approach concerned with the material circumstances of ecologies and the adaptive responses that they evoke (Winterhalder & Alden Smith, 2000). A closely related anthropological approach, dual inheritance theory, emphasizes the interrelated evolution of genes and culture (Durham, 1990, 1992). While all of these perspectives are relevant to life course sociology in varying degrees—for example, for studies of educational and income trajectories inspired by evolutionary theories of kin investment, see Case et al., 2001 and Anderson, 2000—we focus on life history theory, which shares with life course sociology a central interest in the interconnectedness of life's phases.

†For a basic introduction to evolutionary theory, see Mayr (2001); for a specialized treatment, see Futuyma (1998). This summary is based on Futuyma & Mayr (1982) and necessarily glosses over many nuances in evolutionary biology.

observable features of an organism, including behavior. The response to selection refers to changes in the population across generations; these responses may include changes in phenotypes (i.e., the products of genes, including behaviors), genotypes (the genes of an organism), or both. Natural selection favors individuals who are "fit" or "adapted," although in many cases the opposite (i.e., the elimination of the "unfit") may be a more accurate characterization. Thus, adaptive traits "promote fitness," which means that they are positively associated with survival and especially with reproduction.

What is actually selected? According to life-history theory, natural selection determines the balance in trade-offs, which occurs when change in one trait is associated with change in another trait. Life-history recognizes over 45 such trade-offs (Stearns, 1992), including, for example, quantity versus quality of off-spring: as the quantity of off-spring increases, their "quality" (referring to, for example, the likelihood of successful maturation) decreases. Basically, natural selection will favor the right mix of quantity and quality so that reproductive fitness is optimized in a given setting. Thus, the biological life course may be viewed as a complex set of evolved trade-offs surrounding growth, maintenance of the self, and reproduction. In fact, many of the trade-offs link the early and later parts of the life course as one finds, for example, in the trade-off between age of sexual maturity and longevity: as age of sexual maturity decreases, longevity decreases. All of the trade-offs form a relatively cohesive set that describes how the phases of life are coordinated for a species. Thus, one focal point of life history research is the comparison of trade-offs across species (e.g., Charnov, 1993).

Some Darwinians maintain that behaviors were selected for their fitness through the late Miocene and Pleistocene, when humans are thought to have lived as nomadic hunter-gatherers in small groups on dry, cool savannas or perhaps in grassy woodlands. For these scholars, evolution favored features of human life—including body plan, metabolic processes, behaviors, and cognition and emotion—that optimized survival and reproduction to this "environment of evolutionary adaptedness." Life history research generally rejects this notion for several reasons (see Foley, 1995; Irons, 1998). Most importantly, the vast expanse during which humans evolved was characterized by substantial variability in environments both locally and globally (Potts, 1998).

Based on this variability in contexts, life history theory holds that natural selection has favored variability in behavior within a species. Accordingly, *natural selection in humans has produced mechanisms (not finished traits) by which features of context evoke a circumscribed range of adaptive behaviors* from a larger set of behavioral possibilities (Hill & Hurtado, 1996). Based on this supposition, a second focal point of life history research is how the specifics of context, life-history traits, and survival and reproduction are interrelated within a species.

Life History Research: Analytic Strategies and Themes

An example of the first focal point, which addresses between-species differences, comes from studies of the origins of menopause, which is believed to be virtually unique to humans. These studies typically focus on the possible functions of menopause when viewed in the context of other life-history traits. Several hypotheses now attempt to explain menopause in this way. Williams (1957) originally formulated the "grandmother hypothesis," which states that as women age, their evolved "strategy" to pass their genes to subsequent generations shifts from their own reproduction to the care of their children and grandchildren (for a related hypothesis, see Scott Peccei, 2001). That is, at a certain point in life, their reproductive fitness was

optimized if they stopped having children and nurtured their children's children. Williams's hypothesis has been tested in hunter-gatherer societies by measuring the nutritional contributions of grandmothers to their offspring. The evidence is inconclusive at present, however, due to methodological and modeling problems (Blurton Jones, 1999; Hill & Hurtado, 1996). In any event, this line of research illustrates how life history research characteristically posits interactions between the social context—in this case, mode of subsistence and provisioning roles—and biology to explain features of the life course.

Of greater relevance to life course sociology are studies that reflect the second focal point, addressing how life-history traits "work" in specific contexts. This is a question based on the premise that evolution has selected variability and that contexts evoke responses from a range of possibilities. An example of this mode of inquiry concerns reproductive strategies. According to one prominent line of thinking, humans have evolved such that the first 5–7 years of life shape their subsequent pair-bonding and child-rearing behavior (Draper & Harpending, 1982). Specifically, children from families with marital discord, high stress, and inadequate resources are subject to harsh and inconsistent parenting; consequently, they develop an insecure attachment and opportunistic interpersonal orientation, leading to early puberty, early sexual activity, unstable pair bonds, and low levels of parental investment (Belsky, Steinberg et al., 1991). That is, in contexts marked by uncertainty, earlier reproduction is favored. At the other end of the continuum, children from homes characterized by spousal harmony and adequate resources receive responsive, positive parenting, and then develop a secure attachment, leading to later puberty, later sexual activity, long-term bonding, and high levels of parental investment.

Empirical research does not support all of this model's predictions, although there is considerable evidence that a range of stressors in the family are negatively associated with age of menarche (e.g., Moffitt, Caspi et al., 1992; Kim & Smith, 1998; Ellis & Graber, 2000, who also test a "step-father presence" hypothesis). In reviewing research related to this model, Surbey (1998) suggests that contextual cues associated with parental investment and the stability of the context affect the timing of menarche. These cues encompass a wide range of factors that bear on access to resources and environmental stressors, possibly including the presence of biological parents, the quality of the parent–child relationship, socioeconomic status, family size, birth order, and environmental stressors.

Taken together, these lines of research suggest that cohesive patterns of puberty, sexual behavior, and parental investment represent fitness-enhancing responses to earlier contextual cues in the family. Critics note that empirical support for the model is spotty, and some of its predictions run contrary to expectations derived from behavioral endocrinology (e.g., Susman et al., 1989). Nevertheless, the model illustrates an area of research that has been informed by principles of life-history theory, interweaving social roles, family dynamics, context, and biological features of the life course. Other lines of research address issues surrounding multiple aspects of the family cycle (including fertility and parenting), health and well-being, and longevity (see Hill & Kaplan, 1999, for a concise overview).

Integrating the Life History and Life Course Paradigms

Life history theory and the life course share common ground in that life phases and social roles are often intimately tied to biological events or trends. Further, both perspectives share the view that behavior reflects life-long interactions between persons and their contexts.

We can identify three advantages to an awareness of life history research by life course sociologists. First, with its emphasis on natural selection, life history theory alerts life course sociologists that human lives have evolved and thus behaviors may reflect selection pressures. That is, life course sociology should begin to entertain the possibility that behaviors can enhance fitness in response to contextual cues. Second, evolutionary theory can, in principle, serve as a broad framework that integrates diverse findings and leads to the generation of new hypotheses and explanations. Hence, evolutionary theory has the potential to provide an overarching "distal frame" for mid-range research. Life-history research is certainly a lively field of emerging hypotheses that reflect biology-context interactions, and it would be unfortunate if sociologists remain oblivious or uninterested in these developments. Third, life history research often draws on cross-cultural comparisons, which greatly expand the variability to be explained in human lives. Unfortunately, by restricting its attention to contemporary Western societies, life course sociology has ignored significant variability in contexts, behaviors, and the allocation of roles with age (Dannefer, *this volume*).

At the same time, attempts to integrate life history and life course research should proceed with caution. The enhancement of reproductive fitness is a distinguishing feature of all evolutionary explanations based on natural selection, although fitness is in fact rarely measured, perhaps because it is notoriously difficult to define and assess (Beatty, 1992). The problem takes several different forms. For Darwinian theories that posit an environment of evolutionary adaptedness in the Pleistocene epoch, reproductive fitness is probably impossible to measure (e.g., Belsky et al., 1991, p. 649). At the other end of the historical spectrum, for applications of life-history theory to societies that have experienced the demographic transition (involving high levels of contraception), new ways of thinking about fitness are undoubtedly necessary (e.g., Perusse, 1993 and accompanying commentary). Whatever the society being studied, if an enhancement in reproductive fitness has not been empirically addressed in some fashion, then, strictly speaking, a Darwinian theory has not been directly tested. In fact, predictions derived from evolutionary theory but not concerned with fitness are often tested. These predictions can often be derived, however, from other theories that carry fewer assumptions (e.g., Maccoby, 1991 for alternatives to Belsky et al., 1991).

Furthermore, mechanisms not related to natural selection can account for phylogenetic change (Mayr, 1983). Darwinians often assume that behavior has evolved to optimize reproductive fitness, but many behaviors may not be the products of natural selection or, in the alternative, they may be selected but highly imperfect optimizations. Most constraints on selection and optimization reflect the fact that selection acts on holisms, not on a single, atomistic trait. In the context of this holism, selection is a compromise between advantages associated with different organs, portions of the life cycle, and environments. For example, a gene or genes may be naturally selected because of their beneficial effects early in the life course, although they have negative effects in the later course (i.e., antagonistic pleiotropy, which some scholars believe explains menopause, see Scott Peccei, 2001). Furthermore, a specific selected response to an environmental challenge is likely to limit future possibilities for change (i.e., a developmental constraint). Or the selection of one feature may bring with it a whole suite of non-adaptive features (i.e., a hitch-hiking effect). These and other possibilities caution against the simplistic notion that life-history traits have been selected to optimize fitness; more likely, they reflect highly complex combinations of several distinct processes occurring over millions of years (e.g., Vrba, 1990).

These criticisms are not meant to suggest that evolutionary theories of behavior are untestable. In some cases, theories are testable in crucial respects—including claims about reproductive fitness—according to conventions of the behavioral sciences (e.g., Blurton Jones, 1993;

Borgerhoff Mulder, 2000; Hill & Hurtado, 1996; Kaplan et al., 2000). Such tests typically involve the development of mathematical models that suggest optimizations with respect to, for example, age of sexual maturity and longevity, or quantity-quality trade-offs in offspring investment (Hill & Kaplan, 1999). On the other hand, the distinctly Darwinian elements of a theory may be untestable in any sense whatever. In this case, however, selection and optimization could be an "untestable core" that nevertheless leads to unique testable propositions—in much the same way that many nascent theories in the natural sciences have done (e.g., the theory of natural selection before Mendelian genetics). To some scholars, this untestable core is an unnecessary extrapolation; to others, it provides distal mechanisms that, combined with empirical evidence of more proximal processes, offers a holistic explanation.

In the final analysis, the utility of life history theory to the study of lives is an empirical question that is being answered slowly through the development of precise models and the collection of appropriate data. In evaluating evolutionary research, sociologists need to be cautious about what parts of a theory can and cannot be tested, and indeed what claims have and have not been tested (Kacelnik & Krebs, 1997). Ultimately, however, the question is not *whether* evolution has shaped behavior, but *how* (Betzig, 1988). Many subfields are seeking to answer this question and, in the least, life course sociologists should be aware of these developments.

BEHAVIORAL GENETICS AND THE LIFE COURSE

Life-history theory typically does not consider genetic information. In some instances, life history research focuses on variability in behaviors between species (e.g., the timing of puberty and life-span across species of primates), in which case the explanation does not concern genetic variability within species. In other instances, the focal point is variability in behaviors within a species (e.g., the timing of puberty in humans); in these cases, the central inquiry nevertheless remains focused on how context predictably canalizes (or contrains) observed behaviors.

In contrast, behavioral genetics focuses on the association between variation in genotypes (i.e., genetic make-up) and phenotypes (i.e., products of genes, including behaviors) within a population, and the mechanisms that account for these associations. The complex behaviors that are of interest to behavioral genetics can arise from two or more genes working together, from different contexts that evoke different phenotypes from the same genome, and from complex interactions among multiple genes and environments. We begin by sketching basic principles of genetics that are especially relevant to the study of behavior,* and then consider analytic strategies and connections with the life course.

Basic Principles of Genetics and Behavior

The nucleus of the cell contains chromosomes, structures that house the DNA molecule and that become visible to the optical microscope during cell division. The gene is the fundamental unit of heredity, consisting of a length of DNA at a particular chromosomal location (or locus). Genes code for proteins (specific sequences of 20 different amino acids) and these

*For an excellent introduction to behavioral genetics, see Plomin et al., (2000) and Falconer & Mackay (1996).

proteins in turn perform a wide array of functions, operating as enzymes, regulators of other processes, chemical messengers, and as structural components within the cell. (Proteomics, one aspect of the study of genetics, includes the identification and description of proteins and their role in biological systems.) Genes can take on different forms, known as "alleles," which may lead to variations in phenotypes. Behavioral genetics therefore focuses on those genes that exhibit allelic differences within populations.

The link between genes and behaviors is mediated by chains of biological processes that are almost certainly complex and currently not well understood. Nevertheless, much can be learned about associations between genes and phenotypes without a detailed understanding of all of the mechanisms that link them. In a relatively simple case, differences in continuously distributed traits (e.g., height) could arise from a few or many genes, each having an additive (and probably small) effect on the phenotype. Alternatively, dominance occurs when a particular allele exhibits a stronger effect than expected by an additive model.

These connections are likely to be intricate, however, because of complications surrounding genetic expression, which refers to the mechanisms by which specific genotypes are associated with specific phenotypes. One class of mechanisms involves interactions among genes, which occur when a gene's effect is contingent on other genes in non-linear ways. For example, multifactorial phenotypes likely result from the interactions among many genes (McClearn et al., 2001). Even if the complexities of genes themselves were understood, a second class of mechanisms precludes a simple mapping of genotypes onto phenotypes, as acknowledged by the norm of reaction, which refers to the unpredictable nature of phenotypes associated with the same genotype expressed across different contexts (Gottlieb, 1995, 1998).

Thus, genotypes and all levels of the system are likely to interact in nonlinear ways that preclude simple links between genotypes and phenotypes across different environments. Cattell (1963, 1965) was perhaps the first to describe systematically the interactions and correlations that reflect this complex interplay among genotypes, environments, and phenotypes. A gene-environment interaction ($G \times E$) refers to the differential responses of organisms (genotypes) to particular settings, whereas a gene-environment correlation refers to the contextual features that are associated with the genetic predisposition of the individual. Penetrance refers to the probability of expressing a phenotype given a particular genotype. Incomplete penetrance is thought to reflect stochastic processes or interactions such as Cattell identified that are related to differing genetic and environmental backgrounds.

Cattell's approach was simplified to a smaller set of three types of correlations and interactions (Plomin et al., 1977) that are widely referenced today. The passive correlation refers to situations in which children inherit their parents' genes and grow up in settings that have been substantially shaped by those same genetic influences from the parents. In these situations, settings tend not be correlated or less correlated with the child's behaviors once the genotype has been controlled. The reactive correlation refers to situations in which the setting "reacts" to the child in ways consonant with his or her genotype. For example, parents may be more affectionate to children who exhibit warmth. Once again, when the genetic proportions of variance in the children are accounted for (genetic influences on parent and offspring warmth), the parents' degree of affection with the child may not be directly associated with the child's behavior. The active correlation refers to the person actively selecting and molding settings that are congruent with his or her genetic endowment. For example, a person with a genotype favoring high fluid reasoning ability may chose work that is substantively complex, which tends to provide further opportunities for enhanced intellectual functioning. (For extended examples of these correlations as applied to the transition to adulthood, see Plomin et al., 1977; Shanahan et al., 2000). Empirical evidence for particular gene-environment

correlations and interactions, however, is sparse and so these modes of action are presently hypothetical.

Behavioral Genetics: Analytic Strategies and Themes

These complex links between genotypes and phenotypes have been studied from two vantage points that depend on whether or not the genotype has been directly measured. These two approaches yield different insights about links between genotypes and phenotypes. The decomposition of variance approach is used when the genotype has not been measured; this approach focuses on phenotypic associations across groups that vary in degrees of genetic relatedness. Models typically focus on estimating proportions of variance associated with genetic and environmental influences. Because this approach is correlational, results describe populations, not individuals. In contrast, studies involving the measured genotype permit statements about individual development, particularly if interactions with measured environments are considered. We briefly introduce each approach, review its strengths and weaknesses, and note connections with life course sociology.

Decomposition of variance. A population approach seeks to partition variance in behavior according to aggregate genetic and environmental influences, and their interactions. A major focal point of this line of research is heritability (h^2), an estimate of genetic variance associated with phenotypic variance, expressed as the proportion of the total phenotypic variance in a population. *Quantitative genetic analysis* involves the analysis of genetically related individuals to understand total phenotypic variance (P) in a population in terms of heritable (G) and environmental (E) sources of variance, most simply:

$$V_P = V_G + V_E \tag{1}$$

A more complete representation of the decomposition of total reliable phenotypic variance contains additional terms, shown in the variance equation below. With certain samples and designs, genetic variance (V_G) can be decomposed into additive (A), dominance (D), and epistatic (I) sources which represent gene–gene interactions (McClearn, 2001). Environmental variance (V_E) can be separated into common or shared (C) and nonshared (E) components. Additionally, covariance and interaction among these genetic and environmental sources may be included (shown respectively as two simple additional components in the Equation (2), 2Cov_{GE} and V_{GXE}:

$$V_P = V_A + V_D + V_I + V_C + V_E + 2\text{Cov}_{GE} + V_{GXE} \tag{2}$$

All sources of phenotypic variance cannot be accounted for simultaneously in most genetically informative designs (i.e., twin, parent–offspring) and the additional role that time (i.e., development, aging) has on the system of influences must also be considered. Some family designs, however, are more powerful than others for evaluating particular effects (e.g., adoption designs for evaluating common environmental influences) and longitudinal genetic studies permit some understanding of the interactive effects of genes and environment over time. The basic twin design relies on the fact that monozygotic (MZ) twins share 100% of their segregating genes while dizygotic twins (DZ) share 50%, on average. If genetic factors are important for the variability in a trait, the correlation among MZ twins must be higher than among dizygotic twins as seen in comparison of their intraclass correlations. A simple estimate of heritability in twin samples is given as twice the difference between the MZ and

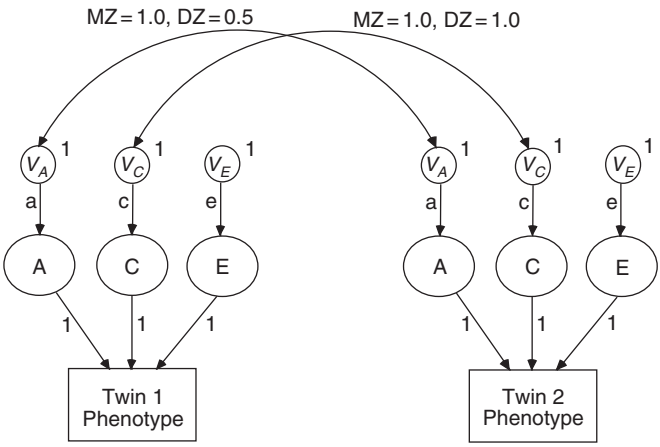

Figure 27-1. The expected correlations among twins.

DZ intraclass correlations [$h^2 = 2(r_{MZ} - r_{DZ})$]. To the extent that shared environment (c^2) is important for a trait, both MZ and DZ intraclass correlations will be positive and higher than expected given the genetic components of variance in the model ($c^2 = r_{MZ} - h^2$).

Maximum likelihood estimation is often used to obtain parameter estimates (e.g., abstract variances of additive genetic, shared and nonshared environment) by maximizing model estimates that make the observed and expected covariances (correlations) among twin pairs as similar as possible. The expected correlations, shown in Figure 27-1, are fixed and based on the expectations for genetic relatedness among twins (A; $r_{MZ} = 1.0$, $r_{DZ} = 0.50$), shared or common environment (C; $r = 1.0$ for both MZ and DZ twins), and nonshared environment (E; $r = 0.0$, path not shown). The paths from A, C, and E are fixed to 1.0 to identify these as variance components. Models such as this one, based on known expectations of genetic relatedness, are easily expanded to extended pedigrees including parents, siblings, half-siblings, and other designs such as adoption studies where siblings are reared apart (Neale & Cardon 1992).

This model and its many variants (e.g., extending into longitudinal models) yield information about the proportion of variance in a behavior that is attributable to genetic variance in a population and to environmental influences that make individuals more similar (C) or more different (E). For the many decades during which the available data did not include direct measurement of the genotype, this approach to the genotype–phenotype link proved immensely popular. Numerous elegant expansions on this basic model have been developed, including models for assortative (nonrandom) mating (Heath, 1985), sex-limitation (differential sex effects), cultural transmission (Eaves, Fulker, & Heath, 1989), and longitudinal models (Eaves, Hewitt, Meyer, & Neale, 1990) including the phenotypic simplex (Boomsa, 1987) and latent growth curve models (McArdle, 1990).

At the same time, weaknesses to this general modeling strategy are widely understood and acknowledged. Three major concerns are worth noting here. First, however it is calculated, heritability is an estimate of the proportion of genetic variance in a population. As with any correlation, a large number of underlying patterns among the genetic and environmental influences can produce an estimate of heritability in a population at a particular time. Care must be exercised in avoiding the ecological fallacy: a heritability estimate does not warrant statements about the genotype–phenotype link at the level of the individual because it says

nothing about genetic expression (Gottlieb, 1995). Second, consistent with the mechanisms of genetic expression discussed earlier, a simple additive model for heritability may be unlikely for many complex phenotypes (McClearn & Vogler, 2001). Third, h^2 has some unusual properties that, if viewed uncritically, can be misleading. For example, low estimates of heritability do not necessarily imply that genes have less influence on particular phenotypes than higher estimates; it may be that these traits have undergone strong evolutionary selection (i.e., there is little genetic variability) but are nevertheless under a high degree of genetic control (Turkheimer, 1998). On the other hand, highly heritable phenotypes (such as height) do not imply that context is unimportant as secular trends in height vividly illustrate: Height is highly heritable, but it has increased substantially in the West through the 20th century because of improvements in nutrition and other factors.

The analysis of related individuals also leads to models of the Mendelian transmission of major gene effects. For example, in an Icelandic study of breast cancer, segregation analysis was used to evaluate the genetic influence on the age of onset of breast cancer (Baffoe-Bonnie, 2000). Segregation analysis draws on pedigrees (related individuals) to estimate the underlying Mendelian inheritance patterns (e.g., allelic frequency, penetrance) most likely to account for familial transmission of the outcome. In this case, a codominance model was found to fit the pedigree data best; such a model implies that the effects of the allelic variants (homozygous, heterozygous) are not additive but exhibit nonlinear risks in terms of age of onset and cumulative risk. The estimated age of onset for high-risk homozygotes, heterozygotes, and low-risk homozygotes was 51.8, 64.0, and 76.3 years, respectively, with the corresponding cumulative risk of breast cancer by age 60 estimated at 32.2%, 16.4%, and 5.0% for these same groups.

Analysis of the measured genotype. A second approach to the genotype–phenotype link has emerged from the increasingly likely situation in which the genotype has been measured directly (i.e., not inferred through degrees of relatedness to other people in the sample). The fundamental aim is to identify regions of the chromosome that contribute to the variation in quantitative traits, where quantitative traits are complex, multifactorial phenotypes. This approach relies on genetic markers, known as quantitative trait loci (QTL), which are markers of regions of a chromosome that contain multiple genes. The analysis of QTL can be performed in experimental populations (McClearn, 1999) or in naturally segregating populations. The availability of increasingly dense maps of the human genome permits detection of genes with weak effects, thus increasing the potential for identifying multiple genetic factors that can influence complex phenotypes.

In it simplest form, the association between particular genetic markers and quantitative traits take the form of regression analysis (or analysis of variance) where phenotypic values are regressed onto the coded values for allelic variants of particular genes or quantitative trait loci (QTL; segregating regions of a chromosome that contain one or multiple genes). Essentially, the analysis focuses on mean differences across classes of the measured genotype where all individuals having a particular allele would be considered to be of the same class (identical by descent or state). The proportion of phenotypic variance accounted for by a particular genetic locus (QTL) would be indicated by the R^2 value.*

*In many cases, the analytic strategy is often much more elaborate than a regression model. For example, genetic association analysis is one approach to map measured genotype on quantitative traits but this method is sensitive to population stratification (subgroups having different frequencies of alleles). Alternatively, linkage analysis (Ott, 1999) makes use of extended pedigree information to overcome these problems. Once a QTL is localized, higher resolution mapping of the QTL segment is usually performed in order to identify the precise location of the chromosome that is related to the quantitative phenotype.

There are numerous challenges to the identification of particular genes for complex outcomes and these are problems that are under intensive study. For example, analyses are typically based on hundreds of genetic markers and this increases the problem of Type I error. Indeed, many QTL-based efforts to understand behavior have been characterized by a lack of replication across studies (e.g., studies of schizophrenia). Further complications include phenocopy (having phenotype but not genotype), incomplete penetrance (having the same genotype but not phenotype), and polygenic traits (heterogeneous genotypes resulting in same phenotype). However, the potential of discovery using both quantitative and molecular genetic approaches is high. In particular, interactive models of measured genotypes, phenotypes, and context will permit unique and valuable contributions for the study of persons in context.

Integrating Behavioral Genetics and the Life Course

Virtually all research on the life course has proceeded without considering the influence of genes on behavior; at the same time, behavioral genetics has proceeded without regard to the sophisticated models of social context that often characterize life course research. Yet, many lines of research have now established that genotypes do not produce behaviors in a simple way (Gottlieb, 1995, 1998). Instead, phenotypes are likely to reflect the cumulative history of the individual's genotype, phenotype, and context. Indeed, there is widespread appreciation among behavioral geneticists that the links between genotypes and phenotypes are often heavily conditioned by social location and personal experiences. One of the forefronts of behavioral genetics—exploring gene–context interactions over many years in the development of behaviors—connects directly to life course interests in long-term continuity and discontinuity. More specifically, however, we can identify a set of problems that provide strategic points of integration between behavioral genetics and the life course.

First, whether the genotype is measured or not, life course sociologists have much to offer in the measurement of contexts, particularly as they capture the richness of prior experience. The direct measurement of dynamic contextual influences within genetically informative designs is crucial for understanding developmental sequences, pathways, transitions, and outcomes across the life course. For example, life course demographers continue to learn about the quality of data that results from various measurement strategies for assessing families and households over time (Brynin & Smith, 1995; Duncan, 1985); similarly, life course researchers have been at the forefront of efforts to measure neighborhoods and communities, family interactions, the workplace, and schools, as well as contextual influences that result from multiple domains (e.g., connections between work-school). The last several decades of life course research have been marked by increasing interest in how these contexts are structured when viewed longitudinally and such a focus is clearly relevant to the description of gene-environment correlations and interactions in development.

These correlations and interactions reflect the person as influenced by context, the context as influenced by person, and the interplay of both. In a life course framework, with its emphasis on dynamic views of context and individual agency, these possibilities become particularly interesting. The active correlation implies that there are periods of development where individuals actively select their environments. Perhaps these periods may be found in transitions between life phases or age-graded social settings. The transition to adulthood may be one such period, raising the issue of how individual differences in genotype, context, and phenotype interact over a period of transition and change. Additionally, an emphasis on the

life course would focus on how adverse, immediate, or novel environmental changes might produce a reorganizing of individual differences, making genetically related individuals more similar if the events were shared and less similar if events differed across individuals. Such questions could be asked with respect to dramatic social changes, but also to comparatively normative transitions (e.g., the transitions from junior high school, to parenthood, and into retirement).

Second, life course sociologists interested in the description of populations can contribute to the study of how different contexts affect heritability. A growing body of research is focusing on the proportion of variance in a phenotype that is associated with genetic, shared environment, and nonshared environment across different populations defined by age or by social settings. Some researchers have hypothesized that heritability will increase with age, reflecting the increasing capacity of the person to select and mold his or her context, which in turn strengthens the active correlation. Studies of aging and heritability estimates typically compare the relative similarity of twins (by way of variance decomposition models) across subgroups defined by age.

Based on this strategy, research on cognitive functioning across the life-span indicates that the heritability of cognitive capabilities is relatively high and stable and may increase with age (Pedersen, 1996; McClearn & Vogler, 2001). However, the preponderance of evidence for age-related changes in the proportions of genetic and environmental influences has been obtained from cross-sectional designs. A limitation of these designs, particularly pertinent to twin studies that require the sampling of intact twin pairs across a wide age range, is nonrandom selection/survival processes leading to age-related population heterogeneity on outcomes of interest. In any event, the age-graded nature of heritability, and the processes that explain observed patterns, represent a strategic point of integration with life course sociology.

Similarly, a growing number of studies have shown that estimates of heritability are sensitive to context (Rose, 2001; Rowe et al., 1999). As Scarr (1974) explains, h^2 is a population average that does not necessarily apply to differences within and between subpopulations. Indeed, sociological theories often emphasize differential exposure to environmental conditions (e.g., stressors) across groups in a population, suggesting that the amount of variance in the phenotype accounted for by heritability will not infrequently vary across subgroups within a broadly defined population. Many, but certainly not all, studies in this area focus on how extreme circumstances can alter heritability, the assumption being that the normal range of settings provides many functionally equivalent opportunities to develop (Scarr, 1992). Further, most studies work from the assumption that strong settings increase the effect of the shared environment (c^2).

Several prominent hypotheses are being investigated along these lines. For example, Scarr (1974) argues that social disadvantages during prenatal and postnatal development can substantially lower the observed IQ among economically and socially marginalized groups, thereby reducing the genotype–phenotype correlation. Another hypothesis, proposed by Bronfenbrenner and Ceci's (1994) bioecological model, maintains that as proximal processes—defined as enduring forms of interaction that characterize a person's immediate setting—become progressively complex and strong, genetic potential is actualized, thereby increasing h^2 and the person's level of functioning. As they explain, "only those genetic predispositions of the individual can find realization for which the necessary *opportunity structures* exist, or are provided…" (p. 575, authors' emphasis). An additional hypothesis is that the regulatory power of values and norms can alter h^2, consistent with theories of social control. For example, Rose and his colleague show that the heritability of alcohol use is much

higher in urban than in rural areas of Finland, because the latter setting is characterized by greater residential stability and increased community monitoring (Dick et al., 2001).

These and related hypotheses can be explored with cohort studies. For example, consistent with the social control hypothesis, Dunne et al. (1997) report that birth cohort moderated the heritability of age of first intercourse in an Australian sample. Specifically, h^2 accounted for 32% and 0% of the variance in age of first intercourse for women and men born between 1922 and 1952, respectively, but 49% and 72% of the variance for women and men born between 1952 and 1965, respectively. The authors suggest that earlier born cohorts were constrained by the higher levels of social control, when compared with the content and force of social controls encountered by youth in later born cohorts.

Third, also in the context of heritability studies, life course sociologists could contribute to the discussion of shared and nonshared environmental influences. Shared environmental influences are nongenetic environmental factors that make family members more similar and may have either transient or long-term effects. Nonshared environmental influences are those influences that are not shared and make family members less alike. Variance decomposition models currently assign weights to these sources of influence by setting the parameters at 1 and 0, depending on whether a twin, for example, is separated at birth or not. As Turkheimer (2000) notes, however, nonshared sources of variance may often result from common environmental influences that make siblings different. These common environmental differences may be related to sibling interaction or differential parental treatment, or the differential effects of processes and events within the family (e.g., marital discord). Surprisingly little research has investigated the details of family life as they relate to the shared–nonshared distinction.

Despite these opportunities, efforts to integrate behavioral genetics and the life course should be cognizant of several caveats. We have already observed that genes do not lead in simple ways to behavior. In fact, given the complexity of gene–gene and gene–context interactions, statements about "the effect" of a gene or genes may well make little sense (McClearn et al., 2001). That is, *the behavioral range associated with a genotype is contingent on the co-presence of other genes and the conditioning effects of context, both of which play out in developmentally complex ways in individuals*. For example, in a study of the lifespan of *drosophila melanogaster* (the fruit-fly), genotype was significantly related to longevity but these effects were highly interactive with sex and environmental influences (Vieira et al., 2000).

Beyond this caution about the complexity of genetic expression, however, is a caveat that is not sufficiently acknowledged by behavioral scientists. Most models of behavior actually focus on the interaction of the phenotype with the environment (Turkheimer & Waldron, 2000). This *developmental phenotypic focus* is a more tractable approach (than a focus that includes genes) for sufficiently understanding development since environmental influences have effects on the phenotype directly. Such a strategy is particularly defensible if genes are a distal causal mechanism and their effects are largely or fully mediated by more proximal, measurable phenomena. For example, Kardia, Haviland, Ferrell, & Sing (1999) examined the link between apolipoprotein E genotypes (apoE) and coronary artery calcification (CAC). Their study concluded that apoE was not associated with CAC after the prediction of CAC was made by a fairly small set of observed risk factors. That is, beyond several readily measurable, phenotypic risk factors, apoE had no significant explanatory value. Similarly, in their study of the development of delinquency and criminality, Sampson and Laub (1993) observe that "there is little, if any, need to introduce biological models of heredity if the direct effect of parental criminality on delinquency is null and instead is mediated by … family functioning."

Thus, knowledge of the genotype may be necessary for a full explanation of a behavior (such as CAC), but not necessary for its prediction.

In the final analysis, genes and context undoubtedly are correlated and undoubtedly interact to produce behaviors. However complex these processes may be can only be resolved through the empirical study of genotypes, phenotypes, and contexts over time. The life course is especially well-suited to contribute to these efforts with its interest in the conceptualization and measurement of dynamic contexts, and with its thematic emphasis on the long-term processes by which people and their contexts are reciprocally interrelated.

BEHAVIORAL ENDOCRINOLOGY AND THE LIFE COURSE

Behavioral endocrinology focuses on the reciprocal links among hormones of the endocrine system, behavior, and context. The basic connections between these processes and evolution and genetics are straightforward. First, hormones have been subject to the forces of evolution (although, perhaps in most cases, the functions of hormones have changed through the millennia, but not their chemical structure). By extension, hormones probably contribute to reproductive fitness. Second, basal hormone levels are heritable and many hormonal effects directly or indirectly modify gene expression in the target cell. Efforts to link the endocrine system with behavior, however, rarely focus on evolutionary or genetic issues. Rather, behavioral endocrinology focuses on the presence, structure, and function of hormones and how they reciprocally interact with behavior and the individual's context.

Principles of Endocrinology

Hormones are chemical messengers that are produced, stored, and released by glands of the endocrine system.* Endocrine glands are located in different regions of the body and are characterized, in part, by their secretion of hormones directly into the blood stream, which is often triggered by a change in the concentration of a substance in the body. In turn, these secreted hormones can travel to specific binding sites (hormone receptors) located on the membrane of or within specific cells throughout the body. The hormone receptors then "translate" the message, initiating biochemical reactions that lead to altered functioning of the cell (e.g., via hormone-influenced DNA-synthesis). Because of the "lock and key" arrangement between specific hormones and their receptors (i.e., the hormone functions as a "key," opening the receptor-"lock" to influence metabolic processes of the cell), even very low concentrations of hormones in the bloodstream may be capable of regulating cellular functions.

In addition to the endocrine system, the study of hormones is also tied to the central nervous system via the neuroendocrine system. These two systems jointly regulate and coordinate bodily functions, such as metabolism or sexual reproduction, and adapt the body to changing short-term and long-term challenges, such as stress. The two systems are closely linked at the hypothalamic-pituitary interface, which is the decisive control center between hormonal and neural regulatory processes. Specifically, the hypothalamus, as a brain structure, and the pituitary gland, as part of the endocrine system, are organizers of the hypothalamic-pituitary axis that controls much of the endocrine system. The endocrine and the nervous

*This summary is based on Bierbaumer and Schmidt (1996), Nelson (2000), and Ojeda and Griffin (2000).

systems are also linked via the enervation of most endocrine glands, and the direct effects of hormones on the central nervous system.

Four classes of hormones are typically recognized. These four classes and their origins and functions are shown in Table 27-1. The table greatly simplifies the origins of hormones, which often involve, for example, complex cycles with other hormones produced by a variety of endocrine glands. The table also simplifies the functions of the listed hormones; in many cases, hormones perform a surprisingly wide array of known functions with the possibility of unknown functions as well. Despite these simplifications, however, Table 27-1 shows that the endocrine system is comprised of numerous glands located throughout the body. Further, hormones affect a wide range of biological processes, some of which, even at first inspection, are likely to correlate with behaviors associated with, for example, reproduction, reactions to stress, and physical and mental health.

Hormones are involved in maintaining homeostasis, which refers broadly to a biochemical balance among many subsystems. Levels of hormones are regulated in several ways. First, hormones are involved in feedback cycles (i.e., most commonly negative, but also possibly positive, or multiple hormone feedback cycles), in much the same way that a thermostat maintains temperature at a set level. For example, following a stressful event, levels of the corticotropin-releasing hormone (CRH) increase, which, in turn, leads to an increase in adrenocorticotrophin (ACTH). Cortisol is then released which, among other things, mobilizes nutrients, modifies the immune response, and regulates glucose concentrations in the blood. When large quantities of cortisol are set free, a negative feedback system causes a reduced output of ACTH and CRH, which, in turn, inhibits the release of cortisol. Second, hormones may also increase or decrease the number of their own receptors—which are continuously generated in response to the internal milieu (up- and down-regulation, respectively)—or regulate the receptors of other hormones (a permissive effect).

Because of these potentially intricate cycles and the interplay among numerous biological subsystems, hormones are often secreted in temporally complex ways. Nevertheless, most hormone secretion shows a high degree of temporal organization. Specifically, secretion of hormone levels is often a pulsatile/episodic rather than a continuous process. Hormones are produced in small amounts, and then released in bursts (or pulsatile secretions) that may be as frequent as every 5–10 min. Other temporal rhythms have also been observed.

Not surprisingly, the measurement of hormones in the study of human behavior is difficult owing to their complex biochemistry, their temporal patterns of secretion, the reactivity of some hormones (e.g., cortisol) to collection procedures, and to ethical and scientific issues surrounding the invasiveness of procedures. In fact, a surprisingly large number of studies do not directly measure hormones (e.g., testosterone or estradiol), relying instead on proxies such as whether a person is taking a medication (e.g., estrogen replacement, or a contraceptive), or whether a person belongs to a group characterized by specific hormone levels (e.g., post-menopausal women, pre- and post menarchal women, or a woman in a certain phase of the menstrual cycle). Direct measures of hormones can be obtained, however, by sampling blood (plasma), urine, or saliva. Increasingly reliable and valid measurement protocols for key hormones in behavioral studies are rapidly emerging (Granger, Schwartz et al., 1999), greatly facilitating the collection of accurate hormone data for social science research.

Analytic Themes and Life Course Research

Behavioral effects of hormones. Two sets of distinctions are often observed among behavioral endocrinologists. The first set concerns the nature of behavioral effects.

TABLE 27-1. Classes and Types of Hormones, Their Primary Origins, and General Functions (Simplified; see also Nelson, 2000)

Class and types (with examples)	Primary origins	General functions of example hormones
1. Steroid hormones		
a. Progestins (e.g., progesterone)	adrenal glands	pregnancy
b. Corticoids (e.g., cortisol)	adrenal glands	body functions
c. Androgens (e.g., testosterone)	gonads	reproduction, secondary sex characters, metabolism
d. Estrogens (e.g., estradiol)	gonads	reproduction, secondary sex characters, metabolism
2. Protein/polypeptide hormones		
a. Hypothalamic (e.g., dopamine)	hypothalamus	neurotransmitter
b. Anterior pituitary (e.g., gonadotropins)	pituitary	reproduction
c. Posterior pituitary (e.g., oxytocin)	pituitary	reproduction, suckling reflex
d. Thyroid (e.g., thyroxine)	thyroid	metabolism, growth/differentiation
e. Parathyroid (e.g., parathyroid hormone)	parathyroid	calcuim metabolism
f. Gut (secretin)	duodenal mucosa	digestive
g. Pancreatic (insulin)	pancreas	glucose regulation
h. Adrenal Medullary (enkephalins)	adrenal medulla	adaptation to stress
3. Monoamine hormones		
a. Adrenal Medullary (e.g., epinephrine)	adrenal medulla	circulatory and metabolic systems
b. Pineal Gland (e.g., melatonin)	pineal gland	puberty onset
4. Lipid-based hormones		
a. Prostaglandins (e.g., E group)	throughout body	reproduction

In the perinatal life-stages (immediately before and shortly after birth), hormones have organizational effects, which refer to direct effects of hormones on the architecture of the brain, body and the distribution of hormone receptors (Buchanan et al., 1992). These organizational effects can in turn expand or limit the range of possible developmental pathways through childhood and into adulthood. In later stages of life, hormones have activational effects, which refer to the initiation of specific behaviors through their contemporaneous influence on neural-based and peripheral processes (Buchanan et al., 1992; Susman, 1997). Activational effects are also considered indirect effects, whereas organizational effects have a direct impact on brain development. Among other things, activational effects can be dependent on earlier organizational effects of hormones on the development of brain structures.

For example, Udry (2000) investigated the long-term consequences of fetal exposure to androgens (masculinizing hormones) during the second trimester, a sensitive period in neurological development. In adulthood, testosterone is thought to be associated with sex dimorphic behaviors (i.e., any behaviors that are differentially distributed in a population based on sex) through its action "on genes in the central nervous system that control the production of neurotransmitters" (p. 444). Udry hypothesized that the effects of post pubertal testosterone (an activational effect) on the sex-typical behaviors of women would be contingent on prenatal masculinization of the brain (an organizational effect). Consistent with this expectation, results showed that as fetal exposure to androgens in the second trimester increased, the effects of adult testosterone (assessed some 30 years later) decreased.

A particularly important class of organizational effects may reflect how experience alters thresholds or sensitivity to subsequent events (Susman, 1993). For example, the kindling model was developed to explain why initial episodes of depression are often linked to stressful events, whereas subsequent depressive episodes are comparatively spontaneous. Similarly, Susman (1993) reports that adolescents have a "trait" of reactivity to potential stressors only under conditions of maximum novelty. This suggests a complex interplay between earlier (possibly organizational) experiences and later reactions to contextual cues. As she concludes, these biological findings underscore the importance of knowing the person's experiential history.

Hormones, context, and behavior over time. A second set of distinctions refers to how hormones and behaviors interrelate over time (Susman, 1997). The basal model (Mazur & Booth, 1998) holds that changes in individual hormone levels lead to changes in behavior. An alternative model holds that behavior causes hormone change. Although these models are now recognized as over-simplifications (Susman, 1997), a great deal of research in behavioral endocrinology is consistent with one or the other of these views, albeit often implicitly. Research in this tradition is typically characterized by relatively limited time frames involving cross-sectional designs or assessments extending across a few hours or days (Mazur & Michalek, 1998). Given historical difficulties in the measurement of hormones and the need for research in non-naturalistic settings, these limitations are understandable.

Nevertheless, these models can have implications for life course phenomena in several important respects. First, cross-sectional studies that collect retrospective life-history data can link life course patterns to hormone data, although they rely on the assumption that the hormone in question is quite stable across the events being studied. For example, Booth and Dabbs (1993) examine the connection between testosterone and marital adversity in a sample of former serviceman. Drawing on a cross-sectional design, they find that testosterone is significantly related to marital adversity, reflecting whether the man ever divorced or separated, had extramarital sex, or had been abusive. The relationship can perhaps be accounted for in part by testosterone's negative effects on socioeconomic attainment and positive effects on the likelihood of unemployment, trouble with the law, and problems with alcoholism.

Second, long-term stability in a link between a behavior and a hormone could produce an accumulation effect. An excellent example concerns the stress response, which is characterized by the activation of the hypothalamus, which triggers the pituitary gland to secrete adrenocorticoid hormone (ACTH), which in turn triggers the secretion of glucocorticoids (e.g., cortisol) by the adrenal glands. This process supports the fast mobilization of the body's energy resources, which may be necessary to neutralize the stressor. If glucocorticoids remain at high basal levels for long periods of time, however, the chances of chronic disease greatly increase, including damage to the nervous system, suppression of the immune and reproductive systems, hypertension, and ulcers. Drawing on this biological model of the stress reaction, Sapolsky, (1992, 2000; Sapolsky, Alberts et al., 1997) has studied the effects of social position on hypercortisolism in baboons, the underlying assumption being that behaviors associated with social position predict hormones (specifically, cortisol). In one study, he examined the effects of "reversal interactions," which refer to interactions indicative of a loss of social status. Sapolsky and his colleagues (1997) report that as reversal interactions with the nearest lower ranking males increase, basal cortisol levels also increase. Although the link between social status and cortisol has not been studied extensively in humans (Decker, 2000), animal models suggest that specific dynamic patterns of social status and basal cortisol could lead to impaired health.

Third, even models of limited duration can inform life course studies of transitions. The biological stress model could certainly apply to some life course transitions, but other hormone models may also be relevant, especially in the family cycle (Booth et al., 2000). For example, animal models suggest that men whose partners are expecting a child may experience changes in hormone levels. Consistent with these models, research shows that the father's testosterone peaks immediately after the birth of his child, and then quickly decreases in the postnatal period; such a pattern is consistent with a protective posture at birth, followed by nurturing, paternal behaviors (Storey et al., 2000). At the same time, prolactin—a hormone associated with parental behaviors in many animals—increases from the early to the late prenatal periods and is high among men who are especially responsive to the cries of infants (Booth et al., 2000).

Research is increasingly focusing on conceptual models that move beyond unidirectional links between hormones and behavior. Thus, the reciprocal model (Mazur & Booth, 1998) holds that the relationship between hormones and behavior is reciprocal (i.e., bidirectionally influencing each other). For example, Udry (1988) found that, among adolescent boys, androgens have a positive indirect effect on sexual behavior through church attendance: androgens decreased church attendance, which normally decreases sexual behavior. An alternative model, however, supports a different (although not mutually exclusive) causal chain: androgens increase sexual behavior, which in turn decreases church attendance. Bi-directional influence, suggested by this model, is also nicely illustrated by a possible feedback loop between assertiveness and individual testosterone levels (Mazur, 1985). Specifically, in puberty, increasing levels of testosterone lead to assertiveness, which, in turn, leads to increasing levels of testosterone.

The most relevant model for life course sociology includes contexts in addition to the reciprocal relationship between hormones and behaviors. This model is consistent with a biosocial modeling strategy, which encourages the merger of sociological and biological models of a given behavior. Udry (1988) demonstrated the potential for this approach with his studies of androgens and social control theory. The generic model posited that androgens affect problem/sexual behaviors both directly and indirectly through pubertal development and social controls. For example, he showed that among adolescent boys with high levels of unbound testosterone, as the number of siblings increased (i.e., a form of social control), problem behaviors decreased (Udry, 1990). Similarly, for adolescent girls who are involved in

sports and live with their biological fathers (again, forms of social control), the link between testosterone and sexual behavior becomes insignificant (Udry, 1988).

The interactive nature of hormones, context, and behaviors is likewise illustrated by Booth's studies of social integration and testosterone. For example, social integration may modify the association between delinquency and testosterone in young male adults. Specifically, young male adolescents who are socially integrated are less likely to be delinquent than young male adults who are not socially integrated (Booth & Osgood, 1993). In another study, Booth and his colleagues (1999) examine a curvilinear (u-shaped) relationship between testosterone and depression among men. They report that marriage and employment "cause the testosterone-depression link to recede to the point where men with above-average testosterone are no more likely to be depressed than men with average levels of testosterone" (p. 137). The authors explain that marriage and employment—as critical sources of social integration—serve as protective factors among high testosterone men.

In her overview of research on context, hormones, and sex dimorphic behaviors, Berenbaum (1998) observes parallels between gene–context correlations and interactions and the influence of hormones. Thus, people with differing levels of hormones may be exposed to different contexts because they chose them and/or because they were allocated to them (hormone–context correlations). For example, Booth and his colleagues (2000) review studies suggesting that testosterone is negatively related to success in school, occupational status, employment status, and marital status and positively associated with divorce and marital conflict. The developmental processes giving rise to these associations are not well understood, but they suggest that testosterone may be linked to the selection and allocation of these roles and career characteristics.

Further, people with different levels of hormones may respond differently to the same context (a hormone–context interaction)—a point illustrated in our discussion of reactivity. This is well illustrated by Udry's (2000) study of fetal exposure to androgens in the second trimester (discussed earlier). He reports that the effects of maternal encouragement of daughters (ages 5 to 15) to be feminine are contingent on androgen exposure during the mother's second trimester. Specifically, maternal encouragement had no effect on daughters exposed to the highest levels of androgens; conversely, encouragement had the maximum effect on daughters exposed to the lowest levels of androgens. As Udry explains, for women with high exposure to androgens, "no matter how much encouragement the mother provides it has little effect, and the daughter remains more masculine than average" (p. 450).

Integrating Behavioral Endocrinology and the Life Course

Developments in behavioral endocrinology have paved the way for collaborations with life course sociologists. First, behavioral endocrinologists recognize that hormones alone typically have modest explanatory value (Susman, 1997); rather, they interact with context in temporally complex ways to explain behavior. Second, although the designs of many earlier studies of hormones and behavior are cross-sectional or of limited duration, *sophisticated conceptual models often call for studies extending across the phases of life. As the preceding section documents, long-term processes include alterations in sensitivity to contexts, long-term sequences of organizational and activational effects involving complex chains of hormones, contexts, and behaviors, and the effects of accumulation processes*. Third, the science of hormones and their interactions with other biological subsystems (e.g., the immune system) continues to progress rapidly, offering much insight into the biological substrates that

interact with context. Finally, emerging technologies are making the measurement of hormones in behavioral studies more efficient, reliable, and valid.

One point of integration seems especially promising: as with behavioral genetics, life course sociologists have much to offer in the assessment of contexts, particularly from a longitudinal perspective. Behavioral human endocrinology has paid little attention to such central concepts as careers (but see Booth et al., 1999), manifold aspects of social position in a hierarchy, pathways, or the social embeddedness of transitions. Understandably, much effort has been devoted to measuring hormones and assessing their basic associations with gross behaviors. Building on these foundations, research is now turning to increasingly sophisticated models of context (Susman, 1997).

At the same time, several caveats should be observed. First, hormones do not cause behaviors in a simple one-to-one coordinate fashion, but rather are expressed in ongoing reciprocal interactions that are significantly modified by context. Second, measurement protocols differ in their reliability and validity and can thus greatly affect observed associations between hormones and behavior (Shirtcliff et al., 2000; Shirtcliff et al., 2001). Third, both hormones and behaviors typically function in highly correlated "webs" of other hormones and behaviors. That is, the likelihood of some degree of spuriousness in observed relationships is quite high. For example, although many studies of reproductive behavior have focused on a few androgens, other classes of hormones need to be considered (Campbell & Udry, 1994). Similarly, a singular focus on a personality trait, intelligence, marital status, or aggression neglects, for example, the many covariates of these factors. Furthermore, because of the complexities involved with assessing biological and social systems through time and with modeling their dynamic interplay, observed associations among hormones, behaviors, and contexts are often not expected to be large in magnitude.

Despite these and related challenges, however, the thematic links between behavioral endocrinology and the life course paradigm are clear and represent excellent opportunities for interdisciplinary research.

CONCLUDING COMMENT: LINKING BIOLOGY AND THE LIFE COURSE

Biological models of behavior and the life course both emphasize interactions between person and context, as well as the importance of studying the development of behaviors across the phases of life. According to life history theory, evolution has produced mechanisms that promote or inhibit specific behaviors depending on cues from the organism's setting. Further, contextual cues influence sets of traits that are highly intercorrelated and that characterize growth and maturation, reproduction, and senescence. For example, if evolutionary forces have influenced the timing of puberty, we would expect that the timing of menarche and spermarche would be sensitive to contextual cues and would be "coordinated" with aspects of growth, reproduction, and aging across the entire life of the species. That is, the timing of puberty could not be understood without reference to setting or to other biological transitions in the life course.

Likewise, the field of behavioral genetics holds that genes do not produce finished traits, but rather interact with environments to constrain or enhance the range of possible behaviors. These interactions begin in the womb and cross the many decades of life. Each new data collection effort thus steps into an ongoing, personal history of passive, reactive, and active gene–environment correlations and interactions. Only through an appreciation of these life-long

transactions can the analyst begin to understand the sources of continuity and possibilities for discontinuity in the present and future. Similarly, sophisticated conceptual models in behavioral endocrinology acknowledge the importance of context and its ongoing reciprocal interactions with behavior and hormones. These interactions may encompass sensitive periods, complex causal chains of organizational and activational effects, alterations in thresholds of reactivity, and processes of accumulation—all of which are also likely to encompass many decades of life. Behaviors observed in adulthood may originate in significant ways in the womb.

Both behavioral endocrinology and genetics recognize this fact and provide conceptual and empirical tools for the study of such long-term phenomena. These concepts acknowledge that people are "subjected" to environments as a matter of their lineage, that people evoke reactions through their own behaviors (including being selected for positions within organizations), and that people actively select and modify their settings. Yet these perspectives have yet to develop sophisticated models of social context. These dynamic interactions—and the accurate conceptualization and measurement of contexts that they require—define the central point of integration between biological and life course models of behavior.

Given these conceptual affinities, biological models and life course sociology are well suited as frameworks for interdisciplinary collaborations. Our review makes clear that life course sociologists have much work to do in acquainting themselves with recent developments in biological models of behavior. Yet such an acknowledgement is only half of the story. Biologists and biological oriented behavioral scientists have much to do in acquainting themselves with good models of social context and their attendant processes. Biological studies of behavior without reference to context are simplistic. But, equally true, life course studies of development must begin to incorporate biological insights. At this stage, the integration of biology and the study of the life course is in its infancy: a world of possibility awaits it and its future can only be suggested in broad terms.

ACKNOWLEDGMENT: The authors wish to acknowledge the helpful suggestions of Alan Booth, Glen H. Elder, Jr., Gilbert Gottlieb, Michael Grant, Kathryn E. Hood, Richard A. Miech, Jeylan T. Mortimer, Elizabeth J. Susman, and Richard Udry. The paper was largely completed while the first author was a professor of Human Development and Family Studies at the Pennsylvania State University.

REFERENCES

Anderson, K. G. (2000). The life-histories of American step-fathers in evolutionary perspective. *Human Nature*, *11*, 307–333.

Baffoe-Bonnie, A. B., Beaty, T. H., Bailey-Wilson, J. E., Kiemeney, L. A. L. M., Sigvaldason, H., Olafsdottir, G., Tryggvadottir, L., & Tulinius, H. (2000). Genetic epidemiology of breast cancer: Segregation analysis of 389 Icelandic pedigrees. *Genetic Epidemiology, 18*, 81–94.

Beatty, J. (1992). Fitness: theoretical contexts. in E. F. Keller & E. A. Lloyd (Eds.), *Keywords in evolutionary biology*. (pp. 115–119). Cambridge, MA, Harvard University Press.

Belsky, J., Steinberg, L. & Draper, P. (1991). Childhood experience, interpersonal development, and reproductive strategy: an evolutionary theory of socialization. *Child Development, 62*, 647–670.

Berenbaum, S. A. (1998). How hormones affect behavioral and neural development: Introduction to the special issue on "Gonadal Hormones and Sex Differences in Behavior". *Developmental Neuropsychology, 14*, 175–196.

Betzig, L. (1988). Mating and parenting in Darwinian perspective. In L. Betzig, M. Borgerhoff Mulder, & P. W. Turke (Eds.), *Human reproductive behavior: A Darwinian perspective*. (pp. 3–20). Cambridge: Cambridge University Press.

Bierbaumer, N. & Schmidt, R. F. (1996). *Biologische Psychologie [Biological Psychology]*. Berlin: Springer.

Blurton Jones, N. (1993). The lives of hunter-gather children: effects of parental behavior and parental reproductive strategy. In M. E. Pereira and L. A. Fairbanks (Eds.), *Juvenile primates: life history, development, and behavior* (pp. 309–338). New York, Oxford.

Blurton Jones, N. (1999). Some current ideas about the evolution of human life history. In P. C. Lee (Ed.) *Comparative primate socioecology*. (pp. 140–166). Cambridge, Cambridge University Press.

Boomsa, D. I., & Molenaar, P. C. M. (1987). The genetic analysis of repeated measures. *Behavior Genetics, 17*, 111–123.

Booth, A., Carver, K. et al. (2000). Biosocial perspectives on the family. *Journal of Marriage and the Family, 62*, 1018–1034.

Booth, A., & Dabbs, J. M., (1993). Testosterone and men's marriage. *Social Forces, 72*, 463–477.

Booth, A., D. Johnson, R., & Granger, D. (1999). Testosterone and men's depression: The role of social behavior. *Journal of Health and Social Behavior, 40*, 130–140.

Booth, A., & D. W. Osgood (1993). The influence on testosterone on deviance in adulthood: Assessing and explaining the relationship. *Crimonology, 31*, 93–117.

Borgerhoff Mulder, M. (2000). Optimizing offspring: The quanity-quality trade-off in agropastoral Kipsigis. *Evolution and Human Behavior, 21*, 391–410.

Bronfenbrenner, U., & Ceci, S. J. (1994). Nature-nurture reconceptualized in developmental perspective: A bioecological model. *Psychological Review, 101*(4), 568–586.

Brynin, M., & R. Smith (1995). Mapping the household. *Journal of Economic and Social Measurement, 21*, 127–144.

Buchanan, C. M., Eccles, J. S., & Becker, J. B. (1992). Are adolescents the victims of raging hormones: Evidence for activational effects of hormones on moods and behavior at adolescence. *Psychological Bulletin, 111*, 62–107.

Cairns, R. B., McGuire, A. M., & Ganepy, J.-L. (1993). Developmental behavior genetics: Fusion, correlated constraints, and timing. In D. F. Hay and A. Angold (Eds.), *Precursors and causes in development and psychopathology*. (pp. 87–122). New York: John Wiley.

Campbell, B. C., & Udry, R. J. (1994). Human reproductive ecology: Interactions of environment, fertility, and behavior. In K. L. Campbell & J. W. Wood (Eds.), *Annals of the New York Academy of Sciences* (pp. 117–127). New York: New York Academy of Sciences, *709*.

Case, A., Lin, I.-F., & McLanahan, S. (2001). Educational attainment of siblings in stepfamilies. *Evolution and Human Behavior, 22*, 269–289.

Cattell, R. B. (1963). The interaction of hereditary and environmental influences. *British Journal of Statistical Psychology, 16* 191–210.

Cattell, R. B. (1965). Methodological and conceptual advances in evaluating hereditary and environmental influences and their interaction. In S. G. Vandenberg (Ed.), *Methods and goals in human behavior genetics* (pp. 95–139). New York: Academic Press.

Charnov, E. L. (1993). *Life history invariants: some explorations of symmetry in evolutionary ecology*. Oxford: Oxford University Press.

Cole, L. (1954). The population consequences of life history phenomena. *Quarterly Review of Biology, 29*, 103–137.

Decker, S. A. (2000). Salivary cortisol and social status among men. *Hormones and Behavior, 38*, 29–38.

Dick, D. M., Rose, R. R., Viken, R. J., Kaprio, J., & Koskenvuo, M. (2001). Exploring gene-environment interaction: Socioregional moderation of alcohol use. *Journal of Abnormal Psychology, 110*(4), 625–632.

Draper, P., & Harpending, H. (1982). Father absence and reproductive strategy: an evolutionary perspective. *Journal of Anthropological Research, 38*, 255–273.

Duncan, G. J. (1985). A framework for tracking family relationships over time. *Journal of Economic and Social Measurement, 13*, 237–243.

Dunne, M. P., Martin, N. G., Statham, D. J., Slutske, W. S., Dinwiddie, S. H., Bucholz, K. K., Madden, D. A. F., & Heath, A. C. (1997). Genetic and environmental contributions to variance in age at first sexual intercourse. *Psychological Science, 8*, 211–216.

Durham, W. H. (1990). Advances in evolutionary culture theory. *Annual Review of Anthropology, 19*, 187–210.

Durham, W. H. (1992). Applications of evolutionary culture theory. *Annual Review of Anthropology, 21*, 331–355.

Eaves, L., Fulker, D. W., & Heath, A. C. (1989). The effects of social homogamy and cultural inheritance on the covariances of twins and their parents. *Behavior Genetics, 19*, 113–122.

Eaves, L., Hewitt, J. K., Meyer, J., & Neale, M. (1990). Approaches to the quantitative genetic modeling of development and age-related changes. In M. E. Hahn, J. K. Hewitt, N. D. Henderson, & R. Benno (Eds.), *Developmental behavior genetics: Neural, biometrical, and evolutionary approaches* (pp. 266–280). New York: Oxford University Press.

Ellis, B. J., & Graber, J. (2000). Psychosocial antecedents of variation in girls' pubertal timing: maternal depression, stepfather presence, and marital and family stress. *Child Development, 71*, 485–501.

Falconer, D. S., & Mackay, T. F. C. (1996). *Introduction to quantitative genetics*. Harlow, England: Addison, Wesley, Longman.

Finkel, D., Pedersen, N. L., Plomin, R., & McClearn, G. E. (1998). Longitudinal and cross-sectional twin data on cognitive abilities in adulthood: The Swedish Adoption/Twin Study of Aging. *Developmental Psychology, 34*, 1400–1413.

Foley, R. (1995). The adaptive legacy of human evolution: a search for the environment of evolutionary adaptedness. *Evolutionary Anthropology, 4*, 194–203.

Ford, D. H., & Lerner, R. M. (1992). *Developmental systems theory: an integrative approach*. Newbury Park, CA: Sage.

Futuyama, D. J. (1998). *Evolutionary biology*. Sunderland, MA: Sinauer Associates.

Gladue, B. A., Boehler, M., & McCaul, K. (1989). Hormonal response to competition among human males. *Aggressive Behavior, 15*, 409–422.

Gottlieb, G. (1995). Some conceptual deficiencies in "Developmental Behavior Genetics". *Human Development, 38*, 131–141.

Gottlieb, G. (1996). Developmental psychobiological theory. In R. B. Cairns, G. H. J. Elder, & E. J. Costello (Eds.), *Developmental science* (pp. 63–77). New York: Cambridge University Press.

Gottlieb, G. (1998). Normally occurring environmental and behavioral influences on gene activity: from central dogma to probabilistic epigenesis. *Psychological Review, 105*, 792–802.

Granger, D. A., Schwartz, E. B., Booth, A., & Areatz, M. (1999). Salivary testosterone determination in studies of child health and development. *Hormones and Behavior, 35*, 18–27.

Heath, A. C., & Eaves, L. J. (1985). Resolving the effects of phenotype and social background on mate selection. *Behavior Genetics, 15*, 15–30.

Hill, K., & A. M. Hurtado (1996). *Ache life history: The ecology and demography of a foraging people*. New York: Aldine De Gruyter.

Hill, K., & H. Kaplan (1999). Life history traits in humans: Theory and empirical studies. *Annual Review of Anthropology, 28*, 397–430.

Irons, W. (1998). Adaptively relevant environments versus the environment of evolutionary adaptedness. *Evolutionary Anthropology, 6*, 194–204.

Kacelnik, A., & Krebs J. R. (1997). Yanomamo dreams and starling payloads: the logic of optimality. In L. Betzig (Ed.). *Human nature: a critical reader*. (pp. 21–35). New York, Oxford University Press.

Kaplan, H., Hill, K., Lancaster, J. & Hurtado, M. (2000). A theory of human life history evolution: diet, intelligence, and longevity. *Evolutionary Anthropology, 9*, 156–185.

Kardia, S. L. R., Haviland, M. B., Ferrell, R. E., & Sing, C. F. (1999). The relationship between risk factor levels and presence of coronary artery calcification is dependent on apolipoprotein E genotype. *Arteriosclerosis, Thrombosis, and Vascular Biology, 19*, 427–435.

Kim, K. and P. K. Smith (1998). Retrospective survey of parental marital relations and child reproductive behavior. *International Journal of Behavioral Development, 22*, 729–751.

Lerner, R. M. (1984). *On the nature of human plasticity*. Cambridge: Cambridge University Press.

Maccoby, E. (1991). Different reproductive strategies in males and females. *Child Development, 62*, 676–681.

Mayr, E. (1982). *The growth of biological thought: diversity, evolution, and inheritance*. Cambridge, MA: Belknap Press.

Mayr, E. (1983). How to carry out the adaptationist program. *American Naturalist, 121*, 324–334.

Mayr, E. (2001). *What evolution is*. New York: Basic Books.

Mazur, A. (1985). A biosocial model of status in face-to-face primate groups. *Social Forces, 64*, 377–402.

Mazur, A. & Booth, A. (1998). Testosterone and Dominance in Men. *Behavioral and Brain Sciences, 21*, 353–363.

Mazur, A., Booth, A., & Dabbs, J. (1992). Testosterone and chess competition. *Social Psychology Quarterly, 55*(1), 70–77.

Mazur, A., & Michalek, J. (1998). Marriage, divorce, and male testosterone. *Social Forces, 77*, 315–330.

McArdle, J. J., & Goldsmith, H. H. (1990). Alternative common-factor models for multivariate biometric analyses. *Behavior Genetics, 20*, 569–608.

McClearn, G. E., & Hofer, S. M. (1999). Genes as gerontological variables: Genetically heterogeneous stocks and complex systems. *Neurobiology of Aging, 20*, 147–156.

McClearn, G. E., & Vogler, G. P. (2001). The genetics of behavioral aging. In J. E. Birren & K. W. Schaie (Eds.), *Handbook of the psychology of aging* (pp. 109–131). San Diego: Academic Press.

McClearn, G. E., Vogler, G. P., & Hofer, S. M. (2001). Environment–gene and gene–gene interactions. In E. J. Masoro, & Austad, S. N. (Eds.), *The biology of aging* (pp. 423–444). San Diego: Academic Press.

Moffitt, T. E., Caspi, A., Bekky, J., & Silva, P. (1992). Childhood experience and the onset of menarche: a test of a sociobiological model. *Child Development, 63*, 47–58.

Nelson, R. J. (2000). *An introduction to behavioral endocrinology*. Sunderland, MA: Sinauer Associates.

Nielsen, F. (1994). Sociobiology and sociology. *Annual Review of Sociology, 20*, 267–303.

Ojeda, S. R., & Griffin, J. E., (2000). Organization of the endocrine system. In S. R. Ojeda (Ed.), *Textbook of endocrine physiology*. Oxford: Oxford University Press.

Ott, J. (1999). *Analysis of human genetic linkage*. Baltimore: Johns Hopkins University Press.
Pedersen, N. (1996). Gerontological behavioral genetics. In J. E. Birren & K. W. Schaie. (Eds.), *Handbook of the psychology of aging*. (pp. 59–77). San Diego: Academic Press.
Peel, J. D. Y. (1971). *Herbert Spencer: The evolution of a sociologist*. New York: Basic Books.
Perusse, D. (1993). Cultural and reproductive success in industrial societies: testing the relationship at the proximate and ultimate levels. *Behavioral and Brain Sciences, 16*, 267–322.
Plomin, R., Defries, J., & Loehlin, J. (1977). Genotype-environment interaction and correlation in the analysis of human development. *Psychological Bulletin, 84*, 309–322.
Plomin, R., DeFries, J., McClearn, & McGuffin (2000). *Behavioral genetics*. New York: W. H. Freeman.
Potts, R. (1998). Variability selection in hominid evolution. *Evolutionary Anthropology, 7*, 81–96.
Rose, R. R., Dick, D. M., Viken, R. J., & Kaprio, J. (2001). Gene-environment interaction: Regional residency moderates longitudinal influences on alcohol use. *Alcoholism: Clinical and Experimental Research, 25*, 637–643.
Rowe, D. C., Jacobsen, K. C., & Van der Dord, E. (1999). Genetic and environmental influences on vocabulary IQ: Parental education level as moderator. *Child Development, 70*(5), 1151–1162.
Rutter, M. (2002). Nature, nurture, and development: From evangilism through science toward policy and practice. *Child Development, 73*(1), 1–21.
Sampson, R. J. & Laub, J. H. (1993). *Crime in the Making: Pathways and turning points through life*. Cambridge, MA: Harvarrd University Press.
Sapolsky, R. M. (1992). Cortisol concentrations and the social significance of rank instability among wild baboons. *Psychoneuroendocrinology, 17*(701–709).
Sapolsky, R. M. (2000). Stress hormones: Good and bad. *Neurobiology of Disease, 7*, 540–542.
Sapolsky, R. M., Alberts, C., & Altmann, J. (1997). Hypercortisolism associated with social subordinance or isolation among wild baboons. *Archives of General Psychiatry, 54*, 1137–1142.
Scarr, S. (1992). Developmental theories for the 1990s: Development and individual differences. *Child Development, 63*, 1–19.
Scott, C. H. (1976). *Lester Frank Ward*. Boston: Twayne Publishers.
Scott Peccei, J. (2001). Menopause: adaptation or epiphenomenon? *Evolutionary Anthropology, 10*, 43–57.
Shanahan, M., Sulloway, F., & Hofer, S. M. (2000). Change and constancy in developmental contexts. *International Journal of Behavioral Development, 24*, 421–427.
Shirtcliff, E. A., G. D. A., Granger, M., Schwartz, E., Curran, M., Booth, A., & Overman, W. H. (2000). Assessing estradiol in biobehavioral studies using saliva and blood spots: Simple radioimmunoassay protocols, reliability, and comparative validity. *Hormones and Behavior, 38*, 137–147.
Shirtcliff, E. A., Reavis, R., Overman, W. H., & Granger, D. A. (2001). Measurement of gonadal hormones in dried blood spots versus serum: Verification of menstrual cycle phase. *Hormones and Behavior, 39*, 258–266.
Stearns, S. C. (1992). *The evolution of life histories*. Oxford: Oxford University Press.
Storey, A. E., Walsh, C. J., Quinton, R. L., & Wynne-Edwards, K. E. (2000). Hormonal correlates of paternal responsiveness in new and expectant fathers. *Evolution and Behavior, 21*, 79–95.
Surbey, M. K. (1998). Parent and offspring strategies in the transition at adolescence. *Human Nature, 9*, 67–94.
Susman, E. J. (1993). Psychological, contextual, and psychobiological interactions: A developmental perspective on conduct disorder. *Development and Psychopathology, 5*, 181–189.
Susman, E. J. (1997). Modeling developmental compexity in adolescence: Hormones and behavior in context. *Journal of Research on Adolescence, 7*, 283–306.
Turkheimer, E. W. (1998). Heritability and biological explanation. *Psychological Review, 105*, 782–791.
Turkheimer, E. W. (2000). Nonshared environment: A theoretical, methodological, and quantitative review. *Psychological Bulletin, 126*, 78–108.
Udry, J. R. (1988). Biological predispositions and social control in adolescent sexual behavior. *American Sociological Review, 53*, 709–722.
Udry, J. R. (1990). Biosocial models of adolescent problem behaviors. *Social Biology, 37*, 1–10.
Udry, J. R. (2000). Biological limits of gender construction. *American Journal of Sociology, 65*, 443–457.
Vieira, C., Pasyukova, E. G., Zeng, Z.-B., Hackett, J. B., Lyman, R. F., & Mackay, T. F. C. (2000). Genotype-environment interaction for quantitative trait loci affecting life span in drosophila melanogaster. *Genetics, 154*, 213–227.
Vrba, E. S. (1990). Life history in relation to life's hierarchy. In C. J. DeRousseau (Ed.), *Primate life history and evolution* (pp. 37–46). New York, Wiley-Liss.
Williams, G. (1957). Pleiotropy, natural selection, and the evolution of senescence. *Evolution, 11*, 398–411.
Winterhalder, B. & Alden Smith, E. (2000). Analyzing adaptive strategies: human behavioral ecology at twenty-five. *Evolutionary Anthropology, 9*, 51–72.