LIFE HISTORY STRATEGIES AND DEFENSE-RELATED PATHOLOGIES

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ABSTRACT

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Existing frameworks of psychopathology have received criticism from evolutionary researchers. It is argued that mental health research lacks theoretical unity and that there is no comprehensive understanding of psychiatric disorders. Evolutionary researchers have posited that a paradigm of psychopathology informed by evolution could accomplish this aim. Drawing from evolutionary biology, life history theory provides such a framework. Life history theory is a subfield of evolutionary biology that examines how organisms allocate limited environmental resources over their lifespan in order to maximize their fitness. The strategies that organisms adopt in response to their environment are referred to as life history strategies. These strategies are graphed on a spectrum between two poles, fast and slow. The fast and slow life history classifications form the basis of the fast-slow-defense activation model of psychopathology which describes causal pathways for mental disorder. Within this framework, sex is a moderating factor between life history strategy and psychopathology. At present, there is little empirical research evaluating the fast-slow-defense activation model. The current research seeks to provide an analysis of the fastslow-defense activation framework by exploring the relationship between life history strategy, sex, and defense activation disorders particularly, depression. It is expected that there will be a causal relationship between life history strategy and depression. Specifically, a fast life history strategy will predict for increased symptoms of depression. In addition to this, it is expected that women with fast life histories experience greater symptoms of depression. This dissertation is

available in open access at AURA, https://aura.antioch.edu/ and OhioLINK ETD Center, https://etd.ohiolink.edu.

Keywords: Evolutionary Psychology, Life History Theory, Psychopathology, Life History Strategies, Depression, Depressive Disorders, Fast-Slow-Defense Activation, Mental Health, Structural Equation Modelling, Confirmatory Factor Analysis, QIDS-SR

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CHAPTER I: OVERVIEW

The existing body of social science research lacks a compelling, coherent theoretical unity that extends beyond the field of social science research. Many assumptions on which the field stands conflict with other scientific disciplines that examine human behavior and ecology. The internal lack of unity is especially pronounced in mental health research, which lacks theoretical harmony and has so far failed to develop a comprehensive understanding of psychiatric disorders (Brune, 2012; Nesse, 2016). Critics of the biomedical psychiatric model presented by the American Psychiatric Association in the Diagnostic and Statistical Manual of Mental Disorders, suggest that advancing a comprehensive model of psychiatric disorders requires a full working model of the mind and its functions (Del Giudice, 2018). It is argued that such a model is not possible based on current trends in mental health research. Instead, it is asserted that research that adopts an evolutionarily informed approach to studying behavior and psychopathology could accomplish this goal.

Evolutionary psychological researchers have been contributing to a growing body of empirical and theoretical literature that uses of life history theory to explain human behavior and mental disorders (Figueredo et al., 2013, Del Giudice, 2014; Hurst & Kavanagh, 2017; Del Giudice, 2018, Kahl et al., 2020; Kahl et al., 2021). Fundamentally, life history theory details how individuals within and between species allocate limited resources across their lifespans to increase their expected reproductive success. The strategies that an individual or species adopts in response to selective pressures are referred to as their life history strategy. Life history strategies range across a continuum from fast to slow. Faster life history strategies are characterized by earlier physical and reproductive development, greater mating effort and lower parenting effort, a greater number of lower quality offspring, and a tendency to discount future rewards. Slower life history strategies are marked by slower physical and reproductive development, reduced mating effort and higher parental investment in offspring, fewer, highquality offspring, and future orientation. At its core, life history theory is an organizing principle for species and individual variation as a function of environmental factors such as risk of mortality and morbidity.

The fast-slow framework has generated an emerging model of psychopathology that offers the possibility of a coherent unified understanding of mental, emotional, and behavioral disorders (Del Giudice, 2014; Del Giudice 2023). Withing this framework, at the most basic level, symptoms, and disorders cluster along the fast to slow continuum. According to Del Giudice (2023) fast spectrum disorders involve issues related to impulse control and antisocial behavior. In contrast, slow spectrum disorders are associated with exaggerated self-control and lower sexual interest.

There is limited direct empirical support for the categorizations of fast and slow type disorders (Del Giudice, 2018). Moreover, the fast-slow model has been criticized because several disorders (e.g., depression and anxiety) appear at either end of the spectrum (Kennair, 2014). This prompted the creation of the defense activation disorder category. Defense activation disorders are a byproduct of dysregulated emotional defenses, rather than direct reproductive strategies. The largely theoretical nature of the original fast/slow model, and the new unexplored category of disorders leaves several hypotheses to be tested. Del Giudice's (2018) model examines the intersection between sex, life history strategy, and mental disorder. For example, this framework predicts that women who have a fast life history strategy would be more likely to experience defense activation disorders than their male counterparts. It is this intersection that

will be the focus of the current study. A comprehensive examination of the intersection between sex, life history, and mental disorder, or even the defense activation category, is beyond the scope of this study. What the present research aims to accomplish is to test two hypotheses. First, it is predicted that fast life history strategies will be associated with greater symptoms of depression. Second it is expected that sex will moderate the relationship between life history strategy and sex such that women will experience greater symptoms of depression. These findings will direct future research investigating the application of life history theory to mental disorders. Insights taken from this study and future life history research will contribute to an overall comprehensive understanding of psychopathology. In doing so, it will refine diagnostic categories and prompt new, efficacious therapeutic protocols for patients seeking mental health treatment as well as increasing an overall understanding of mental disorders.

CHAPTER II: LITERATURE REVIEW

The Importance of Evolutionary Psychology

According to Tooby and Cosmides (2016), the social sciences are an amalgam of contradictory models of human behavior that lack a foundational theoretical unity. It is posited that many of the assumptions on which current social and psychological theories are built are demonstrably false. Moreover, they have limited grounding in natural, scientifically based frameworks. These assumptions are frequently inconsistent with other scientific disciplines such as evolutionary science, information theory, computer science, neuroscience, physics, and biological science. There are social science paradigms that incorporate frameworks from the natural sciences, for example, biology. However, the application of biological concepts to human behavior is often narrow in focus (Brune et al., 2012; Tooby & Cosmides, 2016).

The limitations of existing social science models of human behavior highlight the need for a coherent, internally consistent approach to understanding human behavior and social interaction. More importantly, a comprehensive mode should integrate with the natural sciences (Nesse et al., 2010; Tooby & Cosmides, 2016). This necessity has prompted psychological, behavioral, and social science researchers to apply evolutionary theory to understanding human behavior and social interactions. It is asserted that using an evolutionary approach to examine human behavior would generate a paradigm shift that would result in a greater understanding of the human mind. The result being a single, logically integrated research paradigm for the psychological, behavioral, and social sciences. Such a model would draw equally from the evolutionary biological sciences, information theory, and neuroscience resulting in a synthesis that ongoing research practice requires (Tooby & Cosmides, 1992). In particular, the field of mental health research needs theoretical unity, and it is proposed that it would benefit from the insights of evolutionary researchers (Brune et al., 2012; Nesse, 2013; Ray 2020). To expand on this, Del Giudice (2018) asserts the importance of examining mental health through the lens of evolutionary theory such that a "unified approach to psychopathology would be impossible without an integrated working model of the mind and its functions" (pp. 3). That is, the aim of such a model is not to replace existing models for psychiatric disorders, but to create a logically consistent functional taxonomy (Del Giudice, 2023). The subsequent section explores this line of reasoning in greater detail, and it examines evidence for the utility of an evolutionary paradigm.

Issues with Existing Models of Psychopathology

Brune et al. (2012) argue that there is a crisis in mental health research. They contend that there has yet to be a unified effort to develop a coherent and comprehensive scientific understanding of psychiatric disorders. Brune et al. (2012) contend that even biologically informed practitioners fail to account for the aspects of human behavior and experience that have been formed during their ancestral past. In doing so they neglect the evolutionary origins and significance of social behavior and emotionality. This negligence results in an incomplete understanding of human psychopathology. Moreover, Brune et al. (2012) suggest that extended efforts by reductionist researchers to find reliable biomarkers and specific causes of major mental disorders have so far been unsuccessful.

The application of evolutionary principles to an understanding of psychopathology could provide long-sought answers regarding the etiology of mental disorders (Alcock, 2001; Brune et al., 2012). It is emphasized that an evolutionarily informed perspective of human behavior and emotionality would give rise to a revolution in the field of mental health research of equal magnitude to the revolution that occurred in the study of nonhuman animal behaviors following the application of evolutionary principles. Evolutionary perspectives have also triggered rapid advancements in the field of medicine (Nesse et al. 2010; Stearns, 2012). The rapid growth in animal behavior studies and advancements in medicine is attributed to the supposition that an adequate explanation of any biological, and therefore psychological trait requires a description of its evolutionary history and mechanism (Tinbergen, 2005). To develop a full understanding of mental disease, mental health researchers need to know not only why some individuals develop disorders, but why all members of a given species have traits that are susceptible to failure (Nesse, 2005).

What is highlighted is that evolutionary psychology could address outstanding questions regarding psychopathology. It presents a point from which to appreciate why the human species is susceptible to emotional and behavioral dysregulation. By doing so, it offers mental health researchers a scientifically sound etiology of psychopathology (Nesse, 2005). However, accomplishing this requires an understanding of how natural selection shaped human behavior which first requires an overview of evolutionary theory.

General Evolutionary Concepts

Darwin (1859) identified natural selection as a force that shapes the behavior and the form of a species. Natural selection is considered to have occurred in populations of reproducing individuals if: 1) there is a limit to available resources, thus rendering unconstrained reproduction impossible; 2) individual organisms are different in terms of their morphological, physiological and behavioral traits known has phenotypes; 3) phenotypic traits are associated with an organism's ability to successfully reproduce; and 4) phenotypes are inherited and transmitted to descendants with some reliability. Natural selection predicts that individuals that have greater reproductive success make more descendants. In turn, their offspring carry the traits that favored reproduction in the previous generation (Del Giudice, 2018). As time continues, successful traits spread throughout a population because they improve an organism's odds of reproductive success, or fitness. Traits that increase fitness are considered to be adaptive, whereas those that diminish it are maladaptive. Traits that are neither adaptive nor maladaptive are neutral. Importantly, natural selection can occur through multiple forms of inheritance; it does not rely solely on DNA transmission.

To be emphasized, the basic metric by which an organism's fitness is assessed is not by its survival, but by the number of its offspring that reach reproductive maturity (Del Giudice, 2018). Survival is an important aspect only to the extent that an individual can survive long enough to reproduce. How well an organism's ability to survive is rendered useless if it fails to pass its traits onto descendants. As a result, organisms are required to make trade-offs between longer survival and increased reproduction. Natural selection favors individuals that allocate resources such as time and energy in a way that improves their overall fitness. However, there is no solitary advantageous method of allocating resources. That is, there is more than one way for an organism to increase their fitness. Inclusive fitness theory and the individual-as-maximizing-agent (IMA) are gene-centric understandings of natural selection that explain how individuals can adopt different strategies to increase their fitness.

How Traits Spread and Persist

The process of natural selection constantly weeds out unsuccessful variations producing modifications of existing phenotypes. This process culminates in traits called adaptations which are coordinated to enhance survival and reproductive success. Williams (1966) defines adaptations as inherited phenotypes that reliably develop over time which are selected for because of their causal role in enhancing fitness of the individuals that possess them. Adaptations that have the appearance of purposeful design have biological functions. For example, the heart serves the function of pumping blood. While traits that are products of natural selection are adaptations, not every trait of an organism is an adaptation. Natural selection is only one of the forces that shape the traits of organisms.

Genetic drift is a random process by which neutral or even deleterious traits propagate through a population due to chance fluctuations in the frequency of genes (Williams, 1992). Alternatively, genetic linkages between traits can result in neutral or even weakly maladaptive traits persisting and spreading through a population by hitchhiking on traits that are adaptive. Traits may also proliferate by being byproducts of adaptations. Referring to the previous example, the heart makes noise while it is pumping blood but the noise it makes is not an adaptation. It is a byproduct of an adaptive trait. Finally, traits can be maintained in a population by chance historical contingencies.

These general evolutionary forces shaped every organism that has existed, including the human animal. They offer important insights into the biological and behavioral development of the human species. It is these insights that differentiate evolutionary psychology and the prevailing social science models.

Evolutionary Psychological Approach versus Prevailing Social Science Models

The differences between evolutionary psychology and traditional social science models are often nuanced and subtle; however, there are several fundamental differences (Tooby & Cosmides, 1992; Del Giudice, 2018). The central tenets of evolutionary psychology are among the most significant of these differences. At its core, evolutionary psychology posits that the minds of modern humans originated in the deep historical past, and that they have been shaped over millions of years by the changing demands of human ecology. Following this logic, any biological, and by extension, any behavioral system can be explained in four ways: from the standpoint of mechanism (i.e., *What is the system like? How does it work?*); that of ontogeny/development (i.e., *How does the system change over the course of an organism's life cycle?*); that of phylogeny (i.e., *What is the history of a given system? How has that system changed over evolutionary time? How does the system differ between related species?*); and that of adaptation (*Why did the system evolve into its present form? What evolutionary advantages did it provide?*) (Tinbergen, 1963/2005; Del Giudice, 2018).

Proximate and Ultimate Explanations

The above-noted explanations fit into one of two explanatory categories, proximate and ultimate (Tingbergen, 1963/2005). Proximate explanations are composed of ontogenetic and mechanistic explanations – they describe how an organism functions in the present. Ultimate (i.e., phylogenetic/adaptive) explanations view an organism in reference to the past. They offer a description of the evolutionary forces that shaped an individual's body and behavior. While ultimate and proximate explanations constitute distinct categories, they are not mutually exclusive. Instead, they are complementary and synergistic. Del Giudice (2018) elaborates on the synergy between ultimate and proximate explanations, "Just knowing the adaptive purpose of a mechanism can illuminate its functioning, understanding the mechanics and development of a trait constrains the range of plausible adaptive explanations for its evolution," (pp. 6). The differentiation between proximate and ultimate explanations is the primary line of divergence between the prevailing social science models of behavior and evolutionary psychology. The prevailing models focus almost exclusively on the proximate level of analysis (Del Giudice,

2018). Evolutionary researchers make the next logical step by asking questions about adaptation to understand the purpose of human behavioral systems.

Ultimate explanations give researchers the means to understand the evolved history of adaptations that govern human behavioral systems, that is, the function of those systems. By doing so, they also describe how and why they are vulnerable to dysfunction (Nesse et al., 2010). Ultimate explanations are not the only area of divergence between evolutionary psychological models of behaviors and the current social science models. Evolutionary researchers have also critiqued the current concept of disorder. In doing so, they provide an alternative definition of disorder.

Disorder and Harmful Dysfunction

Defining disorders with a high level of specificity has been a difficult task for mental health researchers (Nesse, 2001; Del Giudice, 2018). The DSM-5-TR defines mental disorders as "a dysfunction in the psychological or developmental processes underlying mental functioning" (American Psychiatric Association, 2022, p. 20). It also requires that distress needs to accompany the dysfunction as well as an impairment in an important domain of life functioning. The DSM-5 definition of dysfunction has been the subject of criticism for failing to clearly outline what constitutes dysfunction (Wakefield, 2016; Del Giudice, 2018). Defining dysfunction as a dysfunction in neural circuits also fails to provide a precise definition of disorder (Cuthbert & Insel, 2013).

In contrast, a prevailing evolutionary model reclassifies mental disorders as harmful dysfunctions (Wakefield, 1992; Wakefield, 1999; Wakefield, 2016). The harmful dysfunction model converges with the DSM-5-TR in the requirement that mental disorders be defined in the context of cultural values; a disorder must be perceived as socially negative, or harmful. Where

the two differ is how the term dysfunction is defined. In the context of evolution, dysfunction occurs when a biological or mental mechanism to fails perform its evolved function. This definition refers to ultimate explanations of traits; identifying dysfunction in a mental mechanism first requires an understanding of its function.

Psychopathology

Applying the concepts of proximate and ultimate explanations of organic systems is the keystone of evolutionary research on mental illness (Nesse, 2011; Nesse, 2016). Proximate explanations of the body's systems describe how they function and by extension they can help identify when these systems are not functioning as designed (Nesse, 2011). Contrariwise, ultimate explanations of the body's systems explain how these systems came to be and by proxy why they are vulnerable to failure. Applied to mental health research, proximate explanations describe psychopathology itself while ultimate explanations explain the etiology of mental dysfunction; they illuminate why mental systems are vulnerable to failure.

Vulnerability itself results from the inherent limitations of the powers of natural selection (Nesse, 2019). To reiterate, the selective forces that drive the development of traits are constrained by laws of physics in addition to being limited to building on existing phenotypes that is, "there is no starting fresh with bodily designs, so substandard aspects abound" (Nesse, 2015, p. 1008). Minding these constraints, Nesse (2016) describes five evolutionary explanations for vulnerability: 1) mismatches with modern environments; 2) coevolution with fast-evolving organisms; 3) trade-offs; 4) advantages for alleles at the expense of health; and 5) defenses that are useful (Nesse, 2015). These explanations are far reaching in evolutionary models of mental disorder. They require individual attention to undergird the subsequent models of mental disorder.

Mismatches with Modern Environments

Li et al. (2018) described evolutionary mismatches as: "...adaptive lag that occurs if the environment that existed when a mechanism evolved changes more rapidly than the time needed for the mechanism to adapt to change" (p. 38). Human behavioral and affective mechanisms have origins in their deep history. As a result, many psychological mechanisms evolved during the 99% of human history where people lived as hunter-gatherers, in close-knit, kin-based groups. It is only in the last 10,000 years - one percent of human history - that human developed agriculture which enabled the formation of larger societies. This was a substantial departure from their deep past. The recent industrial and digital revolutions have only increased this divergence. This has resulted in two possible forms of mismatch, forced and hijacked. Forced mismatch occurs when a novel environment is rapidly imposed on an organism and hijacked mismatch is the result of novel stimuli being favored by a mechanism over the stimuli it evolved to process. It is possible that mismatch could occur on a smaller timescale. Kavanagh and Kahl (2018) suggest that mismatch can occur between an individual's expected environment and their current environment over the course of individual developments (e.g., early development in a hostile environment promoting a fast life history strategy and later living in a predictable safe environment more conducive to having a slow life history strategy).

Coevolution with Fast Evolving Organisms

Coevolution with fast evolving organisms may be a cause of mental disorders (Nesse, 2015). That is, they can emerge as a result of arms races with pathogens and their auto-immune sequelae. There are two implications to this: natural selection cannot remove vulnerabilities to pathogens due to said pathogens evolving faster than the human immune system; and at times, immune defenses can give rise to problems themselves. This assertion is supported by findings

that immune responses have been associated with depressive disorders (Raison & Miller, 2017) and prenatal infection appears to play a role in the development of schizophrenia (Arias et al., 2012).

Trade-offs

Trade-offs are an inevitable outcome of natural selection and as a result, perfection is impossible for any trait (Nesse, 2015). To illustrate, humans could have less anxiety, but at the cost of increasing mortality and injury rates. The underlying concept is another explanation of vulnerability. Natural selection favors traits that increase reproductive success even if said traits come at the expense of health and wellbeing. A gene that increases vulnerability to mental disorder will be selected for if it also increases the expected reproductive success of an individual. Trade-offs can result in defenses that yield a positive impact on reproductive success, while negatively affecting mental wellbeing. This is captured in the smoke detector principle which asserts that excessive anxiety results in an increase to survivability and reproductive success (Nesse, 2005). Despite excessive anxiety being an adaptive defense, it could cause substantial distress for those who experience it.

The salient point is that traits that may cause vulnerability may also have adaptive value. Natural selection prioritizes reproductive success above all else. Accordingly, traits that have a deleterious effect on wellbeing and health can still be categorized as adaptive due to their net positive effect on expected reproductive success. Traits associated with increased aggression exemplify this. Physically aggressive behavior (i.e., fighting) increases chances of injury, but could result in higher reproductive success (e.g., fending off mating rivals). As a result, these traits could be adaptive and are likely to persist due to their net positive benefit on fitness. What is important to recall is that maladaptive and adaptive have different meanings in the context of evolution than they do when used in the prevailing models of mental health research. The focus of the succeeding section explores this difference in greater detail along with examining how adaptive, but harmful behavior is pathologized leading to pseudopathologies (Ellis & Bjorklund, 2012).

Adaptive, Maladaptive, and Pseudopathologies

The distinction between the terms adaptive and maladaptive deserves attention. To reiterate, adaptations are inherited phenotypes that develop and persist over time due to their causal role in enhancing an organism's reproductive fitness (Williams, 1966). This use of adaptive and maladaptive follows the logic of natural selection which promotes reproductive success above all else (Nesse, 2006). A trait or behavior is seen as adaptive if it improves an individual's odds of reproducing, even if the trait and/or behavior is socially undesirable and comes at a cost to the actor's health (Nesse, 2004). The inverse is true when referring to maladaptive behaviors or traits which decrease an actor's reproductive fitness, even if they improve health or mental wellbeing. In contrast, mainstream mental health research defines adaptive behaviors and traits as being prosocial and having a positive effect on wellbeing. Behaviors that are socially undesirable or harmful to an actor are maladaptive. This contrast in how these terms is used has given rise to pseudopathologies (Ellis & Bjorklund, 2012).

Exploitative and aggressive behaviors may be socially negative, and they can cause harm to the individual engaging in them. However, they may also represent an adaptive strategy (Ellis & Bjorklund, 2012). Behaviors that are pathologized from traditional psychological disciplines may not represent a true pathology, only a differential reproductive strategy (Ellis & Del Giudice, 2018). Exploitative and aggressive behaviors, while being socially unacceptable, and therefore harmful, may not be the result of dysfunction. Instead, they may be a behavioral strategy that could improve reproductive success in each environment despite harmful consequences for the actor and those being acted upon. Put simply, there is harm but no dysfunction. Pseudopathologies are illustrative of the distinction between how evolutionary psychological and traditional psychological models' use adaptive and maladaptive.

A fundamental component in pseudopathologies is the relationship between environment and behavior. Understanding this relationship requires that careful attention be given to an individual's developmental environment, as well as the environment in which its traits evolved. The synergy between the two impacts how an individual allocates resources across its lifespan to maximize its fitness (Del Giudice, 2014). The relationship between environment and biology is too complex to fully explicate here. However, there are essential concepts in evolutionary theory that need to be discussed to further an evolutionary perspective of mental illness. Broadly speaking, life history theory (LHT) is a framework from which the relationship between an individual's developmental environment and their biological/behavioral traits is studied. LHT is the backbone for the model of psychopathology that is the focus of the present research.

Life History Theory

Natural selection favors organisms who allocate energy in a way that, on average, results in the greatest inclusive fitness (Del Giudice, 2018). Optimal allocation of energy depends on both the characteristics of the individual and their environment. For instance, healthy individuals are likely to allocate energy differently than those who are infected by a disease. Likewise, an optimal allocation strategy that may be effective in a stable environment may not be effective in circumstances in which the future of the individual is unpredictable.

Bearing in mind the relationship between orgasm and environment, the most important task for an individual is how it allocates resources between competing fitness related activities (Ellis et al., 2009). Life history theory elucidates the strategies organisms employ to optimally apportion time and energy to the diverse activities that encompass their life cycle (Del Giudice, 2014). It is a conceptual framework that explains how, under the constraints of limited environmental resources and time, organisms may allocate their energy resources to tasks and traits across their life cycle to optimize their inclusive fitness (Del Giudice, 2014; Del Giudice et al., 2016). Del Giudice et al. (2016) note: "Individuals can enhance fitness in two primary ways: They can invest either in traits that affect the age-schedule of survival, or in traits that affect the age-schedule of fertility" (p. 88). That is, organisms may either invest in traits that allow for earlier reproductive maturity and thus earlier offspring at the expense of longevity.

The trade-off between bodily growth and reproduction occurs because both processes contribute to an individual's fitness (Del Giudice, 2014). Time spent searching for mates detracts from time spent acquiring food and other resources. Resources must then be divided between somatic effort and reproductive effort because producing offspring reduces the time and energy that can be devoted to increasing somatic capital. The tactics organisms employ to optimally allot time and energy between survival and reproduction demands are their life history strategies. Del Giudice (2018) describes life history strategies as "adaptive solutions to fitness trade-offs within the constraints imposed by physical laws, phylogenetic history, and developmental mechanisms" (pp. 263). The trade-offs between somatic and reproductive efforts fall into several subcategories (Geary, 2002). Activities relating to somatic effort are growth, survival and body maintenance, and developmental activity. Developmental activity is further divided into play, learning, exercise, and other activities that contribute to accruing embodied capital (i.e., strength coordination, skills, and knowledge) (Hill & Kaplan, 1999). Reproductive effort refers to mating

effort, parenting effort, and nepotistic effort. Mating effort involves finding and attracting mates as well as conceiving offspring. In contrast, parenting and nepotistic effort tasks include investing resources in already conceived offspring and investing in other relatives.

To summarize, life history strategies involve decisions regarding trade-offs between current and future reproduction, and between the quality and quantity of offspring. Mating species face a further trade-off between mating and parenting effort (Hill, 1993; Ellis et al., 2009). Different strategies address trade-offs by determining how organisms distribute effort among fitness related traits. By delaying reproduction, an organism accrues resources and/or embodied capital which improves fitness of future offspring. However, this must be weighed against the increased probability of dying before reproducing. Successful reproduction incurs another trade-off between high and low parental investment as well as having few high-quality offspring or many low-quality offspring, respectively. Higher parental investment in offspring increases their embodied capital, improving the long-term fitness of one's descendants; however, this comes at the expense of reducing the mating opportunities. These descriptions may give the impression that life history decisions are made consciously, but it is important to recall that these decisions are made at a genetic level. Of equal importance, LHT involves differences between species as well as variations within a given species.

The Fast/Slow Continuum

The trade-offs that encompass life history strategies are not mutually exclusive. Del Giudice (2014) describes the variations in life history trade-offs as "...show[ing] a general pattern of trait covariation" (pp. 264). The patterns of trait variation can be plotted on a continuum of fast/slow life history strategies. Fast life history strategies are those in which individuals adopt a strategy of rapid bodily growth and early reproduction. Mell et al. (2018) found that the onset of

puberty occurs earlier in organisms that adopt a fast life history strategy relative to their slow life history counterparts. Fast strategies correlate with a shorter lifespan, large numbers of lowquality offspring with minimal parental investment, greater numbers of sexual partners and high rates of juvenile mortality (Ellis et al., 2009, Del Giudice, 2014). Conversely, slow life history strategies are characterized by slow growth with a longer life-span, and delayed reproduction. Slow strategies correlate with greater parental investment, fewer high-quality offspring, fewer sexual partners with more stable relationships and low rates of juvenile mortality.

The fast-slow continuum is not limited to an organism's biological and reproductive development – life history strategies have far-reaching effects on an organism's behavior (Del Giudice, 2014; Han & Chen, 2020). Individuals who adopt a slow strategy bet on future reproduction requiring them to survive long enough to do so. Put differently, they must maximize their survival and health in order to reproduce. As a result, individuals with a slow life history strategy are more likely to be risk averse, and to avoid investment in immediate variable rewards in favor of surer outcomes, even if doing so results in lower returns on average. In contrast, organisms with a fast life history strategy are more likely to disregard future payoffs in favor of more immediate high risk short-term payoffs. Risk aversion is the only domain in which life history strategies affect behavior. They have a far-reaching effect on the diverse suite of emotions and complex social lives of species such as humans.

Life History Strategies in Humans and Pathways to Psychopathology

In humans, life history strategies have a complex interaction with behavior that extends beyond risk-averse behaviors. For example, cooperation, pair-bonding, and reciprocity are behavioral traits that are affected by life history strategies (Hill, & Kaplan, 1999; Del Giudice, 2014). Life history theory is hypothesized to serve as an organizing framework for individual differences in humans. It is expected that the traits of individuals could be plotted along on the fast-slow continuum, depending upon their developmental environment (Del Giudice, 2014). Varying life history strategies result in different profiles regarding self-regulation, interpersonal styles, cognition, and wellbeing.

Belsky et al. (1991) advanced a life history model of human development that explored how factors such as the quality of parent-child and family relationships impact life history decisions that contribute to individual variation in social and sexual behavior as well as the timing of sexual maturity. Harsh parenting, conflictual family relationships and insecure attachments predict the developmental, sexual, and behavioral features of fast life history strategies. This results in earlier sexual maturation, impulsivity, reduced cooperation, and exploitative interpersonal styles. There is evidence in support of this model, particularly regarding sexual and social development. Cui and Lan (2020) found that harsh parental styles were associated with aggressive behaviors in adolescent males. Unstable developmental environments tend result in manipulative behaviors and unstable social relationships (Csathó & Birkás, 2018). Additionally, there were associations between harsh parenting, insecure attachment, and early puberty in girls (Del Giudice, 2014). Further research demonstrated relationships between harsh developmental environments and precocious sexuality, unstable couple relationships and promiscuous mating styles (James et al., 2012; Wang, 2019). Chisholm et al. (2005) reported relationships between insecure attachment, present orientation (i.e., the inability to delay gratification and/or await larger, future rewards), and shorter subjective life expectancy in adult women. Present orientation and shorter expected lifespan further predicted earlier onset of sexual activity, greater number of sexual partners, and younger age at first birth. This evidence is consistent with a fast life history strategy (i.e., earlier reproduction, and high

mating effort) (Laghi, et al., 2009). The relationship between shorter life expectancy and early childbearing has been further demonstrated by epidemiological studies (Copping, Campbell, & Muncer, 2013).

In addition to their associations with fast life history strategy markers, present orientation, impulsivity, and shorter subjective life expectancy are correlated with other features expected of a fast life history strategy. All three factors have relationships with increased risk taking, short subjective life expectancy, reduced cooperation, deviance, antisocial behavior, younger age at first intercourse, and a greater number of sexual partners (Brezina et al., 2009; Chen & Vazsonyi, 2011; Del Giudice, 2014). Psychometric evaluations of life history strategies have demonstrated a direct relationship between poorer perceived parental investment and a fast life history strategy (Hurst and Kavanagh, 2017; Kahl et al., 2020; Kahl et al., 2021). Faster life history strategies were associated with higher levels of aggression and greater symptoms of psychopathology.

Conversely, Figueredo et al. (2013) reported associations between markers of slow life history strategies and several factors including: cognitive and behavioral indicators and covitality. More specifically, slow life history factors were positively correlated with parental investment, social support, romantic partner attachment, general altruism, insight, planning, and executive functioning. In contrast, factors of slow life history negatively predicted short-term mating, sociosexual orientation, escalated male retention tactics, affective and punitive responses, interpersonal aggression, female intrasexual competitiveness, disordered eating behavior, sexual and emotional infidelity, and negative ethnocentrism and androcentrism. With respect to co-vitality, slow life history factors positively predicted for reception of parental and nepotistic effort, reciprocal and mutualistic relationships, general health, wellbeing, and positive affect while being negatively associated with negative affect and healthy symptoms. The findings made by Figueredo et al. (2013), evidence a fast-slow spectrum in humans. The development of fast life history strategies is predicted for by harsh, unpredictable environments and fast life history strategies are associated with earlier sexual development and greater incidence of aggression and psychopathology. Likewise, slow life history strategies are correlated with stable developmental environments and higher levels of parental involvement. Slow life history strategies also are associated with greater wellbeing, future planning, and fewer symptoms of psychopathology. Life history theory is an organizing principle to understand individual differences and a means for explaining why traits covary with each other (Del Giudice, 2014). The predictive power of life history theory creates a pathway for an evolutionary understanding of psychopathology. The upshot is a model that offers causal pathways to psychopathology.

Causal Pathways to Psychopathology

The life history theory framework offers four causal pathways to psychopathology (Del Giudice, 2014). First, life history traits may be regarded as symptoms. Noted before, variations in reproductive strategies may result in pseudopathologies that is, adaptive behaviors that accomplish the goal of increasing reproductive success, while simultaneously causing harm to the individual and others (Ellis & Bjorklund, 2012). Second, life history related traits may also be expressed at maladaptive levels (Del Giudice, 2014). To illustrate, Bergstrom and Meacham (2016) demonstrated how adaptive anxiety behaviors can produce maladaptive anxiety due to individual differences in experience. Third, adaptive strategies may yield maladaptive outcomes. Fourth, life history related traits may increase an individual's vulnerability to dysfunction. Finally, it is believed that differences between an individual's expected environment and their current environment may lead to psychopathology (Kavanagh & Kahl, 2018). That is, disorder

can occur when an individual is placed in an environment that varies significantly from their developmental environment. Notably, the fast-slow continuum is a lens through which symptoms and disorders may be classified. However, when explaining psychopathology there are limitations to the fast-slow continuum alone (Del Giudice, 2018).

The Fast-Slow-Defense Activation Model of Psychopathology

The fast-slow continuum is a principle by which causal pathways to psychopathology can be understood (Del Giudice, 2014). However, it does not provide a complete, coherent theoretical understanding of disorders and their symptoms (Del Giudice, 2018). Del Giudice (2018) posits that a compelling framework should accomplish four main tasks:

(a) explain observed patterns of comorbidity between disorders; (b) address the problem of heterogeneity within diagnostic categories; (c) bridge psychopathology with normative individual differences in personality and cognition: and (d) make sense of the developmental features of mental disorders, including their life course trajectories and early risk factors (pp. 153).

To accomplish this, Del Giudice (2014) introduced the distinction between fast and slow spectrum disorders. This was later expanded to include the new category of defense activation disorders (Del Giudice, 2018).

The distinctions between fast spectrum, slow spectrum and defense activation disorders comprises the fast-slow-defense (FSD) model of psychopathology (Del Giudice, 2018). Life history strategies serve as an organizing principle for individual differences by coordinating variation across a multitude of traits. Accordingly, they offer explanations for the development of psychopathology. Different strategies, or profiles may increase (or decrease) the risk of developing mental disorders. As an individual moves toward either pole of the fast-slow continuum, specific symptoms and disorders are expected to appear at a greater frequency while others should be less likely to occur; disorders cluster at one end or the other. For example, individuals with a fast life history are more likely to develop disorders characterized by impulsivity and antisocial behavior. In contrast, people on the slow end of the spectrum would be expected to develop disorders involving behavioral constraint, exaggerated self-control, or reduced sexual motivation (Del Giudice, 2014). In summary, mental disorders result from broad patterns of individual differences that can be identified as manifestations of life history strategies. Correspondingly, disorders may be categorized based on their connections with different strategies, and profiles within those strategies.

The primary limitations to the to the fast-slow model is that several disorders such as depression and anxiety, appear at both ends of the spectrum (Del Giudice, 2018). This prompted the creation the defense activation category of disorders. To be noted, much of the original fast-slow model remains unaltered and theoretical. This along with the newly created defense activation category provides avenues of quantitative exploration. In particular, the current research will examine the new category.

Defense Activation Disorders

The defense activation category was created because symptoms of psychopathology do not have a straightforward relationship with life history strategies (Del Giudice, 2018). Del Giudice (2018) observed that defense mechanisms are a major source of psychiatric symptoms. Recalling the smoke detector principle, negative emotions such as shame, anxiety, fear, and disgust can serve self-protective functions in response to social and physical threats (Nesse, 2005; Nesse, 2016). Some disorders primarily consist of symptoms that reflect the frequent, strong activation of defenses mechanisms that is, major and persistent depression, generalized and social anxiety, panic, specific phobias, and posttraumatic stress (Del Giudice, 2018). It is uncertain if these defenses have adaptive value. Panic and anxiety can be adaptive; however, errors in activation without the presence of a threat can lead to maladaptive outcomes. More significantly, defense mechanisms can become damaged and/or dysregulated, which can result in harmful dysfunctions (Bergstrom & Meacham, 2016; Meacham & Bergstrom, 2016a; Del Giudice, 2018).

Stress and threats to an individual are environmental factors that contribute to life history decisions. This creates a functional connection between physiological stress and life history strategy. However, these connections do not always map clearly onto the fast-slow continuum (Del Giudice, 2018). By their nature, upregulated defenses are likely to occur at both ends of the fast-slow spectrum (Del Giudice, 2014, 2018). In reference to fast life history strategies, a low threshold for defense activation protects an individual in a threatening and unpredictable environment. On the slow end of the spectrum, upregulated defenses are future oriented. They function to prevent dangerous events and avoid potentially risk situations in the future, even if the present environment is predictable and relatively free of threat. From a slow life-history perspective, avoiding even minor threats has adaptive utility as doing so contributes to long term maintenance of somatic capital.

Although defense activation disorders are more common at either end of the life history spectrum, it is expected that they should occur relatively more frequently in dangerous and unpredictable environments that favor the development of fast life history strategies (Del Giudice, 2018). Fast life history strategies can also have a feedback effect on symptoms. They i.e., risk-taking, impulsivity) increase the likelihood of experiencing stressful events which contributes to upregulated defenses and an increased risk of defense activation disorders.

The relative recency of the defense activation disorder classification presents new hypotheses by which to validate FSD model (Del Giudice, 2018). Relevant to the present study is the prediction that women on the fast end of the life-history spectrum will experience defense activation disorders more frequently than their male counterparts. This prediction will be the focus of the present research. The present hypothesize is that females who are identified as having fast life history strategies will experience more defense activation symptoms than their male counterparts.

Current Research

There is evidence for a relationship between fast life history strategies and emotional and social difficulties, and psychological distress (Dunkel et al., 2013; Figueredo et al., 2013; Hurst & Kavanagh, 2017; Kahl et al, 2020; Kahl et al., 2021). More pertinent to this study, Hurst and Kavanagh (2017) explored the direct relationships between life history strategy and symptoms of psychopathology. Their findings indicated that fast life history strategies were associated with more symptoms of psychopathology overall and greater aggression. They also reported a relationship between perceived parental investment and attachment, and life history strategy such that individuals who perceived less parental investment and poorer attachment relationships responded to life history measures in a manner consistent with a fast life history strategy. Kahl et al. (2020) found that life history traits could predict general factors of psychopathology as well as specific symptom groups. Kahl et al. (2021) expanded on these findings by identifying mediational pathways in which fast life history strategies were correlated with dysfunction schemas which predicted greater depression, anxiety, and obsessive-compulsive symptoms. However, a potential limitation with these findings is their use of psychometric measures of life history.

Recent research has criticized the use psychometric tools to evaluate life history (Međedović, 2020; Sear 2020). In an examination of the application of life history theory to humans, Sear (2020) argued that psychometric measures of life history strategies are conceptually removed from life history theory. Međedović (2020) posited that a possible metric for life history would be indicators more closely associated with evolutionary trades-offs.

Accordingly, the current study used a psychosocial-biodemographic life history model to examine life history traits in relation to defense activation disorders, specifically depression. This study also examined the prediction that sex plays a moderating role between depression and life history. That is, it was predicted that women on the fast end of the life history spectrum should report higher severity of depressive symptoms than their male counterparts. The current research sought to examine the causal relationship between life history strategy and depression as well as the moderating role of sex. It will focus on two hypotheses. First, it is hypothesized that there will be a causal relationship between life history and depression. Second, it is predicted that sex will moderate this relationship. That is, it will amplify the effect of life history strategy.

CHAPTER III: METHOD

Participants

Survey data from an initial total of 318 respondents was collected. Six respondents were excluded because they did not complete one or more entire sections of direct link to a survey. Data from an additional respondent was removed because their reported age was under 18 years. A final total of 311 respondents were included. Demographic characteristics of the sample are displayed in Table 1. The ages of the participants ranged from 18 to 78 years (M = 44.54, SD = 16.03). Most respondents indicated their sex/gender as female or male, with approximately equal representation of women and men in the sample. Most respondents indicated their sexual orientation as straight (n = 247, 79.4%). The majority of respondents identified their race as White (n = 253, 81.4%), and the largest proportion of participants indicated that a Bachelor's degree was the highest level of education they had completed (n = 88, 28.3%).

Table 1

Variable	Frequency	Percent
Sex		
Female	154	49.5
Male	152	48.9
Transmale	3	1.0
Missing/Declined to state	2	0.6
Gender		
Female	151	48.6
Male	150	48.2
Gender Non-Conforming	1	0.3
Nonbinary	7	2.3
Gender Fluid	1	0.3
Missing/Declined to state	1	0.3
Sexual orientation		
Straight	247	79.4
Gay	15	4.8
Lesbian	5	1.6

Sample Demographic Characteristics

Bisexual	33	10.6
Pansexual	7	2.3
Other	4	1.3
D		
Race		
White or Caucasian	253	81.4
Hispanic or Latino	28	9.0
Asian or Asian American	13	4.2
American Indian or Alaska Native	4	1.3
Black or African American	13	4.2
Education		
Did not Graduate High School	4	1.3
High School Diploma, GED, or Equivalent	52	16.7
Some College	61	19.6
Associate's Degree	31	10.0
Bachelor's Degree	88	28.3
Graduate Degree	70	22.5
Other	5	1.6

Procedure

Data were gathered using the survey site, Survey Monkey. Recruitment occurred through several social media platforms (i.e., Facebook, Instagram, Reddit). Respondents were provided with an invite and link to the survey. Upon following the link, respondents read the informed consent document as well as a brief description of the study. Respondents were then asked for demographic information before proceeding to the survey. At the completion of the survey, respondents received a letter including widely accessible mental health resources.

Measures

Life History

Međedović (2020) identified several psychosocial-biodemographic indicators of life history strategy – environmental harshness and instability, mating, and reproduction. The indicators suggested by Međedović (2020) were applied as a measure of life history in the current study. Respondents were asked about the socio-economic conditions, supportiveness, and stability of their developmental environment using multiple choice and Likert-type scales. Respondents answered yes-or-no questions about the composition of their developmental family. Respondents were also asked about their mating history as well as their attitudes regarding reproduction and childrearing using open ended, yes-or-no, and Likert-type scales.

Depression

Depression was measured using the Quick Inventory of Depressive Symptomatology, self-rated (QIDS-SR) (Rush et al., 2003). The QIDS-SR measures the severity of depressive symptoms in adults 18 and older. There are 16 measures selected from the Inventory of Depressive Symptomology (IDS, 2000). Users respond on a 4-point Likert-type scale to assess their behaviors and mood over the course of the past week. It takes five to seven minutes to complete the screener. It is important to note that this measure is a screener, rather than a diagnostic tool. This measure has acceptable reliability ($\alpha = .86$).

Data Analysis

Structural Equation Modeling

Structural equation modeling (SEM) refers to an array of widely used statistical analysis techniques such as path analysis, factor analysis, multiple regression etc. It can be employed to analyze structural models containing latent variables and can be used to evaluate complex causal relationships between multiple variables (Gamst & Guarino, 2013; Fan et al., 2016). Structural equation modeling is composed of a measurement model and a structural model (Fan et al., 2016), "A measurement model measures the latent variables or composite variables, while the structural model tests all hypothetical dependencies based on path analysis" (p. 03). In the

current research, life history indicators will be used to comprise a single latent variable of life history strategy. This will be the measurement model. The structural model will measure the extent of the relationship between life history strategy and depression and sex.

Evaluating Model Fit

The chi-square (χ^2) likelihood ratio tests the differences between the theoretical and the observable model (Fan et al., 2016). A significant χ^2 indicates that the theoretical model does not fit the observable data. A non-significant χ^2 suggests indicates a good fit between the two. The χ^2 will be used to determine the fit of this model.

Sample Size

There is little consensus regarding appropriate sample size for SEM (Wolf et al., 2013; Schumacker & Lomax, 2016). Moreover, rules of thumb can pose issues. Wolf et al. (2013) notes that there is a high level of variability in the sample size requirements for SEM. Because of this, it is preferable for researchers to consider sample sizes estimates within a specific range. They suggest that minimum sample size requirements range from 30-460 cases.

CHAPTER IV: RESULTS

Data Preparation

The dataset contained an initial total of 318 responses. Data from six respondents were removed because the respondents did not complete one or more entire sections of the survey. An additional respondent was excluded because their reported age was under 18 years. A final total of 311 respondents were included. All remaining missing data was handled using full information maximum likelihood estimation in the confirmatory factor analysis and structural equation model.

Items pertaining to life history were numerically coded such that higher values of the variables reflected slower life history. To achieve this for all variables, survey questions 14 (number of siblings), 17 (number of children), 19 (having children should be spontaneous), and 20 (planned to have first child) were reverse coded. Responses to open-ended numerical items were screened for validity by removing non-numerical responses and replacing participant-reported ranges with the midpoint of the range. These items also were screened for extreme values. Specifically, for survey question 15 (age sexually active), values less than 5 and values greater than the participant's reported age were removed. For survey question 16 (duration of longest relationship), values reflecting a duration greater than 50% of the participant's reported age were removed. For the purposes of the analysis, the responses to questions 21 (age they had first child) and 22 (age they desire to have their first child) were combined into a single item reflecting the age at which participants either had or desired to have their first child. Finally, composite scores were calculated for depression following the scoring instructions for the QIDS-SR.

Descriptive Statistics

Demographic characteristics of the sample are displayed in Table 1. The ages of the participants ranged from 18 to 78 years (M = 44.54, SD = 16.03). Most participants indicated their sex/gender as female or male, with approximately equal representation of women and men in the sample. Most participants indicated their sexual orientation as straight (n = 247, 79.4%). The majority of participants identified their race as White (n = 253, 81.4%), and the largest proportion of participants indicated that a Bachelor's degree was the highest level of education they had completed (n = 88, 28.3%).

The largest proportion of participants indicated that their family had occasional financial difficulties. Approximately 46% of participants reported that their family relationships were supportive or highly supportive (n = 143), and most participants indicated that their family was moderately or highly stable (n = 163, 52.4%). Approximately 27% of participants grew up in single parent households (n = 84), and 24% grew up with a stepparent (n = 75). The majority of participants had one or more siblings (n = 260, 83.6%). Most participants had one or more children (n = 185, 59.5%). The majority of participants completely agreed that it is important to plan before having children (n = 189, 60.8%), whereas 10.9% of participants completely agreed that having children should be spontaneous (n = 34). Approximately 39% of participants said that they planned to have their first child (n = 120). Thirty percent of participants indicated that they were highly able to financially support a child (n = 94), and 35% completely agreed that raising children is one of the most important things in life (n = 110).

Variable		Percent
Family's financial situation		
Missing/Declined to state	7	2.3
My family had frequent financial difficulties.	79	25.4
My family had occasional financial difficulties.	102	32.8
My family had infrequent financial difficulties.	55	17.7
My family did not have financial difficulties.	68	21.9
Family relationships		
Missing/Declined to state	6	1.9
1. Not at all Supportive and Distant	28	9.0
2. Somewhat Supportive and Distant	72	23.2
3. Somewhat Supportive and Close	62	19.9
4. Supportive and Close	71	22.8
5. Highly Supportive and Very Close	72	23.2
Family stability		
Missing/Declined to state	6	1.9
1. Highly Unstable	29	9.3
2. Moderately Unstable	47	15.1
3. Somewhat Stable	66	21.2
4. Moderately Stable	73	23.5
5. Highly Stable	90	28.9
Grew up in single parent household	0	2.0
Missing/Declined to state	9	2.9

Table 2Descriptive Statistics for Categorical Life History Variables

Yes	84	27.0
No	218	70.1
Grew up with stepparent		
Missing/Declined to state	4	1.3
Yes	75	24.1
No	232	74.6
Grew up with stepsiblings or half-siblings		
Missing/Declined to state	6	1.9
Yes	77	24.8
No	69	22.2
N/A	159	51.1
Number of siblings		
Missing/Declined to state	3	1.0
I don't have any siblings and/or stepsiblings.	48	15.4
1	76	24.4
2	88	28.3
3	38	12.2
4	27	8.7
5 or More	31	10.0
Number of children		
I do not have children	121	38.9
1	53	17.0
2	57	18.3
3	37	11.9
4	19	6.1

5	11	3.5
6 or More	8	2.6
Missing/Declined to state	5	1.6
Important to plan having children		
Missing/Declined to state	3	1.0
1. Completely Disagree	7	2.3
2. Somewhat Disagree	11	3.5
3. Neither Agree or Disagree	32	10.3
4. Somewhat Agree	69	22.2
5. Completely Agree	189	60.8
Having children should be spontaneous		
Missing/Declined to state	7	2.3
1. Completely Disagree	107	34.4
2. Somewhat Disagree	72	23.2
3. Neither Agree or Disagree	61	19.6
4. Somewhat Agree	30	9.6
5. Completely Agree	34	10.9
Planned to have your first child		
Missing/Declined to state	8	2.6
Yes	120	38.6
No	64	20.6
I do not have children	119	38.3
Could financially support children		
Missing/Declined to state	13	4.2

1. Not at all Able	43	13.8
2. Barely Able	20	6.4
3. Somewhat Able	51	16.4
4. Moderately Able	90	28.9
5. Highly Able	94	30.2
Raising children is important		
Missing/Declined to state	12	3.9
1. Completely Disagree	30	9.6
2. Somewhat Disagree	35	11.3
3. Neither Agree or Disagree	59	19.0
4. Somewhat Agree	65	20.9
5. Completely Agree	110	35.4

Table 3

Descriptive Statistics for Categorical Life History Variables

Variable	п	М	SD	Min	Max
Age became sexually active	255	18.03	4.46	5	40
Duration of longest relationship (months)	242	75.80	86.61	0	444
Age had/desire to have first child	228	25.61	9.20	0	58

On average, participants reported becoming sexually active at approximately 18 years of age (SD = 4.46). Participants' longest relationships, on average, were 75.80 months in duration (SD = 86.61). On average, participants either had or desired to have their first child at 25.61 years of age (SD = 9.20).

Finally, descriptive statistics were computed for the depression (QIDS-SR) composite scores. The depression scores ranged from 0 to 27 with a mean of 12.90 (SD = 5.65, n = 266). These scores indicate that, on average, members of the sample experienced moderate depression.

Confirmatory Factor Analysis

Selected life history variables were submitted to a confirmatory factor analysis to determine a set of items to use as indicators for the latent life history variable. The analysis was conducted in Mplus software using full information maximum likelihood estimation. The initial measurement model included 16 indicators loading on a single latent factor: financial difficulties, family relationships, family stability, single parent household, step-parent, step siblings, number of siblings, age became sexually active, longest relationship duration, number of children, important to plan for children, having children should be spontaneous, planned to have first child, age had/desire to have first child, able to financially support children, and raising children is important. Multicollinearity was assessed by examination of R^2 values for each indicator in the initial measurement model. One indicator had a high R^2 value (stepparent, $R^2 = .95$), indicating possible multicollinearity. Bivariate correlations were computed between the stepparent variable and all other indicators to further examine the degree of multicollinearity. The magnitude of the correlations ranged from .00 to .73. As no bivariate correlations exceeded .9 in magnitude, the degree of multicollinearity was retained.

The chi-square test of model fit for the initial measurement model was significant, $\chi^2(59)$ = 110.50, *p* < .001. The value of the Akaike Information Criterion (AIC) was 18922.33, and the value of the Bayesian Information Criterion (BIC) was 19105.58. Table 4 displays the standardized loadings for each indicator. Indicators with weak loadings (i.e., standardized loading magnitude less than .32) were removed from the model. Indicators that were removed included financial difficulties, family relationships, family stability, age became sexually active, longest relationship duration, important to plan for children, and age had/desire to have first child. The final measurement model with the weakly loading indicators removed was significant, $\chi^2(60) = 110.90$, p < .001. The value of the AIC increased to 18932.76 suggesting a worse fit than the initial model, and the value of the BIC decreased to 19089.83 suggesting a better fit than the initial model. Table 4 displays the standardized loadings for each indicator in the final measurement model.

Table 4

	Initial Model		Final Model	
Indicator	Standardized	р	Standardized	р
	Loading		Loading	
Financial difficulties	0.21	<.001		
Family relationships	0.01	.835		
Family stability	0.10	.123		
Single parent household	0.77	<.001	0.74	<.001
Stepparent	0.98	<.001	0.98	<.001
Stepsiblings	0.86	<.001	0.86	<.001
Number of siblings	0.33	<.001	0.33	<.001
Age sexually active	0.04	.568		
Longest relationship	0.13	.073		
Number of children	0.45	<.001	0.45	<.001
Important to plan for children	0.14	.021		
Children should be spontaneous	0.44	<.001	0.43	<.001
Planned to have first child	0.35	<.001	0.35	<.001
Age had/desire to have first child	0.23	.003		
Able to financially support children	-0.32	<.001	-0.36	<.001
Raising children is important	-0.35	<.001	-0.37	<.001

Structural Equation Model

To test the relationship between life history and depression, a structural equation model was computed. In this analysis, life history was a latent variable measured using the indicators determined from the confirmatory factor analysis. Life history served as the independent variable in this model. The depression composite score served as the dependent variable in this model. Participant sex was included as a moderating variable in this model; a life history × sex interaction term was included in the model. This analysis only included participants who identified their sex as female or male. The model consisted of regression paths from the life history latent variable, sex, and the life history × sex interaction to depression.

The chi-square test of model fit for the structural model was significant, $\chi^2(25) = 63.49$, p < .001. Table 5 displays the results of the model regression estimates. The regression path from life history to depression was not significant ($\beta = -0.14$, p = .516), indicating that there was no significant relationship between life history and depression scores. The regression path for the life history × sex interaction was not significant ($\beta = -0.03$, p = .719), indicating that sex did not significantly moderate the relationship between life history and depression scores.

Table 5

Path From	Path To	Standardized Estimate	р
Life history	Depression	-0.14	.516
Sex [Male]	Depression	-0.04	.491
Life history × Sex	Depression	-0.03	.719

Results of Model Regression Estimates

Summary

Data from 311 participants were analyzed to develop a measurement model for life history and test the relationships between life history, sex, and depression. Sixteen life history indicators were submitted to a confirmatory factor analysis, and nine indicators were found to load strongly on a single latent factor: single parent household, stepparent, stepsiblings, number of siblings, number of children, having children should be spontaneous, planned to have first child, able to financially support children, and raising children is important. A structural equation model was then created to test the relationship between the life history latent variable and depression, with sex included as a moderating variable. The results showed that there was no significant relationship between life history and depression, and there was no significant moderating effect of sex.

CHAPTER V: DISCUSSION

The broad aim of this study was to empirically examine the FSD model of psychopathology developed by Del Giudice (2018), specifically defense activation disorders. Defense activation disorders, as a category, are believed to be a product of dysregulated defense mechanisms which may appear at either end of the fast/slow continuum. The current study gave particular focus to the causal relationship between life history strategies and defense disorders, as well as the moderating role of sex. The FSD model predicts that women with fast life history strategies should experience greater symptoms of defense activation disorders than their male counterparts. However, exploring the entire category of defense activation disorders was beyond the scope of the present research. Instead, the primary disorder of interest was depression. Accordingly, this study sought to test two hypotheses:

- a. There is a causal relationship between life history strategy and depression.
- b. Sex has a moderating role in the relationship between life history strategy and depression.
 It is predicted that women on the fast end of the life history spectrum experience greater symptoms of depression that male with fast life history strategies.

Prior findings demonstrated identified mediational pathways between fast life history strategies and predicted dysfunctional schemas which predicted greater depressive, anxious, and obsessive-compulsive symptoms (Kahl et al., 2021). There is also a significant body of evidence to suggest that life history strategies are associated with social, mental, and emotional distress (Hurst & Kavanagh, 2017; Csathó & Birkás, 2018; Cui & Lan, 2020; Kahl et al, 2020). Surprisingly, the current research did not demonstrate a causal relationship between life history strategies and depressive symptoms. Additionally, the relationship between sex and depression has been well supported by existing research (Cryanowski et al., 2000; Baxter et al., 2014). Contrarily, the current research found that sex did not play a moderating role in the relationship between life history strategy and depression.

A fundamental difference between the current study and previous research is how life history strategies were measured. The findings reported by Hurts and Kavanagh (2017), and Kahl et al., 2021, relied on psychometric tools to evaluate life history strategy. However, measures such as the Arizona Life History Battery (Figueredo, 2007) have been criticized for over-emphasizing the cognitive aspects of LHT (Međedović, 2020). Moreover, it has been argued that such tools have strayed from the conceptual framework of LHT (Sear, 2020). Sear (2020) asserted that, in order, to keep life history research grounded in the framework of LHT, greater emphasis should be placed on life history trade-offs. The life history strategy an individual adopts ultimately arises because their life history traits are either associated with or are trade-offs against each other.

Resultingly, the current research attempted to measure life history theory by using psychosocial-biodemographic indicators of life history strategy that were grounded in somatic, resource, and reproductive trade-offs. However, the measure applied in this study had not been previously validated. It was assumed that the 16 life history indicators used in the survey would load onto a single latent factor and a confirmatory factor analysis was applied. Although 9 indicators did strongly load onto a single latent variable, the lack of prior validation is likely to have created issues. It is possible that the latent variable that was applied to the structural equation model may partially explain the lack of a relationship between depression and life history strategies. A similar issue may have arisen when sex was implemented as a moderating variable between life history strategies and depression, that is if the life history variable did not properly capture life history trade-offs and strategies, the role of sex may not have been

applicable. Notably, on average participants reported moderate depression. However, due to the aforementioned issues, it is unclear whether their depressive symptoms could be attributable to life history strategies or trade-offs. Additionally, this researcher did not conduct a careless and inattentive responding analysis (CIR) (Curran, 2016). A CIR is typically applied to ensure that participants are not responding in a careless or inattentive manner by providing cut-offs to determine whether a specific participant responded accurately and attentively. Additionally, in an attempt to reduce the possibility of participant attrition, this researcher composed a brief study. The upshot to this is that the indicators of life history strategy were limited in scope. Finally, and more significantly, there are significant challenges to measuring trade-offs in human life histories (Bolund, 2020). A survey approach to such measurements may fall short of doing so.

Directions for Future Research

The use of an unvalidated questionnaire to measure life history strategies posed challenges to the current research. While this study attempted to approach life history strategies using psychosocial-biodemographic indicators of life history strategy, a number of the indicators did not load on a single latent factor as anticipated. Recommendations for future research in this domain are twofold. It is advised that a more robust measure of life history trade-offs be developed. The current measure screened for indicators, but additional items pertaining to the psychosocial-biodemographic indicators of life history strategies could be of use. Second and more importantly, this approach needs a well-validated measure of life history trade-offs.

Critics of the application of LHT to human developmental psychology have argued that human LHT research has diverged into several branches: evolutionary anthropology, evolutionary developmental psychologists, and evolutionary personality psychologists (Sear, 2020). Within the evolutionary social sciences, LHT is often used interchangeably with life history strategies. Due to this, a rift has formed between LHT programs in evolutionary biology and the evolutionary social sciences. In a literature review, Nettle and Frankenhuis (2019) observed that the citations between the two bodies of research seldom overlap. Additional confusion has arisen as both research paradigms apply the same terminology for conceptually different research schemas. Moreover, the use of LHT in the evolutionary social sciences is not consistent, with some researchers grounding their application of LHT in an evolutionary biological framework whereas others work completely in the in the social science paradigm. Bolund (2020) notes the inherent challenges of interdisciplinary work:

...if different disciplines venture into the domains of each other without much communication, there is a risk of misapplying theories or methods that have been developed for decades between one field, before being applied in a new contact in another field of enquiry (p. 502).

Although, this challenge is seldom addressed, it impacts and leads to confusion between fields.

The field of evolutionary psychology is relatively young and will face growing pains. Although evolutionary psychological researchers are confident that a framework grounded in evolutionary sciences would provide a theoretical unity of human ecology and behavior within the social sciences and between various disciplines (Tooby & Cosmides, 2016), at present this is not the case. With particular regard to LHT psychological research, the field would benefit from interdisciplinary collaboration, as well as collaboration between evolutionary social science researchers. Doing so may foster a greater understanding of human life history strategies as well as produce novel methods of measuring them in humans. Carrying along this line, future research may need to adopt new methods of evaluating life history strategies (Sear, 2020; Bolund, 2020). Bolund (2020) outlines several possible methods for evaluating human LHT that would be grounded in evolutionary biology. Doing so would accomplish the aforementioned aim of integrating with other disciplines, but it could also yield a more complete model of LHT and mental disorder.

Conclusion

Although, the current research did not find a causal relationship between life history strategies and depression, it did provide an initial examination of the relationship between psychosocial-biodemographic indicators of life history strategies and depression. It also yielded potential directions for future human LHT research to accomplish the end goal of developing a coherent taxonomy of human behavior and mental disorder. Research of this type is expected to result in a more complete understanding of mental disorders. Further, it is hoped that the development of a new paradigm may aid in the treatment of mental disorders, offering clinical practitioners novel treatment approaches.

REFERENCES

American Psychiatric Association. (2022). *Diagnostic and statistical manual of mental disorders* (5th ed.) https://doi.org/10.1176/appi.books.9780890425596

Alcock, J. (2001). The triumph of sociobiology. Oxford University Press.

- Arias, I., Sorlozano, A., Villegas, E., de Dios, L., McKenny, K., Cervilla, J., Gutierrez, B., & Gutierrez, J. (2012) Infectious agents associated with schizophrenia: A meta-analysis.
 Schizophrenia Research, 136, 128-136. https://doi.org/10.1016/j.schres.2011.10.026
- Bailey, K. (1987). Human Paleopsychology: Applications to aggression and pathological processes. Lawrence Erlbaum Associates. https://doi.org/10.4324/9780203781937
- Baumard, A; Belsky, J., Steinberg, L., & Draper, P. (1991). Childhood experience, interpersonal development, and reproductive strategy: An evolutionary theory of socialization. *Child Development*, 62, 647-670. https://doi.org/10.1111/j.1467-8624.1991.tb01558
- Bergstrom, C.T., Meacham, F. (2016). Depression and anxiety: Maladaptive byproducts of adaptive mechanisms. *Evolution Medicine & Public Health*, 214-218. https://doi.org/10.1093/emph/eow019
- Bolund, E. (2020) The challenge of measuring trade-offs in human life history research. *Evolution and Human Behavior*, *41*, 502-512.

https://doi.org/10.1016/j.evolhumbehav.2020.09.004

- Brezina, T., Tekin, E., & Topalli, V. (2009). Might not be a tomorrow: A multimethods approach to anticipated early death and youth crime. *Criminology*, 47, 1091-1129. https://doi.org/10.1111/j.1745-9125.2009.00170
- Brune, M., Belsky, J., Fabrega, H., Feierman, J.R., Gilber, P., Glantz, K., Poliment, J., Price, J.S., Sanjuan, J., Sullivan, R., Troisi, A., Wilson, D.R. (2012). The crisis of

- psychiatry: Insights and prospects from evolutionary theory. *World Psychiatry*, 11(1), 55-57. https://doi.org/10.1016/j.wpsyc.2012.01.009
- Buss, D.M. (2016). Foundations of evolutionary psychology. In D.M. Buss (Ed.), *The handbook of evolutionary psychology, Vol. 1: Foundations* (2nd ed., pp. 3-88). Wiley & Sons.
- Chen, P., Vazsonyi, A.T. (2011). Future orientation, impulsivity, and problem behaviors: A longitudinal moderation model. *Developmental Psychology*, 47, 1633-1645. https://doi.org/10.1037/a0025327
- Chisholm, J.S., Quinlivan, J.A., Petersen, R.W., & Coall, D.A. (2005). Early stress predicts age at menarche and first birth, adult attachment, and expected lifespan. *Human Nature*, 16, 233-265. https://doi.org/10.1007/s12110-005-1009-0
- Copping, L.T., Campbell, A., & Muncer, S. (2013). Violence, teenage pregnancy, and life history: Ecological factors and their impact on strategy-driven behavior. *Human Nature*, 24(2), 137-157. https://doi.org/10.1007/s12110-013-9163-2
- Cosmides, L., Tooby, J. (2016). The theoretical foundations of evolutionary psychology. In D.M.
 Buss (Ed.), *The handbook of evolutionary psychology, Vol. 1: Foundations* (2nd ed., pp. 3-88). Wiley & Sons.
- Csathó, A., & Birkás, B. (2018) Early-life stressors, personality development, and fast life history strategies: An evolutionary perspective on malevolent personality features. *Frontiers in Psychology*, 9, 1-6. https://doi.org/10.3389/fpsyg.2018.00305
- Cui, G., & Lan, X. (2020) The associations of parental harsh discipline, adolescents' gender, and grit profiles with aggressive behavior among chinese early adolescents. *Frontiers in Psychology*, 11, 1-14. https://doi.org/10.3389/fpsyg.2020.00323

- Curran, P.G., (2016) Methods for the detection of carelessly invalid responses in survey data. Journal of Experimental Psychology, 66, 4-19. https://doi.org/10.1016/j.jesp.2015.07.006
- Cuthbert, B.N., & Insel, T.R. (2013). Toward the future of psychiatric diagnosis: The seven pillars of RDoC. *BMC Medicine*, 11, 126. https://doi.org/10.1186/1741-7015-11-126

Darwin, C. (1859). On the origins of species by natural selection. Murray.

Dawkins, R. (1976) The selfish gene. Oxford University Press.

- Del Giudice, M. (2014). An evolutionary life history framework for psychopathology. *Psychological Inquiry*, 25, 261-300.
- Del Giudice, M., & Ellis, B.J. (2016). Evolutionary foundations of psychopathology. In D. Cicchetti (Ed.), *Developmental Psychopathology*.
- Del Giudice, M. (2018). Evolutionary psychopathology: A unified approach. Oxford University Press.
- Del Giudice, M., Gangestad, S.W., & Kaplan, H.S. (2016). Life history theory and evolutionary psychology. In D.M. Buss (Ed.), *The handbook of evolutionary psychology, Vol. 1: Foundations* (2nd ed., pp. 3-88). Wiley & Sons.
- Del Giudice, M. (2023) An integrative evolutionary framework for psychopathology. Development and Psychology, 35(1), 1-11. https://doi.org/10.1017/S0954579421000870
- Dunkel, C. S., Mathes, E., & Beaver, K. M. (2013). Life history theory and the general theory of crime: Life expectancy effects on low self-control and criminal intent. *Journal of Social*, *Evolutionary, and Cultural Psychology*, 7(1), 12–23. https://doi.org/10.1037/h0099177
- Ellis, B.J., & Bjorklund, D.F. (2012) Beyond mental health: An evolutionary analysis of development under risky and supportive environmental conditions: An introduction to the

special section. *Developmental Psychology*, 48 (3), 591-597. https://doi.org/10.1037/a0027651

- Ellis, B.J., & Del Giudice, M. (2019). Developmental adaptation to stress: An evolutionary perspective. *Annual Review of Psychology*, 70, 111-139. https://doi.org/10.1146/annurevpsych-122216-011732
- Ellis, B.J., Figueredo, A.J., Brumbach, B.H., & Schlomer, G.L. (2009). Fundamental dimensions of environmental risk: The impact of harsh vs unpredictable environments on the evolution and development of life history strategies. *Human Nature*, 20, 204-268. https://doi.org/10.1007/s12110-009-9063-7
- Ellis, B.J., & Bjorklund, D.F. (2012) Beyond mental health: An evolutionary analysis of development under risky and supportive environmental conditions: An introduction to the special section. *Developmental Psychology*, 48 (3), 591-597.

Field, A.F. (2013). Discovering statistics using ibm spss statistics. Sage Texts.

- Figueredo, A.J., Cabeza de Baca, T., & Woodley, M.A. (2013). The measurement of human life history strategies. *Personality and Individual Differences*, 55(3), 251-255. https://doi.org/10.1016/j.paid.2012.04.033
- Figueredo, A. J., Garcia, R. A., Menke, J. M., Jacobs, W. J., Gladden, P. R., Bianchi, J., Patch, E. A., Beck, C. J., Kavanagh, P. S., Sotomayor-Peterson, M., Jiang, Y., & Li, N. P. (2017).
 The K-SF-42. *Evolutionary psychology: an international journal of evolutionary approaches to psychology and behavior*, 15(1)
 https://doi.org/10.1177/1474704916676276

- Figueredo, A. J., Vásquez, G., Brumbach, B. H., & Schneider, S. M. R. (2004). The heritability of life history strategy: The K-factor, covitality, and personality. *Social Biology*, 51, 121– 143. https://doi.org/10.1080/19485565.2004.9989090
- Figueredo, A.J., Vásquez, G., Brumbach, B.H., Schneider, S.R., Sefcek, J.A., Tal, IR., Jacobs,
 W.J. (2006). Consilience and life history theory: From genes to brain to reproductive strategy. *Developmental Review*, 26, 243–275.
- Figueredo, A. J., Wolf, P. S. A., Olderbak, S. G., Gladden, P. R., Fernandes, H. B. F., Wenner, C., Hill, D., Andrzejczak, D. J., Sisco, M. M., Jacobs, W. J., Hohman, Z. J., Sefcek, J. A., Kruger, D., Howrigan, D. P., MacDonald, K., & Rushton, J. P. (2014). The psychometric assessment of human life history strategy: A meta-analytic construct validation. *Evolutionary Behavioral Sciences*, 8(3), 148–185. https://doi.org/10.1037/h0099837
- Geary, D.C. (2002). Sexual selection and human life history. *Advances in Child Development* and Behavior, 30, 41-101. https://doi.org/10.1016/s0065-2407(02)80039-8
- Giosan, C (2006). High-K strategy scale: A measure of the high-k independent criterion of fitness. *Evolutionary Psychology*, 4, 394–405.

https://doi.org/10.1177/147470490600400131

- Hamilton, W.D. (1964). The genetical evolution of social behaviour. *Journal of Theoretical Biology*, 7, 1-52. https://doi.org/10.1016/0022-5193(64)90038-4
- Han, W., & Chen, B.-B. (2020). An evolutionary life history approach to understanding mental health. *General Psychiatry*, 33(6), 1–9. https://doi.org/10.1136/gpsych-2019-100113
- Hill, K. (1993). Life history theory and evolutionary anthropology. *Evolutionary Anthropology*, 2, 78-88. https://doi.org/10.1002/evan.1360020303

Hill, K., & Kaplan, H. (1999). Life history traits in humans: Theory and empirical studies.
 Annual Review of Anthropology, 28, 397-430.
 https://doi.org/10.1146/annurev.anthro.28.1.397

- Hurst, J. E. & Kavanagh, P. S. (2017). Life history strategies and psychopathology: the faster the life strategies, the more symptoms of psychopathology. *Evolution and Human Behavior*, 38(1), 1–8. https://doi.org/10.1016/j.evolhumbehav.2016.06.001
- James, J., Ellis, B.J., Schlomer, G.L., & Garber, J. (2012). Sex specific pathways to early puberty, sexual debut, and sexual risk taking: Tests of an integrated evolutionarydevelopmental model. *Developmental Psychology*, 48, 687-702. https://doi.org/10.1037/a0026427
- Kahl, B.L., Kavanagh, P.S., Gleaves, D.H. (2021) Extending a life history model of psychopathology: Expectations schemas and mechanisms. *Evolutionary Psychological Science*, 158–173 (2022). https://doi.org/10.1007/s40806-021-00300-1
- Kahl, B.L., Kavanagh, P.S. & Gleaves, D.H. (2022). Testing a life history model of psychopathology: A replication and extension. *Current Psychology*, 41, 6233–6246. https://doi.org/10.1007/s12144-020-01062-y
- Kaplan, H., Lancaster, J., & Robson, A. (2003). Embodied capital and the evolutionary economics of the human life span. *Population & Development Review*, 29(1), 152–182. https://doi.org/10.1111/j.1728-4457.2003.00113
- Kavanagh, P.S., & Kahl, B.L. (2018). Are expectations the missing link between life history strategy and psychopathology? *Frontiers in Psychology*, 89(9), 1-7. https://doi.org/10.3389/fpsyg.2018.00089

Kennair, L., (2014) Evolutionary psychology and life history: A clinician's perspective.*Psychological Inquiry*, 25, 346-351. https://doi.org/10.1080/1047840X.2014.915707

- Kuzawa, C. W., & Bragg, J. M. (2012). Plasticity in human life history strategy: Implications for contemporary human variation and the evolution of genus homo. *Current Anthropology*, 53(Number S6), S369–S382. https://doi.org/ 10.1086/667410
- Laghi, F., D'Alessio, M., Pallini, S., & Baiocco, R. (2009). Attachment representations and time perspective in adolescence. *Social Indicators Research*, 90, 181-194. https://doi.org/10.1007/s11205-008-9249-0
- Li, N.P., Van Vugt, M., & Colarelli, S.M. (2018). The evolutionary mismatch hypothesis: Implications for psychological science. *Current Directions in Psychological Science*, 27 (1), 38-44. https://doi.org/10.1177/0963721417731378
- Međedović, J. (2020). On the incongruence between psychometric and psychosocialbiodemographic measures of life history. *Human Nature*, 31, 341-360. https://doi.org/10.1007/s12110-020-09377-2
- Meacham, F., Bergstrom, C.T. (2016). Adaptive behavior can produce maladaptive anxiety due to individual differences in experience. *Evolution Medicine & Public Health*, 270-285. https://doi.org/10.1093/emph/eow024
- Mell, H., Safra, L. Algan, Y, Baumard, N and Chevallier, C. (2018) Childhood Environmental Harshness Predicts Coordinated Health and Reproductive Strategies: A Cross-sectional Study Of A Nationally Representative (France) Sample'. Evolution and Human Behavior, 39(1), 1-8.
- Nesse, R.M. (2005). Maladaptation and natural selection. *Quarterly Review of Biology*, 80(1), 62-70. https://doi.org/10.1086/431026

Nesse, R.M. (2011). Ten questions for evolutionary studies of disease and vulnerability. *Evolutionary Applications*, 264-277. https://doi.org/10.1111/j.1752-4571.2010.00181

- Nesse, R.M. (2013). Tinbergen's four questions organized: A response to bateson and laland. *Trends in Ecology and Evolution*, 28(12), 681-682. https://doi.org/10.1016/j.tree.2013.10.008
- Nesse, R.M. (2016). Evolutionary psychology and mental health. In D.M. Buss (Ed.), *The handbook of evolutionary psychology, Vol. 2: Integrations* (2nd ed., pp. 1007-1026).
 Wiley & Sons.
- Nesse, R.M. (2019). Good reasons for bad feelings: Insights from the frontier of evolutionary psychiatry. Penguin.
- Nesse, R. M., Bergstrom, C. T., Ellison, P. T., Flier, J. S., Gluckman, P., Govindaraju, D. (2010). Making evolutionary biology a basic science for medicine. *Proceedings of the National Academy of Sciences*, 107(Suppl_1), 1800–1807. https://doi.org/10.1073/pnas.0906224106
- Neter, J., Kutner, M., Nachtsheim, & Wasserman, W. (1996). *Applied linear statistical models*. McGraw Hill.
- Nettle, D., & Frankenhuis, W.E. (2019) The evolution of life history theory: Bibliometric analysis of an interdisciplinary research area. *Proceedings of the Royal Society of Biological Sciences*, 286 (1899). https://doi.org/10.6084/m9.figshare.c.4440197.v2
- Raison, C.L., & Miller, A.H. (2017). Pathogen-host defense in the evolution of depression:
 Insights into epidemiology, genetics, bioregional differences and female preponderance.
 Neuropsychopharmacology, 47, 5-27. https://doi.org/10.1038/npp.2016.194

- Ray, W.J. (2020). Psychopathology from an evolutionary perspective. In L. Workman, W.
 Reader & J.H. Barkow (Eds.) *The cambridge handbook of evolutionary perspectives on human behavior* (pp. 395-408). University Printing House.
- Rush, A. J., Trivedi, M. H., Ibrahim, H. M., Carmody, T. J., Arnow, B., Klein, D. N., Markowitz, J. C., Ninan, P. T., Kornstein, S., Manber, R., Thase, M. E., Kocsis, J. H., & Keller, M. B. (2003). The 16-Item Quick Inventory of Depressive Symptomatology (QIDS), clinician rating (QIDS-C), and self-report (QIDS-SR): a psychometric evaluation in patients with chronic major depression. *Biological psychiatry*, *54*(5), 573–583. https://doi.org/10.1016/s0006-3223(02)01866-8
- Schumacker, R.E., & Lomax, G.L. (2016). *A beginners guide to structural equation modeling* (4th ed.). Routledge.
- Sear, R. (2020). Do human 'life history strategies,' exist? *Evolution and Human Behavior*, 41(6), 513-526. https://doi.org/10.1016/j.evolhumbehav.2020.09.004
- Spielberger, C. D. (1989). *State-trait anxiety inventory: Bibliography* (2nd ed.). Consulting Psychologists Press.
- Stearns, S.C. (2012). Evolutionary medicine: Its scope, interest and potential. Proceedings of the Royal Society B: Biological Sciences, 279(1746), 4305-4321. https://doi.org/10.1098/rspb.2012.1326
- Tinbergen, N. (2005). On aims and methods of ethology. *Animal Biology*, 55(4). https://doi.org.10.1163/157075605774840941
- Tooby, J., & Cosmides, L. (1992). The psychological foundations of culture. In J. Barkow, L. Cosmides & J. Tooby (Eds.), *The adapted mind: Evolutionary psychology and the generation of culture* (pp. 19-136). Oxford University Press.

- Tooby, J., & Cosmides, L. (2016). The theoretical foundations of evolutionary psychology. InD.M. Buss (Ed.), *The Handbook of Evolutionary Psychology, Vol 1: Foundations* (2nd ed., pp. 3-87). Wiley & Sons.
- Wang, M. (2019). Harsh parenting and adolescent aggression: Adolescents' effortful control as the mediator and parental warmth as the moderator. *Child Abuse & Neglect*, 94, 104021. https://doi.org/10.1016/j.chiabu.2019.05.014
- Wakefield, J.C. (1992). The concept of mental disorder: On the boundary between biological facts and social values. *American Psychologist*, 47, 373-388. https://doi.org/10.1037//0003-066x.47.3.373

Wakefield, J.C. (1999). Evolutionary versus prototype analyses of the concept of disorder.

Journal of Abnormal Psychology, 108, 374-399. https://doi.org/10.1037/0021-843X.108.3.374

- Wakefield, J.C. (2016). Biological function and dysfunction: Conceptual foundations of evolutionary psychology. In D.M. Buss (Ed.), *The handbook of evolutionary psychology, Vol. 2: Integrations* (2nd ed., pp. 988-1006). Wiley & Sons.
- White T.D., Asfaw, B., Beyene, Y. Haile-Selassie, Y., Lovejoy, C.O., Suwa, G., & Wolde, G.
 (2009). Ardipithecus ramidus and the paleobiology of early hominids. *Science*, 326, 75-86. https://doi.org/10.10.1126/science.1175802

Williams, G.C. (1966). Adaptation and natural selection. Princeton University Press.

- Williams, G.C. (1992). Natural selection: Domains levels and challenges. Oxford University Press.
- Wolf, E. J., Harrington, K. M., Clark, S. L., & Miller, M. W. (2013). Sample Size Requirements for Structural Equation Models: An Evaluation of Power, Bias, and Solution Propriety.

Educational and psychological measurement, 76(6), 913–934.

https://doi.org/10.1177/0013164413495237

Workman, L., & Reader, W. (2016). Evolution and Behavior. Routledge.