DET PSYKOLOGISKE FAKULTET

Ψ

Why Does Depression Exist? A Review With New Predictions From Evolutionary Theories

HOVEDOPPGAVE

profesjonsstudiet i psykologi

Thomas Haarklau Kleppestø

Vår 2014

Veileder: Leif Edward Ottesen Kennair

Acknowledgments

I would like to express my gratitude and appreciation to my supervisor Leif Edward Ottesen Kennair. Thank you for believing in me.

I also want to thank Paul J. Watson for helpful conversations and comments on an earlier draft of the Social Navigation Hypothesis, and David Buss for planting seeds in my brain on the sex differences in depression.

Abstract

Depression is highly prevalent, but its evolutionary origin is poorly understood. Evolutionary psychology generates hypotheses on the computational and functional design architectures of the human mind. Evolutionary medicine and evolutionary psychopathology study the design characteristics of adaptations that make humans vulnerable to diseases. These perspectives are necessary when attempting to answer why complex traits exist in living organisms. Some theoreticians postulate that depression is an adaptation that solves particular problems, such as reducing the risk of being socially excluded, elicit help from others, or to analyze complex problems. Others view depression as an evolutionary side-effect of pathogen host defense, and others still view depression as dysfunctions of adaptive mood-mechanisms. The purpose of this paper is to discuss predictions from all evolutionary theories, and make new testable predictions from them. I will also discuss predictions from evolutionary theories on why women are more likely to be depressed than men (~2:1). I argue that research on normal sadness, or "low-mood" states are likely to illuminate our understanding of when depressed mood is working as designed and when it is malfunctioning.

Keywords: evolution; depression; sadness; mood; disorder; dysfunction; sex differences; evolutionary psychology; evolutionary psychopathology; theories; hypothesis

Sammendrag

Depresjon er svært utbredt, men dens evolusjonære opprinnelse er uklar. Evolusjonspsykologi genererer hypoteser om sinnets informasjonsprosesserende og funksjonelle design. Evolusjonsmedisin og evolusjonspsykopatologi studerer designkarakteristikkene til menneskets tilpasninger og hvordan disse kan gjøre mennesket sårbar for sykdommer. Disse perspektivene er nødvendige for å kunne svare på hvorfor komplekse trekk eksisterer i levende organismer. Noen teoretikere postulerer at depresjon er en tilpasning som løser spesifikke problemer, som å redusere risiko for å bli sosialt eksludert, utløse hjelp fra andre, eller analysere komplekse problemer. Andre ser på depresjon som en evolusjonær side-effekt av menneskers immunologiske forsvar mot patogener, mens andre ser på depresjon som dysfunksjoner av adaptive mekanismer som styrer stemningsleie. Hensikten med denne artikkelen er å diskutere prediksjoner fra alle evolusjonære teorier om hvorfor depresjon eksisterer og danne nye testbare prediksjoner fra teoriene. Jeg vil også diskutere prediksjoner fra evolusjonære teorier om hvorfor kvinner har større sjanse for å bli deprimerte enn menn (~2:1). Jeg argumenterer for at forskning på det evolusjonære designet til normal tristhet (eller lavt stemningsleie) vil forbedre vår forståelse av når depresjon og tristhet fungerer som designet av seleksjon og når disse tilstandene har blitt dysfunksjonelle.

Stikkord: evolusjon; depresjon; tristhet; stemningsleie; sykdom; dysfunksjon; kjønnsforskjeller; evolusjonspsykologi; evolusjonspsykopatologi; teorier; hypoteser

Table of Contents

Human depression is an evolutionary paradox. Depression is deeply debilitating and costly to its sufferers, yet it is highly prevalent. The lifetime estimate for Major Depressive Disorder (MDD) in North-America is 16.2% (Kessler et al., 2003). Depression is estimated (C. Murray & Lopez, 1997) to become the world's greatest cause of, only surpassed by heart disease, disability and mortality in 2020. Sufferers of depression typically lose all interest in pleasant activities; thinks and moves slowly; have trouble concentrating; experience either weight loss or gain; either insomnia or excessive sleep; feels sad, worthless, guilty and have thoughts about death and suicide (American Psychiatric Association, 2013; World Health Organization, 1992).

However, clinical depression aside, most humans are capable of experiencing *the blues.* Melancholia is a recurrent theme in the great literature, painting and music of our world. Sadness is ubiquitous—it is found, and recognized (Ekman et al., 1987), in all cultures. For example, most humans experience a dramatic, yet normal, grief and sadness at the loss of loved ones (Bowlby, 1980/1996), including our pets (Adrian, Deliramich, & Frueh, 2009; Gosse & Barnes, 1994). Depression, too, has been observed in traditional hunter-gatherer societies such as the !Kung of the Kalahari desert in southern Africa (Howell, 1979/2000), suggesting it has been a part of human nature for a long time. Indeed, Darwin (1872/1998) noted that human and non-human apes portrayed similar facial expressions in situations where they were depressed, which strongly suggests that sadness is an ancient phylogenetic trait (cross-species adaptation), likely to have evolved in the common ancestors of the great apes. Sadness might be evolutionary useful (Nesse, 2000). Or as Forgas (2007) put it, there might be "evolutionary advantages of not being too happy" (p. 107). Indeed, in four experiments conducted by Forgas (2007), the induction of sadness made people produce more concrete, persuasive, and higher-quality arguments, which was more likely to change other people's attitudes. In another study (Forgas & East, 2008) sadness was shown, relative

to neutral and happy mood, to improve accuracy in the detection of deceptive communication. There are many examples in the scientific literature (e.g. Raghunathan & Pham, 1999) of how sadness improves motivation, interpersonal behavior, memory, and judgment in particular ways (for a review, see Forgas, 2013). Keller and Nesse (2005) have argued that natural selection shaped subtypes of low mood, and they found evidence that specific situations lead to the different "symptoms" of low mood that they predicted, such as crying under social losses and fatigue when one failed to reach a goal.

The findings above suggest that transient sadness, or low-mood, have particular effects on information-processing in humans that might be useful. However, we know little on the evolutionary function, if any, of sadness and the relationship it might have with clinical depression.

If normal sadness is useful, what about mild and severe depression? In the scientific literature, depression has been associated with many different traits, which makes depression very complex and multifaceted. Depression, in both mild and major forms, independently predicts earlier death in elderly people (Schulz et al., 2000), it predicts cardiovascular disease and suicide (Mykletun et al., 2007), affective disorders cause 25 work loss days per month per 100 workers (Kessler & Frank, 1997), Major depression is associated with neurophysiological abnormalities in the medial and prefrontal cortex (Drevets, 2000), and cognitive deficits (Austin, Mitchell, & Goodwin, 2001; Hammar, Lund, & Hugdahl, 2003). Furthermore, depression is highly correlated with guilt and negative social comparison (O'Connor, Berry, Weiss, & Gilbert, 2002), and shame and rumination (Cheung, Gilbert, & Irons, 2004). Depression is associated with elevated body temperatures (Rausch et al., 2003) and reductions in bodily iron storage (Maes et al., 1996), and other inflammatory markers (see Raison, Capuron, & Miller, 2006), and is associated with decreased appetite (Paykel, 1977) but also with carbohydrate-cravings (Wurtman & Wurtman, 1995). And depression is

more common in women, than in men (~2:1) (Lopez, Mathers, Ezzati, Jamison, & Murray, 2006).

Some of these factors are obviously detrimental to survival and reproduction, so why does depression exist at all? Why has not natural selection eliminated the risk-alleles that make so many humans vulnerable to depression? Is depression an evolutionary function executed by the brain in specific situations? Or is depression the result of dysfunctional mood systems? Either way, when is depression a dysfunction, and when can we safely consider depression as an emotional system working as designed by natural selection?

The evolutionary hypotheses described in this article try to address these questions, albeit with very different, and sometimes contradictory, predictions. They also have unique problems. For example, adaptationist theories must explain why depression is so costly for the individual (Sloman, 2000), and nonadaptive theories must explain why depression is so prevalent.

All traits that we observe in biological organisms (phenotypes) can be divided into four categories. 1) The trait can be the workings of an adaptation (e.g. eyes, muscles, fear in the presence of predators); 2) be side-effects of adaptations (e.g. human belly-buttons, see Buss, Haselton, Shackelford, Bleske, & Wakefield, 1998); or 3) the trait can simply be the product of fitness-irrelevant, genetic "noise" (Confer et al., 2010), and 4) a trait can be a dysfunction of an adaptation, caused by, for example mutations or abnormal development. The evolutionary theories of depression can be divided into these categories: adaptive, side-effect, noise and dysfunction. Although no theory described here postulates that depression can be categorizes as fitness-neutral "noise"—it is either viewed as adaptive, as a side-effect of adaptations or as a dysfunction.

Some theories claim that depression, including MDD, has evolved to solve complex problems and to elicit help from others (Andrews & Thomson, 2009; Hagen, 2003; Watson &

Andrews, 2002). In other words, depression is hypothesized to be an adaptation that has, in certain contexts, helped our ancestors to survive and reproduce, and is now a part of human nature. Other theories hypothesize that depression is a side-effect of other adaptations, such as defending against pathogens (Raison & Miller, 2012). Lastly, some evolutionary theories view depression as nonadaptive: As a continuum around an adaptive peak of neuroticism (Nettle, 2004), or that major depression is best explained as a manifestation of the hundreds of mutations that all individuals carry, many of which affect the brain (Keller & Miller, 2006)

Opinions on the function of depression vary widely, from mainstream notions in psychiatry that all depression, even low mood, is an illness, to the view that MDD is a normal, functional reaction (Nesse, 2000). It might be unreasonable to expect that one theory should account for the existence of all types of melancholic states, from mild to severe, in addition to all the fitness-damaging traits described above. The complexity suggests that depression is unlikely to be one single phenomenon. Nevertheless, the existence of depression is still a scientific mystery, which is not yet understood under a coherent whole (Hagen, 2003). And more empirical work on the existing theories is needed before we can tell.

The purpose of this article

The present purpose is to discuss predictions from all evolutionary hypotheses on why depression exists and why it differs between the sexes. The predictions are numbered and collected in the appendix. Most of those predictions are discussed in the text, and are referred to by their number. The predictions are either directly derived from the relevant articles, while others are new for this article, made with the logic of the theories in mind. This article starts by presenting relevant principles from evolutionary psychology, evolutionary medicine and evolutionary psychopathology. Then we move on to discuss predictions from the adaptive theories, and the theories on the sex difference in depression. This is followed by a

discussion of the side-effect hypotheses of depression, and finally the dysfunction views.

The term “depressives” is often used in this article, this term is solely for the sake of simplicity. Its meaning is an individual who currently, or has in the past, experienced some of the symptoms of depression as defined by the ICD or DSM diagnostic systems. It does not mean that depressed individuals are always experiencing depression, or that depression is a category of humans. Another term I use is “adaptive”. In this article, “adaptive” always refer to evolutionary adaptations. Not, as it is sometimes used in psychological literature, to describe individuals who favorably adjust to some unfortunate situation.

Evolutionary Psychology

Is it not reasonable to anticipate that our understanding of the human mind would be aided greatly by knowing the purpose for which it was designed? (Williams, 1966/1996, p. 16).

Evolutionary psychology (EP) is the attempt to understand the architecture of the mind by understanding the evolutionary processes that shaped it (Pinker, 2009). EP is the marriage of cognitive science and modern evolutionary biology: The brain is a complex information-processor and it has been shaped by natural selection (Cosmides & Tooby, 2013). In EP, complex human behavior is viewed as the functional output from psychological mechanisms (also known as Darwinian algorithms, modules, psychological adaptations) that evolved in our ancestors. These psychological mechanisms are, in turn, dependent on internal and environmental inputs for their development, expression and activation (Buss, 1995a; Confer et al., 2010; Tooby & Cosmides, 2005). Evolutionary psychology, then, is the “missing link” (Cosmides & Tooby, 1987) between evolution and behavior: The functional psychological mechanisms that generates behavior based on input from the internal or external environment.

Darwin (1859/1998) ingeniously observed that organisms *vary* in their abilities to

survive and reproduce and that "there is a struggle for existence leading to the *preservation* [italics added] of each profitable deviation of structure or instinct" (Darwin, 1998, p. 346). Hence, the occasional useful change in an organism's genetic code that came up during the deep time of evolution, or "good trick" as Dennett (1995) has called it, which made an organisms slightly more effective in replicating their genes (see Dawkins, 1986/2008) will, by definition, eventually spread themselves throughout a population. This means that the genes responsible for these fitness-enhancing traits (the good tricks) reach *fixation*. Eventually these traits—adaptations—are all "preserved" in what we would recognize as the nature of a species. In the observable universe, this process is the only known causal mechanism behind complex biological functionality, or the apparent "design" of life (Buss, 1995a; Dawkins, 1986/2008; Kennair, 2002).

Proximate and ultimate causation. Darwin's insights, then, allows us to ask penetrating *how* and *why* questions on the traits we observe in biological organisms (e.g. flight, echolocation, altruism, mating rituals). "How questions" investigate what biologists and evolutionary psychologists call *proximate* factors, this is the immediate causes—how does this trait work and develop? "Why questions", on the other hand, investigate what is called *ultimate* factors (sometimes called distal or simply evolutionary)—why does this trait exist at all? (Confer et al., 2010).

Nikolaas Tinbergen (1963) famously formalized proximate and ultimate causation into two proximate, and two ultimate questions. Nesse (1990), in turn, has exemplified Tinbergen's questions with emotions: 1) What is the physiological, psychological, and behavioral mechanisms of the emotion? Which cues elicits the emotion?; 2) what is the ontogeny of the emotion (how does it develop over a lifetime)?; 3) what was the function of the emotional capacity that increased fitness over evolutionary time? In what situations (such as threats, opportunities) is it adaptive? What is the adaptive significance of the changes in

physiology, cognition and behavior?; and 4) what is the phylogeny of the emotional capacity? That is, the interspecial evolutionary history of the emotion. Question one and two is proximate (how) questions, while three and four is ultimate (why) questions. For a full understanding of a complex biological trait, all these four questions must be answered. And, importantly, proximate and ultimate explanations are complimentary; they never compete with each other. Traditional psychological and neuroscientific perspectives often produce sophisticated understandings of proximate mechanisms, but they lack half of the story. A description of only the proximate mechanisms that were involved in behavior (such as neurotransmitters, activation of an emotion, peer-pressure and so on—the immediate factors) is insufficient. Hence, it has been argued by many that a mature psychological science must include the ultimate level of explanation (Barkow, Cosmides, & Tooby, 1992; Buss, 1995a, 2005; Confer et al., 2010; Haselton & Nettle, 2006; Kennair, 2002).

David Marr, an advocate of the computational theory of the mind, emphasized that "an algorithm is likely to be understood more readily by understanding the nature of the problem being solved than by examining the mechanism (and the hardware) in which it is embodied" (1982/2010, p. 27). Hence, answers to ultimate questions is not explanatory ornamentation on existing psychological theories, nor is it the evolutionary perspective a subfield of psychology. It is a fundamentally integrating, non-reductionist, true biopsychosocial *perspective*; a meta-theory, which can generate hypotheses and bind together anything in psychology in a theoretically coherent framework (Buss, 1995a; Kennair, 2002), just as it has in the other life-sciences for over 150 years (Tooby & Cosmides, 1992). Clinical psychology is no exception to this. Brüne (2008) argued that it is a common misconception in psychiatry that to understand early development and the neurochemistry of the brain is sufficient to understand the pathology of cognitive, emotional and behavioral systems. That provides only the answers to ontogeny and physiology. It is essential to add the

complementary ultimate level of analysis (Brüne, 2008). It is not untrue to say that men are, on average, taller than women because men have taller leg-bones. However, we also have to ask why this is the case. Similarly, it might also be correct to say that depression is associated, for example, with reduced monoamine activity, or the experience of loss, but "why" is that the case?

Multimodularity. A good pump requires some amount of mechanical force, a good oxygenator requires a large surface area in which to exchange gases. That is why the heart (a muscle) pumps blood, and the lungs oxygenate the blood: neither can effectively do both. They solve different evolutionary problems. The body, then, can be described as *mutimodular*: Evolutionary problems are not solved effectively with one tool (Cosmides & Tooby, 1994b; Nesse & Williams, 1996). This is also true for the human brain. It can be seen as many organs (Kurzban, 2012), which evolved, like the heart, liver, immune systems and lungs, to solve specific adaptive problems during human evolutionary history (Nesse & Williams, 1996; Williams & Nesse, 1991), that is, during the Environment of Evolutionary Adaptedness (EEA). The visual system of humans, for example, process contours, texture, brightness, and so on differently (Marr, 1982/2010)—specific solutions to specific problems. How many, and how specific, the minds modules are, is still an open question which is debated and empirically explored within cognitive science and evolutionary psychology. For example, are all modules highly domain-specific? Or are some domain-general, like fluid intelligence? (See Barrett & Kurzban, 2006, for a review of the debate). However, due to the flexibility and complexity of human behavior, we can be reasonably confident that natural selection armed the human brain with more than just a few domain-general psychological mechanisms (e.g. operant conditioning). If we want an information-processing system, for example a computer, or a smartphone, to be flexible and complex—to solve many problems, it has to contain, maybe somewhat counterintuitively, many programs (or "instincts",

"algorithms", "mechanisms", or "modules") in their software (Barrett, 2012; Kurzban, 2012). A logic that William James (James, 1890/2011, p. 236) early pointed out: "a creature which has few instinctive impulses, or interests, practical or æsthetic, will dissociate few characters, and will, at best, have limited reasoning powers; whilst one whose interests are very varied will reason much better". The behavior that is produced by the human nervous system is obviously complicated and flexible, which requires a certain richness of psychological mechanisms; humans have a *nature* (Kurzban, 2012; Pinker, 2003). Is depression an evolved module, or modules?

Evolutionary psychology as a prediction-generating tool. Like any good scientific theory, evolution is an excellent generator of testable hypotheses. Evolutionary theories are hierarchically organized and contain several levels of explanation (Simpson & Campbell, 2005). Evolution by selection is the overarching theory from which "middle-level" evolutionary theories such as reciprocal altruism (Trivers, 1971), parental investment (Trivers, 1972), parent-offspring conflict (Trivers, 1974) are formulated. This, in turn, leads to specific predictions. For example, from Parental-investment theory we can hypothesize that if males sometimes contribute resources to offspring, females will select mates based on their willingness to contribute resources to their offspring; women will be more "choosy" when picking mates (Buss, 1995a). This hypothesis, in turn, can lead us to three specific predictions (Buss, 1995a): 1) Women have evolved preferences for high-status men, 2) Women have evolved preferences for men who portray cues of willingness to invest resources in offspring, and 3) Women will divorce men who fail to deliver the expected resources, or who diverts them to other women or their children (Buss, 1995a). Indeed, Buss (1989) found, amongst other things, that, in the 37 cultures that he studied, males prefer young and physically attractive (fertile) mates; and women prefer cues to resource acquisition (such as ambition and industriousness) (see also Grøntvedt & Kennair, 2013). With the use of

evolutionary logic from middle-level theories, EP has grown to be a mature, empirical science (Buss, 2009). An example of a well-established empirical finding is in human reasoning about social exchanges. It has been empirically supported that humans have cognitive (Cosmides, 1989; Cosmides & Tooby, 1992) and neural adaptations (Cosmides & Tooby, 2005; Stone, Cosmides, Tooby, Kroll, & Knight, 2002) for content-specific detection and punishment (Delton, Cosmides, Guemo, Robertson, & Tooby, 2012) of cheaters in social exchanges across cultures (Sugiyama, Tooby, & Cosmides, 2002). Another example is a psychological mechanism for kin-detection and incest-avoidance (Debra Lieberman, Tooby, & Cosmides, 2003, 2007). There are also several evolutionary psychological hypotheses which have been refuted (Buss, 2008), for example the hypothesis that male homosexuality was maintained through kin selection (inclusive fitness) has now been falsified (Bobrow & Bailey, 2001; Rahman & Hull, 2005). When data do not support the predictions from an evolutionary middle-level hypothesis, like in the rest of science, we must return to the drawing board to attempt a better translation of the middle-level hypothesis, or reject it altogether (Ketelaar & Ellis, 2000).

What is an adaptation?

"Evolutionary adaptation is a special and onerous concept that should not be used unnecessarily, and an effect should not be called a function unless it is clearly produced by design and not by chance" (Williams, 1966/1996, p. V).

Adaptations can be defined this way: "Adaptations are mechanisms or systems of properties "designed" by natural selection to solve the specific problems posed by the regularities of the physical, chemical, ecological, informational, and social environments encountered by the ancestors of a species during the course of its evolution" (Tooby & Cosmides, 1990, p. 383). Now, Williams quote above illustrate an important point on adaptations: They are, by definition, overwhelmingly unlikely to be the product of chance. A

human eye, for example, is clearly adapted for detecting the light that reflects off objects in the world. The likelihood for such a complex organ too arise by chance, is practically zero (Dawkins, 1986/2008). Adaptations must show *evidence of design*. However, this does not mean that everything about an organism, even the ones that look useful, is an adaptation. It can also be side-effects, noise or malfunctions. However, many psychological traits show evidence of design. For example, humans do not choose partners to cooperate with, or to mate with, at random: It is particular traits that are desired, suggesting that it is the functional output from some information-processing unit housed in the brain—chance cannot explain it. If we suspect that there has been some adaptive problem in our evolutionary past which has produced some particular, complex designs in the brain—an evolutionary hypothesis—we can test whether modern humans indeed are capable of efficiently solving the problem(s) that our hypothesis predicts. As Randy Thornhill (1997, p. 4) points out "If selection created a trait, that trait will be functionally designed for the ecological problem generating the selection - the selective force - that created it."

Hence, we can reverse-engineer the trait that we hypothesize to have evolved. If a trait is efficient and economical, specialized and reliably develops in all members of a species (Thornhill, 1997; Williams, 1966/1996), it is likely an adaptation. If depression (or indeed any other trait) is an adaptation, then, we would expect the adaptation to portray four hallmarks (Nettle, 2004): 1) *lack heritable variation*. A disclaimer is appropriate here. Genes produce proteins, which in turn produce design in information-processing neural structures (psychological mechanism); all psychological design is therefore genetic design (Kennair, 2002). However, because adaptations quickly spread and become species-typical (everyone have the adaptive gene variants), adaptations should lack "heritability" as traditionally defined by behavioral geneticists. There are no phenotypic *differences* attributable to *differences* in gene-variants (alleles, polymorphisms) therefore the adaptation has "0"

heritability. The only exceptions to this is when adaptive genetic variance is maintained by balancing selection, for example negative frequency-dependent selection, or the adaptation is still in the process of reaching fixation; 2) As mentioned, an adaptation should show *evidence of good design,* such as signs of specialization, efficiency and so on; 3) be *evoked by appropriate triggers* (reliably elicited by the circumstances it evolved to solve); and 4) *fitness is reduced when the trait is lacking*. An example of this point is incongenital sensitivity to pain—a rare condition where individuals are born with an inability to experience physical pain (see Pinsky & DiGeorge, 1966; Rosemberg, Nagahashi Marie, & Kliemann, 1994), which is likely caused by mutations (Indo et al., 1996; Mardy et al., 1999), sufferers of this condition often have unusual self-inflicted damage to their bodies (Protheroe, 1991). Indeed, Nesse (1999) has suggested that one way evolutionary hypotheses on the function of low mood can be tested (and this principle surely applies to the adaptationist hypotheses on major depression too) is too "find people who lack a capacity for low mood and identify the disadvantages they experience" (p. 262).

Evolutionary medicine and evolutionary psychopathology

Medicine benefits hugely from physiology and biochemistry, which studies how organs work and what they normally do. However, most medical research has studied proximate factors only (Williams & Nesse, 1991). This may be because disease is necessarily understood as abnormal, and to study them from an evolutionary perspective might seem preposterous (Nesse & Williams, 1996). However, the ultimate perspective in medicine does not study the evolution of the disease itself, but the design characteristics that make humans vulnerable to disease (Nesse & Williams, 1996). Natural selection, importantly, does not care about our happiness, it promotes health only when it is in the "interest" of our genes (Kurzban, 2012; Nesse & Williams, 1996). If tendencies for anxiety and physical pain (or, for that matter, heart disease and skin cancer) are associated with reproductive success it will be

selected for; if it is detrimental to reproductive success, it will strongly be selected against.

An evolutionary perspective is necessary in order to not confuse defenses with disease states. For example, host defenses, such as cough and fever, are evolved defenses against disease-causing pathogens (Kluger, Kozak, Conn, Leon, & Soszynski, 1998; Nesse & Williams, 1996). The use of drugs that reduces the functioning of these adaptations (for example fever-reducing medicine during infections) can, therefore, be dangerous (Nesse & Williams, 1996). And, of course, the *dysfunctioning* of evolved defenses (inappropriately high fever for instance) can also be dangerous. A fine-tuned understanding of evolved function and dysfunction will therefore greatly inform clinical judgment when patients have fever. The task of explaining why humans are vulnerable to mental disorders, is not different, conceptually, from explaining why we are vulnerable to physical diseases (Nesse, 2009). The field of understanding mental disorder from an evolutionary perspective, has been called evolutionary psychopathology (Baron-Cohen, 1997a; Gilbert, 1998; Kennair, 2003).

The complexities of brain mechanisms in its normal operation, is often hidden from sight (Tooby & Cosmides, 1992), leading to an "instinct blindness" (Cosmides & Tooby, 1994a). For example children, given some environmental input, automatically acquire incredibly complex languages and speak them effortlessly (Pinker, 2003). The rare cascading failures of such biological complexity are often illuminating (Csete & Doyle, 2002), the failures of psychological mechanisms often reveal complexities of the mind that might else be taken for granted (e.g. autism Baron-Cohen, 1997b).

Most clinicians would agree that sadness and grief can be normal responses, still much of the scientific literature assumes depression is an illness. And it is hard to blame anybody for it. As Nesse (2008, p. 525) put it:

> How can it possibly be helpful to feel hopeless, worthless, and lacking all motivation? In general, it is not. Much depression is a disease. However, depression is not a

disease like diabetes or cancer, it is more like chronic pain, a dysregulation of a response that can be useful in some situations.

If this is true, what mechanisms have become dysregulated? Some instances of depression are undoubtedly disorders, and extremely unlikely to be an adaptation. For example a chronic major depression that seems to be unrelated to social and environmental circumstances (Horwitz & Wakefield, 2007). Where do we draw the line? Jerome Wakefield has attempted to answer this question.

Harmful dysfunction. Natural selection shape mechanisms that work, so how can it help understand why the mind fails (Nesse, 2005)? To understand mental disorders, Jerome Wakefield (1992, 1999, 2005) has argued, it is necessary to understand dysfunction in light of evolved, normal biological function. The modules of the mind can fail, and this is sometimes harmful (Cosmides & Tooby, 1999)—if both of those criteria are meet, a condition would meet Wakefield's criteria of a Harmful Dysfunction (HD), and hence can safely be called a "disease" (Wakefield, 1992, 1999, 2005, 2007). The first criteria, biological dysfunction, is simply mechanisms that do not properly perform its evolved function. To define a mental disorder, Wakefield argues, we also need to understand whether the condition is *harmful*, that is, a value judgment regarding the usefulness of the condition (or the suffering it maintains in individuals). Many dysfunctions can be harmless, or even desirable (especially in modern environments), and hence not a disorder. And, many traits can be functional but still harmful (e.g. jealousy), and hence not a disorder, in the HD sense (but might still prove to be a "treatable condition") (Cosmides & Tooby, 1999).

Judgments regarding psychological normality and disorder, even before Darwin, was, in fact, judgments of evolutionary design. If the heart fails to pump blood, everybody agrees that it is dysfunctioning. Clinical psychology is not generally armed with these understandings of normally functioning cognitive architecture, which makes diagnostic

systems arbitrary (Nesse & Jackson, 2011; Nesse & Stein, 2012). Scientific progress, then, in our understanding of etiology, diagnosis and treatment of mental disorders may largely depend on progress in evolutionary psychology (Wakefield, 2005): The normal, cognitive architecture (functions) of the human mind must be understood before dysfunction can be properly understood.

At this point, that is also the problem with the harmful dysfunction analysis: It depends on future mental health research, which will provide an evolutionary taxonomy on functions (Kennair, 2010). However, tests of evolutionary predictions on normal sadness will greatly inform our understanding of their dysfunctions (severe mood disorders).

Mismatches. One important aspect from evolutionary medicine is the effect of *mismatches.* This happens when the environment of evolutionary adaptedness (EEA)—the environment that provided the selection pressures for the adaptations in a species—is significantly different than the current environment. The EEA concept is very useful in evolutionary medicine in order to understand why there are so many "diseases of civilization", like coronary heart disease, diabetes, obesity, asthma, allergies, and so on. One dramatic example is diet (see Cordain et al., 2000; Eaton, Eaton, Konner, & Shostak, 1996). The human digestive system evolved (like most human adaptations), in a hunter-gatherer context in the pleistocene. Human diets have diverged radically after the first agricultural revolution (~10000 years ago) and industrial revolution (~250 years ago), which have led to a mismatch between the foods the human digestive system evolved to process, and many current foods, creating an array of human health problems (Cordain et al., 2005; Durant, 2013; Daniel Lieberman, 2013). For example, the high availability of vegetable (seed) oils rich in omega-6 polyunsaturated fatty acids (which has been cheap and easy to produce after the industrial revolution), leads to an abnormally high—for humans—omega6/omega3 ratio, which are involved in the pathogenesis of cardiovascular disease, cancer and inflammatory

and autoimmune diseases (Simopoulos, 2002).

Some argue that depression, too, is a disease of modernity (Hidaka, 2012), caused by evolutionary mismatches, in not only diet—such as modern omega3 deficiencies (see Chilton et al., 2011; Lin & Su, 2007)—but many other arenas such as sleep and sunlight deficiency, sedentary lifestyles, loneliness and social inequality (for a discussion on these factors, see Hidaka, 2012). If the mismatch hypothesis of depression is true, we would expect increases in depression as societies are industrialized, (or "westernized", "modernized"), There is evidence that depression is recently increasing in incidence (Compton, Conway, Stinson, & Grant, 2006), but this issue is unsettled, overdiagnosis and other factors might contribute to this trend (Horwitz & Wakefield, 2007). Ilardi (2009) has developed a treatment, based on evolutionary mismatch ideas, which attempts to change life-style factors (healthy diet, sufficient sleep, social activities and so on) in depressives. This approach might prove promising.

However, mismatch need no imply that *disorders* have increased, it can also mean that normally functioning adaptations are activated to a higher degree. So if we propose that depression is caused, for example, by social alienation in modern societies, it could simply explain the upsurge of normally functioning sadness-mechanisms, not an upsurge of brain disorders (Horwitz & Wakefield, 2007). Although the mismatch hypothesis is highly relevant for understanding proximate causes, and treatments for depression—for example, if it is true, we would expect that hunter-gatherers (or people with an EEA-similar diet and activity level) rarely experience MDD—it is relatively agnostic on ultimate function: It reveals, and predict nothing on *why* sadness and/or depression exist in the first place.

Adaptive Theories

Social Competition Hypothesis and Rank Theory

The social competition hypothesis (also called rank theory), first postulated by John

Price (1967) and later by several authors (Gilbert, 1992; Price, Sloman, Gardner, Gilbert, & Rohde, 1994; Sloman, 2000) postulate that depression is an unconscious and involuntary subordinate strategy in social competition. Depression, it is argued, is an adaptation that is activated whenever one is *losing* rank. Price developed this hypothesis because he noticed similarities between depressed patients and animals who lose in hierarchical encounters (Sloman, 2000). Indeed, Price (1967) argues that hierarchical struggles in groups requires certain behavior patterns from their members: Irritability towards inferiors, anxiety towards superiors (see prediction 1.1), elation on going up hierarchy and depression on going down (prediction 1.2). Expanding on this, Price et al (1994) hypothesized that mammalian depression executes an evolutionary function by activating specific psycho-biological response patterns that mediate the behavioral variation observed after losing or winning at social competition. In Gilbert's (2000, p. 149) words: "The potential for involuntary, subordinate self-appraisal and its co-assembled affects and behaviors, evolved (perhaps) precisely to inhibit an animal from challenging for breeding resources in situations that it could not win". The social competition hypothesis, then, predicts that depression is a ritual losing behavior which produces psychological incapacity (low self-esteem) that signals submission to the winner, but preserves the loser without physical damage.

Many social animals have evolved complex hierarchical rank systems (Sapolsky, 2005; Watts, 2002). Indeed, the mammalian selection pressures that gave rise to the "three peaks" of extremely large brains, namely toothed wales, humans and elephants, were mutual dependency based on external threats from predators and conspecific (same-species) groups (Connor, 2007). Social dependency and complexity produce a large neocortex (Dunbar, 1993, 1998, 2007). Rank-sensitive neural circuitry's in social mammals (chimpanzees, macaques, baboons, lions, dolphins, birds and many others) estimate relative rank amongst their group, and responds accordingly (e.g. Raleigh, McGuire, Brammer, Pollack, & Yuwiler, 1991). It is

likely, therefore, that the human brain is equipped with phylogenetically ancient rank-mechanisms (see Barrett, 2012), maybe dating back to the common ancestors of humans and reptiles 250 million years ago (Price, 2000). For humans in particular, we have both cooperated (Axelrod, 2006; Cosmides & Tooby, 2005) and predated each other (Tooby & Cosmides, 1988), which is likely to have provided important selection pressures for the evolution of particularly sensitive rank-systems. Successful orientation in the complex rank-relations of human cultures are no easy task, particularly when descending the hierarchical ladder, which often entails aggression from superiors (Price & Sloman, 1987; Sloman & Gilbert, 2000).

In the conflicts that arise when animals compete for important resources such as food, territory and sexual opportunity, organisms face the choice of either employing escalating strategies, that is, trying harder and use more aggressive tactics, or de-escalate, meaning going for damage limitation, retreating or giving up (Gilbert, 1992; Sloman, 2000). These strategies are called *hawk and dove strategies*, respectively. A dove strategy enables an organism to avoid getting into, or prolonging, a struggle one will lose. Whereas a hawk strategy facilitates better access to resources. Employing the right strategies can be complex, and have far-reaching consequences (Sloman, 2000). In social competition, an animal's strategy is based on the *Resource-holding potential* (RHP), this variable holds, and expresses, an animal's knowledge of its own fighting capacity, based on size, strength, skill, previous success, weapons and allies. Amongst humans, the SCH views the RHP as the equivalent of self-esteem. Thus, the RHP's relative value allows an organism to either execute an escalating (hawk) or de-escalating (dove) strategy in agonistic (fighting) encounters. When the RHP is low, a contestant is more likely to yield (flee or submit) rather than to attack (Price et al, 1994).

Maynard Smith's (1982) evolutionary game theories shed light on the evolution of

dove and hawk strategies. These "games" mimic the evolutionary process by having computer-simulated strategies repeatedly compete against each other. Over time, the differential effectiveness of the strategies are reflected in their prevalence in the population of competing strategies. That is, their 'fitness'. Maynard Smith and Price (1973) showed that pure hawk strategies were not "evolutionary stable", a pure hawk strategy can only be effective in low numbers (negative frequency-dependent selection), mixed hawk/dove strategies, however, infiltrates such populations and stabilizes.

Smith's theoretical games suggest that it is not unlikely, in principle, that natural selection could favor "limited war" tactics, rather than, tactics of uninhibited aggression. Depression might be an evolved dove strategy meant for losses in agonistic encounters. However, this has not been tested empirically on humans. It is not clear either, in clinical terms, if it can account for both mild, and more severe depressive states. Even so, if depression is a "limited-war" tactic, is it a strategy that is activated in all humans under the right circumstances, or is it an evolved strategy that is maintained in only some humans?

Depression, then, from the SCH perspective, is viewed as the human version of a dove strategy, or an *Involuntary Subordinate Strategy* (ISS). The ISS is hypothesized by Price (1994) to have three main functions. Firstly, it holds an executive function by inhibiting aggressive behavior from rivals and superiors (but not towards dependents) (prediction 1.4b and 1.1) and, in addition, creating a subjective sense of incapacity. Secondly, Price and his colleges (1994, p. 309) writes that the ISS holds a "communicative function that signals 'no threat' to rivals and 'out of action' to any kin or supporters who might wish to push the individual back into the arena to fight on their behalf" (prediction 1.4a), a "go-down stay put" program (Gilbert, 1992). The final function, Price and his colleges argue (1994), is facilitative, meaning that it encourages acceptance, and yielding behavior that signals acceptance of the outcome of competitive struggles ("I lost—I am harmless"). This final

stage leads to reconciliation and the termination of the conflict that triggered the ISS. If the involuntary yielding behavior (the depression) is "blocked" in a particular situation, the ISS may become intense and prolonged and thus be recognized as depressive disorder (Gilbert, 2000; Gilbert & Allan, 1998) (see prediction 1.4a-d). This last point is a common view in the later writings from SCH theoreticians (e.g. Gilbert, 1998, 2000; Gilbert & Allan, 1998; Sloman, Farvolden, Gilbert, & Price, 2006). They suggest more of a "dysregulation" view: Major depression is a dysregulated form of adaptive "escape" behaviors from conflict. If the conflict is not resolved, it turns into a disorder. As Sloman, Farvolden, Gilbert and Price (2006, p. 97) argue: "when there is a failure of reconciliation, or when flight is not possible, the mechanisms associated with low positive affect or high negative affect may go into overdrive and become maladaptive by operating at a greater intensity and/or over a prolonged period of time."

Nevertheless, it is somewhat unclear whether the SCH attempts to explain the continuity from low mood to reactive major depressive disorders. Hence, tests of of the SCH's on both normal "low-mood", as well as clinical populations will illuminate this. For example, is low-mood an effective communicator of "I am out of action", or "I yield", while, major depression—which is known to cause interpersonal problems (see Segrin & Abramson, 1994)—is not?

Can loss of social status explain human depression?

The RHP concept in the SCH involves social comparison processes. It is well-known that humans compare themselves with other members of their group (e.g. Festinger, 1954). If an individual perceive its RHP (self-esteem) as high relative to others, either due to cues from the environment or manipulation by drugs, that person might challenge and defend resources (act as a high-status individual, and hence *not* be depressed). The SCH, thus, might be able to explain why social inequality (see Pickett & Wilkinson, 2010) and low socioeconomic status

is related to depression (see Everson, Maty, Lynch, & Kaplan, 2002; Lorant et al., 2003): Perceptions of one's own subordination in society maintains the depression. However, if the SCH is true, it would mean that inequality *cause* depression in anyone, not that depression cause low income. Attempts to entangle the causal directions of this association will probably prove revealing.

Because many of the writers in the SCH-camp propose that subordination strategies may be phylogenetically ancient, ethological and cross-species (comparative) research is appropriate. This might shed valuable light on the evolutionary origins of sad mood and depression. However, this focus might also be its weakness: Have human rank-mechanisms and depressive behaviors remained unchanged since its phylogenetic origin? The SCH does not account for the social complexities of depression: Why, for instance, would losses of loved ones induce depression? Does that involve rank-loss? One possible answer is that social losses have, for humans and other primates, entailed the loss of an ally (Hagen, 2011). But, it is unclear why depression would, evolutionary speaking, "help" in this scenario. Another weakness, both functionally and dysfunctionally, is suicide. As Hagen (2011, p. 718) asked "why would submission to dominants, which protects a person from harm, be associated with killing oneself?". There are other things which are left unclear by the SCH: How do humans calculate their social roles in groups? Which proximate factors are predicted to activate depressive states in humans (marriages, siblings, parents, work, societal factors)? If the SCH is true, depression is indeed the best strategy when descending the hierarchical ladder: Depression works, It actually inhibits aggression from high-ranking humans, at least in EEA-similar environments (prediction 1.7). And conversely, if the SCH is true, manic, high-self esteem strategies, (or neutral mood) is not. This must be explored empirically.

Behavioral Shutdown Mechanism, Disengagement from Incentives and Resource-conservation

Henriques has postulated an adaptationist hypothesis called "behavioral shutdown mechanism" (BSM). Meaning that an individual should decrease its "behavioral expenditure in response to chronic danger, stress or consistent failure to achieve one's goals" (Henriques, 2000). This is similar to Klinger's (1975) analysis where depression is viewed as "disengagement from incentives"—the ultimate function of depression is to disengage from unobtainable goals in order to conserve resources. Nesse (2000) has expressed similar ideas. These models share aspects with the Social Competition Hypothesis, and they are included in the Social Risk Hypothesis (discussed in detail later). However, the disengagement and BSM views predict, uniquely, that depression should be activated when an individual is unable to disengage from an unreachable goal (prediction 2.1). And that depression should be frequent in people who are "anxious, duty-bound, ambitious or lacking alternatives because such individuals are especially likely to get themselves into situations in which they are unable to give up a major goal" (Nesse, 2000, p. 18) (prediction 2.2).

The Third Ventricle Hypothesis

Colin Hendrie and Alasdair Pickles (Hendrie & Pickles, 2010) argue that depression is an adaptation that ensures group membership, and reduces aggression, when social status is lowered. Hence, they posit the same ultimate functions as the SCH. However, they note that many brain mechanisms involved in producing the behavioral cluster of depression are bordering the third ventricle in the brain. For example, the pineal is involved in sleep/wake cycles; the hypothalamus in food and sex behaviors; and the amygdala in social affiliation and fear and defensive behaviors, whose main output, the stria terminalis, they note, pass through the third ventricle (Hendrie & Pickles, 2010). Based on these observations, Hendrie and Pickels (2010) postulate a proximate mechanism: Depression is produced by the release

of a toxin or toxins (e.g. cytokines) into the third ventricle, or, alternatively, by the inhibition of suppresion of toxins already present in the cerebrospinal fluid (prediction 3.2). They also predict that the toxin is likely to damage many cells (particularly glia cells, because they constitute 90% of the human brain) bordering the third ventricle that are *unrelated* to depressive behavior (prediction 3.2).

The Third Ventricle Hypothesis can potentially explain findings in postmortem studies of depressives, such as the volume-reductions, in the mammilary bodies and fornix (Bernstein et al., 2012) and in the external pallidum in major depressives (Bielau et al., 2005). It can also explain the finding that some major depressives had reduced sizes of the amygdala in MRI scans (Sheline, Gado, & Price, 1998). However, both structural and blood-flow abnormalities have been observed in the frontal lobes of depressives (Drevets, 2000; Soares & Mann, 1997), which is left unexplained by the Hendrie and Pickels.

Analytical Rumination Hypothesis

Analytical Rumination Hypothesis (ARH) postulates that depression is an adaptation that is designed to solve complex fitness-relevant problems and maintain attention on those problems. This idea was originally included in Watson and Andrews (Watson & Andrews, 2002) Social Navigation Hypothesis (which is a combination of the ARH and Bargaining theory). Here, I will discuss the ARH as later described by Andrews and Thomson (2009). They structure the postulations of the ARH by four main claims: 1) complex problems trigger depressed affect, 2) depression coordinates changes in body systems that promotes sustained analysis of the triggering problem, 3) depressive rumination helps people solve the triggering problem, and 4) depression reduces performance on laboratory tasks because depressive rumination takes up limited processing resources.

First claim: Complex problems trigger depressed affect. Because the ARH postulates that depressive rumination evolved to effectively solve problems, Andrews and

Thomson (2009) expect that the treatment of depressive *symptoms*, rather than the triggering problem, should be ineffective in the long-term (see prediction 4.1a and b). Indeed, evidence suggests that antidepressant medications often fail to prevent relapse once the treatment stops, especially if the medication is discontinued before 6 months (Hollon, Thase, & Markowitz, 2002). Indeed, Andrews and colleges (2012) have argued elsewhere that antidepressants do more harm than good because antidepressants have adverse effects on bodily systems which are regulated by serotonin. According to the ARH, problem solving therapies like Interpersonal Therapy (IPT) and Cognitive-Behavioral Therapy (CBT), should be more effective relapse preventer's, because patients learn to solve interpersonal problems (which Andrews and Thomson suggests depression evolved to do). Tentatively, as predicted by the AR hypothesis, CBT and IPT do indeed have lower relapse-rates compared to antidepressants (Hollon et al., 2002). The ARH generates more testable predictions on treatment: Psychotherapies focusing on identification and solutions of social problems in the depressives life, should be more effective, than depression-therapies that do not (prediction 4.2a, b and 4.3). Removing the depression symptoms without solving the underlying problem, Andrew and Thomson (2009) argue, would be like "curing" infection with aspirin: It removes the fever, but not the infectious agents (see Nesse & Williams, 1996). Andrew and Thomson (2009) therefore predict that a therapeutic focus on changing cognitions are ineffective (prediction 4.1a & 4.2a). They support this claim by pointing our attention to a study by Castonguay and colleges (1996). Here a positive correlation was found between depressive symptoms after treatment and CBT therapists focus on the link between distorted thoughts and negative emotions. Andrews and Thomson (2009) raise the possibility that this correlation exists "because patients could perceive CBT as dismissive of their real troubles" (2009, p. 626). Thus, Andrews and Thomson (2009) suggests that the behavioral activation component is more effective, compared to the automatic thoughts component, in CBT.

Indeed, they point to one study which found that chronic, severely depressed patients responded better to behavioral-activation therapy, than CBT (Coffman, Martell, Dimidjian, Gallop, & Hollon, 2007). From an ARH perspective this finding makes sense because behavioral-activation might facilitate real-life problem-solving rather than alteration of, or distraction from, rumination. However, it is not known whether severely depressed patients automatically engage in active problem-solving when they are encouraged to do behavioral activation. Clearly, behavior activation is not problem-solving. Furthermore, Wells (2009) found that, using Metacognitive therapy—which enables patients to actively *interrupt rumination*—was effective in treating four patients with chronic, major depression after 6 month follow-up. Although the sample was small, this result opposes the idea that encouragement of the depressive's rumination is necessary to terminate the depression.

Interpersonal therapy focuses on strategies for solving social problems, and Andrews and Thomson (2009) mention one study that suggest that IPT, like Behavioral activation, might be more effective than CBT (see Cuijpers, van Straten, Andersson, & van Oppen, 2008). Further, Andrews and Thomson (2009) claim that the reason why therapies such as CBT and IPT are effective treatments of depression is because they have a common feature of *solving the problems* which depressives face. Problem-solving therapy (PST) is an effective treatment of depression and suicide potential (Eskin, Ertekin, & Demir, 2008). A meta-analysis concluded that PST was significantly more effective than "treatment as usual", and attention placebos, but not more effective compared to other *bona fide* treatments presented specifically in other studies (Malouff, Thorsteinsson, & Schutte, 2007). Watson (2008) hypothesize that the reason why PST treatments are not remarkably more effective than other treatments is because standard PST focuses on general, rational problem-solving skills: "Standard PST does not offer a concentrated effort to identify specific social barriers to major fitness-enhancing revisions of the patients socioeconomic niche, that is, the socially

cause fitness-hindrances that the SNH proposes to be the main context for adaptive major depression". A new problem-solving therapy, based on the SNH, then, should be more effective in treating depression. Wampold and his colleges (1997), in a meta-analysis, found that cognitive therapy was more effective than non-bona fide treatments (therapies not based on principles in psychological science, nor given by a professional), but not more effective than other bona fide treatments. This is contrary to what is expected from the ARH because cognitive therapy, which contain at least some problem-solving ingredients, are expected to be more efficient than therapies that do not. Nevertheless, the ARH/SNH provides a rich source of predictions on clinical effectiveness which are in need of testing (see predictions 4.1, 4.2 & 4.3).

To elaborate on the assertion that "complex problems trigger depressed affect", Andrews and Thomson (2009) point to the *content* of depressed people's thoughts. Depressives often report that they face severe complex problems that are difficult to solve. Further, depressives report little confidence in solving those problems, and, also, that they focus more than before on their problems (Andrews & Thomson, 2009; Lyubomirsky, Tucker, Caldwell, & Berg, 1999). Indeed, the *content* of thoughts; the inner melancholic voices, *what* depressives ruminate on, is an important, interesting, even radical aspects of the ARH: Depressive thoughts are not pathological, irrelevant side-effects of a disease of the mind, they are a cognitive call-to-arms for finding solutions to fitness-relevant social problems. See prediction 4.4. It should be noticed, however, that Lyubomirsky et al (1999) found that ruminating depressives were less willing to solve their problems, both personal and hypothetical. This appears in contrast to what we would expect if the ARH was true. Lyubomirsky (1999) raise the possibility that depressives are attempting to solve their personal problems, which can explain the poor hypothetical problem-solving. However, rumination has also been described as a vicious-cycle which prolongs, even worsens the

depression, and that people who ruminate when they experience depression, might get worse than people who distract themselves (e.g. seeNolen-Hoeksema, 1991). This seems to contrast with the ARH. It might also contradict with the ARH that several treatments for depression, like cognitive therapies, especially metacognitive therapy, which terminates rumination, or teaches the uselessness of it, can be effective. However, Watson (personal communication) points out that the rumination alone might not terminate the depression—only perceived solutions to the fitness-relevant problem, which rumination is not guaranteed to find, is hypothesized to terminate depression (see the bargaining section). Tests of the treatment-predictions (prediction 4.1-4.3), should control for this effect.

To test whether depression evolved to solve *complex* problems, we must ask what makes a social problem complex. And, Andrews and Thomson (2009) claim that the complexity of a social problem is dependent on the number of people involved. They call such problems fitness-relevant *dilemmas.* They argue that a human worrying about a social dilemma cannot a priori know what strategy is effective to pursue. Especially when the number of people involved is high, because then the number of viable strategies also increase (which makes the problem *complex.* See point 1 in prediction 4.5). This is not obvious. Heuristic solutions to many complex problems can, and have, evolved. For example, male humans seek out reliable cues to fertility, youth and health, such as low ratio in hips to waist, symmetrical face, clear skin and so on (see Buss, 1989, 1994a, 1994b; Singh, 1993; Symons, 1979). Nevertheless, Andrews and Thomson (2009) argue that all the potential solutions to social dilemmas are non-obvious and tend to differ in their effectiveness. Hence, they argue that it is worth the time and cost to figure out which strategy to pursue, rather than using heuristics.

Andrews and Thomson view findings such as the 25-fold increased risk of major depression when unhappily married (Weissman, 1987) and that conflict with close social

partners (e.g. closer friends) is associated with higher levels of depression if the relationships otherwise are characterized by cooperation (Major, Zubek, Cooper, Cozzarelli, & Richards, 1997) as good evidence that conflicts within cooperative relationships are depressogenic. It can be predicted from the ARH, then, that the number, and closeness, of people involved in a problem are part of what determines the intensity of depressive episodes (see point 2 in prediction 4.5).

Another important part of the first claim is that depression prevents recurrences of avoidable stressors. As some social problems are avoidable, organisms are under selection to prevent them from happening (Andrews and Thomson, 2009). One reason why avoidable stressors happen to humans is a lack of causal understanding of the situation (Roese & Olson, 1997). Mechanisms of operant conditioning associates, for many organisms, negative outcomes with recurrent environmental stimuli. This allows the organism to avoid these negative events in the future. Similarly, people may, when exposed to avoidable stressors, devote some cognitive effort to understand how the unfortunate event can be avoided in the future (Andrew and Thomson, 2009). Such thoughts, Andrew and Thomson (2009) remind us, are called *upward counterfactual thoughts* (see Roese & Olson, 1997). These thoughts are *counterfactual* in the sense that they attempt to simulate "how could my situation turn out differently if I chose a different course of action". They are also *upward* counterfactual thoughts because they tend to focus on how to make the situation turn out *better* next time. Avoidable stressors tend to induce negative affect, which in turn trigger upward counterfactual thoughts. Depressives tend to have more upward counterfactual thoughts about recent avoidable stressors (Markman & Weary, 1996). Andrews and Thomson (2009) cite three different studies (Nasco & Marsh, 1999; Page & Colby, 2003; Roese, 1994), and the common finding among those studies is that upward counterfactual thinking may help prevent avoidable stressors repeating themselves. Nasco and Marsh (1999) found that

students' upward counterfactual thoughts were associated with higher perceptions of control in students. This, subsequently, led to better grades. Such studies suggest that counterfactuals might, at least in some cases, be useful. But, did depression (or low-mood) evolve to activate counterfactuals in specific situations? Testing needs to be done in the AR context: Do upward counterfactual thoughts in depressives, in fact, enhance efficient problem-solving of complex fitness-relevant social dilemmas (see prediction 4.6a and b)?

Andrews and Thomson (2009, p. 628) provide a prediction: "experimentally manipulating the analytical difficulty of the dilemma by changing the degree to which trade-offs must be made between cooperating and pursuing self-interest, by changing the number of people to keep track of, and so on, will induce depressed affect" (prediction 4.9). Andrews (2007) performed an experiment testing some of these ideas. Their participants were asked to solve questions from the Raven's Advanced Progressive Matrices (RAPM) (Raven, Court, & Raven, 2003) test (a nonverbal IQ test). Their prediction was that these analytically challenging problems would induce depressed affect in individuals with low levels of pre-existing depression. Their prediction was supported. This suggests that depressed affect is induced whenever there is a complex task at hand (prediction 4.8b), and that sadness is induced in people exposed to an analytically challenging problem (prediction 4.8a). Andrews et al (2007) also found that the depressive reactions of their subjects were positively related to their performance.

Andrews et al. (2007) study above suggests that depressed affect is induced in humans, with no pre-existing depression, when simply exposed to a complex problem, such as the RAPM IQ test. And, that it actually enhances the performance on it. However, The AR hypothesis claims that depression evolved to solve fitness-relevant problems, not modern IQ tests. It remains to be tested then, if this effect is stronger for interpersonal problems (see prediction 4.5, 4.6 and 4.9). It is important to test too, whether *clinically depressed*

individuals solve complex social-dilemmas differently than individuals with experimentally induced sadness. According to the ARH, sadness-intensity will positively correlate with performance. The more sadness the better. In general, the studies Andrews and Thomson discuss, are on non-clinical populations. Sad college students, mostly. Hence, researchers attempting to falsify these hypotheses on clinical populations will shed a bright light on depression's status as a problem-solving adaptation. The sadness-inducing experiments however, are useful because they seem to suggest that *normal levels of sadness* evolved to solve complex problems. The ARH, and SNH, of course, postulate that even MDD can be an adaptive problem-solving strategy; it is the response to the most complex problems. This begs for testing.

Second claim: Depression coordinates changes in body systems that promote sustained analysis of the triggering problem. Andrews and Thomson (2009) suggest that depression changes cognition and bodily functions in order to *hinder diversion* from the time-consuming analytical rumination. These hindrances are threefold 1) depressed affect activates neurological mechanisms responsible for attentional control, which gives problem-related information prioritized access to limited cognitive resources, and keeps the ruminations intrusive and resistant to distraction (Andrews & Thomson, 2009). 2) Anhedonia, which reduces the desire to engage in activities that disrupt problem-related processing. 3) Psychomotor retardation, which hinders exposure to stimuli that disrupt processing.

Andrews and Thomson (2009) argue that analysis of a complex problem requires the use of working memory (WM) because analytical problems are predicted to be broken down into smaller parts, and studied sequentially, hence the results of the analysis must be kept active in WM in order to solve the larger problem (see prediction 4.10) (Andrews & Thomson, 2009). When WM load is high, such as when analyzing complex social problems, the WM tasks become vulnerable to disruption. This is another reason why, from the AR

perspective, depression evolved. Finding cost-effective solutions to reproductively relevant social problems (e.g. group membership, access to resources) are vital for a social mammal like a human. Hence, depression evolved to make nothing *but* the social problem linger in the mind. It makes sense, from the ARH perspective, if depressives with a high WM-functioning solve analytical social problems faster. And thus they will experience less depression compared to individuals who have a low WM-function, and solve social problems less effectively (prediction 4.10).

The left Ventrolateral Prefrontal Cortex (VLPFC) is continually activated when fMRI subjects solve analytical tasks (Andrews & Thomson, 2009; D'Esposito, Postle, & Rypma, 2000). Andrews and Thomson (2009) include other neuroimaging studies which found that individuals with experimentally induced sadness, and outpatients with major depression episodes, usually have highly activated VLPFCs (Drevets, 1999; George et al., 1995), especially in the left hemisphere. Andrews and Thomson (2009) suggest, then, that brain areas that play a role in attentional vigilance are more activated in sad people and depressives than in non-depressed controls. Maybe, they point out, because activation of VLPFC is necessary to avoid distraction from fitness-relevant problems. However, neural correlates of attention in depressives brain, is not necessarily evidence for adaptation, but the ARH produces novel hypotheses here.

Andrews and Thomson (2009) claim that “Behavioral depression” in rodents cause sustained release of serotonin in the rodent homologue of the VLPFC. They therefore predict that brain 5-HT (serotonin) in those regions is *high,* not low, in depressed humans (prediction 4.11b). However, see Hendrie and Pickels (2009) critique of using rodents as a model-species for depression, they argue that the social selection pressures in most rodents evolutionary history are too different to be applicable to humans (2009). Anyhow, Andrews and Thomson (2009) predict that the VLPFC level of activation correlates with the severity of a patients

depressive episode (prediction 4.11c). They also predict that the "sustained release of 5-HT to the VLPFC should promote the production of astrocytic lactate, sustain neuronal firing, and reduce apoptosis by supporting the clearance of synaptic glutamate". The logic behind this prediction is that if the VLPFC is continually activated in depressed individuals, many of these cortical neurons will release glutamate. And, a high level of glutamate in the synapse is toxic because it induces apoptosis (cell suicide) in the neurons. So if it is true that sustained activation of the VLPFC is necessary to prevent disruption of analytical rumination, it should come with the risk of apoptosis. However, serotonin often acts as a modulator on glutamate (see Ciranna, 2006), which is why Andrews and Thomson predict that an increase in serotonin in depression has evolved to decrease the risk of apostosis. Further, astrocytes take up glutamate from the synapse and convert it to glutamine (cellular energy), which is stored in the neuron. Astrocytes (glial cells) also provide neurons lactate, which is also used for energy. Under continued neural firing, the energy-costs becomes acute. Thus, to continue firing, neurons rely on this astrocytic lactate as an energy source. This is relevant for the AR hypothesis because Andrews and Thomson predict that depressives have increased production of astrocytic lactate (prediction 4.11a). The sustained activation of the glutamergic neurons in depressives VLPFC makes this likely. Prediction-wise, it is useful to acknowledge that the ARH predicts that a depressed brain should, contrary to conventional understanding, be marked by an increase in serotonergic firing. And, be marked by increased production of astrocytic lactate in order to provide sufficient energy for continued firing of VLPFC neurons.

This might also provide an explanation for the connection between depression and carbohydrate-cravings (see Wurtman & Wurtman, 1994, 1995). The essential aminoacid tryptophan constantly flows in the bloodstream, and is converted into serotonin in the brain. The insulin-secretion during intake of carbohydrates makes peripheral tissues, like muscles,

take up most aminoacids in the bloodstream. Trypthopan is an exception, it is not affected by insulin. Hence, during intake of carbohydrates, tryptophan has less competition in entering the blood-brain barrier (Wurtman & Wurtman, 1994). This makes sense in the AR perspective: Maybe depressives crave carbohydrates because it enhances the production of serotonin, which is necessary for the continued activation of attention systems such as the VLPFC (see prediction 4.11d).

Anhedonia, the inability to experience pleasure, too, the ARH proposes, reduces disruption of depressive rumination. Attending to immediate rewards, such as sex, eating and companionship, would interfere with attempts to solve the social problems in the depressives life (Andrews and Thomson, 2009). Thus, "Anhedonia should promote uninterrupted rumination by reducing the motivation to engage in hedonic activites" (Andrews & Thomson, 2009, p. 623). A relevant prediction then is that the degree to which a depressives are anhedonic, predicts the degree to which they have a focused and uninterrupted rumination, and hence solve a social problem faster (Prediction 4.12a and b). If this is true, we would expect that anhedonic patients that are treated with simple behavioral activation (with no problem solving), would experience longer, more intense depressive episodes. Given that a meta-analysis (Cuijpers, Van Straten, & Warmerdam, 2007) found that behavioral activation is effective for depression, this is a challenge to ARH. If the ARH is true, the efficacy of behavioral activation is due to enhanced problem-solving. Given that behavioral activation often consists of simple scheduling of pleasant activities (see Cuijpers et al., 2007), this is by no means evident.

Andrews and Thomson (2009) also interpret the psychomotor changes that are typically seen in depressed patients as a disruption-avoidance strategy. Fatigue, changes in appetite and a preference for solitude, all contribute to sustained analysis of the triggering social problem by avoiding distracting stimuli (Andrews and Thomson, 2009). This generates

a similar prediction as for anhedonia: Psychomotor retardation is expected to be associated with more intense rumination (Andrews and Thomson, 2009, p. 632). The authors also argue that Anhedonia should be triggered, in a dose-dependent manner, by analytically difficult problems, especially those who are evolutionary relevant (Andrews and Thomson, 2009). See prediction 4.12a and b.

Third claim: Depressive rumination helps people solve the triggering problem. Andrews and Thomson (2009) argue that future research should investigate how depressive cognition influences the *triggering* problem. They argue that this is best tested experimentally. Individuals with pre-existing depression might have different problems, in experiments, however, the analytical problems are carefully designed and equal to all participants. Induce groups with different affects (and their intensity), then see how they do when solving the same problems. Andrews and Thomson do not address it, but this has the same problem of assuming that sad mood is simply a continuous enhancer of social problem-solving. That might not be true (see prediction 4.14a). Severe cases of sadness, which are hard and unethical to induce experimentally, might not enhance reasoning on social problems. Despite of Andrews and Thomson (2009) preference for experiments, another way of testing could be by interviewing people who have been depressed, but not anymore. What made them depressed, and did their depression in fact disappear after ruminating their way to a solution to their problem?

Experimental studies seem to suggest that depressed (or, at least, normal "bad mood") individuals indeed make better decisions. One example is Au et al (2003) who manipulated mood by asking participants to read mood-laden or neutral statements, and listening to either sad or upbeat music. Thereafter, the study participants were asked to participate in a trading-market game. Traders in a good mood lost more money. People in a bad mood behaved more conservatively, had more accurate judgments, and hence profited the most. Pleasant mood

seemed to reduce peoples accuracy, while increasing confidence in their judgment (Au et al., 2003). Other experiments have shown that depressed people are more sensitive to costs of cooperating than are nondepressed people, and they are more likely to defect when it is costly to cooperate (Hertel, Neuhof, Theuer, & Kerr, 2000). In other words, depressives tend to make more "rational" decisions in these circumstances (see prediction 4.14b). One can argue then that a "good-mood" strategy might be an ineffective strategy when trying to solve complex, high-risk and fitness-relevant, social problems. But this is clearly a premature conclusion. In the study by Hertel and colleges (2000) they found that positive mood promoted a heuristic processing style. Furthermore, they found that negative mood lead to longer decision latencies, which they interpreted as evidence for a deliberate cognitive style. This indeed lead sad participants to more economically rational decisions in the dilemma that they studied (chicken dilemma). However, depressive's failure to make quick decisions can also be viewed as manifestation of an unadaptive illness, not an adaptation for solving complex problems in rational ways. People who are depressed might make "careful" decisions, not because of enhanced rationality, but because depressive illness lead people to *stop making* decisions. Future experiments must explore this. The notion that depressive thinking is rational thinking has been raised before ("depressive realism" see Alloy & Abramson, 1988). In a review of the evidence for depressive realism Ackermann and Derubeis (1991) write: "although many studies have generated evidence consistent with the depressive realism hypothesis, almost as many have provided evidence inconsistent with this view." (p. 565). There is a danger then, that Andrews and Thomson (2009) discussion is biased by cherry-picking of compatible evidence. Even so, the experimental evidence mentioned (Au et al., 2003; Hertel et al., 2000) seem to suggest that sad people make more conservative, careful decisions. The question remains, as discussed earlier, if this is true for clinical depression, and especially major depressive disorder. Experiments on normal

populations insufficiently tests the ARH. Trials that compare neutral mood, experimentally induced sadness *and* patients diagnosed with mild depressive episode, *and* MDD, surely will illuminate how these states affect social problem-solving skills. Also, experimental studies could test whether there is a content-effect. Are there particular problems that sad participants and clinical depressives are better or worse at? Of course, the ARH predicts that depressives are better at solving problems that are interpersonal in nature. Given such a content-effect, is it dose-dependent, meaning more sadness, better social-problem solving? Or, perhaps, is the relationship an inversed-u-curve pattern, sadness being effective, but severe depression not? All are empirical questions, and the ARH predicts that the dose-dependent effect would be true (See prediction 4.14a). Another relevant prediction is this: if depressive rumination is helping to solve the triggering social problem, expressive writing should enhance long-term outcomes of psychotherapy (prediction 4.15). Andrews and Thomson (2009) cite a controlled study which supports this (see Graf, Gaudiano, & Geller, 2008).

Fourth claim: Depression reduces performance on laboratory tasks because depressive rumination takes up limited processing resources. Andrews and Thomson (2009) interpret the evidence that depressed individuals perform badly on laboratory tasks (e.g Austin et al., 2001) in the following way: performance-decrements happen because depressives cognitive resources are allocated to something else, probably the depression-triggering problem. They cite evidence that support their interpretation. When research participants are asked to read statements such as "I feel a little down today" (see Seibert & Ellis, 1991), it reduces their performance on laboratory tasks (Ellis, Ottaway, Varner, Becker, & Moore, 1997). The ARH therefore predicts that depressive rumination will interfere with performances on laboratory tasks. And that depressed individuals will perform just as well as nondepressed individuals *if this rumination is temporarily stopped* (prediction 4.16a & b). This prediction is supported by an experimental study (Watkins & Brown, 2002). Here, the

difference in outcome on a random number-generation task between depressed and nondepressed people were wiped out after both groups performed a distraction task (thinking about a black umbrella for five minutes). Suggesting that depressives apparent decrement in executive functioning might be due to other cognitive processes (rumination, problem-solving) that use limited cognitive resources. As we have seen, the ARH claims that this is one thing depression evolved to do: hinder diversion from problem-solving rumination. This leads the ARH to an interesting set of predictions: Temporary alleviation of rumination will increase performance on laboratory tasks, but prolonged alleviation of rumination will increase the length of depressive episodes (See prediction 4.16, 4.1a and b). The fourth claim also receives support from a study by Andrews and colleges (2007). Here, their "magnet metaphor" was supported (prediction 4.16a): people with pre-existing depression experience a decrease in depressed affect when given a distracting task. In the absence of a distracting task however, their depressed affect increases. The steel ball (the depressive) must be pulled away from its magnetic source (their problem) in order to temporarily reduce the magnetic force (depressed affect).

Social Motivation Function and Bargaining Hypothesis.

In addition to the analytical rumination function, Social Navigation Hypothesis (Watson & Andrews, 2002) postulated a *social motivation function*: Depression is hypothesized to serve the function of persuading others to help the depressive (2002). Similar ideas have been expressed by Hagen (2003; Hagen, Watson, & Thomson) in his Bargaining Hypothesis (BH). Both theories attempt to account for depression in general. Hagen has also hypothesized about bargaining in relation to postpartum depression (this is discussed in the sex differences section). The idea of help-elicitation it also a part of Suarez and Gallup's (1985) Reproductive Failure Theory and the Social Risk Hypothesis. The notion of depression as help-eliciting is therefore important to test, as they will throw light on several

evolutionary hypothesis for depression.

The logic of Watson and Andrew's (Watson & Andrews, 2002) social motivation function is twofold: The depressed state forces a fitness cost not only on the depressive herself, but to her relatives and other allies. It is an *honest signal* of need (this is based on the handicap principle, see Zahavi, 1975, 1977). Since the depression is honest it motivates others to help due to *fitness extortion:* it forces members of the depressives' social network to make investments they would not otherwise make (Watson & Andrews, 2002). If the depressive state was easily manipulated, it would not be an honest signal, hence the very point of depression, the SNH and BH predict, would be to provide an enormous fitness cost on the depressive herself. People who are interested in the depressives fitness might do best by intervening, rather than ignoring or manipulating the depressive. A straightforward prediction, then, is that genuine (honest) help from significant others would relieve, or stop entirely, the depressive episode (prediction 5.1b). From this perspective, MDD is a form of manipulation, therefore the SNH predicts that depressives are met with negative reactions (prediction 5.2b). Furthermore, Hagen and Watson (2004) emphasize that MDD will be induced in individuals if that person has a need for social assistance *and* has strong conflicts with important social partners (prediction 5.2a). Negative life events in general, are not predicted to induce MDD (Hagen & Thomson, 2004). It is predicted, as with the ARH, that depressive symptoms are reduced to the degree the triggering problem is solved (prediction 5.5).

Watson and Andrews (2002) divide the fitness extortion in two *motivation hypotheses*. Depression is designed to either motivate whole networks, the "niche change" function of depression, or to motivate specific partners. This generates more predictions.

Watson and Andrews (2002) define an individual's social niche as the reciprocal exchange contracts they have with each person in their social network. "A person's social

niche", Watson and Andrews (2002) write, "may vastly under-utilize their capacities for maximizing inclusive fitness". The authors go on to postulate that niche changes require assistance from people in their network (political favors, skill training, capital investment). If an individual motivated to change its social niche experiences drawbacks due to the expectations and attitudes of their group, this problem might best be solved by motivating the social network *as a whole* through severe depression (Watson and Andrews, 2002). Thus, the prediction is that depression should be designed to resist convictions of staying in the status quo, and, in fact "contingently worsen to a point where the costs imposed on the depressive's social partners overwhelm their resistance to the bid for niche change". It could be predicted then that the numbers of, and degree to which, a depressives family members and friends try to resists the depressives attempts of niche change (e.g. starting a new job or education, moving away from home) are positively related to the strength of the depressives symptoms, including suicide (Watson & Andrews, 2002) (prediction 5.3a and 5.7, respectively). If the niche-change idea is true, psychotherapy for depression should be effective if the therapist is sensitive to the opportunity costs the patients perceives ("I have much to gain by switching social niche") and help the patient to develop an idiosyncratic plan for achieving the niche-change, or alternatively, help the patient abandon the desire for niche-change (Watson, personal communication). In both of these cases the depression should be relieved (prediction 5.3b). However, there are still no empirical tests of these ideas.

It is also possible that depression is designed to motivate specific partners. Watson and Andrews (2002) suggest that this could be tested by "showing variation in symptomatology that preferentially imposes costs on those partners with whom the depressive is in conflict, and that the costs were imposed to overcome their reluctance to help". In other words, the "specific partners" motivational hypothesis predicts that depressives symptoms will idiosyncratically correspond to motivate a specific significant

other (see prediction 5.4). Watson and Andrews (2002) point out that both motivational hypotheses predict that people should become more depressed when they are in conflict with partners who have a greater positive fitness interest in them, defined as people who have helped the depressive in the past (prediction 5.6a and b). Complimentary, Watson and Andrews (2002) propose an adaptive function for suicidal behavior (see prediction 5.7). Suicide has, albeit differently, been hypothesized as adaptive before (as a kin-selected mechanism, see de Catanzaro, 1995). Under the SNH perspective, the risk of death is an honest signal which informs others of the severity of their need. The reason for the heightened risk of suicide after hospitalization (Bostwick & Pankratz, 2000), Watson and Andrews (2002) predict, is that such practices removes the depressive from their social group, and hence, failing to motivate the absent social partners, suicidal behaviors escalates into louder and louder signals, which ends in death (Watson & Andrews, 2002).

Do the Analytical Rumination Hypothesis and the Bargaining Hypothesis Show Evidence of Design?

The ARH views depression as a coordinator of bodily and cognitive systems which facilitates creative solutions to real-life, complex, fitness-relevant social problems. And, furthermore, the bargaining model and the social motivation function predict that depression evolved to elicit help from social partners. Nettle (2004) has critiqued adaptive hypothesis, but especially the Social Navigation Hypothesis, which consists of the Analytical Rumination and Bargaining Hypothesis. The reason for this is that they propose the boldest claim: Major depression can be adaptive.

As I discussed in the introduction, adaptations must lack heritable variation; show evidence of good design; have appropriate evocation (Nettle, 2004), and; individuals who lack the adaptation (in, or close to, the ancestral environment), must, either by chance or experimental manipulation, have reduced fitness (Nettle, 2004). Since the SNH, and hence

the ARH, and the BH, postulate that depression, both minor or major, can be an adaptation, Nettle (2004) argues that "any individuals who lack the capacity to become depressed when there are appropriate cues in the environment should suffer reduced fitness". And this is the core of Nettle's (2004) criticism of the SNH (ARH and BH): some people are more at risk, due to genetic variance, for depression than others, which means that depression fails to make the criteria for an adaptation. Although environmental triggers have been shown to be important in depression, their effects seem to be moderated by genetic risk-factors (Caspi et al., 2003; Kendler, Karkowski, & Prescott, 1999; Kendler et al., 1995; Kessler, 1997). This supports a classical stress-diathesis view; genetic predispositions are activated by environmental triggers. In other words, and Nettle (2004) stress this point: There are many people who experience the life-stressors that depressives do, but *do not* become depressed, which suggests that depression is not a species-typical adaptation activated by appropriate environmental triggers (this point is discussed further under the mutation-selection part). Additionally, Nettle (2004) points out, the best predictor for depression is *an earlier depressive episode*, and as Varga (2012, p. 49) has remarked, while assuming, for thought-experimentation, that depression solves complex problems (ARH):

> Knowing that every depressive episode dramatically increases the risk of another episode, this means that individuals facing complex problems are more likely to face such problems again. So even if it were true that depression is an adaptation to allow optimal functioning, it certainly does not seem to be effective.

At the face of it, depression does not seem to solve the evolutionary problem which the SNH and the Bargaining Hypothesis predicts it to solve, which, of course, is a requirement for an adaptation: It must be shown to actually solve the problems it was designed to solve. If we predicted, and tested in a laboratory, that men are equipped with a psychological adaptation to solve the adaptive problem of identifying fertile mates, but the

men's mate choices were found to be completely random and unrelated to fertility cues (such as a 0.7 waist-to-hip ratio), it would led us to strongly suspect that men do not have a psychological mechanism to solve this problem. This is the situation with depression too: There is no evidence—which may, of course, be due to lack of proper empirical testing—that depressives solve a set of adaptive problems better than non-depressives (especially in the clinical range). Indeed, Nettle (2004) argues that depression instead has "all the hallmarks of a progressively deteriorating and socially disastrous dysfunction of the central nervous system rather than an adaptation that has achieved its goal".

The Social Risk Hypothesis

The Social Risk Hypothesis (SRH) postulates that depression evolved to minimize risk in social interaction. That is, to minimize the risk for group exclusion. Depression down-regulates positive affect and confident engagement in the world because it evolved, in Badcock and Allen's words (2011), to "facilitate a risk-aversive approach to social interactions in situations where individuals where typically at risk of exclusion from social contexts (dyadic relationships or groups) that were vital to dealing with adaptive, socio-reproductive challenges" (p. 131). Altogether, Allen and Badcock (2003) postulate that depression serves the function of decreasing risk of exclusion by first being hypersensitive to signals of social threat from others, and, based on those perceptions, send signals that reduce risk and elicit care, and additionally, inhibit confident risk-seeking behavior. Social activities such as competition and cooperation with non-kin, are viewed as high-risk activities. Depression, then, from the SRH perspective, is a shift in an individual's desire for competition and non-kin cooperation, which is viewed as high-variance and risky, to less risky care-elicitation from close, communal-type relations (Allen & Badcock, 2003) (see prediction 6.1).

Importantly, Allen and Badcock (2003) specify that their theory attempts to explain

"mild (and predominantly transient) depressive states of the type that are experienced by most persons from time to time" (p. 888), and they argue, as many others have (Gilbert, 1992; Hagen, 2011; Horwitz & Wakefield, 2007), that extreme states of depression, are best understood as pathologies.

If the SRH is correct, mild and transient depressed mood preserves social relationships (prediction 6.4b), and also, reduces the risk that an individual will be socially excluded (Allen and Badcock, 2003) (prediction 6.4a). Another straightforward prediction from the SRH is that sad and mildly depressed individuals are more sensitive to social risk when such risks are present (prediction 6.3b). However, the *reverse* causal pattern is also plausible: heritable personality factors (Jang, Livesley, & Vemon, 1996) are associated with depression, for example neuroticism and interpersonal sensitivity (see Boyce & Mason, 1996), this can also explain the interpersonal sensitivity of depressives. If the SRH is true, we would not expect that any normal human being will fail to be depressed, regardless of personality structure, given a history of problematic interpersonal encounters, such as exclusions, and certainly not during the exclusion process.

Allen and Badcock (2003) intentions with the SRH, they write, is to analyze the "specific socially embedded selection pressures that would likely have led to the evolution of depressed states—that is, the avoidance of social exclusion by controlling the balance between an individual's social value to others and his or her social burden on others" (p. 887). Here, the social value refers to resources: If an individual is considered to be low in resources, or burdensome, it might be advantageous for the other members of the group to exclude that individual (Allen & Badcock, 2003). The ratio then, between resources that an individual provides to its group (social value) and the cost to others (social burden), is critical. In order to *avoid* social exclusion, Allen and Badcock (2003) propose an evolved mechanism by which an individual can judge its own social value. They call it *social*

investment potential (SIP)—this is the calculated ratio between social burden and value—and phenomenologically the SIP is experienced as low self-esteem (Allen & Badcock, 2003). This mechanism enables a human being to internally measure their social value, such as attractiveness, talents, skills and resources, relative to others (Allen & Badcock, 2003). This is similar to Leary's "sociometer" theory, where self-esteem is linked to social exclusion (Leary & Baumeister, 2000; Leary, Schreindorfer, & Haupt, 1995; Leary, Tambor, Terdal, & Downs, 1995), however, Allen and Badcock (2003) argue that the SIP is a mechanism that is directly relevant to behavior. The factors leading to depression, they postulate, do so by affecting the SIP (Allen & Badcock, 2003). However, contrary to the SRH, Leary's (1995, p. 297) view is that self-esteem "are best viewed as reactions to real, anticipated or imagined rejection rather than as consequence of self-esteem per se" (p. 297). The SRH predicts, then, that negative interpersonal experiences should cause lower self-esteem, and depression is then predicted to come *afterwards* (prediction 6.7c).

It is appropriate to mention Spoor and Williams' (2007) model, where the painful responses that humans feel when they are ostracized (see MacDonald & Leary, 2005; Wesselmann, Bagg, & Williams, 2009) is the workings of an evolved ostracism-detection device. Buss (1990) too has argued that the evolutionary dangers of ostracization, such as the loss of crucial reproductive opportunities, has been one of the selection pressures behind the evolution of anxiety. Kurzban and Leary (2001) have written about the other side of the fence: the evolution of psychological mechanisms behind exclusion and avoidance. Humans, Kurzban and Leary (2001) argue, are motivated to avoid poor social exchange partners, join cooperative groups, and avoid contact with people more likely to carry pathogens. Allen and Badcock (2003) are hence, not alone when arguing that group-processes such as exclusion, have been important in the evolution of humans. As humans have evolved a desire to increase their social investment potential (SIP), depression can be viewed as a strategy, when their SIP

is critically low. The SRH predicts, then, that the loss, or dissolution of significant interpersonal relationships, or experiences that are associated with low or inadequate social rank, induces depression (Allen & Badcock, 2003). The Social Competition Hypothesis predicts the same thing. However, Allen and Badcock (2003) go on to say that it is *not only loss or defeat* that induces depression, but all "socially relevant experiences that indicates (or has indicated through evolutionary history) to an individual that his or her ability to successfully invest in socially implicated endeavors is dangerously low" (p. 894). What cues from the environment can reveal social status? Allen and Badcock (2003) provide examples: negative interpersonal experiences (losses, rejections); failure of important social goals; perceived loss of rank or status; perception of lack of control in social situations. Furthermore, they include facial behavior, voice characteristics/verbal content and eye gaze/direction as relevant input from the social world. All these cues are relevant for testing the SRH, they should all contribute to depression because they have probably been reliable indicators of social exclusion (see prediction 6.7).

Is depression reducing social risk?

The SRH hypothesize that the evolutionary task of depression has been to *reduce the variance* in social outcomes until a more secure social position is established (Allen & Badcock, 2003). That is, Allen and Badcock (2003) argue that it has been adaptive for humans, when in danger of ostracization, to abstain from complex social endeavor's such as competition and cooperation (which they view as highly variable), and instead shift their focus on eliciting support from their immediate circle of family and friends (reliable, low-variance care). The SRH theory is inspired by work done in risk-sensitive foraging (Allen & Badcock, 2003; Caraco, Martindale, & Whittam, 1980; Hagen, 2011; Stephens, 1981). Hagen (2011) provides an example of how a wild animal might solve its energy-dilemmas: Imagine an animal that can either harvest strawberries or blueberries, where both have the same *mean*

energy return. But the return of strawberries is highly variable. If the animal has plenty of energy stored in its body fat or in its food reserve, it does not matter if the animal forages for strawberries or blueberries—both berries return, on average, the same energy. Now, imagine if the animal is slightly above its energy threshold; the body fat or food reserve are barely adequate for surviving the night. In this scenario, the wise strategy is to be risk-averse and forage for blueberries. Due to strawberries varied energy-return, the chances of coming home empty-handed is too great.

This is what Allen and Badcock (2003, p. 893) hypothesize for human depression. "Just as in the financial context in which those with high levels of resources are able to tolerate the risk (i.e., the potential losses) associated with making more uncertain (but potentially higher pay-off) investments", those who perceive their social value to exceed their social burden (that is, their SIP is high), are able, they continue, to "safely tolerate the risk associated with more speculative social investments" (p. 893). Opportunity-seeking strategies are risky, Allen and Badcock (2003; 2011) argue, and depressive behavior is not. Although this is certainly a possibility, it is not obvious. It is equally plausible that humans evolved a tendency to behave *more* riskfully under the threat of exclusion (Nettle, 2009). Consider an even more deprived situation for Hagen's (2011) animal: The food reserve (and/or body fat) is well-below the danger-threshold. Here, the animal should forage on high-risk strawberries, because it at least provides a chance to reach the critical threshold, and hence avoid starvation (Hagen, 2011). The same logic could be applied to the down-regulation of mood in humans: if the threat of group-exclusion is dangerously high, do something drastic (risky-strategies, with potential large payoffs). Indeed, Nettle (2009, p. 5) writes: "…if low mood had been associated with exactly the opposite symptoms (increased energy and optimism, greater risk-taking), that would have been equally easy to formulate an adaptive logic for". The foraging literature can provide some hints. Caraco's (1980) laboratory experiments found that the

food-preferences of yellow-eyed junkos (a North American bird) changed based on their energy-budget. Interpreting Caracos results, Stephens (1981) suggests a rule-of-thumb, that the Junkos operate by: be risk-averse if your expected 24-h energy budget is positive, be risk-prone if your expected energy budget is negative. If we apply this logic to human sociality, it is expected that individuals threatened with exclusion should be risk-prone. However, the SRH's predicts that when a human has low social value and self-esteem (low SIP), this individual should be *risk-averse* (depressed) in social situation. However, it might be the case that a risk-prone strategy, like it is for food-deprived Junkos, is more adaptive when your social value is low. Hence, the adaptive logic behind the SRH is not watertight. Of course, it is possible that depressive behaviors raises social value—the selection pressures behind sadness may be completely different than the evolution of risk-sensitive foraging. But, if so, how? Nettle (2009) tested this idea by using the same theoretical, mathematical paradigm used in the foraging literature (Stephens, 1981). Nettle's (2009) model predicted that an organism should prefer the risky strategies when in a good state (that is, far away from the "dangerous" threshold), and, as the SRH predicts, prefer less risky behavior, in a step-by-step manner, when the state is approaching the dangerously low state (see fig 1 in Nettle (2009)). However, contrary to the SRH's predictions, as the state deteriorates over a critical stage the strategy of choice becomes more, and more risky. The risky strategies are chosen, roughly, in a U-pattern: the ones in a really good state should choose the *same* risky strategies as the ones in a really bad state (Nettle, 2009). Analogously, there is evidence that both rats (Boakes, 2007) and humans (Fessler, 2002; Holtkamp et al., 2003) become agitated under food-restriction. This makes sense evolutionary, Fessler (2002, p. 381) argues, because starving humans "have little to lose, and much to gain, by pursuing the promise of immediate resource acquisition regardless of potential cost". Indeed, the excessive running and workout anorexic patients often do, is probably a misfiring of such mechanisms (Fessler, 2002). So,

evolutionary speaking, why would a human that finds itself dangerously close to social exclusion "choose" a low-variance depression strategy? Nettle's (2009) results indicate, at least theoretically, that the SRH's prediction that low-variance, risk-averse strategies is adaptive in dire situations, such as an unfavorably low value/burden ratio (prediction 6.1), is inadequate at best. Nettle's (2009) results does not falsify the SRH, but they strongly indicate that the theory is incomplete: When a depressives social situation increasingly worsen, the "depression strategy" should end in favor of a risky strategy.

Interestingly, Nettle (2009) suggests that the increased energy and disinhibition in mania is a potential candidate for a high-risk, high-variance strategy that is activated in extremely dire situations. Another candidate is agitated depression (Nettle, 2009) (See the predictions based on Nettle (2009): 6.10 and 6.11). There is evidence that mania can be activated by dire social situations, such as the death of loved ones, known as "funeral mania" (Krishnan, Swartz, Larson, & Santoliquido, 1984; Rickarby, 1977). However, these studies are done on small samples, mainly on patients diagnosed with bipolar disorder, which, clearly, is no evidence of species-typical design. Especially given the evidence that some patients with schizophrenia and manic-depressive disorder have specific mutations, not shared by the general population or parkinson patients, in a gene (NURR1) relevant for the dopamine system (Buervenich et al., 2000). However, it is not known whether adversity-caused mania is part of human nature, or if only individuals with heritable mutations have this ability. If the former is true, it would strongly suggests dysfunction rather than function (Kennair, 2010; Wakefield, 2005). Nettle's (2009) predictions are novel and interesting nevertheless, and can potentially update, rather than falsify the SRH (prediction 6.10 and 6.11).

It is to be expected, if the SRH is true, that people, and organisms in general, who lack the ability to calculate social value and burden, should either lack, or show less evidence

of, depressive behavior (prediction 6.8a). Other mammals, human children, or humans with specific brain/cognitive pathologies (which show evidence of deficits in value/burden calculations) should be less depressed (prediction 6.8b) (Allen & Badcock, 2003). Indeed, Allen and Badcock predict that amongst individuals who have impairments in social cognition, such as autism, are less likely to be depressed because of a lack of ability to estimate social value and burden (Allen & Badcock, 2003). However, one comorbidity study (Leyfer et al., 2006) found that ten percent of *children with autism* met the DSM-IV criteria for major depression. Which is might be evidence against this notion. However, Baron-Cohen and his colleges (see Baron-Cohen, 1997b; Baron-Cohen, Leslie, & Frith, 1985) have found evidence that people with autism lack the ability to make "theories of mind", that is, the ability to impute beliefs, and predict the behavior of others. Baron-Cohen (1997b) calls it "mindblindness". Although this is obviously a serious deficit in social cognition, it is possible that individuals with autism do not lack the ability to estimate social burden and value. If future scientific investigations show that autistic individuals *lack* the ability to estimate their own social value and burden, and at the same time, are often sad and *minorly* depressed, which is what the SRH attempts to explain, it would be a challenge to the SRH.

The SRH predicts that mild depressives have faced several problematic interpersonal encounters in their history (Badcock & Allen, 2011). Presumably, from the SRH perspective, such experiences have activated the risk-aversive depressive mechanisms that are part of the normal evolved human architecture. In other words, the SRH predicts that anybody can become mildly depressed in specific contexts, for example while being socially excluded. This is contrary to data which indicate that some people, due to genetic vulnerability, are more likely than others to become depressed, even when exposed to the same stressors (Caspi et al., 2003; Kendler et al., 1999). However, it is unclear if this data would be different for the normal sadness and mild depression in which the SRH hypothesize on (see prediction 6.14

and 6.15). Hence, if it is shown that mild depressives have experienced no more social stress during their life, than the general population, the SRH is probably false. Noteworthy, it is known that the depressed state can alter the *retrieval* of past social failures. Memory retrieval is mood-dependent—sad people remember sad events, happy people remember happy events (Bower, 1981; Josephson, 1996). Potential mood-dependencies are important to acknowledge when using methods such as interviews and questionnaires when testing whether mild depressives have experienced more social stress/exclusion, for example by inquiring about bad interpersonal experiences when the study participants are in a neutral mood.

The SRH also predicts that the lack of appetitive functions in depression, such as desire for food and sex, are activated because those symptoms reduce the depressives social burden (prediction 6.9) (Allen & Badcock, 2003).

Tests of the Social Risk Hypothesis

Three hypotheses derived from the SRH has been tested by Dunn, Whelton and Sharpe (2012). They asked full-time working Canadians from several professions to fill out surveys that measure the evolutionary-relevant variables for the SRH (and the SCH), such as the Defeat Scale (Gilbert & Allan, 1998) and the Social Comparison Scale (Allan & Gilbert, 1995). The first hypothesis they tested is that attachment, social comparison and defeat would predict depressed mood (Prediction 6.12). Indeed, all of those factors predicted depressed mood, especially defeat, which was their strongest predictor (Dunn et al., 2012). This finding supports the Social Competition Hypothesis—perceptions of lost status are expected to induce depressed mood. Secondly, they predicted that self-esteem (SIP) should mediate the roles of attachment, social comparison and defeat (prediction 6.13). This was only partially supported, the link was only observed through the defeat variable: "when self-esteem is low, the impact of depression is magnified in those who are experiencing defeat in the social realm" (Dunn et al., 2012, p. 756). The third hypothesis Dunn, Whelton and Sharpe (2012)

tested was that depression should lead to the inhibition of social risk-taking behavior, submission and interpersonal sensitivity (prediction 6.14). They found, consistent with the SRH, that depression predicted interpersonal sensitivity and submission (Dunn et al., 2012). An obvious limitation of this study is that it found associations; the causal variables cannot be determined. Another model could potentially explain the data equally well (Dunn et al., 2012).

Relevant for the SIP, Leary et al (1995) tested his Sociometer Hypothesis by directly manipulating, in experiments, social inclusion and exclusion variables (See study 3 and 4 in Leary, Tambor, et al., 1995), they found that individuals who were deliberately excluded, had their self-esteem significantly reduced, indeed the authors write that they were "surprised that respondents responded as strongly as they did to being excluded by the group" (p. 524), although this strongly suggest that perceived exclusion induces averse emotions, Leary and his colleges (1995) did not test how this related to clinically depressed individuals, let alone whether mild depression is a part of humans evolved cognitive architecture.

Badcock and Allen themselves (2003) have also tested aspects of their hypothesis. They did mood-induction experiment (with music). Both the depressed-mood and the neutral-mood group were asked to respond on Wason's selection tasks (Wason, 1966) with themes on attachment, social competition, truth detection and cheater-detection (See Cosmides & Tooby, 1992). The depressed-mood group had significantly more correct responses on the social competition selection task, but they failed to show significant superiority on the three other Wason selection tasks. The failure by the depressed-mood group to show superiority on truth-detection and cheater-detection was as Badcock and Allen (2003) predicted (there are evidence that cheater-detection is a cognitive adaptation, and is not necessarily expected to be mood-dependent). However, Badcock and Allen (2003) did also predict that members of the depressed group would outperform the neutral-mood group on the attachment selection task,

this was not supported. Overall, these findings provide strongest support, as Badcock and Allen (2003) point out themselves, for the Social Competition Hypothesis (Price et al., 1994): Humans in a depressed mood may have rank-related attentional biases (Badcock & Allen, 2003).

Given a human at the risk of exclusion, why is it useful to activate depressives behaviors, such as psychomotor retardation, suicidal cognition and so on? Is it not possible that a strategy could evolve where an individual invests *more* in social relationships, that is, for example, by temporarily cooperating and giving without expecting reciprocation from others, and thereby alter the value/burden ratio in their favor, instead of being depressed, and hence, be unable to provide efficiently because of psychomotor retardation, fatigue and so on? The SRH, as mentioned, is an attempt to explain sadness and mild depression. Severe strong psychomotor retardation, suicidal ideation and so on, is often more associated with major depression, but the SRH does not address how, and when, depression starts to dysfunction (is suicidal ideation, for instance, part of depressions design?). And, it is not obvious how mild sadness elevates social value. This needs to be tested further. Nevertheless, humans have been a very social mammal during the EEA, and have a strong desire for interpersonal attachments (Baumeister & Leary, 1995). This makes it plausible that ostracism-detection and social risk-taking have been involved in the evolution of affective states such as sadness and depression, but little is known. Tests of the SRH's predictions might prove promising in furthering our understanding of normally functioning sadness and depression.

Ultimate Theories on the Sex Difference in Depression

Unipolar depression is typically twice as prevalent among women around the world (Lopez et al., 2006; Nolen-Hoeksema, 1987; Weissman & Klerman, 1977). This finding is consistent, and it is a genuine difference, not caused by measurement artifacts (Weissman et

al., 1993). Although, worthy of notice, the gender difference might be smaller in developing countries (Culbertson, 1997). Depression, then, like height, is sexually dimorphic, which requires an explanation (D. Buss, personal communication). In evolution, gender-differences are intriguing. Natural selection tends to decrease phenotypic variance in a population. This happens because higher-fitness traits spread more rapidly, eventually leading to fixation, and thus reduce variance (See Barkow et al., 1992). Most adaptations are species-typical—all members of a species reliably develop the adaptation. A large gender difference in any species, therefore, suggests that males and females have reliably faced different selection pressures in their evolutionary history (Buss, 1995a, 1995b). Is this the case the with human depression? Why does this difference exist? We know that females start getting more depressed than males at the age of 13 (Hankin & Abramson, 2001; Hyde, Mezulis, & Abramson, 2008; Thapar, Collishaw, Pine, & Thapar, 2012). The fact that the difference starts emerging at the onset of puberty, and not earlier or later, suggests that proximate factors such as gender-specific hormones, and the genes they activate, might play a role in the development of depression.

Hormonal changes in the reproductive systems affect neuronal systems (especially serotonergic ones), which affect mood (Noble, 2005). Sex hormones seem to be important because semen in women's reproductive tracts have been shown to negatively correlate with depressive symptoms (not explained by sexual activity per se), suggesting that the estrogen, testosterone, and prostaglandins in the semen, which is absorbed into the bloodstream (Benziger & Edelson, 1983), play a role in modulating mood (Burch & Gallup, 2006; Gordon Jr, Burch, & Platek, 2002). This supports Ney's hypothesis where he suggested that "a woman through her vagina absorbs sufficient quantities of the prostaglandins from her partner's semen, to affect her mood" (1986, p. 221). Adding to this, using a random sample of 9792 people in the UK, Bebbington et al. (1998) found evidence that the gender difference

is strongest during women's reproductive years. And that there was a "clear reversal of the sex difference in prevalence of depression in those over age 55" (Bebbington et al., 1998, p. 9). Postmenopausal women, it seems, are not more likely to be depressed than men of the same age. And this could not be explained, Bebbington et al. (1998) claim, by differences in marital status, child care, or employment.

Another, probably not mutually exclusive, proximate factor that can account for the increase in adolescent depression, particularly amongst girls (Thapar et al., 2012), is the difficult changes and challenges in the social and sexual arena (Petersen et al., 1993). The cognitive and affective changes, too, during adolescence (Steinberg, 2005), such as capacities for metacognition, and affective states such as shame (Reimer, 1996), might also play a role in the increase of depression amongst adolescents generally, and amongst girls particularly. There are many other proximate factors involved in the pubertal emergence of the gender difference, and many have been discussed in the literature, such as rumination, temperament, negative life events, and genes (See Hyde et al., 2008, for a good discussion and integration of these). However, we do not know *why* women are more depressed. I will now go on to discuss the predictions on the sex-difference in depression from the Reproductive Failure Theory (Suarez & Gallup, 1985) and from some of the theories that have already been discussed for depression in general: the Social Competition Hypothesis, the Bargaining Hypothesis, the Analytical Rumination Hypothesis and the Social Risk Hypothesis, and, lastly, predictions from Sexual Selection Theory.

Reproductive Failure Theory.

Suarez and Gallup (1985, p. 280) argued that women are oftener depressed because their reproductive stakes are higher:

> The fact that women are normally fertile for only a few days each month, release only a limited number of eggs, and thus have far fewer opportunities to enhance their

fitness than men, who are continuously fertile and produce hundreds of thousands of sperm on a daily basis, may be a contributing factor to the sex difference in the incidence of depression.

Men experience less depression than women, Suarez and Gallup (1985) proposes, because men have more reproductive possibilities throughout life. Suarez and Gallup (1985) also point out that mothers can be certain that their offspring share 50% of their genes, while males, due to cuckoldry and rape, perpetually live under the evolutionary shadow of paternity-uncertainty. Thus, Suarez and Gallup (1985) argue that females are, to a larger degree, able to "measure" their reproductive success at any point in time. Furthermore, "females", Suarez and Gallup (1985, p. 280) reminds us, "bear a much greater biological burden (e.g. pregnancy, childbirth, breastfeeding, and child rearing)". Indeed, maternity-certainty and paternity-uncertainty have been used successfully to test evolutionary predictions on human sex differences in the past (See Buss, 1989, 1994a; Symons, 1979).

Because of women's measurement ability and the biological cost just mentioned, Suarez and Gallup (1985) argue that reproductive failure is more obvious to females, and thus are more prone to depression (see prediction 7.1). Females have more to lose. Moreover, Suarez and Gallup (1985) point out that depression is typically triggered by acute personal crisis such as the loss of a child, spouse, job, home, pet and so on. Presumably, women's evolved depressive mechanisms are more easily triggered after the loss of a child (they do not discuss the other examples in detail), due to menopause and greater biological investment in offspring. Another important prediction from the Reproductive Failure theory (Suarez & Gallup, 1985) is that women who already have children should be less depressed in response to child loss, relative to women who are childless (prediction 7.2). Connolly and Edelmann (1988) used data from an infertility clinic to test prediction 7.1 and 7.2. They found that females scored significantly higher than males on their overall measures of distress after

experiencing a reproductive failure (providing preliminary support for prediction 7.1). However, this finding might simply be due to the fact that women are more depressed in the populations as a whole, not necessarily because of differences in reaction to reproductive failure (Connolly & Edelmann, 1988). The second prediction did not receive support: There was no significant difference in distress between childless women and women with children. This was also the case with the men (Connolly & Edelmann, 1988).

Noteworthy, according to Dominian (1977; cited in Suarez & Gallup, 1985), ovariohysterectomy, the surgical removal of the uterus and ovaries, or, as Suarez and Gallup (1985, p. 281) refers to it "surgically produced menopause", tend to induce depression. This lead Suarez and Gallup (1985) to predict that sterilization induces depression, and that it should be inversely proportional to the number of existing offspring (prediction 7.3). Of course tests of these predictions must control for potential depressive rumination a sterilization procedure might induce because rumination has been found as one mediator for why women are more depressed (Nolen-Hoeksema, Larson, & Grayson, 1999). It remains to be tested whether testicle removal show the same signs in men. Notably, Bailey and Hurd (2005) found that men with more feminine finger length ratios, show a positive correlation with levels of depression, which indicates that lower prenatal exposure to the sex hormone testosterone, is associated with depression in men. Suarez and Gallup (1985) predicts that, depending on family size and availability to resources, pregnancy should counteract depression (prediction 7.4). Although it has been argued that postpartum depression is not a distinct diagnosis (Whiffen, 1991, 1992), and thus might not even exist, it is unclear whether childbirth can *counteract* depression. Even so, they predict that the hospital practice of removing the child from the mother after birth simulates child loss, and hence produce postpartum depression (prediction 7.5). It should be expected that PPD should be more prevalent in hospitals that enforce this practice. Furthermore, Suarez and Gallup (1985)

predict, using a somewhat similar logic as Hamilton's inclusive fitness (Hamilton, 1964), that the severity of a woman's depression at menopause should be greater in childless woman, less so in mothers, and even less in grandmothers (see prediction 7.7). And, we would also expect that, given the lack of future reproductive oppurtunities, menopaused aged-women will experience more depression after child loss, than younger women that still have their reproductive years ahead of them (prediction 7.6).

The ultimate question, in regards to all of the above, still remains: *Why* does the gender difference exist? Suarez and Gallup (1985) postulate two possibilities for the ultimate function of depression.

The first one is a learning capacity. Infant loss, and other forms of reproductive failure, they argue, may have been a byproduct of inappropriate parenting or other behaviors. As such, the individuals that experienced depression will likely not repeat the behaviors that lead to reproductive failure. Depression, here, will function like the nausea and vomiting experienced by an uncareful eater of poisonous food - that food will be avoided in the future.

The second, not mutually exclusive, function Suarez and Gallup (1985, p. 285) propose is that the depressed person "may have been more likely to become the object of aid giving, assistance, and solace, thus partially offsetting the loss". This function is similar to the Bargaining Hypothesis and Social Navigation Hypothesis (Hagen, 2003; Watson & Andrews, 2002).

It is not clear how these functions explain the gender difference in depression, as the authors do not attempt to address this in detail. Presumably, men and women possess the same adaptations for depression, but women have been under heavier selection pressures to have these mechanisms more easily activated by environmental triggers such as the loss of a child. Females' fitness may have suffered more from parenting-mistakes, and have more to gain by help-elicitation from others. Suarez and Gallup provide more predictions in their

paper, which deserve attempts of falsification. The depression experienced after reproductive failure, should be reduced by the reproductive success of one's other children (prediction 7.8), and that foster children, even pets, would activate the mothers' child-rearing mechanisms and hence reduce depression (prediction 7.9). This is an interesting prediction, as it can be tested in a psychotherapeutic context: Misfiring of child-rearing mechanisms (such as pet ownership) relieves depression, particularly amongst women. I am unaware of any direct tests of this, but it has been found (Krause-Parello, 2012, p. 199) that "pet attachment support acted as a coping resource between loneliness and depressed mood". Furthermore, Suarez and Gallup note (as cited in Dominian, 1977) that 37% of the divorces in the United States happened during the menopausal state of the women. This lead them to predict that the divorce rates should be higher in couples who have experienced infertility, miscarriages, stillbirth, neonatal death, or menopause (Suarez & Gallup, 1985) (prediction 7.10). Importantly, Suarez and Gallup do not make it clear *why* depression would help a woman in such situations, is it a manifestation of the first (learning) or second (help-elicitation) ultimate function, or both? Although it is a possibility, there is no direct evidence that depression serve such functions, neither in hunter-gatherer societies or modern environments, for women who have failed reproductively.

Reproductive failure is, of course, by definition selected against in evolution. If depression genes are associated with reproductive failure, why would they prevail? Implicit in Suarez and Gallup's (1985) hypothesis is the fact that the world is unpredictable, and hence a learning mechanism (depression) evolved so as to reduce the chances of repeating behaviors that led to the reproductive loss. Depression is activated in these situations (just as physical pain, depression acts as psychic pain) to avoid repetition of maladaptive behavior.

A last point worth mentioning from Suarez and Gallup (1985) is a phylogenetic one. They predict that the degree to which depressive behavior is displayed after reproductive

failure in a particular species, is dependent on its history of K versus R selection. That is, the quality vs quantity of offspring; higher and lower parental investment (Prediction 7.11). Suarez and Gallup (1985) provide a few interesting examples to their readers. Prosimians (e.g. Lemurs), which fall on the R-selected continuum, largely ignore their dead infants (Cowgill, 1969; as cited in Suarez & Gallup, 1985), while the more K-selected great apes, such as the gorilla and chimpanzee, show more "elaborate depressive behaviors in response to dead conspecifics" (Suarez & Gallup, 1985, p. 284).

The Social Competition Hypothesis and the Sex Difference in Depression

The fact that women are more likely to be depressed than men, seems to contradict the SCH. The SCH postulates that depression is an involuntary losing strategy after agonistic behavior (e.g. fighting). If this is true, it seems unlikely that women have been *more* involved in such situations than men. In the article where the Social Competition Hypothesis is postulated (Price et al., 1994), the sex difference is only briefly mentioned, but they hint at one interesting, novel prediction: When women have equal opportunities (presumably referring to power and social roles), the "female excess of depression disappears" (Prediction 8.1) (Price et al., 1994, p. 312). Their prediction is vague, as "equal opportunities" is not operationalized, or discussed further by Price et al. (1994). If their prediction is true, however, the sex difference in depression should be sensitive to equality in different societies. Hunter-gatherer societies often enjoy a great equality of wealth (Woodburn, 1982), which would lead us to expect that the sex difference would be smaller, or disappear altogether, in these societies (prediction 8.2).

The Analytical Rumination Hypothesis and the Sex Difference in Depression

Andrews and Watson discuss the sex difference in depression in their 2009 article on the ARH (See section on the ARH). Their hypothesis is that social strain can reduce young women's ability to acquire resources, support, and protection. Men, then, have not been under

the same selection pressures, which explain the lower rate of depression. Hence, Andrews and Thomson (2009) point out, it may be adaptive to down-regulate reproductive functioning. This can explain women's reduced fertility and pregnancy-outcomes when depressed. It has been observed too, that counterfactual analysis mediates the sex difference (See Treynor, Gonzalez, & Nolen-Hoeksema, 2003). This suggests that women spend more analytical resources to *prevent* interpersonal problems (Andrews and Thomson, 2009). As mentioned in the introduction to the sex-difference section, the sex difference develops after the onset of sexual maturity, further suggesting it is an adaptation that reliably develops in women when resources, support and protection are needed the most. This leads to the prediction that two risk factors for depression in women is 1. Raising children alone, 2. inadequate resources to raise children (prediction 9.2). This is similar to Hagen's (1999) defection hypothesis (discussed soon). The number of family or friends that helps with raising children, and higher income should decrease the chances of a woman developing a depressive episode (see prediction 9.2). These effects should not be observed in men, or at least, to a smaller degree, because men have probably not raised children alone in the evolutionary past, and hence, have not been under the same selective pressures. Further, Andrews and Thomson (2009, p. 638) hypothesize that women have been under greater selective pressures to avoid exposure to social stress, and hence predict that "women spend more effort analyzing the future consequences of different decisions (promoted by depressed affect) and being vigilant for signs of potential stress (promoted by anxious affect)". Hence, it is predicted that women have been selected to ruminate *more*, than men. Which lead us to expect that women tend to avoid and solve social problems more effectively than men (prediction 9.1).

The Bargaining Hypothesis and the Sex Difference in Depression

Hagen, Watson and Thomson (2004) writing from the perspective of the Bargaining Hypothesis, predict that women are more depressed than men, because women have more

often been victims of social manipulation. This happens, they propose, because women in the EEA have more often resided with their husbands relatives (not their own kin), which has led to more within-group conflict (Hagen et al.) (prediction 10.1). And, they postulate, because physical aggression has been a more costly conflict-resolution strategy for women. For these two reasons, MDD has been a safer but slower strategy for women (prediction 10.2a). Also, women experience more MDD because their reproductive potential is scarce compared to men, which makes it worth the effort to control and obtain resources through the use of MDD. If this is true, women across cultures are expected to actually receive more resources when they are depressed compared to men (prediction 10.2b).

Hagen, importantly, has hypothesized that postpartum depression is a bargaining strategy (Defection Hypothesis).

Postpartum Depression: The Defection Hypothesis

It is costly, both in terms of time and calories, to be pregnant (at least 80000 kcal, see Thomson & Hytten, 1961), and to lactate (A. M. Thomson, Hytten, & Billewicz, 1970). Additionally, human children are extraordinarily dependent on their caregivers. And, unlike many other mammals, humans provide biparental care for their offspring. Given the costs involved, human women are likely to have been under selective pressures to make good reproductive decisions: to weigh the costs and benefits. It is expected, then, that ancestral human mothers have evolved sensitive detection-devices for whether a man will invest in her children. And, evolve a manipulative strategy when the father, or others, indeed do not cooperate by providing resources. This idea Hagen (1999) calls the *defection hypothesis:* Depression is an adaptation that informs mothers that they are suffering a fitness cost. In addition to the Bargaining idea, this is derived from the psychic pain idea, which, similarly to physical pain, tells an organism that “what you are doing can damage your fitness”, hence the mother will take action to decrease the psychic pain. When mothers are *majorly* depressed

postpartum, Hagen (1999) propose that the ultimate function of this is to negotiate greater levels of social support.

Hagen's (1999) ideas are based upon two important aspects of theoretical evolutionary biology. The first is Trivers' (1974) Parental-investment (PI) theory. Parents and children, particularly in species which invest highly in their offspring (traditionally referred to as K-selection, now, more generally as life-history theory), have conflicting needs and priorities. For example, children are selected for acquiring an abundance of resources, for the longest possible time from their parents. Parents, on the other hand, are selected for diverting their resources and attention towards *all* their offspring (and, particularly the children who are most likely to have reproductive success) or other mating opportunities. The benefit-cost ratio differs depending on whether you view it from the parent or the newborns perspective (that is, their genes perspective). Hence, parents do not automatically invest in new offspring: A human mother, and other K-selected species, must balance survival, growth and development on one hand and reproduction at the other (Hagen, 1999). As predicted by PI theory, the food and care supplied to Yanomamö children during a short-term food crisis, caused by the 1998 El Niño, as measured by subcutaneous fat and skin flea infections, were distributed unequally (Hagen, Hames, Craig, Lauer, & Price, 2001). Hagen (1999) points out that PI conflict makes it clear how a mother could, in the absence of resources and infant problems, neglect, abandon or kill her offspring. It does not make clear, however, why these circumstances leads to motherly depression (Hagen, 1999).

Secondly, Hagen (1999) points to Axelrod and Hamilton's (see Axelrod, 2006; 1981) game-theoretic models of cooperation (prisoners dilemma). Their game models show that cooperation can only evolve if it is *possible to defect from it*. Natural selection does not favor blind cooperation - that would leave an organism vulnerable to exploitation. This is where Hagen (1999) draws the analogy to human childrearing: If a woman's costs of raising her

new child exceeds the evolutionary benefits, depression activates a "defection" strategy: cooperate with me or I will stop mothering. The insights from PI theory and the evolution of cooperation, provides a hypothesis-generating framework. Is PPD a strategy for calculating investments in offspring and/or a defection strategy? Although Hagen's (1999) hypothesis is based on sound evolutionary logic, the very existence, as mentioned under the Reproductive Failure theory, of postpartum depression is non-obvious. Indeed, while reviewing the evidence for the role of endocrine factors in PDD, Bloch, Daly and Rubinow (2003) note that there might be hormonal changes during the postpartum period that can trigger depression in a *subgroup* of women (thus probably not part of a universal human nature), and, they also warn that "in the general population, the prevalence of depression in the postpartum period has not been convincingly shown to be increased over that seen in periods unrelated to childbirth". Hence, the phenomenon that the Defection Hypothesis is trying to explain might, in the future, prove to be an example of an adaptive hypothesis which is not only wrong, but attempts to explain something that does not exist. Although PDD is common (13% according to a metanalysis O'hara & Swain, 1996), Whiffen (1992) has argued that PDD is indistinguishable from non-childbearing depression: It is predicted by the same variables (see also Whiffen, 1991). Nevertheless, this issue is still unsettled.

In Hagen's (1999) analysis, motherly depression works as a labor strike (this is also true for the Bargaining Hypothesis generally). The ultimate function Hagen (1999) postulates is that Postpartum Depression (PPD) is designed to reduce or eliminate investments in offspring under certain circumstances, and to negotiate investment from others (Hagen, 1999). Concretely, depression is activated when the mother perceives either 1) a lack of social support and/or 2) infant problems (see prediction 11.1, 11.5, 11.15a and b). Hagen's postpartum theory is similar the Bargaining Theory (Hagen, 2003) and the Social Motivation Function theory of the SNH (Watson & Andrews, 2002), hence, the predictions from those

theories are also relevant for the defection hypothesis, and vice versa. The difference, here, lies in the specificity: The context in which depression is activated (after childbirth).

The Defection Hypothesis predicts four etiological factors for PPD (Hagen, 1999). The first two are straightforward: PPD is induced in a woman if there is insufficient investment from the father or others (prediction 11.1). Hagen (1999) points to two meta-analysis that found "social support", amongst other predictors, to be linked with PDD (Beck, 1996; O'hara & Swain, 1996). The second etiological factor Hagen (1999) predicts is that PPD is activated when infant liability is low. That is, if there are problems with pregnancy, birth or with the infant itself (See predictions 11.2 and 11.6). Adverse environmental conditions such as insufficient resources, should also induce PDD in mothers, and also increase the chance that mothers kill their infants (prediction 11.2, 11.3, 11.6). The last etiological factor Hagen (1999) postulates regards opportunity costs. Rather than investing in a newborn, a mother might have more to gain by 1) investing in existing offspring, 2) the mothers own survival (mothering can be dangerous, especially if resources are scarce) or 3) finding a better mate. Hagen (1999) therefore predicts PDD will be activated in these situations (prediction 11.4).

Hagen (1999) provides us with more predictions for testing the defection hypothesis. Some expand on the etiological factors sketched above: Lack of social support, low infant viability and poor environments should predict negative affect (prediction 11.5, 11.6 & 11.7, respectively). Hagen conducted a study (2002) in which he tested some of the predictions from the defection hypothesis. He predicted that mothers who face social constraints on abortion from their husband—mothers unable to simply defect from their pregnancy—would be at greater risk for PPD. This was supported. Also, as predicted, husbands constrains on abortion correlated with the mothers PDD. Hagen (2002) also found support for his prediction regarding fathers: sexually successful men, but not women, are at risk for PPD

under socially imposed monogamy, even when controlling for relationship quality (Hagen, 2002). This suggest that men with other sexual opportunities are at risk for PPD (although maternal depression has been found to be the best predictor of paternal postpartum depression Goodman, 2004). Hagen (2002) also found support for the prediction that fathers PPD levels correlated positively (rs = .35) with mothers investment in childcare. Mothers PPD levels, too, were correlated (r = .41) with increased investment by fathers. This suggests that both mothers and fathers can use depression as a bargaining strategy postpartum: Depression might increase investment in the non-depressed partner. Furthermore, Hagen (2002) found that women over 35 with one child, had fewer postpartum symptoms than women under 35 with one child, supporting the notion that women with reproductive possibilities in the future have more to gain by defecting from parenting (also consistent with the Reproductive Failure theory, prediction 7.13). Noteworthy, Hagen's (2002) study found correlations, other tests of the Defection Hypothesis predictions might be able to falsify the causal factors. Hagen (1999) is not clear on when the Defection Hypothesis predicts fatherly depression. If postpartum depression turns out to be just as likely in men, or show the same sex-ratio as the general population, the Defection Hypothesis alone cannot explain why women are more depressed in general. That is, depressions unrelated to childbirths.

Importantly, Hagen (1999) also predicts that PPD should be cross-culturally universal (prediction 11.8). Nancy Howell's (1979/2000) observation that some hunter-gatherer !Kung women experience postpartum depression and anxiety, might provide some preliminary support for this. Additionally, Hagen and Barrett (2007) asked Shuar mothers (from a village in the Ecuadorian Amazon) about sadness during pregnancy. They reported several reasons for being sad (see table 3 and 5 in Hagen and Barret, 2007 for details): "problems with relationship", "husband left" (lack of social support); "poor health prior to and during pregnancy" (poor maternal health); "didn't want child because got pregnant too soon due to

short interbirth interval" (oppurtunity costs).

Hagen (1999) also emphasizes that PPD should not be a hormonal byproduct (prediction 11.9). An adaptation, of course, is expected to have hormonal, neural, or other physiological correlates. The presence or absence, therefore, of particular hormonal activity in depressed mothers is neutral to the Defection Hypothesis (Hagen, 1999).

If the defection hypothesis is correct, an *investing father* and a *healthy infant* should dramatically decrease the risk for PPD (see prediction 11a & b). As a logical extension, PPD should be more prevalent in societies with more single-moms, especially where they do not receive monetary benefits from the state (or anyone else) (prediction 11.16).

The Social Risk Hypothesis and the Sex Difference in Depression

Allen and Badcock (2003) suggest that the increase in depression amongst women around puberty is explained by social-cognitive developments that occur in early adolescence. For example, they suggest, it is possible that women develop a capacity for empathic accuracy or shame, more than it has to do with "strictly biological maturation" (Allen & Badcock, 2003, p. 906). Although this produces useful predictions on proximate factors, Allen and Badcock (2003) are not clear on *why* women would develop more accurate shame and empathic tendencies, not how it is evolutionary useful, let alone why shame and empathy should be *more* useful for women than for men.

Allen and Badcock (2003) share the view of McGuire and Troisi (1998; 2001) who have proposed that the gender difference in depression can be explained by the gender difference in the attainment, and prioritization of biological goals (such as reproduction, survival, resource acquisition, kin investment), and the stress that is caused by impediments of these goals. The capacity to achieve biological goals without social assistance, Allen and Badcock (2003) argue, is greater for males than females, which "should place a greater selective pressure on females to maintain a more risk-averse approach to many types of

relationships, including but not limited to mating relationships" (p. 904) (see prediction 12.1a-c). This predicts that females are more sensitive to social threats (prediction 12.2). This presumes that depressed behavior—risk-averse strategies—actually help women attain their biological goals to a greater degree than what risk-prone strategies do. And, also, that this has been *particularly useful* for women during the EEA, which explains why women are more often depressed.

The evolved modules of the mind, are likely to be highly specialized, and content-specific (Barkow et al., 1992; Barrett & Kurzban, 2006; Kurzban, 2012). Biological goals, then, such as resource acquisition, are unlikely to be as general as Allen and Badcock (2003) describe. Nevertheless, they might have been important differential selection pressures in these domains, and testing of prediction 12.1a-c and 12.2, might illuminate the nature of the gender difference in sadness and depression.

Sexual Selection Theory and the Sex Difference in Depression

It has been argued that differential sexual selection pressures on women and men during evolutionary history shape the emergence of "internalizing" disorders, such as depression and anxiety, in adolescent girls, and "externalizing" disorders, such as ADHD, during childhood in boys (Martel, 2013). The average sex differences in dispositional traits, which have likely been shaped by sexual selection, such as sensation-seeking (more in males Cross, Copping, & Campbell, 2011) and neuroticism (more in females Jorm, 1987) might interact with risk-alleles such as the 5-HTT for depression in producing depression (Martel, 2013) (prediction 13.1 and 13.2). Intrasexual selection during females evolutionary history regarding mate-choice, social support, pregnancy and child-rearing, might have equipped women with a sensitivity to the cohesion of interpersonal relationships (Martel, 2013) (see prediction 13.4a). The sexually selected sex-specific genes, then, which produce sex-dependent exposure to sex hormones during development and during puberty might produce

psychological mechanisms that process information from the environment differently—sensitivity to interpersonal stress, for instance—depending whether you are a girl or a boy (prediction 13.4b). Which, in turn, it is predicted, contribute to the group-level sex difference of depression in adulthood. Hence, depression, at least in severe forms is a dysfunction in this view: Adaptive sexually selected traits interact with genetic vulnerability and developmental stressors to produce depression in some people, particularly in women.

Side-effect Theories

Infection-Prevention and Depression

According to Raison and Miller's (2012) Pathogen Host Defense Hypothesis (PATHOS-D) depression exists because it shields from pathogenic attacks. PATHOS-D shifts the "adaptive context of depression risk alleles from relations with conspecifics to relations with the microbial world" (p. 1). Raison and Miller (2012) collected genetic data from PubMed and Ovid MEDLINE and found support for their hypothesis: Several risk alleles for depression contribute to immunological and behavioral defenses from pathogens. They point to several genes that are linked to both depression and immunological functions. The PATHOS-D hypothesis predicts that allelic risk variants for depression should be located in genes with known immune effects (prediction 14.1a), increase signaling in inflammatory defense pathways (prediction 14.1b), and increase survival in the context of infection (prediction 14.1c).

To humans, pathogens are deadly predators. They are microorganisms that, due to short generational life-span, rapidly evolve sophisticated methods of securing a place within their host and spreading to others. These invasive intruders have, and still do, play a lead role in the evolution of the mammalian immune system. In fact, DNA sequences for host defense existed before the plants and animals diverged (Janeway, Travers, Walport, & Shlomchik, 2001) 1576 ± 88 million years ago (Wang, Kumar, & Hedges, 1999). Like the ever-evolving

swiftness of the attacking lion and fleeing gazelle, pathogens and immune systems are in an evolutionary arms race, giving rise to complex adaptations at both sides. Before humans invented effective defenses like vaccines, antibiotics and water treatments, bacteria and viruses were even more dense and dangerous. Indeed, as Jared Diamond (1997) describes in *Guns, germs and steel*, pathogens have claimed more lives than any war, and have profoundly shaped human history.

Interestingly, and consistent with PATHOS-D, the short version of the previously mentioned 5-HTTLPR gene protects against sudden infant death syndrome, which has been associated with death caused by infection (See Opdal, Vege, & Rognum, 2008). Furthermore, the frequency of the short allele around the world correlates with the historical burden of disease-causing pathogens in that area (See M. Murray & Murray, 1979; Raison & Miller, 2012). Despite the manifold way that, as Raison and Miller (2012, p. 1.) argue, "depression impairs Darwinian fitness", the depressive risk-alleles have not been wiped out by natural selection because these genes also code for the immunological and behavioral responses to infection from pathogens.

The average fitness benefits that the risk-alleles provide in fighting pathogens, Raison and Miller argue, are greater than the cost of depression. Moreover, the immune system is not fully developed during infancy and infection has been the primary cause of death for infants during evolutionary history (Raison & Miller, 2012). This produces even stronger selection pressures for pathogen defense: Death is particularly detrimental to fitness at prepubescent age. The failure to survive infancy is an evolutionary catastrophe, adult melancholy a darwinian misfortune.

This is a vital point in PATHOS-D: Risk-alleles for depression are adaptive not because of any purpose sadness *per se* may serve (indeed, despite of), but because the risk-alleles *also* code for pathogen host defense. Thus, risk-alleles influence on social functioning

are considered side effects. This raises the question of why genes for depression and host defense evolved to be connected in the first place. Raison and Miller (2012, p. 9) address this issue and suggest that there is no a priori "reason why these antipathogen effects should overlap with the depressogenic effects of these risk alleles. That they do so is powerful evidence, we would suggest, for the primacy of immune defense in the pathogenesis of depression, regardless of the environmental adversity that initiates the disorder in individual cases". Other theorizers (Anders, Tanaka, & Kinney, 2013) on depression and infection-prevention, have suggested that depression itself might be linked to the "behavioral immune system" (see Schaller & Duncan, 2007; Schaller & Park, 2011), and hence do not consider depression as merely a side-effect of pathogen defense, but an adaptive defense in its own right. This does not contradict Raison and Miller's (2012) hypothesis, as they also emphasize the adaptive behavioral role that depression might have, but Anders, Tanaka and Kinney (2013) and Kinney and Tanaka (2009) also stress the cognitive factors (such as lack of concentration) as part of the adaptive response.

Raison and Miller (2012) presents four ways elements of depression are adaptive: 1) Fever and hypoferremia (iron-depletion), 2) conservation withdrawal, 3) hypervigilance and, 4) anorexia. As all four are relevant for understanding the adaptive arguments in PATHOS-D, I discuss them below, along with some of the evidence.

The mammalian inflammatory response produces a set of symptoms known as *sickness behavior* (See Dantzer & Kelley, 2007), this includes fever and hypoferremia (iron-deficiency). As exemplified in the introduction, fever and iron-depletion are part of an adaptive central motivational state evolved to promote pathogen defense (See Kluger et al., 1998; Raison & Miller, 2012). Fever is induced by inflammatory mechanisms, but the elevated body-temperature itself hinders further replication of pathogens, decreasing the chances of death. Raison and Miller (2012) argue that "if depressive symptoms aid in

pathogen defense, and fever and hypoferremia are important in this regard, one would expect individuals with MDD to have elevated temperatures and reduced bodily stores of iron, even in individuals with no evidence of an infectious process" (p. 9) (prediction 14.5a). As the authors reference themselves, there is indeed evidence that patients with depression have elevated body temperatures (Rausch et al., 2003) and reductions in bodily iron storage (Maes et al., 1996). Raison and Miller (2012) point out that PATHOS-D predicts these findings and that their absence would strongly argue against its validity. As they put it, referring to iron depletion and fever: "… If depression is simply a nonadaptive phenomenon, why would such ancient, highly conserved and highly complex physiological responses be a hallmark of the disorder?" (p. 9). Both non-adaptive theories and theories focusing on social benefits of depression, fail to explain these findings. Perhaps unsurprisingly, Raison and Miller (2012) think of symptoms of sickness and depression (particularly MDD) as largely overlapping concepts. That is, as symptoms promoted by the mammalian inflammatory response under infection. Although the distinction still remains unclear, the similarity of MDD and sickness behavior has likely not escaped clinicians notice.

As well as fever and hypoferremia, proinflammatory cytokines induces *conservation-withdrawal*, this is the behavioral effect of the sickness behavior: depressed mood, anhedonia (inability to experience pleasure), psychomotor retardation, fatigue, social avoidance and anorexia (Dantzer, O'Connor, Freund, Johnson, & Kelley, 2008; Raison & Miller, 2012). In addition to the well-established purpose of allocating metabolic resources to tissue repair and fever-generation during infection, Raison and Miller (2012) postulate that conservation-withdrawal is adaptive by attenuating infectious exposure by reducing interpersonal contact. This is also a primary focus of Kinney and Tanaka (2009). This is in direct contrast to the Bargaining Hypothesis which predict that family-members are forced to help a depressive (which presumably involves some interpersonal contact). The typical sickness *behaviors* are

also considered adaptive under the PATHOS-D perspective. Using the logic of Hamilton's (1964) inclusive fitness, Raison and Miller (2012) suspect that social withdrawal enhances fitness by reducing the risk of infecting kin. Better to—speaking from the 'selfish' gene's point of view—leave some copies of myself in the bodies of kin, than risk death from infectious diseases of every copy. Some preliminary support for this might come from a study which found that participants injected with proinflammatory cytokines experienced feelings of social disconnection (Eisenberger, Inagaki, Mashal, & Irwin, 2010). This suggests a two-way causality between inflammation and depressive thought. Also noteworthy, Kinney and Tanaka (2009) hypothesize not only that withdrawal behaviors reduce the risk of infecting kin, but that it *signals* to family members that "the infected individual is potentially contagious, so kin should minimize contact with the individual" (p. 2) (see prediction 14.12a and b). Importantly, Kinney and Tanaka (2009) also hypothesize why sexual inhibitions are often seen in depressed patients. Loss of sexual desire, they claim, hinders exposure to sexually transmitted pathogens (prediction 14.13).

The third 'survival promoting' aspect of depression and sickness behaviors is hypervigilance. Challenging the notion of conservation-withdrawal, MDD patients often have metabolically expensive symptoms such as anxiety and insomnia (Raison & Miller, 2012). This is paradoxical. Why would a depressed patient, with activated pathogenic defense systems, also show costly symptoms of agitation and sleeplessness? Raison and Miller (2012, p. 11) contend that "inflammatory processes-especially when chronic—might promote hypervigilant behavior that, while shunting energy away from fighting infection, would nonetheless serve adaptive purposes by protecting against environmental dangers engendered by sickness". In other words, agitation is activated under chronic, long-term inflammation (prediction 14.5b). According to Raison and Miller (2012) this allows the infected person to pursue reproductively relevant resources (mates, children, food acquisition) before it is too

late. Along with colleges, Raison (2010) and Miller themselves conducted a study where patients infected with the Hepatitis-C virus were administered interferon-alpha (to promote inflammatory responses) over 12 weeks. Supporting their notion, patients in the treatment group, measured at 12 weeks, showed an increase in wake after sleep onset (WASO), decreased sleep efficiency and reduced stage 3/4 sleep. This suggests that hypervigilant behavior, at least in terms of wakefulness, kicks in after prolonged secretion of pro-inflammatory cytokines.

Anorexia, lastly, is thought to enhance survival during infection by spending available metabolic energy on inflammation and fever rather than food procurement and digestion (see prediction 14.5c). Additionally, Raison and Miller (2012) postulate, it limits the exposure to food-borne pathogens. In support of their proposition they cite Murray and Murray (1979) who force-feed infected mice and found increased mortality rates.

The PATHOS-D framework can explain why depressive symptoms, including MDD, are associated with inflammatory responses. Additionally, Raison and Miller (2012) argue, it explains why the depression-alleles have not spread to every human being (fixation). Because inflammation-causing alleles can, among other dangers, cause tissue damage and chronic disease, they are expected to show an intermediate prevalence reflecting their benefit and costs in different environments.

There are some noteworthy limitations of the PATHOS-D perspective. If depression is a side effect of inflammatory responses to infection, why are psychosocial stressors predicting depressive episodes? Raison and Miller (2012, p. 7) propose that this link exists because the “vast majority of stressors in mammals over evolutionary time boiled down to risks inherent in hunting, being hunted or fighting conspecifics in dominance hierarchies for reproductive access/status, it is not surprising that these states are also circumstances in which the risks of pathogen invasion-and subsequent death from infection-was greatly

increased as a result of traumatic opening of the protective skin barrier from wounding". Thus, the PATHOS-D model predicts that the long-lasting statistical relationship between the fight/flight response and open wounds, created an evolutionary shotgun-solution: Inflammatory cytokines are released whenever there is a stressor in the environment (see prediction 14.4 and 14.10). Raison and Miller (2012) do not make it clear what "psychosocial stress" entails. Presumably, the PATHOS-D predicts that the reduced mood observed in humans after "psychosocial stress" such as the loss of a mate or family member are to be considered side-effects of pathogen defense. This weakness propagates: Raison and Miller typically examine MDD, without explicitly discussing whether their hypothesis also attempts to explain the existence of low-mood, chronic depression (dysthymia), uni and bipolar depression, and so on. Further, inflammatory markers are not present in all patients diagnosed with MDD. Raison and Miller (2012) suggests that this might be because MDD patients with elevated inflammation represent a distinct evolutionary subset in which selection for host defense happened primarily for individuals with the risk-alleles. The case is clearly not settled here. Also, noting that mania and psychosis are associated with depression, Raison and Miller (2012) find it hard to imagine that these states are adaptive for pathogen host defense. I find it hard to disagree. It is worth noting, too, that depressive episodes have been connected to cognitive impairment (Austin et al., 2001; Hammar & Årdal, 2009), and that some impairment remains even six months after the depressive symptoms have lifted (Hammar et al., 2003). These findings are equally hard to prescribe an adaptive function in this perspective. However, it could be argued that even though depression can cause, or at least is related to, cognitive impairment, the fitness benefits strengthening host defense might outweigh any costs of impaired cognitive abilities. In the infection-prevention perspective it is unclear too (not even mentioned), why women would be more depressed than men.

All of the findings above shed valuable light on the validity of the PATHOS-D

hypothesis. Nevertheless, it is still necessary that skilled researchers and clinicians design experiments to test predictions explicitly derived from the PATHOS-D. Relying on existing data that fits the hypothesis is not sufficient; many hypothesis are easily verified, fewer stand after attempts of falsification.

Nonadaptive Theories

Nettle's Affective Reactivity Model

Nettle's (2004) model is simple: complex, fitness relevant traits are likely to vary between people. Hence, there is an adaptive peak (a normal distribution of fitness) where most individuals have the perfectly functioning or close-to-perfectly-functioning adaptive trait, while some individuals fall on both extremes of the optimal range. Nettle (2004, p. 98) provide the example of height in men, based on some of his earlier work (Nettle, 2002): "reproductive fitness increases steeply with increasing height, until a certain point, above which the prevalence of musculo-skeletal and other health problems becomes very much greater". Nettle (2004) argues that this is true also of the personality trait neuroticism, which is strongly associated with depression: "increasing neuroticism is selected for, because of its beneficial effects on striving in interpersonal contexts, until the point where the negative effects of mental and physical illness outweigh the marginal benefits". In this view, depression (at least severe) is maladaptive variation. If this hypothesis is true, we would expect that many people suffer fitness-cost caused by too little, or too much neuroticism (prediction 15.1).

Mutation-Selection Balance and Major Depression.

All the complex behavioral traits that vary amongst humans (e.g. personality, intelligence) that have been studied so far, are heritable to some degree (Turkheimer, 2000). Behavioral genetics can explain why humans differ, but its research methods (e.g. twin studies) is limited because they cannot explain universal human nature (Pinker, 2003). The

study of universal human nature has been the purview of evolutionary psychology. As we have seen, species-typical adaptations are, of course, heritable, but most adaptations have low "heritability", because there is no genetic or phenotypic variation in the population, and hence, there are no particular genes that can correlate with variance in particular traits. The phenotypic variation that we observe amongst humans can be caused by mutations, and mutations are common and often harmful, as Keller (2007, p. 6) writes:

> most people reading this chapter carry one or more new mutations that impair fitness, that pervade every cell in the body, and that were not inherited from either parent. Sometimes these mutations are catastrophic to the phenotype, causing, for example, skull malformation and digit fusion (Apert's syndrome) or short-limbed dwarfism (Achondroplasia). But most new, deleterious mutations have minor, perhaps unnoticeable, phenotypic effects, such as causing one to be a little less bright, attractive, or athletic.

Even if deleterious mutations have a very small effect on fitness, it is evolutionary relevant (Keller, 2007): depending on how much, or little, the mutation effects reproductive rates, it will—and this is only a matter of time—disappear from the population (Garcia-Dorado, Caballero, & Crow, 2003; John Maynard Smith, 1993). This is why inbreeding is maladaptive: If individuals with the same mutations reproduce, the mutation rates are likely to clump up in their offspring, which makes them vulnerable to disorders (Miller, 2001).

Can mutations explain why major depression exists? Many genes affect the human nervous system, and mutations are relatively common, and heritable—which makes it inevitable that some mutation will affect the brain, and hence, produce phenotypes which we recognize as harmful mental disorders. This leads, Keller and Miller (2006) to argue that mutation-balancing selection best explains common and heritable mental disorders, including major depression. It is important to acknowledge that Keller and Miller (2006) mean *major*

depression. Indeed, Keller & Nesse (2005) have argued that humans are capable of low-mood because it has been evolutionary useful, and is activated by adverse situations. If this is true, we would expect low-mood to be universal (the genes involved in its development have fixated), and it is plausible that it would prove to be universal. However, if *major depression* was an adaptation, we would expect the same thing: no heritability, and context-sensitivity. This is not the case—major depression is heritable (Sullivan, Neale, & Kendler, 2000), which fits well with Keller and Miller's (2006) mutation-selection analysis. Applied to human mental disorders, mutation-selection models imply that "if a mental disorder appears maladaptive, maybe it really is maladaptive - and always have been" (Keller & Miller, 2006, p. 397). This line of reasoning fits well in the Harmful Dysfunction analysis: Psychological adaptations can malfunction, for example due to heritable mutations (and if it is also judged as *harmful* by the individual, or the culture at large, it would be considered a disorder in the Harmful Dysfunction analysis). Keller and Miller's (2006) view contradicts hypotheses such as the Bargaining hypothesis and Analytical Rumination Hypothesis, where major depression is seen as adaptive in certain situations. Even though all evolutionary theorists agrees that any complex adaptation (including psychological ones) can dysfunction (Horwitz & Wakefield, 2007), Keller and Miller's (2006) analysis is the first to explicitly discuss mutations, and reconcile the potential adaptations of low mood and dysfunctional depression with modern behavioral genetics. The heritability of major depression requires an explanation, and Keller and Miller (2006) argue that mutation-selection is a good candidate for this. For example, it has been shown that unipolar depression (and stroke, epilepsy, cancer and schizophrenia etc.) is more common in genetically isolated islands (due to inbreeding) (Rudan et al., 2003). Furthermore, major depression, and other psychiatric conditions, is much more common in patients with traumatic brain injuries, than the general population (Rao & Lyketsos, 2000; Schoenhuber & Gentilini, 1988). Higher incidences of major depression due to inbreeding

and brain damage strongly suggest that major depression is a dysfunction. If major depression is an adaptation produced by selection (and depression's risk-alleles hence are maintained by balancing selection, or on their way to fixation), depression is not expected to work better after brain injury. This is of course true of any complex adaptive trait, as Keller and Miller (2006, p. 401) put it "receiving a blow to the head, for example, should not lead to higher intelligence or attractiveness". If the mutation-selection balance perspective is true, we would expect that individuals with major depression show evidence of a high mutational load in their phenotype, such as bodily asymmetry (prediction 16.1) (this has been found in schizophrenia, see Yeo, Gangestad, Edgar, & Thoma, 1999), we would also expect that the children of individuals with major depression are more likely to have mutation-related disorders, especially if both parents have major depression (prediction 16.2).

Keller and Miller (2006) provide an important point of view, which has largely been ignored by the authors of adaptive theories. Adaptive theories have, often implicitly, assumed that the risk-alleles for major depression were neutral amongst our ancestors (ancestral neutrality model) or maintained by balancing selection (frequency-dependency), or ignored the heritability problem altogether. Keller and Miller focus on the role of many, minorly damaging mutations (which ultimately affects phenotype and fitness), and how these might be connected to heritable and harmful mental disorders such as major depression, bipolar disorders and schizophrenia. Keller & Miller, of course, are *not* saying that mutations explain all instances of normal sadness and low-mood. Rather, if low-mood is an adaptation (which it, like other emotions, probably is), mutations might explain when someone *fail* to be sad in situations where they are expected to be sad (like someone who fails to be anxious when they are being attacked by a predator—the adaptation is not working). Hence, Keller and Miller's analysis might prove to illuminate the dysregulation view that Nesse (2000), Gilbert (1992) and others have suggested for major depression: At low levels, reactive sadness and

depression are likely to be part of an evolved human nature. But it can, like any complex adaptation, including emotional ones, become dysfunctional, either by genetic mutations or developmental disruptions. Thus, it is not surprising that mutations can cause mood disorders. Future research must address how normal, reactive sadness develops and works. This will probably require both the lens of evolutionary psychologists (test hypotheses on universal human nature) and behavioral geneticists (explain *differences*, rather than similarities, between people) (Miller, 2010; Pinker, 2003). Although evolutionary psychology must, and has indeed begun (e.g see Buss, 2009; Buss & Hawley, 2010), to combine the two into a scientific marriage of both universals and individual differences in human nature. Both of these fields are likely to further our understanding of why normal sadness exists, and why and when this emotion turns into a dysfunction.

Conclusion

In this article I have discussed evolutionary theories on the ultimate function of depression and why it differs between the sexes. That is, the theories that attempts to provide testable answers to the question "why does depression exist?" and "why are there sex differences in depression?" Many of the theories overlap and share ideas (e.g. the "social" theories), while some directly contradict each other, such as the Analytical Rumination Hypothesis and the Mutation-selection balancing view, or the Bargaining Hypothesis and the PATHOS-D. This may be caused by several things. For example, the complexity of depression suggests that depression is a multifaceted phenomenon, which might make it unreasonable to put it on *one* continuum. The severity of depression, too, such as mild and major, might entail qualitatively different states (Wolpert, 2006). And depression could also be best understood as different taxonomies of functional depression (Kennair, 2003)—depressed mood can be multimodular. If depressed mood is an adaptation, it might have several functions, and, if depressed mood is a dysfunction, it might dysfunction in several

discrete ways. Hence, one ultimate theory might be unable to explain why all forms of depression exist.

It seems likely that major depression, as suggested by the mutation-selection, affective reactivity, and dysregulation models, is a dysfunction, and not an adaptation. The adaptation concept may have been put to use prematurely by some theories. Nevertheless, the evolved function, if any, of normal sadness and milder forms of reactive depression is not yet understood either, basic research on depressed mood will inform our understanding of pathological mood.

I hope empirical tests of the predictions in the appendix in this paper could serve as an empirical kickstart in furthering our understanding of why depression exists. Especially if researchers test the predictions by using many different methods from fields such as anthropology, paleoarcheology, biology, genetics, computer science, cognitive science, medicine and cognitive neuroscience. The field of evolutionary psychopathology is clearly not testing its ideas sufficiently; this field will only prove to be as useful as the empirical research it produces. We still do not know why depression exists. Fortunately, predictions exist and they have to be tested empirically. I am confident that tests of the evolutionary theories on depression will also inspire new scientific questions through serendipity.

It is reasonable to conclude that the evolutionary lens is necessary when fully trying to understand when, how and why the functions of the mind fails to perform optimally, and furthermore, when the mind is functioning as designed. In the future, when mental health researchers are armed with a high-resolution map of the evolved cognitive and emotional architecture of human nature, we will be better equipped to understand why sadness and depression are prevalent, how they are triggered, and when it becomes a dysfunctional disease for individuals. This understanding is important not only for developing better psychotherapeutic and pharmacological treatments, but also for understanding why the

human capacity for sadness, ranging from the everyday-blues to full-blown major depression, exist in the first place; to expand our understanding of human nature.

References

Ackermann, R., & DeRubeis, R. J. (1991). Is depressive realism real? *Clinical psychology review, 11*(5), 565-584.

Adrian, J. A. L., Deliramich, A. N., & Frueh, B. C. (2009). Complicated grief and posttraumatic stress disorder in humans' response to the death of pets/animals. *Bulletin of the Menninger Clinic, 73*(3), 176-187.

Allan, S., & Gilbert, P. (1995). A social comparison scale: Psychometric properties and relationship to psychopathology. *Personality and Individual Differences, 19*(3), 293-299.

Allen, N. B., & Badcock, P. B. (2003). The social risk hypothesis of depressed mood: evolutionary, psychosocial, and neurobiological perspectives. *Psychological bulletin, 129*(6), 887.

Alloy, L. B., & Abramson, L. Y. (1988). Depressive realism: Four theoretical perspectives. In L. B. Alloy (Ed.), *Cognitive processes in depression* (pp. 223-265). New York: Guilford Press.

American Psychiatric Association. (2013). *The Diagnostic and Statistical Manual of Mental Disorders: DSM 5*. Arlington, VA: American Psychiatric Publishing.

Anders, S., Tanaka, M., & Kinney, D. K. (2013). Depression as an evolutionary strategy for defense against infection. *Brain, behavior, and immunity, 31*, 9-22.

Andrews, P. W., Aggen, S. H., Miller, G. F., Radi, C., Dencoff, J. E., & Neale, M. C. (2007). The functional design of depression's influence on attention: A preliminary test of alternative control-process mechanisms. *Evolutionary Psychology, 5*(3), 584-604.

Andrews, P. W., & Thomson, A. J. (2009). The bright side of being blue: depression as an adaptation for analyzing complex problems. *Psychological review, 116*(3), 620-654.

Andrews, P. W., Thomson, A. J., Amstadter, A., & Neale, M. C. (2012). Primum non nocere: an evolutionary analysis of whether antidepressants do more harm than good. *Frontiers in psychology, 3*, 117.

Au, K., Chan, F., Wang, D., & Vertinsky, I. (2003). Mood in foreign exchange trading: Cognitive processes and performance. *Organizational Behavior and Human Decision Processes, 91*(2), 322-338.

Austin, M.-P., Mitchell, P., & Goodwin, G. M. (2001). Cognitive deficits in depression Possible implications for functional neuropathology. *The British Journal of Psychiatry, 178*(3), 200-206.

Axelrod, R. (2006). *The evolution of cooperation: revised edition*. New York: Basic Books.

Axelrod, R., & Hamilton, W. D. (1981). The evolution of cooperation. *Science, 211*(4489), 1390-1396.

Badcock, P. B., & Allen, N. B. (2003). Adaptive social reasoning in depressed mood and depressive vulnerability. *Cognition & emotion, 17*(4), 647-670.

Badcock, P. B., & Allen, N. B. (2011). Evolution, Social Cognition, and Depressed Mood. Exploring the Relationship Between Depression and Social Risk Taking. In J. P. Forgas, M. G. Haselton & W. v. Hippel (Eds.), *Evolution and the social mind: Evolutionary psychology and social cognition* (pp. 125-145). New York: Psychology Press.

Bailey, A. A., & Hurd, P. L. (2005). Depression in men is associated with more feminine finger length ratios. *Personality and Individual Differences, 39*(4), 829-836.

Barkow, J. H., Cosmides, L., & Tooby, J. (Eds.). (1992). *The adapted mind: Evolutionary psychology and the generation of culture*. New York: Oxford University Press.

Baron-Cohen, S. (1997a). *The maladapted mind: Classic readings in evolutionary psychopathology*. Hove: Psychology Press.

Baron-Cohen, S. (1997b). *Mindblindness: An essay on autism and theory of mind*. Cambridge, MA: MIT press.

Baron-Cohen, S., Leslie, A. M., & Frith, U. (1985). Does the autistic child have a "theory of mind"? *Cognition, 21*(1), 37-46.

Barrett, H. C. (2012). A hierarchical model of the evolution of human brain specializations. *Proceedings of the national Academy of Sciences, 109*(Supplement 1), 10733-10740.

Barrett, H. C., & Kurzban, R. (2006). Modularity in cognition: framing the debate. *Psychological review, 113*(3), 628.

Baumeister, R. F., & Leary, M. R. (1995). The need to belong: desire for interpersonal attachments as a fundamental human motivation. *Psychological bulletin, 117*(3), 497.

Bebbington, P. E., Dunn, G., Jenkins, R., Lewis, G., Brugha, T., Farrell, M., & Meltzer, H. (1998). The influence of age and sex on the prevalence of depressive conditions: report from the National Survey of Psychiatric Morbidity. *Psychological medicine, 28*(1), 9-19.

Beck, C. T. (1996). A meta-analysis of predictors of postpartum depression. *Nursing research, 45*(5), 297-303.

Benziger, D. P., & Edelson, J. (1983). Absorption from the vagina. *Drug metabolism reviews, 14*(2), 137-168.

Bernstein, H.-G., Klix, M., Dobrowolny, H., Brisch, R., Steiner, J., Bielau, H., . . . Bogerts, B. (2012). A postmortem assessment of mammillary body volume, neuronal number and densities, and fornix volume in subjects with mood disorders. *European archives of psychiatry and clinical neuroscience, 262*(8), 637-646.

Bielau, H., Trübner, K., Krell, D., Agelink, M. W., Bernstein, H. G., Stauch, R., . . . Bogerts, B. (2005). Volume deficits of subcortical nuclei in mood disorders. *European archives of psychiatry and clinical neuroscience, 255*(6), 401-412.

Bloch, M., Daly, R. C., & Rubinow, D. R. (2003). Endocrine factors in the etiology of postpartum depression. *Comprehensive Psychiatry, 44*(3), 234-246.

Boakes, R. A. (2007). Self-starvation in the rat: running versus eating. *The Spanish journal of psychology, 10*(2), 251-257.

Bobrow, D., & Bailey, J. M. (2001). Is male homosexuality maintained via kin selection? *Evolution and Human Behavior, 22*(5), 361-368.

Bostwick, J. M., & Pankratz, V. S. (2000). Affective disorders and suicide risk: a reexamination. *American Journal of Psychiatry, 157*(12), 1925-1932.

Bower, G. H. (1981). Mood and memory. *American Psychologist, 36*(2), 129.

Bowlby, J. (1980/1996). *Loss: Sadness and depression*. London: Random House.

Boyce, P., & Mason, C. (1996). An overview of depression-prone personality traits and the role of interpersonal sensitivity. *Australasian Psychiatry, 30*(1), 90-103.

Brüne, M. (2008). *Textbook of evolutionary psychiatry: The origins of psychopathology*. New York: Oxford university press, inc.

Buervenich, S., Carmine, A., Arvidsson, M., Xiang, F., Zhang, Z., Sydow, O., . . . Ross, R. G. (2000). NURR1 Mutations in cases of schizophrenia and manic-depressive disorder. *American journal of medical genetics, 96*(6), 808-813.

Burch, R. L., & Gallup, G. G. (2006). The psychobiology of human semen. In S. M. Platek & T. K. Shackelford (Eds.), *Female Infidelity and Paternal Uncertainty.*

Evolutionary Perspectives on Male Anti-Cuckoldry Tactics (pp. 141-172). Cambridge: Cambridge University Press.

Buss, D. M. (1989). Sex differences in human mate preferences: Evolutionary hypotheses tested in 37 cultures. *Behavioral and brain sciences, 12*(1), 1-49.

Buss, D. M. (1990). The evolution of anxiety and social exclusion. *Journal of Social and Clinical Psychology, 9*(2), 196-201.

Buss, D. M. (1994a). *The evolution of desire: Strategies of human mating*. New York: Basic books.

Buss, D. M. (1994b). The strategies of human mating. *American Scientist*, 238-249.

Buss, D. M. (1995a). Evolutionary psychology: A new paradigm for psychological science. *Psychological inquiry*, *6*(1), 1-30.

Buss, D. M. (1995b). Psychological sex differences: Origins through sexual selection. *American Psychologist*, *50*(3), 164-168.

Buss, D. M. (2008). *Evolutionary psychology: The new science of the mind* (3rd ed.). Boston, MA: Allyn & Bacon.

Buss, D. M. (2009). How can evolutionary psychology successfully explain personality and individual differences? *Perspectives on Psychological Science*, *4*(4), 359-366.

Buss, D. M. (Ed.). (2005). *The handbook of evolutionary psychology*. New Jersey: John Wiley & Sons.

Buss, D. M., Haselton, M. G., Shackelford, T. K., Bleske, A. L., & Wakefield, J. C. (1998). Adaptations, exaptations, and spandrels. *American Psychologist*, *53*(5), 533.

Buss, D. M., & Hawley, P. H. (2010). *The evolution of personality and individual differences*. New York: Oxford University Press.

Caraco, T., Martindale, S., & Whittam, T. S. (1980). An empirical demonstration of risk-sensitive foraging preferences. *Animal Behaviour*, *28*(3), 820-830.

Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., . . . Braithwaite, A. (2003). Influence of life stress on depression: moderation by a polymorphism in the 5-HTT gene. *Science*, *301*(5631), 386-389.

Castonguay, L. G., Goldfried, M. R., Wiser, S., Raue, P. J., & Hayes, A. M. (1996). Predicting the effect of cognitive therapy for depression: a study of unique and common factors. *Journal of Consulting and Clinical Psychology*, *64*(3), 497.

Cheung, M.-P., Gilbert, P., & Irons, C. (2004). An exploration of shame, social rank and rumination in relation to depression. *Personality and Individual Differences, 36*(5), 1143-1153.

Chilton, F. H., Lee, T. C., Willard, S. L., Ivester, P., Sergeant, S., Register, T. C., & Shively, C. A. (2011). Depression and altered serum lipids in cynomolgus monkeys consuming a Western diet. *Physiology & behavior, 104*(2), 222-227.

Ciranna, L. (2006). Serotonin as a modulator of glutamate-and GABA-mediated neurotransmission: implications in physiological functions and in pathology. *Current neuropharmacology, 4*(2), 101.

Coffman, S. J., Martell, C. R., Dimidjian, S., Gallop, R., & Hollon, S. D. (2007). Extreme nonresponse in cognitive therapy: Can behavioral activation succeed where cognitive therapy fails? *Journal of Consulting and Clinical Psychology, 75*(4), 531.

Compton, W., Conway, K., Stinson, F., & Grant, B. (2006). Changes in the prevalence of major depression and comorbid substance use disorders in the United States between 1991–1992 and 2001–2002. *American Journal of Psychiatry, 163*(12), 2141-2147.

Confer, J. C., Easton, J. A., Fleischman, D. S., Goetz, C. D., Lewis, D. M., Perilloux, C., & Buss, D. M. (2010). Evolutionary psychology: Controversies, questions, prospects, and limitations. *American Psychologist, 65*(2), 110.

Connolly, K. J., & Edelmann, R. J. (1988). Depression and reproductive failure: a comment on Suarez & Gallup. *Journal of social and biological structures, 11*(2), 215-217.

Cordain, L., Eaton, S. B., Sebastian, A., Mann, N., Lindeberg, S., Watkins, B. A., . . . Brand-Miller, J. (2005). Origins and evolution of the Western diet: health implications for the 21st century. *The American journal of clinical nutrition, 81*(2), 341-354.

Cordain, L., Miller, J. B., Eaton, S. B., Mann, N., Holt, S. H., & Speth, J. D. (2000). Plant-animal subsistence ratios and macronutrient energy estimations in worldwide hunter-gatherer diets. *The American journal of clinical nutrition, 71*(3), 682-692.

Cosmides, L. (1989). The logic of social exchange: Has natural selection shaped how humans reason? Studies with the Wason selection task. *Cognition, 31*(3), 187-276.

Cosmides, L., & Tooby, J. (1987). From evolution to behavior: Evolutionary psychology as the missing link. In J. Dupre (Ed.), *The Latest on the Best: Essays on Evolution and Optimality*. Cambridge, MA: MIT Press.

Cosmides, L., & Tooby, J. (1992). Cognitive adaptations for social exchange. In J. H. Barkow, L. Cosmides & J. Tooby (Eds.), *The adapted mind* (pp. 163-228). New York: Oxford University Press.

Cosmides, L., & Tooby, J. (1994a). Beyond intuition and instinct blindness: Toward an evolutionarily rigorous cognitive science. *Cognition, 50*(1), 41-77.

Cosmides, L., & Tooby, J. (1994b). Origins of domain specificity: The evolution of functional organization. In L. A. Hirschfeld & G. A. Susan (Eds.), *Mapping the mind: Domain specificity in cognition and culture* (pp. 85-116). Cambridge: Cambridge University Press.

Cosmides, L., & Tooby, J. (1999). Toward an evolutionary taxonomy of treatable conditions. *Journal of abnormal psychology, 108*(3), 453-464.

Cosmides, L., & Tooby, J. (2005). Neurocognitive adaptations designed for social exchange. In D. M. Buss (Ed.), *The handbook of evolutionary psychology* (pp. 584-627). New Jersey: John Wiley & Sons.

Cosmides, L., & Tooby, J. (2013). Evolutionary psychology: New perspectives on cognition and motivation. *Annual Review of Psychology, 64*, 201-229.

Cowgill, U. M. (1969). Some observations on the prosimian Perodicticus potto. *Folia Primatologica, 11*(1-2), 144-150.

Cross, C. P., Copping, L. T., & Campbell, A. (2011). Sex differences in impulsivity: a meta-analysis. *Psychological bulletin, 137*(1), 97.

Csete, M. E., & Doyle, J. C. (2002). Reverse engineering of biological complexity. *Science, 295*(5560), 1664-1669.

Cuijpers, P., van Straten, A., Andersson, G., & van Oppen, P. (2008). Psychotherapy for depression in adults: a meta-analysis of comparative outcome studies. *Journal of Consulting and Clinical Psychology, 76*(6), 909.

Cuijpers, P., Van Straten, A., & Warmerdam, L. (2007). Behavioral activation treatments of depression: A meta-analysis. *Clinical psychology review, 27*(3), 318-326.

Culbertson, F. M. (1997). Depression and gender: an international review. *American Psychologist, 52*(1), 25.

D'Esposito, M., Postle, B. R., & Rypma, B. (2000). Prefrontal cortical contributions to working memory: evidence from event-related fMRI studies. *Experimental Brain Research, 133*(1), 3-11.

Dantzer, R., & Kelley, K. W. (2007). Twenty years of research on cytokine-induced sickness behavior. *Brain, behavior, and immunity, 21*(2), 153-160.

Dantzer, R., O'Connor, J. C., Freund, G. G., Johnson, R. W., & Kelley, K. W. (2008). From inflammation to sickness and depression: when the immune system subjugates the brain. *Nature Reviews Neuroscience, 9*(1), 46-56.

Darwin, C. (1859/1998). *The origin of species by means of natural selection: or, the preservation of favored races in the struggle for life*. Ware: Wordsworth.

Darwin, C. (1872/1998). *The expression of the emotions in man and animals*. New York: Oxford University Press.

Dawkins, R. (1986/2008). *The blind watchmaker: Why the evidence of evolution reveals a universe without design*. London: Folio Society.

de Catanzaro, D. (1995). Reproductive status, family interactions, and suicidal ideation: Surveys of the general public and high-risk groups. *Ethology and Sociobiology*, *16*(5), 385-394.

Delton, A. W., Cosmides, L., Guemo, M., Robertson, T. E., & Tooby, J. (2012). The psychosemantics of free riding: Dissecting the architecture of a moral concept. *Journal of personality and social psychology*, *102*(6), 1252.

Dennett, D. C. (1995). *Darwin's dangerous idea*. New York: Simon & Schuster.

Diamond, J. (1997). *Guns, Germs and Steel: The Fates of Human Societies*. New York: W.W Norton & Company.

Dominian, J. (1977). The role of psychiatry in the menopause. *Clinics in obstetrics and gynaecology*, *4*(1), 241-258.

Drevets, W. C. (1999). Prefrontal Cortical-Amygdalar Metabolism in Major Depression. *Annals of the New York Academy of Sciences*, *877*(1), 614-637.

Drevets, W. C. (2000). Neuroimaging studies of mood disorders. *Biological psychiatry*, *48*(8), 813-829.

Dunbar, R. I. (1993). Coevolution of neocortical size, group size and language in humans. *Behavioral and brain sciences*, *16*(04), 681-694.

Dunbar, R. I. (1998). The social brain hypothesis. *brain*, *9*(10), 178-190.

Dunbar, R. I. (2007). The social brain hypothesis and its relevance to social psychology. In J. P. Forgas, M. G. Haselton & W. v. Hippel (Eds.), *Evolution and the social mind: Evolutionary psychology and social cognition* (pp. 21-31). New York: Psychology Press.

Dunn, J. C., Whelton, W. J., & Sharpe, D. (2012). Retreating to safety: testing the social risk hypothesis model of depression. *Evolution and Human Behavior, 33*(6), 746-758.

Durant, J. (2013). *The Paleo Manifesto: Ancient Wisdom for Lifelong Health*. New York: Random House LLC.

Eaton, S. B., Eaton, S. B., Konner, M. J., & Shostak, M. (1996). An evolutionary perspective enhances understanding of human nutritional requirements. *The Journal of nutrition, 126*(6), 1732-1740.

Eisenberger, N. I., Inagaki, T. K., Mashal, N. M., & Irwin, M. R. (2010). Inflammation and social experience: an inflammatory challenge induces feelings of social disconnection in addition to depressed mood. *Brain, behavior, and immunity, 24*(4), 558-563.

Ekman, P., Friesen, W. V., O'Sullivan, M., Chan, A., Diacoyanni-Tarlatzis, I., Heider, K., . . . Ricci-Bitti, P. E. (1987). Universals and cultural differences in the judgments of facial expressions of emotion. *Journal of personality and social psychology, 53*(4), 712.

Ellis, H. C., Ottaway, S. A., Varner, L. J., Becker, A. S., & Moore, B. A. (1997). Emotion, motivation, and text comprehension: The detection of contradictions in passages. *Journal of Experimental Psychology: General*, *126*(2), 131.

Eskin, M., Ertekin, K., & Demir, H. (2008). Efficacy of a problem-solving therapy for depression and suicide potential in adolescents and young adults. *Cognitive Therapy and Research*, *32*(2), 227-245.

Everson, S. A., Maty, S. C., Lynch, J. W., & Kaplan, G. A. (2002). Epidemiologic evidence for the relation between socioeconomic status and depression, obesity, and diabetes. *Journal of Psychosomatic Research, 53*(4), 891-895.

Fessler, D. M. (2002). Pseudoparadoxical impulsivity in restrictive anorexia nervosa: A consequence of the logic of scarcity. *International Journal of Eating Disorders*, *31*(4), 376-388.

Festinger, L. (1954). A theory of social comparison processes. *Human relations, 7*(2), 117-140.

Forgas, J. (2007). The strange cognitive benefits of mild dysphoria: On the evolutionary advantages of not being too happy. In J. P. Forgas, M. G. Haselton & W. v. Hippel (Eds.), *Evolution and the social mind: Evolutionary psychology and social cognition* (pp. 107-121). New York: Psychology Press.

Forgas, J. P. (2007). When sad is better than happy: Negative affect can improve the quality and effectiveness of persuasive messages and social influence strategies. *Journal of Experimental Social Psychology, 43*(4), 513-528.

Forgas, J. P. (2013). Don't worry, be sad! On the cognitive, motivational, and interpersonal benefits of negative mood. *Current Directions in Psychological Science, 22*(3), 225-232.

Forgas, J. P., & East, R. (2008). On being happy and gullible: Mood effects on skepticism and the detection of deception. *Journal of Experimental Social Psychology, 44*(5), 1362-1367.

Garcia-Dorado, A., Caballero, A., & Crow, J. F. (2003). On the persistence and pervasiveness of a new mutation. *Evolution, 57*(11), 2644-2646.

George, M. S., Ketter, T. A., Parekh, P. I., Horwitz, B., Herscovitch, P., & Post, R. M. (1995). Brain activity during transient sadness and happiness in healthy women. *American Journal of Psychiatry*, *152*(3), 341-351.

Gilbert, P. (1992). *Depression: The evolution of powerlessness*. Hove: Psychology Press.

Gilbert, P. (1998). Evolutionary psychopathology: Why isn't the mind designed better than it is? *British Journal of Medical Psychology, 71*(4), 353-373.

Gilbert, P. (2000). Varieties of submissive behavior as forms of social defense: Their evolution and role in depression. In P. Gilbert & L. Sloman (Eds.), *Subordination and defeat: An evolutionary approach to mood disorders and their therapy* (pp. 3-45).

Gilbert, P., & Allan, S. (1998). The role of defeat and entrapment (arrested flight) in depression: an exploration of an evolutionary view. *Psychological medicine*, *28*(3), 585-598.

Goodman, J. H. (2004). Paternal postpartum depression, its relationship to maternal postpartum depression, and implications for family health. *Journal of advanced nursing*, *45*(1), 26-35.

Gordon Jr, G., Burch, R. L., & Platek, S. M. (2002). Does semen have antidepressant properties? *Archives of Sexual Behavior*, *31*(3), 289-293.

Gosse, G. H., & Barnes, M. J. (1994). Human grief resulting from the death of a pet. *Anthrozoos: A Multidisciplinary Journal of The Interactions of People & Animals*, *7*(2), 103-112.

Graf, M. C., Gaudiano, B. A., & Geller, P. A. (2008). Written emotional disclosure: A controlled study of the benefits of expressive writing homework in outpatient psychotherapy. *Psychotherapy Research*, *18*(4), 389-399.

Grøntvedt, T. V., & Kennair, L. E. O. (2013). Age preferences in a gender egalitarian society. *Journal of Social, Evolutionary, and Cultural Psychology*, *7*(3), 239.

Hagen, E. H. (1999). The functions of postpartum depression. *Evolution and Human Behavior*, *20*(5), 325-359.

Hagen, E. H. (2003). The bargaining model of depression. *Genetic and cultural evolution of cooperation*, 95-123.

Hagen, E. H. (2011). Evolutionary theories of depression: a critical review. *Canadian journal of psychiatry. Revue canadienne de psychiatrie*, *56*(12), 716.

Hagen, E. H., & Barrett, H. C. (2007). Perinatal sadness among Shuar women: support for an evolutionary theory of psychic pain. *Medical Anthropology Quarterly, 21*(1), 22-40.

Hagen, E. H., Hames, R. B., Craig, N. M., Lauer, M. T., & Price, M. E. (2001). Parental Investment and Child Health in a Yanomamö Village Suffering Short Term Food Stress. *Journal of biosocial science, 33*, 503-528.

Hagen, E. H., & Thomson, A. J. (2004). Social navigation hypothesis of depression revisited. *Journal of affective disorders, 83*(2), 285-286.

Hagen, E. H., Watson, P. J., & Thomson, A. J. (2004). *Love's labour's lost: Depression as an evolutionary adaptation to obtain help from those with whom one is in conflict*.

Hamilton, W. D. (1964). The genetical evolution of social behaviour. I. *Journal of Theoretical Biology, 7*(1), 1-16. doi: http://dx.doi.org/10.1016/0022-5193(64)90038-4

Hammar, Å., Lund, A., & Hugdahl, K. (2003). Long-lasting cognitive impairment in unipolar major depression: a 6-month follow-up study. *Psychiatry research, 118*(2), 189-196.

Hammar, Å., & Årdal, G. (2009). Cognitive functioning in major depression–a summary. *Frontiers in human neuroscience, 3*.

Hankin, B. L., & Abramson, L. Y. (2001). Development of gender differences in depression: An elaborated cognitive vulnerability–transactional stress theory. *Psychological bulletin, 127*(6), 773.

Haselton, M. G., & Nettle, D. (2006). The paranoid optimist: An integrative evolutionary model of cognitive biases. *Personality and social psychology Review, 10*(1), 47-66.

Hendrie, C., & Pickles, A. (2009). Depression as an evolutionary adaptation: Implications for the development of preclinical models. *Medical Hypotheses, 72*(3), 342-347.

Hendrie, C., & Pickles, A. (2010). Depression as an evolutionary adaptation: anatomical organisation around the third ventricle. *Medical Hypotheses, 74*(4), 735-740.

Henriques, G. (2000). Depression: Disease or behavioral shutdown mechanism. *Journal of Science and Health Policy, 1*, 152-165.

Hertel, G., Neuhof, J., Theuer, T., & Kerr, N. L. (2000). Mood effects on cooperation in small groups: Does positive mood simply lead to more cooperation? *Cognition & emotion*, *14*(4), 441-472.

Hidaka, B. H. (2012). Depression as a disease of modernity: explanations for increasing prevalence. *Journal of affective disorders*, *140*(3), 205-214.

Hollon, S. D., Thase, M. E., & Markowitz, J. C. (2002). Treatment and prevention of depression. *Psychological Science in the Public Interest*, *3*(2), 39-77.

Holtkamp, K., Herpertz-Dahlmann, B., Mika, C., Heer, M., Heussen, N., Fichter, M., . . . Schweiger, U. (2003). Elevated physical activity and low leptin levels co-occur in patients with anorexia nervosa. *Journal of Clinical Endocrinology & Metabolism*, *88*(11), 5169-5174.

Horwitz, A. V., & Wakefield, J. C. (2007). *The loss of sadness: How psychiatry transformed normal sorrow into depressive disorder*. New York: Oxford University Press.

Howell, N. (1979/2000). *Demography of the Dobe Kung* (2 ed.). Hawthorne, New York: Aldine De Gruyter.

Hyde, J. S., Mezulis, A. H., & Abramson, L. Y. (2008). The ABCs of depression: integrating affective, biological, and cognitive models to explain the emergence of the gender difference in depression. *Psychological review*, *115*(2), 291.

Ilardi, S. S. (2009). *The depression cure: The 6-step program to beat depression without drugs*: Da Capo Press.

Indo, Y., Tsuruta, M., Hayashida, Y., Karim, M. A., Ohta, K., Kawano, T., . . . Matsuda, I. (1996). Mutations in the TRKA/NGF receptor gene in patients with congenital insensitivity to pain with anhidrosis. *Nature genetics*, *13*(4), 485-488.

James, W. (1890/2011). *The principles of psychology* Retrieved from http://www.bahaistudies.net/asma/principlesofpsychology.pdf

Janeway, C. A., Travers, P., Walport, M., & Shlomchik, M. J. (2001). *Immunobiology*. New York: Garland Science.

Jang, K. L., Livesley, W. J., & Vemon, P. A. (1996). Heritability of the big five personality dimensions and their facets: a twin study. *Journal of personality, 64*(3), 577-592.

Jorm, A. (1987). Sex differences in neuroticism: a quantitative synthesis of published research. *Australian and New Zealand Journal of Psychiatry, 21*(4), 501-506.

Josephson, B. R. (1996). Mood regulation and memory: Repairing sad moods with happy memories. *Cognition & emotion, 10*(4), 437-444.

Keller, M. C. (2007). The role of mutations in human mating. *Mating intelligence: Sex, relationships, and the mind's reproductive system*, 173-192.

Keller, M. C., & Miller, G. F. (2006). Resolving the paradox of common, harmful, heritable mental disorders: which evolutionary genetic models work best? *Behavioral and brain sciences, 29*(04), 385-404.

Keller, M. C., & Nesse, R. M. (2005). Is low mood an adaptation? Evidence for subtypes with symptoms that match precipitants. *Journal of affective disorders, 86*(1), 27-35.

Kendler, K. S., Karkowski, L. M., & Prescott, C. A. (1999). Causal relationship between stressful life events and the onset of major depression. *American Journal of Psychiatry, 156*(6), 837-841.

Kendler, K. S., Kessler, R. C., Walters, E. E., MacLean, C., Neale, M. C., Heath, A. C., & Eaves, L. J. (1995). Stressful life events, genetic liability, and onset of an episode of major depression in women. *American Journal of Psychiatry, 152*(6), 833-842.

Kennair, L. E. O. (2002). Evolutionary psychology: An emerging integrative perspective within the science and practice of psychology. *Human Nature Review, 2*, 17-61.

Kennair, L. E. O. (2003). Evolutionary psychology and psychopathology. *Current Opinion in Psychiatry, 16*(6), 691-699.

Kennair, L. E. O. (2010). The Problem of Defining Psychopathology and Challenges to Evolutionary Psychology Theory. In D. M. Buss & P. H. Hawley (Eds.), *The evolution of personality and individual differences* (pp. 451). New York: Oxford University Press.

Kessler, R. C. (1997). The effects of stressful life events on depression. In S. E. Hyman (Ed.), *Depression: The science of mental health*. New York: Routledge.

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Koretz, D., Merikangas, K. R., . . . Wang, P. S. (2003). The epidemiology of major depressive disorder: results from the National Comorbidity Survey Replication (NCS-R). *Jama, 289*(23), 3095-3105.

Kessler, R. C., & Frank, R. G. (1997). The impact of psychiatric disorders on work loss days. *Psychological medicine, 27*(04), 861-873.

Ketelaar, T., & Ellis, B. J. (2000). Are evolutionary explanations unfalsifiable? Evolutionary psychology and the Lakatosian philosophy of science. *Psychological inquiry, 11*(1), 1-21.

Kinney, D. K., & Tanaka, M. (2009). An evolutionary hypothesis of depression and its symptoms, adaptive value, and risk factors. *The Journal of nervous and mental disease, 197*(8), 561-567.

Klinger, E. (1975). Consequences of commitment to and disengagement from incentives. *Psychological review, 82*(1), 1.

Kluger, M. J., Kozak, W., Conn, C. A., Leon, L. R., & Soszynski, D. (1998). Role of fever in disease. *Annals of the New York Academy of Sciences, 856*(1), 224-233.

Krause-Parello, C. A. (2012). Pet ownership and older women: the relationships among loneliness, pet attachment support, human social support, and depressed mood. *Geriatric Nursing*, *33*(3), 194-203.

Krishnan, K. R., Swartz, M. S., Larson, M. J., & Santoliquido, G. (1984). Funeral mania in recurrent bipolar affective disorders: Reports of three cases. *Journal of Clinical Psychiatry*.

Kurzban, R. (2012). *Why everyone (else) is a hypocrite: Evolution and the modular mind*. New Jersey: Princeton University Press.

Kurzban, R., & Leary, M. R. (2001). Evolutionary origins of stigmatization: the functions of social exclusion. *Psychological bulletin*, *127*(2), 187.

Leary, M. R., & Baumeister, R. F. (2000). The nature and function of self-esteem: Sociometer theory.

Leary, M. R., Schreindorfer, L. S., & Haupt, A. L. (1995). The role of low self-esteem in emotional and behavioral problems: Why is low self-esteem dysfunctional? *Journal of Social and Clinical Psychology*, *14*(3), 297-314.

Leary, M. R., Tambor, E. S., Terdal, S. K., & Downs, D. L. (1995). Self-esteem as an interpersonal monitor: The sociometer hypothesis. *Journal of personality and social psychology*, *68*(3), 518.

Leyfer, O. T., Folstein, S. E., Bacalman, S., Davis, N. O., Dinh, E., Morgan, J., . . . Lainhart, J. E. (2006). Comorbid psychiatric disorders in children with autism: Interview development and rates of disorders. *Journal of autism and developmental disorders*, *36*(7), 849-861.

Lieberman, D. (2013). *The Story of the Human Body: Evolution, Health, and Disease*. New York: Random House LLC.

Lieberman, D., Tooby, J., & Cosmides, L. (2003). Does morality have a biological basis? An empirical test of the factors governing moral sentiments relating to incest. *Proceedings of the Royal Society of London. Series B: Biological sciences, 270*(1517), 819-826.

Lieberman, D., Tooby, J., & Cosmides, L. (2007). The architecture of human kin detection. *Nature, 445*(7129), 727-731.

Lin, P.-Y., & Su, K.-P. (2007). A meta-analytic review of double-blind, placebo-controlled trials of antidepressant efficacy of omega-3 fatty acids. *Journal of Clinical Psychiatry, 68*(7), 1056-1061.

Lopez, A. D., Mathers, C. D., Ezzati, M., Jamison, D. T., & Murray, C. (2006). Global and regional burden of disease and risk factors, 2001: systematic analysis of population health data. *The Lancet, 367*(9524), 1747-1757.

Lorant, V., Deliège, D., Eaton, W., Robert, A., Philippot, P., & Ansseau, M. (2003). Socioeconomic inequalities in depression: a meta-analysis. *American journal of epidemiology, 157*(2), 98-112.

Lyubomirsky, S., Tucker, K. L., Caldwell, N. D., & Berg, K. (1999). Why ruminators are poor problem solvers: clues from the phenomenology of dysphoric rumination. *Journal of personality and social psychology, 77*(5), 1041.

MacDonald, G., & Leary, M. R. (2005). Why does social exclusion hurt? The relationship between social and physical pain. *Psychological bulletin, 131*(2), 202.

Maes, M., Van de Vyvere, J., Vandoolaeghe, E., Bril, T., Demedts, P., Wauters, A., & Neels, H. (1996). Alterations in iron metabolism and the erythron in major depression: further evidence for a chronic inflammatory process. *Journal of affective disorders, 40*(1), 23-33.

Major, B., Zubek, J. M., Cooper, M. L., Cozzarelli, C., & Richards, C. (1997). Mixed messages: implications of social conflict and social support within close relationships for adjustment to a stressful life event. *Journal of personality and social psychology, 72*(6), 1349.

Malouff, J. M., Thorsteinsson, E. B., & Schutte, N. S. (2007). The efficacy of problem solving therapy in reducing mental and physical health problems: A meta-analysis. *Clinical psychology review, 27*(1), 46-57.

Mardy, S., Miura, Y., Endo, F., Matsuda, I., Sztriha, L., Frossard, P., . . . Andria, G. (1999). Congenital Insensitivity to Pain with Anhidrosis: Novel Mutations in the TRKA (NTRK1) Gene Encoding A High-Affinity Receptor for Nerve Growth Factor. *The American Journal of Human Genetics, 64*(6), 1570-1579.

Markman, K. D., & Weary, G. (1996). The influence of chronic control concerns on counterfactual thought. *Social Cognition, 14*(4), 292-316.

Marr, D. (1982/2010). *Vision: A computational investigation into the human representation and processing of visual information*. New York: University Press Group Limited.

Martel, M. M. (2013). Sexual selection and sex differences in the prevalence of childhood externalizing and adolescent internalizing disorders.

McGuire, M. T., & Troisi, A. (1998). Prevalence differences in depression among males and females: Are there evolutionary explanations? *British Journal of Medical Psychology, 71*(4), 479-491.

Miller, G. F. (2001). *The mating mind: How sexual choice shaped the evolution of human nature*. United States of America, New York: Anchor books.

Miller, G. F. (2010). Are Pleiotropic Mutations and Holocene Selective Sweeps the Only Evolutionary-genetic Processes Left for Explaining Heritable Variation in Human

Psychological Traits? In D. M. Buss & P. H. Hawley (Eds.), *The evolution of personality and individual differences*. New York: Oxford University Press.

Murray, C., & Lopez, A. D. (1997). Alternative projections of mortality and disability by cause 1990–2020: Global Burden of Disease Study. *The Lancet, 349*(9064), 1498-1504.

Murray, M., & Murray, A. (1979). Anorexia of infection as a mechanism of host defense. *The American journal of clinical nutrition, 32*(3), 593-596.

Mykletun, A., Bjerkeset, O., Dewey, M., Prince, M., Overland, S., & Stewart, R. (2007). Anxiety, depression, and cause-specific mortality: the HUNT study. *Psychosomatic medicine, 69*(4), 323-331.

Nasco, S. A., & Marsh, K. L. (1999). Gaining control through counterfactual thinking. *Personality and Social Psychology Bulletin, 25*(5), 557-569.

Nesse, R. M. (1990). Evolutionary explanations of emotions. *Human nature, 1*(3), 261-289.

Nesse, R. M. (1999). Testing evolutionary hypotheses about mental disorders. *Evolution in health and disease*, 261-266.

Nesse, R. M. (2000). Is depression an adaptation? *Archives of General Psychiatry, 57*(1), 14-20.

Nesse, R. M. (2008). Evolution: medicine's most basic science. *The Lancet, 372*, S21-S27.

Nesse, R. M. (2009). Explaining depression: neuroscience is not enough, evolution is essential. *Understanding Depression: A Translational Approach*, 17-35.

Nesse, R. M., & Jackson, E. D. (2011). Evolutionary foundations for psychiatric diagnosis: making DSM-V valid. *Maladapting Minds: Philosophy, Psychiatry, and Evolutionary Theory*, 167-191.

Nesse, R. M., & Stein, D. J. (2012). Towards a genuinely medical model for psychiatric nosology. *BMC medicine, 10*(1), 5.

Nesse, R. M., & Williams, G. C. (1996). *Evolution and healing: The new science of Darwinian medicine*. New York: Phoenix.

Nettle, D. (2002). Height and reproductive success in a cohort of British men. *Human nature, 13*(4), 473-491.

Nettle, D. (2004). Evolutionary origins of depression: a review and reformulation. *Journal of affective disorders, 81*(2), 91-102.

Nettle, D. (2009). An evolutionary model of low mood states. *Journal of Theoretical Biology*.

Ney, P. (1986). The intravaginal absorption of male generated hormones and their possible effect on female behaviour. *Medical Hypotheses, 20*(2), 221-231.

Noble, R. E. (2005). Depression in women. *Metabolism, 54*(5), 49-52.

Nolen-Hoeksema, S. (1987). Sex differences in unipolar depression: evidence and theory. *Psychological bulletin, 101*(2), 259.

Nolen-Hoeksema, S. (1991). Responses to depression and their effects on the duration of depressive episodes. *Journal of abnormal psychology, 100*(4), 569.

Nolen-Hoeksema, S., Larson, J., & Grayson, C. (1999). Explaining the gender difference in depressive symptoms. *Journal of personality and social psychology, 77*(5), 1061.

O'hara, M. W., & Swain, A. M. (1996). Rates and risk of postpartum depression-a meta-analysis. *International review of psychiatry, 8*(1), 37-54.

O'Connor, L. E., Berry, J. W., Weiss, J., & Gilbert, P. (2002). Guilt, fear, submission, and empathy in depression. *Journal of affective disorders, 71*(1), 19-27.

Opdal, S. H., Vege, Å., & Rognum, T. O. (2008). Serotonin transporter gene variation in sudden infant death syndrome. *Acta Paediatrica, 97*(7), 861-865.

Page, C. M., & Colby, P. M. (2003). If only I hadn't smoked: The impact of counterfactual thinking on a smoking-related behavior. *Psychology & Marketing, 20*(11), 955-976.

Paykel, E. (1977). Depression and appetite. *Journal of Psychosomatic Research, 21*(5), 401-407.

Petersen, A. C., Compas, B. E., Brooks-Gunn, J., Stemmler, M., Ey, S., & Grant, K. E. (1993). Depression in adolescence. *American Psychologist, 48*(2), 155.

Pickett, K., & Wilkinson, R. (2010). *The spirit level: Why greater equality makes societies stronger*: Bloomsbury Publishing USA.

Pinker, S. (2003). *The blank slate: The modern denial of human nature*. New York: Penguin.

Pinker, S. (2009). *How the mind works*. New York: Norton.

Pinsky, L., & DiGeorge, A. M. (1966). Congenital familial sensory neuropathy with anhidrosis. *The Journal of pediatrics, 68*(1), 1-13.

Price, J. (1967). The dominance hierarchy and the evolution of mental illness. *The Lancet, 290*(7509), 243-246.

Price, J. (2000). Subordination, self-esteem and depression. In P. Gilbert & L. Sloman (Eds.), *Subordination and defeat: An evolutionary approach to mood disorders and their therapy* (pp. 165-177). New Jersey: Lawrence Erlbaum Associates.

Price, J., & Sloman, L. (1987). Depression as yielding behavior: An animal model based on Schjelderup-Ebbe's pecking order. *Ethology and Sociobiology, 8*, 85-98.

Price, J., Sloman, L., Gardner, R., Gilbert, P., & Rohde, P. (1994). The social competition hypothesis of depression. *The British Journal of Psychiatry, 164*(3), 309-315.

Protheroe, S. (1991). Congenital insensitivity to pain. *Journal of the Royal Society of Medicine, 84*(9), 558.

Raghunathan, R., & Pham, M. T. (1999). All negative moods are not equal: Motivational influences of anxiety and sadness on decision making. *Organizational Behavior and Human Decision Processes, 79*(1), 56-77.

Rahman, Q., & Hull, M. S. (2005). An empirical test of the kin selection hypothesis for male homosexuality. *Archives of Sexual Behavior, 34*(4), 461-467.

Raison, C. L., Capuron, L., & Miller, A. H. (2006). Cytokines sing the blues: inflammation and the pathogenesis of depression. *Trends in immunology, 27*(1), 24-31.

Raison, C. L., & Miller, A. H. (2012). The evolutionary significance of depression in Pathogen Host Defense (PATHOS-D). *Molecular psychiatry, 18*(1), 15-37.

Raison, C. L., Rye, D. B., Woolwine, B. J., Vogt, G. J., Bautista, B. M., Spivey, J. R., & Miller, A. H. (2010). Chronic interferon-alpha administration disrupts sleep continuity and depth in patients with hepatitis C: association with fatigue, motor slowing, and increased evening cortisol. *Biological psychiatry, 68*(10), 942-949.

Raleigh, M. J., McGuire, M. T., Brammer, G. L., Pollack, D. B., & Yuwiler, A. (1991). Serotonergic mechanisms promote dominance acquisition in adult male vervet monkeys. *Brain research, 559*(2), 181-190.

Rao, V., & Lyketsos, C. (2000). Neuropsychiatric sequelae of traumatic brain injury. *Psychosomatics, 41*(2), 95-103.

Rausch, J., Johnson, M., Corley, K., Hobby, H., Shendarkar, N., Fei, Y., . . . Leibach, F. (2003). Depressed patients have higher body temperature: 5-HT transporter long promoter region effects. *Neuropsychobiology, 47*(3), 120-127.

Raven, J., Court, J. H., & Raven, J. (2003). Raven's Progressive Matrices. In R. S. McCallum (Ed.), *Handbook of Nonverbal Assessment* (pp. 223-237): Springer US.

Reimer, M. S. (1996). "Sinking into the ground": The development and consequences of shame in adolescence. *Developmental Review, 16*(4), 321-363.

Rickarby, G. A. (1977). Four cases of mania associated with bereavement. *The Journal of nervous and mental disease, 165*(4), 255-262.

Roese, N. J. (1994). The functional basis of counterfactual thinking. *Journal of personality and social psychology, 66*(5), 805.

Roese, N. J., & Olson, J. M. (1997). Counterfactual thinking: The intersection of affect and function. *Advances in experimental social psychology, 29*, 1-59.

Rosemberg, S., Nagahashi Marie, S. K., & Kliemann, S. (1994). Congenital insensitivity to pain with anhidrosis (hereditary sensory and autonomic neuropathy type IV). *Pediatric neurology, 11*(1), 50-56.

Rudan, I., Rudan, D., Campbell, H., Carothers, A., Wright, A., Smolej-Narancic, N., . . . Deka, R. (2003). Inbreeding and risk of late onset complex disease. *Journal of Medical Genetics, 40*(12), 925-932.

Sapolsky, R. M. (2005). The influence of social hierarchy on primate health. *Science, 308*(5722), 648-652.

Schaller, M., & Duncan, L. A. (2007). The behavioral immune system: Its evolution and social psychological implications. In J. P. Forgas, M. G. Haselton & W. v. Hippel (Eds.), *Evolution and the social mind: Evolutionary psychology and social cognition* (pp. 293-307). New York: Psychology Press.

Schaller, M., & Park, J. H. (2011). The behavioral immune system (and why it matters). *Current Directions in Psychological Science, 20*(2), 99-103.

Schoenhuber, R., & Gentilini, M. (1988). Anxiety and depression after mild head injury: a case control study. *Journal of Neurology, Neurosurgery & Psychiatry, 51*(5), 722-724.

Schulz, R., Beach, S. R., Ives, D. G., Martire, L. M., Ariyo, A. A., & Kop, W. J. (2000). Association between depression and mortality in older adults: the Cardiovascular Health Study. *Archives of Internal Medicine, 160*(12), 1761-1768.

Segrin, C., & Abramson, L. Y. (1994). Negative reactions to depressive behaviors: a communication theories analysis. *Journal of abnormal psychology, 103*(4), 655.

Seibert, P. S., & Ellis, H. C. (1991). A convenient self-referencing mood induction procedure. *Bulletin of the Psychonomic Society*.

Sheline, Y. I., Gado, M. H., & Price, J. L. (1998). Amygdala core nuclei volumes are decreased in recurrent major depression. *Neuroreport, 9*(9), 2023-2028.

Simopoulos, A. P. (2002). The importance of the ratio of omega-6/omega-3 essential fatty acids. *Biomedicine & pharmacotherapy, 56*(8), 365-379.

Simpson, J. A., & Campbell, L. (2005). Methods of Evolutionary Sciences. In D. M. Buss (Ed.), *The handbook of evolutionary psychology* (pp. 119-144). New Jersey: John Wiley & Sons.

Singh, D. (1993). Adaptive significance of female physical attractiveness: role of waist-to-hip ratio. *Journal of personality and social psychology, 65*(2), 293.

Sloman, L. (2000). How the Involuntary Defeat Strategy Relates to Depression. In L. S. Paul Gilbert (Ed.), *Subordination and defeat: An evolutionary approach to mood disorders and their therapy* (pp. 47-67). New Jersey: Lawrence Erlbaum Associates.

Sloman, L., Farvolden, P., Gilbert, P., & Price, J. (2006). The interactive functioning of anxiety and depression in agonistic encounters and reconciliation. *Journal of affective disorders, 90*(2), 93-99.

Sloman, L., & Gilbert, P. (Eds.). (2000). *Subordination and defeat: An evolutionary approach to mood disorders and their therapy*. New Jersey: Lawrence Erlbaum Associates.

Smith, J. M. (1993). *The theory of evolution* (3rd ed.). Cambridge: Cambridge University Press.

Smith, J. M., & Price, G. (1973). The Logic of Animal Conflict. *Nature, 246*, 15.

Soares, J. C., & Mann, J. J. (1997). The anatomy of mood disorders—review of structural neuroimaging studies. *Biological psychiatry, 41*(1), 86-106.

Spoor, J., & Williams, K. D. (2007). The evolution of an ostracism detection system. In J. P. Forgas, M. G. Haselton & W. v. Hippel (Eds.), *The evolution of the social mind: Evolutionary psychology and social cognition* (pp. 279-292). New York: Psychology Press.

Steinberg, L. (2005). Cognitive and affective development in adolescence. *Trends in cognitive sciences, 9*(2), 69-74.

Stephens, D. W. (1981). The logic of risk-sensitive foraging preferences. *Animal Behaviour, 29*(2), 628-629.

Stone, V. E., Cosmides, L., Tooby, J., Kroll, N., & Knight, R. T. (2002). Selective impairment of reasoning about social exchange in a patient with bilateral limbic system damage. *Proceedings of the national Academy of Sciences, 99*(17), 11531-11536.

Suarez, S. D., & Gallup, G. G. (1985). Depression as a response to reproductive failure. *Journal of social and biological structures, 8*(3), 279-287.

Sugiyama, L. S., Tooby, J., & Cosmides, L. (2002). Cross-cultural evidence of cognitive adaptations for social exchange among the Shiwiar of Ecuadorian Amazonia. *Proceedings of the national Academy of Sciences, 99*(17), 11537-11542.

Sullivan, P. F., Neale, M. C., & Kendler, K. S. (2000). Genetic epidemiology of major depression: review and meta-analysis. *American Journal of Psychiatry, 157*(10), 1552-1562.

Symons, D. (1979). *The evolution of human sexuality*. New York: Oxford University Press.

Thapar, A., Collishaw, S., Pine, D. S., & Thapar, A. K. (2012). Depression in adolescence. *The Lancet, 379*(9820), 1056-1067.

Thomson, & Hytten. (1961). Calorie requirements in human pregnancy. *Proceedings of the Nutrition Society, 20*(01), 76-83.

Thomson, A. M., Hytten, F. E., & Billewicz, W. (1970). The energy cost of human lactation. *British journal of Nutrition, 24*(02), 565-572.

Thornhill, R. (1997). The concept of an evolved adaptation *Characterizing human psychological adaptations* (pp. 4-22). Chichester: John Wiley & Sons.

Tinbergen, N. (1963). On aims and methods of ethology. *Zeitschrift für Tierpsychologie, 20*(4), 410-433.

Tooby, J., & Cosmides, L. (1988). The evolution of war and its cognitive foundations. *Institute for Evolutionary Studies Technical Report, 88*(1), 1-15.

Tooby, J., & Cosmides, L. (1990). The past explains the present: Emotional adaptations and the structure of ancestral environments. *Ethology and Sociobiology, 11*(4), 375-424.

Tooby, J., & Cosmides, L. (1992). The psychological foundations of culture. In J. Tooby, L. Cosmides & J. H. Barkow (Eds.), *The adapted mind: Evolutionary psychology and the generation of culture* (pp. 19-136). New York: Oxford University Press.

Tooby, J., & Cosmides, L. (2005). Conceptual foundations of evolutionary psychology. In D. M. Buss (Ed.), *The handbook of evolutionary psychology* (pp. 5-67). New Jersey: John Wiley & Sons.

Treynor, W., Gonzalez, R., & Nolen-Hoeksema, S. (2003). Rumination reconsidered: A psychometric analysis. *Cognitive Therapy and Research, 27*(3), 247-259.

Trivers, R. L. (1971). The evolution of reciprocal altruism. *Quarterly review of biology*, 35-57.

Trivers, R. L. (1972). Parental investment and sexual selection.

Trivers, R. L. (1974). Parent-offspring conflict. *American zoologist, 14*(1), 249-264.

Troisi, A. (2001). Gender differences in vulnerability to social stress: a Darwinian perspective. *Physiology & behavior, 73*(3), 443-449.

Turkheimer, E. (2000). Three laws of behavior genetics and what they mean. *Current Directions in Psychological Science, 9*(5), 160-164.

Varga, S. (2012). Evolutionary psychiatry and depression: testing two hypotheses. *Medicine, Health Care and Philosophy, 15*(1), 41-52.

Wakefield, J. C. (1992). Disorder as Harmful Dysfunction: A Conceptual Critique of DSM-III-R's Definition of Mental Disorder. *Psychological review, 99*(2), 232-247.

Wakefield, J. C. (1999). Evolutionary versus prototype analyses of the concept of disorder. *Journal of abnormal psychology, 108*(3), 374.

Wakefield, J. C. (2005). Biological Function and Dysfunction. In D. M. Buss (Ed.), *The handbook of evolutionary psychology* (pp. 878-903). New Jersey: John Wiley & Sons.

Wakefield, J. C. (2007). The concept of mental disorder: diagnostic implications of the harmful dysfunction analysis. *World Psychiatry, 6*(3), 149.

Wampold, B. E., Mondin, G. W., Moody, M., Stich, F., Benson, K., & Ahn, H.-n. (1997). A meta-analysis of outcome studies comparing bona fide psychotherapies: Empiricially," all must have prizes.". *Psychological bulletin, 122*(3), 203.

Wang, D. Y.-C., Kumar, S., & Hedges, S. B. (1999). Divergence time estimates for the early history of animal phyla and the origin of plants, animals and fungi. *Proceedings of the Royal Society of London. Series B: Biological sciences, 266*(1415), 163-171.

Wason, P. C. (1966). Reasoning. In B. Foss (Ed.), *New horizons in psychology* (pp. 131-151). Harmondsworth: Penguin Books.

Watkins, E., & Brown, R. (2002). Rumination and executive function in depression: An experimental study. *Journal of Neurology, Neurosurgery & Psychiatry, 72*(3), 400-402.

Watson, P. J. (2004). [Letter to the editor of the Journal of Affective Disorders]. from http://biology.unm.edu/Biology/pwatson/public_html/WatsonResponse.pdf

Watson, P. J. (2008, 04.11.2008). Predictions of the Social Navigation Hypothesis in Relation to Major Depression. Retrieved 26.11.2013, 2013, from http://biology.unm.edu/biology/pwatson/public_html/SNH_predictions1.html

Watson, P. J., & Andrews, P. W. (2002). Toward a revised evolutionary adaptationist analysis of depression: the social navigation hypothesis. *Journal of affective disorders, 72*(1), 1-14.

Watts, D. P. (2002). Reciprocity and interchange in the social relationships of wild male chimpanzees. *Behaviour, 139*(2-3), 2-3.

Weissman, M. M. (1987). Advances in psychiatric epidemiology: rates and risks for major depression. *American Journal of Public Health, 77*(4), 445-451.

Weissman, M. M., Bland, R., Joyce, P. R., Newman, S., Wells, J. E., & Wittchen, H.-U. (1993). Sex differences in rates of depression: cross-national perspectives. *Journal of affective disorders, 29*(2), 77-84.

Weissman, M. M., & Klerman, G. L. (1977). Sex differences and the epidemiology of depression. *Archives of General Psychiatry, 34*(1), 98.

Wells, A., Fisher, P., Myers, S., Wheatley, J., Patel, T., & Brewin, C. R. (2009). Metacognitive therapy in recurrent and persistent depression: A multiple-baseline study of a new treatment. *Cognitive Therapy and Research, 33*(3), 291-300.

Wesselmann, E. D., Bagg, D., & Williams, K. D. (2009). "I Feel Your Pain": The effects of observing ostracism on the ostracism detection system. *Journal of Experimental Social Psychology, 45*(6), 1308-1311.

Whiffen, V. E. (1991). The comparison of postpartum with non-postpartum depression: a rose by any other name. *Journal of Psychiatry and Neuroscience, 16*(3), 160.

Whiffen, V. E. (1992). Is postpartum depression a distinct diagnosis? *Clinical psychology review, 12*(5), 485-508.

Williams, G. C. (1966/1996). *Adaptation and natural selection: a critique of some current evolutionary thought*. New Jersey: Princeton University Press.

Williams, G. C., & Nesse, R. M. (1991). The dawn of Darwinian medicine. *Quarterly review of biology*, 1-22.

Wolpert, L. (2006). *Malignant sadness: The anatomy of depression*. London: Faber and Faber.

Woodburn, J. (1982). Egalitarian societies. *Man*, 431-451.

World Health Organization. (1992). *The ICD-10 classification of mental and behavioural disorders: clinical descriptions and diagnostic guidelines* (Vol. 1): World Health Organization.

Wurtman, R. J., & Wurtman, J. J. (1994). Carbohydrates and depression. *The Neurotransmitter Revolution: Serotonin, Social Behavior, and the Law*, 96.

Wurtman, R. J., & Wurtman, J. J. (1995). Brain Serotonin, Carbohydrate-Craving, Obesity and Depression. *Obesity Research*, *3*(S4), 477S-480S.

Yeo, R. A., Gangestad, S. W., Edgar, C., & Thoma, R. (1999). The evolutionary genetic underpinnings of schizophrenia: the developmental instability model. *Schizophrenia research*, *39*(3), 197-206.

Zahavi, A. (1975). Mate selection—a selection for a handicap. *Journal of Theoretical Biology*, *53*(1), 205-214.

Zahavi, A. (1977). The cost of honesty: further remarks on the handicap principle. *Journal of Theoretical Biology*, *67*(3), 603-605.

Appendix

List of all Predictions From the Evolutionary Theories on Why Depression Exists

1.0. Predictions From the Social Competition Hypothesis

1.1. Depressed patients will have hostile thoughts towards inferiors, but subordinate thoughts towards superiors (Price, 1967); depressives will also be fearful of superiors.

1.2. Losing in competitive social struggles will activate the involuntary defeat strategy and, if the conflict is not resolved, results in clinical depressive behavior, thoughts and affects.

1.3. Exclusion from social groups (f.x bullying) activates involuntary defeat and thus terminates ambitions for the future (friendship, sexual partners, promotions). Later inclusion and acceptance in a social group reinstates ambitions.

1.4a. Depression is an effective social signal that is interpreted by others as “no threat” and “out of action”.

1.4b. Depression (and “low mood”) actually inhibits aggressive behavioral from rivals and higher-ranking individuals (”superiors”).

1.4c. Depression puts an individual into a “giving up” state of mind, which encourages acceptance (”yielding”) of the outcomes of social competitions, both cognitively and behaviorally, this behavior from the depressive leads to reconciliation of the conflict (Price et al., 1994).

1.4d. If the depressives “yielding” is blocked and/or prolonged, the Involuntary Defeat Strategy becomes intense and is turn into the clinical picture of depression (Price et al., 1994).

Proposed SCH predictions.

1.5. Individual humans inhibit/remove subjective goals (sex, friendships, promotions,

education etc) in depressive states, but not outside of depressive states.

1.6. Sudden loss of social rank (losing a professional authorization, pension) mediates negative thoughts about the self, and positive thoughts about high-ranking humans.

1.7. Subordinate behavior, as a response to losing in social conflict in hunter-gatherer societies, lower the chance for death and/or physical damage.

2.0. Predictions From Behavioral Shutdown Model and Incentive-Disengagement Theory

2.1. Depression is activated in individuals that are unable to, or does not want to, disengage from an unreachable goal (Klinger, 1975; Nesse, 2000).

2.2. Depression is frequent in people who lack alternatives to an unreachable goal, and who are anxious, duty-bound and ambitious (Nesse, 2000).

3.0. Predictions From the Third Ventricle Hypothesis.

3.1. If an individual loses social status, toxin(s) are released in their third ventricle, which produces depressive behaviors (Hendrie & Pickles, 2010).

3.2. The toxin that is released in depression (especially prolonged depressive episodes) is likely to damage cells in proximity to the third-ventricle that are unrelated to depressive behavior (Hendrie & Pickles, 2010).

4.0. Predictions From the Analytical Rumination Hypothesis

First claim (complex problems trigger depressed affect).

4.1a. Psychotherapeutic techniques which interrupts or avoid problem-solving thoughts will be less effective than therapy that encourages depressogenic problem-solving thoughts; encouragement of rumination should be more effective in relieving depressive symptoms than distraction.

4.1b. Antidepressives should be less effective relapse-preventer's than problem-solving therapies because symptom-reduction alone hinders the problem-solving properties of

depressive rumination.

4.2. Psychotherapy that explicitly focuses on the identification, and solution to, the fitness-hindering social problems that triggered patients depression, should be more effective, in the long-term, compared to other "active intervention" therapies, which do not have this focus.

4.3. Therapies where patients are encouraged to actively solve social problems in their life (regardless of the content of their rumination) are more effective than thought-pattern changing therapies.

4.4. The content of intrusive thoughts in depression should almost always concern social problems in the depressive's life, not general worries on nonsocial issues.

4.5. Depressive episodes are activated by the presence of complex social problems. The intensity of an episode (minor-major) is mediated by 1. The number of present problems, 2. The complexity of these problems (That is, the number of people involved and how close/important the involved individuals are) and 3. The avoidability of this problem (degree to which the depressive could have controlled the situation).

4.6. Depressives that engage in upward counterfactual thoughts solve complex fitness-relevant social dilemmas more efficiently than those who do not.

4.7. Depression and sad mood enhances efficient problem-solving of complex fitness-relevant social dilemmas.

4.8a. Depressed individuals solve complex fitness-relevant social dilemmas analytically (split a problem into subproblems and solve them in a step-by-step manner). Because of this, depressives solve social dilemmas faster than non-depressives.

4.8b. Feelings of sadness and depression are induced in non-depressed people when exposed to an analytically difficult problem.

4.9. The degree of analytical difficulty of a social dilemma should correlate positively

with the intensity of the depressed affect. Hence, the more there are tradeoffs between cooperating and pursuing self-interest and the number of people to keep track of, the more depressed affect will be induced (Andrews and Thomson, 2009).

Second claim (bodily changes to avoid disruption of analysis).

4.10. People with a high Working Memory (WM) function, will solve analytical social problems faster than individuals with a lower WM function. Hence, WM scores predicts length of depressive episodes (negative correlation).

4.11a. Because of a prolonged activation of glutamate neurons in the VLPFC (area involved in attentional control), depressives have increased production of astrocytic lactate.

4.11b. The activity of the serotonergic neurons in the VLPFC should be high, rather than low, in depressives.

4.11c. The VLPFC level of activation correlates positively with the severity of a patients depressive symptoms.

4.11d. The longer a depressive episode lasts (and hence the activation of VLPFC), the more likely the patient will start craving food rich in carbohydrates.

4.12a. The more anhedonic behaviors a depressive engage in, the more uninterrupted the depressives rumination are likely to be. Hence, anhedonia will predict how fast a depressive solve a complex social problem (Andrews and Thomson, 2009).

4.12b. Anhedonia are induced in a dose-dependent manner, according to the complexity of the evolutionary relevant analytical task an individual are trying to solve (Andrews and Watson, 2009).

4.13. Psychomotor retardation should be positively associated with rumination (Andrews and Watson, 2009). Hence depressives with strong psychomotor retardation will solve social problems faster than depressives without (given that the social problem they deal with are of similar complexity).

Third claim (depressive rumination solves the triggering problem).

4.14a. Severely depressed people (e.g. diagnosed with MDD) will solve their triggering social problem faster than people with a less severe depressive state (e.g diagnosed with a minor depressive episode)

4.14b. In experiments where mood is manipulated, participants effectiveness on solving fictitious social problems, will be determined by the strength of their melancholic mood (e.g. as measured on a depression scale). A very-sad group will outperform a sad group and so on.

4.15. Expressive writing, where the focus is on writing down thoughts and feelings, should enhance the long-term outcome of psychotherapeutic treatments of depression because it enhances the problem-solving nature of rumination.

Fourth claim (depression reduces performance on laboratory tasks because depressive rumination takes up limited processing resources).

4.16a. Depressives will perform similarly too nondepressives on laboratory (e.g. neuropsychological assessments) tasks if their rumination is temporarily stopped.

4.16b. Depressive rumination is very resistant to attempts of distraction, which leads depressives to perform badly at nonsocial tasks. However, depressives should outperform controls when the task at hand involves identification and solution of complex social problems

5.0. Predictions From the Social Motivation Function (Social Navigation Hypothesis) and Bargaining Hypothesis.

5.1a. Individuals with Major Depressive Disorder elicit help from a broad set of important social partners (Watson, 2008).

5.1b. When genuine, honest help is provided to a depressive, the depressive episode will end.

5.2a. MDD is induced in an individual if that person has 1) a need for social for assistance and 2) conflicts with important social partners.

5.2b. Because MDD serves as a strategy to manipulate others, depressives are often met with negative reactions from their social group (Hagen & Thomson, 2004; Watson, 2004). The depression is usually resistant to those negative reactions.

5.3a. Motivational hypothesis, Niche change: The number of family and friends who resist a depressives attempts to change their social niche (and to which degree), are positively correlated with the strength of the depressives symptoms.

5.3b. If a depressive start to perceive that niche-changes is unnecessary or unfeasible, the depressive symptoms lifts (Watson, 2008).

5.4. Motivational hypothesis, specific partner: A depressives symptomatology differentiates based on whom it is designed to lay costs upon, and hence to motivate that particular person to help.

5.5. Depressive symptoms (in the long-term) are always reduced to the degree that the triggering problem is solved (degree of help extorted from allies and kin), or perceived to be solved.

5.6a. Depression only helps to motivate people who are positively interested in the depressives fitness (relatives, partners).

5.6b. People become more depressed when they are in conflict with partners who have a greater positive fitness interest in them (as measured by help received in the past). (Watson and Andrews, 2002)

5.7. The lethal risk of suicidal behavior in a depressive increases depending on how effective the suicidal behavior is in recruiting help from their significant others (Watson & Andrews, 2002).

6.0. Predictions From the Social Risk Hypothesis

6.1. Depression is a low-variance, risk-averse strategy which is activated in dire social situations. That is, situations where the depressives value/burden ratio is unfavorably low.

6.2. Phenomenologically, a human that perceives herself as having a high social burden and low social value (low SIP), will experience low self-esteem and portray the typical "symptoms" of sadness and mild depression.

6.3a. Perceptions, and threats, of social exclusion (e.g. from dyadic relationships, groups) leads to depressed affect.

6.3b. The depressed state leads to a hypersensitivity of social loss (and more correct identifications when social risks are present), and not hypersensitivity to non-social matters.

6.4a. When an individual is in danger of social exclusion, sadness, and mild depression decreases the likelihood that the individual is indeed excluded from their social group (Allen & Badcock, 2003).

6.4b. Mild depressed mood increases the likelihood that the depressed individual preserves their social relationships.

6.5. Any organism that lack the ability to calculate social value and burden should be less depressed (e.g. children, adults with pathologies, non-human species without this ability) (Allen & Badcock, 2003).

6.6. Sad and mildly depressed individuals are more sensitive to social risk when such risks are present.

6.7a. Social cues, such as facial behavior, voice characteristics, verbal content and eye gaze can signal exclusion and therefore induces mild depression in humans across cultures.

6.7b. People who are mildly depressed have had more negative interpersonal encounters in their history than others (e.g. experienced exclusion, the social cues from 7a).

6.7c. Negative interpersonal experiences always lower self-esteem first, which might

develop into mild depression later (self-esteem mediates depression).

6.8a. People, and organisms in general, who lack the ability to calculate social value and burden, should either lack, or show less evidence of depressive behavior. Human children, or humans with specific brain/cognitive pathologies (which show evidence of deficits in value/burden calculations) should be less depressed.

6.8b. Non-human species (most likely mammals with past social selection pressures) that are able to calculate social value and burden have a capacity for depressive behavior patterns.

6.9. The lack of appetitive functions in depression (desire for food, sex and so on) reduces the depressives social burden, which makes other people less likely to exclude the depressive from their group.

Nettle's (2009) Predictions Relevant for the Social Risk Hypothesis.

6.10. Mania and agitated depression is activated under extremely dire social situations, and therefore functions as a "risky" strategies when the social burden (low SIP - danger of exclusion) is dangerously high.

6.11. If an individual perceives its situation as absolutely dire, it is more likely to have an agitated depression, or have symptoms of mania, if the individual perceives it's situation to be merely "poor", however, it is more likely to have retarded depression (slow psychomotor activity and thinking). (Nettle, 2009)

Dunn, Whelton and Sharpe's predictions (2012) on the Social Risk Hypothesis.

6.12. Insecure attachment, unfavorable social comparison and social defeat should predict depressed mood.

6.13. Insecure attachment, unfavorable social comparison, and defeat as predictors of depression would be mediated by self-esteem.

6.14. Depression should predict and mediate three consequences in the social realm:

inhibited social risk-taking behavior, submission, and interpersonal sensitivity.

Proposed predictions for the Social Risk Hypothesis.

6.14. Individuals in a hunter-gatherer environment (as close as possible to the EEA), who fail to become mildly depressed when threatened with exclusion, are more likely to actually be ostracized, relative to individuals who react with mild depression.

6.15. Socially reactive sadness and mild depression are not explained by genetic mutation or heritability (the genes predisposing for this are fixated; part of human nature), however, major depression (especially non-reactive) can be explained by mutation/genetic heritability.

7.0. Predictions From the Reproductive Failure Theory and the Sex Difference in Depression (from Suarez & Gallup, 1985, p. 285)

7.1. Because of greater genetic assurance, limited reproductive potential, and being required to make a greater biological commitment (e.g. pregnancy and child rearing), females should be more prone to become depressed in response to reproductive failure than males.

7.2. Women who have had previous children should be less depressed in response to child loss (neonatal death, stillbirth, abortion, etc.) than childless females.

7.3. Sterilization should produce depression in females, but be inversely proportional in severity to the number of surviving offspring.

7.4. Within limits imposed by family size and availability of resources, pregnancy should tend to antagonize depression.

7.5. Postpartum depression should be related to the extent that hospital and obstetrical practices simulate child loss.

7.6. Since with increasing age women tend to remarry less often than men, depression in response to the death of a spouse should be higher in females than males of the same age, and should be more severe for childless widows.

7.7. The incidence and severity of depression during menopause should be inversely proportional to the number of surviving offspring and, other things being equal, should be less common in females who have become grandmothers by the time menopause occurs.

7.8. Depression in response to reproductive failure should be tempered by the reproductive success of kin.

7.9. In cases of infertility, child loss, or menopause, the adoption of a child, bringing foster children into the home, or even obtaining a pet as a child surrogate may have therapeutic value in alleviating depression since they allow one to act out the proximate roles of parenting and/or grandparenting.

7.10. Divorce rates ought to be higher in couples who have experienced infertility, miscarriages, stillbirth, neonatal death, or menopause.

7.11. The occurrence of depression as a response to reproductive failure should be a function of the extent to which a species is R- vs K-selected.

Proposed complimentary predictions of the Reproductive Failure theory.

7.12. The death of a child produces stronger depressive symptoms in women, compared to men. Particularly if 1. The child dies after menopause, 2. The dead child is the parent's only child, and 3. If they have no grandchildren. The same pattern should be observed in men, albeit to a lesser degree.

7.13. The loss of a child induces less severe depressive symptoms in younger women, than in post-menopause aged women.

7.14. Women born without a uterus and ovaries, are relatively more depressed than women in general.

7.15. Ovariohysterectomy, the surgical removal of the uterus and ovaries in women, induces depression (the severity, again, should negatively correlate with the number of children and grandchildren), the surgical removal of the testicles, orchiectomy, too, induces

depression in men. However, to a lesser degree than in women (men, too, show the same negative correlation pattern with children, albeit a much weaker one). This pattern should be observed cross-culturally.

7.16a. Men with more feminine personality traits, and less circulating testosterone in their blood stream, should be more prone to developing depression in general, and particularly after reproductive loss.

7.16b. Testosterone-agonists (metabolic steroids) have short-term antidepressive effects on both men and women.

16c. Men and women on testosterone-agonists show less severe depressive symptoms after reproductive loss compared to men and women not on testosterone-agonists.

Proposed tests of Suarez and Gallup's (1985) ultimate functions (learning and help-elicitation.

17. Females in hunter-gatherer societies who are depressed due to reproductive failure, receive more help from kin, than those who are not depressed (relevant for Bargaining Hypothesis too).

18. Compared to males, females will be less likely to repeat the parenting behaviors that led to child loss (Females show a greater degree of behavioral flexibility after child loss).

19. If a parent's behavior was related to the loss of a child, such as failure to provide sufficient attention to dangers, protection, food and so on, the female's changed behavior will be more related to future fitness, than if the male changes parenting strategies.

8.0. Predictions From the Social Competition Hypothesis and Women's Depression

8.1. If women have equal social opportunities as men, the sex difference disappear (Price et al., 1994).

8.2. Less economic and social inequality (e.g. hunter-gatherer societies) between the sexes reduces the sex difference in depression.

9.0. Predictions From the Analytical Rumination Hypothesis and the sex difference in depression

1. Women, relative to men, spends more cognitive resources on avoiding social stressors (rumination), and are expected to be more depressed. However, in turn, they avoid, and solve social problems more effectively than men.

2. Inadequate resources and absent support and protection from family and friends, particularly perinatally, should increase the rate of depressive episodes *to a larger degree* in women than in men.

10.0. Predictions From the Bargaining Hypothesis and the sex difference in depression

10.1. Women are more often victims of social manipulation. Especially in EEA-similar (hunter-gatherer) environments where women resides with their husbands relatives which should lead to more conflict, this induces depression in order to solve the conflict and elicit help.

10.2a. Depression (including MDD) is a safe and slow strategy for conflict-resolution for women, but to a lesser degree for men (Hagen et al., 2004).

10.2b. Women receive more resources when they are depressed compared to men.

11.0. Predictions From the Defection Hypothesis and the sex difference in depression (postpartum depression)

Hypothesized etiological factors of PPD (From Hagen, 1999, p. 333).

11.1. There is insufficient investment from the father, family or others to successfully raise the offspring

11.2. There are problems with pregnancy, birth, or with the infant that indicate that this offspring may have low viability, that is, is unlikely to survive to reproductive age.

11.3. Environmental conditions are poor for raising an offspring (e.g. harsh winter, insufficient resources)

11.4. There are large opportunity costs—investment in the offspring precludes investment in other beneficial activities. In this case, investments directed towards offspring would be more profitably directed toward:

11.4A.existing offspring

11.4B. The mothers own survival, growth and development, and thus her ability to invest in future offspring

11.4C. Finding a better mate

Defection hypothesis, part one (Hagen, 1999):

11.5. Lack of social support should predict negative affect

11.6. Low infant viability should predict negative affect

11.7. Poor environments should predict negative affect

11.8. PPD should be universal

11.9. PPD is not a hormonal byproduct

Defection hypothesis, part two (reduce cost).

11.10. Mothers with PDD are 1. More likely to have thoughts of harming their infant (Hagen, 1999) and 2. Mothers with PDD are more likely to kill their infants. This risk increases if the PDD has not elicited social support, or if the infant is unhealthy/has deformities.

11.11. Unwanted pregnancy predicts PPD.

11.12. Mothers with PDD lose interest in their newborn child, but often not in existing children.

11.13. Mothers with PPD mother less: caring *behaviors* decrease.

Defection hypothesis, Part three (negotiation).

11.14. PDD predicts greater investments from the depressed woman's social group, particularly from the father.

Proposed group-level predictions of the Defection Hypothesis:

11.15a. The variables *investing father* and *Healthy infant* should both be causal factors in decreasing the risk for PPD in all mothers.

11.15b. Conversely, the variables non-investing father and unhealthy infant strongly predicts PDD in mothers.

11.16. Societies where single-motherhood is more common, have more incidences of postpartum depression, particularly if these mothers do not receive benefits from the state (or anyone else).

12.0 Predictions From the Social Risk Hypothesis and the Sex Difference in Depression

12.1a. Social thwarting of important, evolutionary biological goals (reproduction, survival, resource acquisition, kin investment and so on) activates risk-averse social strategies (manifested as depression) in humans;

12.1b. These depressive, risk-averse social strategies help an individual achieve their biological goals (Relative to humans who respond by non-depression strategies when their biological goal are thwarted);

12.1c. These strategies are particularly useful for women - women are more likely to attain their biological goals with risk-averse social strategies than men.

12.2. Women are more likely to perceive social threats, and make negative self-evaluations on the basis of it (Allen & Badcock, 2003).

13.0 Predictions From Sexual Selection Theory and the Sex Difference in Depression (from Martel, 2013, p. 1239)

13.1. Adolescent stressors will have a particularly prominent effect on adolescent-onset internalizing disorders and associated sexually selected traits (i.e., negative emotionality, empathy, rumination) for females compared to males (sex as moderator).

13.2. Rising levels of ovarian hormones will interact with the HPA axis and cortisol and oxytocin (moderators) to increase sensitivity to the interpersonal environment during adolescence via increasing negative emotionality, empathy, and rumination (mediators; mediated moderation), particularly for females (sex as moderator; moderated mediation).

13.3. Ovarian hormones directly and indirectly via interactions with cortisol and oxytocin will influence amygdala function and serotonergic neurotransmission, with particularly adverse effects on negative emotionality, empathy, rumination, and adolescent-onset internalizing disorders in response to interpersonal stressor exposure, particularly for those with genetic risk influencing the serotonergic neurotransmission system.

13.4a. Females will be differentially susceptible to stressors that provide information about the cohesion of interpersonal relationships (e.g., social support, intimacy) at puberty, compared to males, because they have a large impact on intrasex competition, mate choice and retention, pregnancy, and child-rearing, and these stressors will impact the sexually selected traits of negative emotionality, empathy, and rumination, traits that can facilitate the development of interpersonal relationships and increase risk for adolescent-onset internalizing disorders.

13.4b. Thus, interpersonal stressors (IV) will influence negative emotionality, empathy, and rumination (mediators), particularly in females compared to males (sex as a moderator), and these traits will increase risk for adolescent-onset internalizing disorders (mediated moderation model).

13.5. Culture will play a particularly important role in influencing the types, intensity, and chronicity of interpersonal stressors encountered during puberty and adolescence and will exert strong effects on the sexually selected traits of negative emotionality, empathy, and rumination, as well as prevalence of adolescent-onset internalizing disorders.

14.0. Predictions From the Infection-Prevention Perspectives.

From Raison and Miller (2012):

14.1a. Allelic risk variants for depression should be located in genes with known immune effects.

14.1b. Allelic risk variants for depression should Increase signaling in inflammatory defense pathways.

14.1c. Allelic risk variants for depression should increase survival in the context of infection.

14.2. Depression should be associated with increased inflammation and inflammatory activation should induce depression.

14.3. Allelic variants that increase the risk for MDD should enhance host defense mechanisms in general and innate immune inflammatory responses in particular

14.4. Genes promoting inflammatory responses following psychosocial stress should decrease in prevalence in societies where associations between stress and infection are reduced (because of health practices etc)

14.5a. MDD patients have elevated body temperature and reduced bodily iron stores.

14.5b. Vigilant behavior (and stress hormones) is only activated in depressives when the depressive, inflammatory response has been prolonged over a long time.

14.5c. Anorexia in depression help to defend against infections more effectively; anorexic depressives , relative to non-anorexic depressives, are less likely to die from infectious agents.

Proposed predictions of the infection-prevention views.

14.6. Humans diagnosed with MDD are more likely to have an infection, but less likely to die from infection, compared to infected non-depressed individuals.

14.7. Depressed people are more likely to have an infection; but non-infected

depressed individuals are less likely to suffer from infectious diseases; depressive behaviors reduce the chance of being infected.

14.8a. Individuals from populations with relatively higher prevalence of infectious disorders (e.g. Malaria in sub-saharan Africa) are more likely to show depressive symptoms.

14.8b. Individuals from populations where depression is relatively more prevalent (due to other reasons than pathogen-defense) have less chance to die from infection (after controlling for pathogen-load in the environment).

14.8c. People with immunological deficiencies are less likely to be depressed, than people with normally functioning pathogen host defense.

14.9. Body temperature and iron-depletion increases under a depressive episode, and down again at the end of a depressive episode. This correlation relationship is seen both within and across individuals, and is observable in all forms/severities of depression.

14.10. Environmental stressors for humans in the EAA were reliably associated with open wounds, which still produces the current association between psychosocial stress and depression.

14.11a. Antibacterial drugs alleviates depression.

14.11b. Increased handwashing will indirectly lower the prevalence of depression in populations.

Predictions from Kinney and Tanaka (2009)

14.12a. Sons and daughters that perceive “sickness behaviors” in their mother should avoid her presence, and resume it when the sickness behavior ends.

14.12b. Mothers are motivated to avoid their children’s proximity whenever sickness behaviors are activated.

14.13. Depressed individuals should be highly motivated to avoid sexual contact, and thus avoid sexually transferable pathogens.

15.0. Predictions From Nettle's (2004) Affective Reactivity Model

15.1. Depression is caused by variation around the adaptive peak of neuroticism, hence, very low and very high neuroticism is associated with fitness-costs.

16.0. Predictions From Mutation-Selection Balance and Major Depression

16.1. Individuals with major depression have higher-than-average mutational load. Hence, they are more likely to have other hallmarks of mutation (such as physical asymmetry).

16.2. Individuals with major depression are more likely to have children who have disorders caused by genetic mutation, especially if the spouse also has major depression.