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Empathy and depression: the moral system on overdrive

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4.1 Introduction

This chapter describes the intimate connection between empathy and depression, the epidemic of our modern world. While depression has been described as a ‘disorder of the self’, it may be more accurately characterized as a disorder of ‘concern for others’. People who are depressed most often have normal or elevated levels of empathy; however, their affect-directed, automatic causal interpretations of pain in others are often disturbed, leading to non-conscious assertions of blame, usually placed on themselves. Empathy, a socially organizing neural system, allows us to share others’ feelings, to mimic without awareness, and forms the basis of our relationships and our social learning (Decety & Jackson, 2004).

A sophisticated Theory of Mind (ToM), or the ability to know what others are thinking, is sometimes considered a prerequisite for true empathy. The capacity for empathy, present in infants from the first days of life, may be independent of cognitive maturity and a developed ToM. Healthy empathy, however, requires an understanding of causality, undeveloped in very young children and affectively distorted in depression. The empathic reaction in depressives often leads to great distress because they tend to unrealistically blame themselves for pain felt by others. Thus, in mood disorders, the empathy system may be functional; however, an overly active and automatic moral system, connected to the empathic experience, tends to misinterpret attribution, and the guilt

felt at believing that you have caused pain in another leads to empathic distress, an exaggerated reaction.

Ubiquitous in mammalian species (Preston & de Waal, 2002), empathy is reflexive and non-conscious, occurring without awareness. The empathy system is active in all interpersonal encounters. Through a system of mirror neurons, people react to witnessing motor actions as well as to emotions, as if they themselves were having the same experiences and feelings. In empathic responses, people are literally feeling others’ feelings, while maintaining a separate sense of self. While watching others, people mirror them as they engage in social conversations, experience emotions such as pain and distress, or participate in positive and pleasurable feelings and activities that bond people together, such as smiling, laughing, touching affectionately, reconciling after a break in connection, and forgiving after being harmed by another (Berry et al., 2005; Farrow et al., 2001; Keltner et al., 2006). Blair (1997) observed that psychopaths have a deficit in the empathy system, leading to a lack of normal moral judgement. In contrast, people afflicted with depression are empathic, and yet they often fail to make normal moral assessments and this may be a fundamental dysfunction characterizing mood disorders.

The limbic and paralimbic system structures, found active in emotional empathy, automatic moral decision-making and guilt, are, broadly considered, those also found active or hyperactive in people suffering from depression. A deficit of empathy appears in a few mental disorders that have low prevalence rates, namely sociopathy, autism, Asperger’s and some psychotic disorders, and in those with injury resulting from strokes, or other forms of damage. In contrast, people suffering from mood disorders are marked by functional and structural neural changes in the neural circuit associated with empathic responses; however, they exhibit a normal degree of empathy, or even a surplus in some cases. Depressives are rarely thinking exclusively about the self; instead, they are often dwelling on how they might endanger others, or on their beliefs—often false—that they have harmed others in the past. Depression is highlighted by excessive empathy-based guilt, and in our laboratory, we have repeatedly found empirical evidence of the connection (O’Connor et al., 2002). Presently, multiple lines of evidence are converging to support the connection between depression, empathy and an overly active or misattributing moral system.

Advances in social, clinical and personality psychology, along with findings from neuroscience, psychiatry, molecular biology and psychopharmacology support a biological perspective of normal and abnormal sociability, with empathy at the centre of attachments. Empathy is perhaps the heart of mammalian development, limbic regulation and social organization (MacLean, 1985). The loss of attachments often initiates the onset of depression. Depressed individuals are
eager to maintain relationships and to be of help when needed. However, they may fail in efforts to help and to remain socially connected, and often lose the affection needed for biological regulation. Their limited capacity to effectively help others is mirrored by their failure to help themselves or to routinely act in their own best interests, due to passivity, a symptom of depression. Failures in efforts to help and failures in relationships increase the severity of depression. Adding insult to injury, people suffering from depression interpret these failures as further evidence of their ‘moral inferiority’.

Depressed patients often appear withdrawn and reclusive, their worries about others remaining silent and internal. Although it seems that no one understands the depth of their despair, their fear and sadness are reflected in the empathy system of their caretakers. However, instead of feeling more alert to the pain of those suffering from depression, caretakers may react like the depressive, by feeling overly responsible and guilty, resulting in their own withdrawal, and sometimes blaming depressives for their troubles. Like other pervasive, negative emotional states, depression is thus often contagious, first touching and then frustrating empathy in others. As multidisciplinary research continues to increase our knowledge of this isolating, chronic and relapsing illness, mental health providers, families and friends may be better able to maintain their natural empathy towards those afflicted with depression.

4.2 Depression: prevalence, costs and theories of aetiology

Depression is the most common mental disorder on our contemporary landscape, affecting millions of people worldwide. The World Health Organization has estimated that by 2020 depression will be the major source of disability in the developed world, and the second most important cause internationally (Ingleby, 2004). The prevalence of depression has been rising steadily since the middle of the last century; the rate in the United States has escalated from 2% of the population in the 1960s to 25% in the 1990s and 28.8% of Americans will experience a mood disorder sometime during their lifetime (Kessler et al., 2005). It has been estimated that over 19 million people in the United States experience depression yearly, accounting for 4.7 million office visits and costing over $30 billion annually. Untreated depression costs billions annually. Suicide is the second leading cause of death for young people today, eclipsed only by motor vehicle accidents.

Rates of depression are consistently higher in women, with 12% affected by mood disorders each year compared to 7% of men. Mood disorders are afflicting our children and adolescents as well, with rates ranging from 2.5% to 8.3% in the United States. The criteria identifying depression in children have broadened, and
now include the angry and defiant behaviours previously diagnosed as conduct and attention deficit hyperactivity disorders. Depression in the geriatric population is estimated to affect 1% to 2% of people over the age of 65, and 13% to 24% of the elderly suffer from subclinical depression, placing them at risk for major depression and suicide (Kessler et al., 2003).

The diagnosis of depression is complex, as clinicians are confronted with symptoms indicative of unipolar depression, bipolar I, bipolar II, or dysthymic disorder, with each diagnosis frequently associated with symptoms of anxiety disorders. Bipolar I and II are often misdiagnosed as unipolar depression and then undertreated, or treated with the wrong medications, resulting in rapid cycling or a worsening of depression (Strakowski, 2002). Patients suffering from bipolar disorders have also often been misdiagnosed as personality disordered and provided no psychopharmacological treatment, thus leading to a worsening of their condition.

The personal, social and economic costs of depression have resulted in international research efforts focused on etiology, underlying biological and social mechanisms, effective treatments and prevention. Multiple biological factors have been associated with depression, including genetic heritability (Neumeister et al., 2004) and distinct brain dysfunctions (Caetano et al., 2004; Drevets, 2001; Goldapple et al., 2004). Recent studies suggest that levels of brain-derived neurotrophic factor (BDNF), which functions to protect neurons, are lower in depressed people. Neuroticism, significantly associated with the BDNF gene, is a personality factor consisting of attributes such as high sensitivity to negative emotions, fear of rejection and proneness to worry. Heritable by 40% to 60%, neuroticism is also associated with depression, which is heritable by 35% to 50%. One of the two variants of the BDNF gene, the Val allele, is associated with higher scores on neuroticism and depression (Sen et al., 2003). The Val allele is also implicated in bipolar disorder, impulse control syndromes, schizophrenia and addiction. BDNF protects hippocampal neurons in chronic stress conditions and in depression, which, if left untreated, results in neuronal death, with shrinkage of the hippocampus and prefrontal cortex. The other BDNF variant, Met, is associated with higher levels of BDNF, lower scores in Neuroticism and a significantly lower risk of depression. The BDNF gene appears to affect both serotonin and dopamine. While treatment with selective serotonin reuptake inhibitors (SSRIs) raises BDNF, lower levels are associated with the destruction of dopamine, central to the reward pathways. Using a scale consisting of attributes derived from psychiatrists’ interviews of patients, we found high scores on the ‘Low-Dopamine’ subscale of the Neurotransmitter Attributes Questionnaire (NAQ; O’Connor et al., 2005) to be associated with depression, along with high scores on neuroticism, and empathic guilt. This connection with high neuroticism supports
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the BDNF theory while the findings related to guilt provide the link to empathy (O'Connor et al., 2005).

Various treatments, such as the SSRIs, monoamine oxidase (MAO) inhibitors, mood stabilizers including lithium and valproic acid, electroconvulsive therapy (ECT) and physical exercise, increase levels of BDNF (Russo-Neustadt et al., 2001). Other genes related to the dopaminergic and serotonergic systems have been implicated in vulnerability to mood disorders, suggesting that they are polygenetic illnesses. Twin and sibling studies and research in molecular genetics, psychiatry and psychopharmacology are contributing to our understanding of the heritability of depression. Patients' genetic profiles in combination with symptoms will likely be the basis of diagnosis in the future. Stressors on pregnant women have been shown to have negative effects on the neural system in developing infants, including a lack of emotional support and traumatizing experiences during pregnancy such as terrorism and war. Neurotoxin exposure (Masters, 2001), and pre- and post-natal bacterial and viral infections are also associated with vulnerability to depression. Even a single neurotoxin may be implicated; for example, high blood mercury levels in dentists are associated with low BDNF, along with symptoms of mood disorders (Heyer et al., 2004).

Negative childhood experiences are also related to the etiology of depression (Westen, 1998). Family stressors, disturbed or embattled parents, and poor socioeconomic conditions are found significant, and environmental factors are known to influence gene expression. Environmental conditions connecting high levels of empathy and depression were initially studied by developmental researchers (Eisenberg, 2000; Zahn-Waxler, 2000) and a few clinicians (Niederland, 1961; Modell, 1971; Weiss, 1993). Some practitioners continue to consider childhood experiences primary in the etiology of depression, while minimizing biological factors. Others explain depression as a function of an egocentric, anti-social and maladaptive unconscious mind, a fundamental construct in psychoanalytic theory. Biased by the belief in a maladaptive unconscious, it is easy to mistake the flat, anxious and passive responses of depressed patients as self-centred.

Depressed patients may also be seen as egocentric because they often fail to disclose their worry about others, and instead describe themselves as selfish. Clinicians from differing perspectives, including some who practise evidence-based therapy and biological psychiatrists dispensing medication, may refer to 'resistance to treatment', viewing depressed patients as hostile and lacking in empathy. However, as cognitive social neuroscience provides an empirically based replacement for the Freudian unconscious with the 'new unconscious', an adaptive and social mental framework will more likely guide the treatment of depression in the future (Fassin et al., 2005; Kihlstrom, 1987).
4.3 Empathy and developmental pathways to depression

Over 30 years ago, developmental psychologists began to identify the links between high sensitivity to others’ distress, proneness to worry, empathic guilt and vulnerability to depression. From observations of infants and their mothers in naturalistic and laboratory settings, associations between empathy, guilt and later proneness to depression, particularly in females, were noted (Nolen-Hoeksema et al., 1999; Zahn-Waxler, 2000). Research designed to help parents teach their children empathic and prosocial responses to distress in others led to the discovery of significant links between guilt, empathy and moral development (Hoffman, 1975, 2000). Rosenfeld et al. (2000) reported a correlation between empathy for distress in others and depression. Murray (2004) found that runaway adolescents in foster care had significantly more empathy-based guilt compared to non-troubled adolescents. Hay and Pawlby (2013) found that prosocial children worried about their families’ well-being and suffered more than their less-worried peers from internalizing problems.

Zahn-Waxler (2000) described ‘the presence of an early developmental pathway where surges of empathy, as well as guilt can place individuals at risk for later depression’ (p. 226). The capacity to respond to others’ distress appears early. Only one day after birth, infants react to the distress cry of other newborns with greater intensity than to the cry of a 5-month-old baby, a white noise, a synthetic cry, or a recording of their own distress cries (Martin & Clark, 1982; Sagi & Hoffman, 1976). From 4 months, infants attempt to engage depressed mothers (Cohn et al., 1990); and between 12 and 18 months, they make overt efforts to help others in distress (Zahn-Waxler et al., 1979). This sensitivity to people’s emotions continues into adulthood. For example, Lane and DePaulo (1999) found that depressives exhibited heightened sensitivity to emotional dishonesty; the depressed subjects were better able to detect deception in dishonest feedback than the non-depressed sample.

Zahn-Waxler (2000) noted that early dysregulation of the moral system occurs when biologically vulnerable infants fail to engage depressed mothers, who appear to elicit a heightened sense of responsibility for others’ emotions, along with chronic guilt. High empathy in children is a risk factor for later depression, if they are over-involved, self-blaming and distressed, with unregulated negative affect (Klimes-Dougan & Boiger, 1998). Recent findings suggest that the acquisition of a Theory of Mind in girls at an unusually early age is a predictor for high empathy-based guilt by adolescence, and may be associated with depression later (L. Rasco, personal communication, 2004).

The problems about which depressed patients feel like failures are ordinarily beyond their control; they can neither prevent nor resolve the distress they witness,
but are feeling guilty for imaginary crimes. Despite intense concern for others, in depression empathic responses may fail to result in effective action even when it is possible. Symptoms such as passivity and withdrawal, common in depression, often inhibit altruistic behaviour and the ability to act in general. Nevertheless, in empirical studies we have found that empathy-based guilt, though associated with depression, is also associated with acts of altruism towards family members, friends and strangers (Crisostomo et al., 2005). Figure 4.1 provides a structural model of these relationships.

Depression may render people unable to think clearly about helpful strategies or to carry out plans to come to the aid of others; thus, the connection between empathic concern and acts of altruism may sometimes be severed. Some suggest

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**Figure 4.1.** Structural model: this presents a structural model illustrating that guilt (based on worry about others) appears to have a significant and positive influence on engaging in altruistic behaviours except when it leads to empathic distress and neuroticism. Neuroticism, a marker for brain-derived neurotrophic factor (BDNF) and a high risk factor for depression, appears to inhibit altruistic actions. The cumulative fit index = 0.96. All path coefficients were statistically significant at the 0.001 level.
that excessive distress on witnessing distress in others results from overidentification and failure to distinguish self from others and, therefore, is not authentic empathy. However, it seems more likely that cognitive limitations accompanying depression have an impact on effective action at all levels: shrinkage of the hippocampus after a flood of excess cortisol and other stress hormones affects memory adversely. Furthermore, overactivity of the amygdala resulting from misunderstood input may lead to hyper-emotionality further disturbs cognitive processing. The dysfunctions of the prefrontal and orbitofrontal cortex found in depressives may impact other cognitive capacities, including planning and decision-making. Orbitofrontal and prefrontal cortex dysfunctions may increase inhibitions, in contrast to the uninhibited behaviour observed after brain-damaging accidents affecting the orbitofrontal cortex. Thus symptoms of withdrawal and passivity in part reflect the temporary decrease in cognitive competence in depression, and the associated inability to be effective contributes to the perception that depressed people are selfish. Despite these changes that affect cognition, the depressive remains highly attuned to others, but unable to effectively help them.

4.4 Survivor guilt and depression

While clinical literature often fails to mention empathy and its connection to depression, the association between guilt and depression has been long acknowledged, and guilt is a criterion for major depressive disorder in the Diagnostic and Statistical Manual of Mental Disorders, 4th edn. Text Revision. The failure to note the connection between guilt and empathy follows from the Freudian perspective on guilt. In psychoanalytic theory guilt is viewed as a manifestation of unconscious hostility and rivalry, beginning with the child's desire to kill his same-sex parent in the oedipal struggle and ending with an internalization of the avenging parent intent on castration. This theory, so pervasive in the middle to late twentieth century, continues to be influential throughout our culture, affecting psychological explanations of both normal and abnormal processes. However, current research characterizes guilt as a prosocial emotion tied to empathy and the desire to maintain social bonds (Baumeister & Leary, 1995; O'Connor, 2000). The connection between empathy and depression is highlighted in survivor guilt.

Survivor guilt was observed by Darwin and Freud as each described the guilt one feels following the death of a loved one. The term 'survivor guilt' came to life when Niederland (1961) studied the severe depression and anxiety in survivors of Nazi concentration camps. He found them suffering from guilt, simply for being alive while their families had all been killed by the Nazis, as if their own survival had somehow caused the death of their families. Modell (1971) expanded the construct to include the guilt people feel when they believe they are harming others, by being
successful, or happy. He noted that depressed patients often held the belief that if they had good fortune, success, or happiness, it was at the expense of other family members, who might then be less fortunate because there was a limit on how much ‘good’ could be had in a family. Weiss et al. (1986) followed with extensive clinical observations linking survivor guilt to depression and other psychological problems, forming the foundation for his theory of psychopathology and treatment, with altruism, the fundamental but often hidden human motivation, replacing the antisocial wishes and feelings held central by the Freudians. He proposed that people feel unconsciously compelled to help family and social group members, even when helping is at their own expense or personally costly. In line with these observations emphasizing human strengths rather than weaknesses, and in contrast to the psychodynamic perspective, Weiss conveyed a positive view of human nature. He also observed that people think and plan non-consciously, much as they do consciously, which heralded discoveries in social cognitive neuroscience and positive psychology in the last decade.

Weiss et al. (1986) observed that patients enter psychotherapy with an unconscious plan to test grim and inhibiting pathogenic beliefs warning them not to pursue normal goals, for fear of surpassing someone in the family and thus making them feel inadequate by comparison. He proposed that patients’ plans to test and change pathogenic beliefs were purposeful. Arguing that patients were not gratified by their problems, as proposed in analytic theory, Weiss held that patients were guided by the adaptive unconscious and determined to overcome their problems. In collaboration with Samuelson, Weiss et al. (1986) supported his clinical observations upon which he built this more positive theory, with empirical single-case design psychotherapy research (Weiss et al., 1986). Following Weiss, and in the context of the paradigm shift occurring in psychological science, we developed our programme of research to empirically test hypotheses derived from this new model of the mind and motivation (O'Connor et al., 1997).

In our empirical research, we found significant associations between survivor guilt, empathy and depression. Survivor guilt is an empathic emotion often occurring without conscious awareness. We feel survivor guilt – albeit just slightly unconsciously – when we get a promotion or hear that a paper has been accepted for publication, while a friend just heard he was laid off or that he was going to be refused tenure. We feel survivor guilt when we are healthy and a friend calls one evening to tell us that she has been diagnosed with breast cancer. Survivor guilt is unusual in that we often fail to recognize it when we feel it most acutely; nor is it perceived by others watching our facial expressions or bodily movements. However, the presence of survivor guilt is often marked by submissive behaviour; we may put ourselves down and act as if we are lower in status than the person for whom we feel sorry. Survivor guilt may come to our attention only after we get
self-destructive, in an effort to reduce its impact, by trying to 'level the playing field' and 'make things equal'.

The self-damaging behaviours resulting from survivor guilt may be self-defeating, but at the same time they are empathic acts of altruism aimed at preventing feelings of inadequacy in those perceived as less fortunate. The net result is a self-destructive but common cycle. The show of submissive behaviour often elicits negative reactions from others who then reevaluate the social ranking of the guilt-prone person. As witnesses look down on people who respond to survivor guilt with submission, they treat them as inferior. Though perhaps reducing guilt in the guilt-prone person, these social interactions are emotionally dysregulating and enhance feelings of depression if a mood disorder is already present or set off an episode in a vulnerable person. Thus, people who become submissive in an effort to help others often unwittingly become altruistic martyrs.

4.5 Programme of research: empathy, guilt and depression

Our programme of research on empathy, altruism, guilt and depression was initiated to test our biological, relational and affective theory of psychopathology and psychotherapy (Lewis et al., 2000; O'Connor, 2000; Weiss, 1993). In line with our social and adaptive perspective on the mind, we hypothesized that people who suffer from depression and other common psychological problems score significantly higher in interpersonal guilt than those free of depression. In contrast to popular opinion, we also hypothesized that depressives are equal to or higher in empathy than non-depressed people.

We began our series of studies with the development of a measure designed to operationalize the constructs found useful in clinical work and central to our theory, placing altruism as a fundamental motivation, often manifested in survivor, separation, and overly responsible, omnipotent guilt. In prior single-case studies, it was found that survivor and separation guilt were the primary focus of each empirically developed case formulation and that interventions successful in reducing guilt were predictive of immediate and longer term positive outcome. The Interpersonal Guilt Questionnaire (IGQ-67: O'Connor et al., 1997) was initiated by collecting statements typical of guilt-prone patients. These were categorized into four subscales: Survivor, Separation/Loyalty, Omnipotent Responsibility Guilt and Self-Hate. The first three subscales are other-focused, assessing guilt related to worry about others. The fourth subscale, Self-Hate, consists of negative self-focused statements, similar to items in cognitive measures of depression. The reliability and validity of the IGQ-67 was established in studies using several other validated measures of guilt, including scales of adaptive (Tangney et al., 1992) and other measures of ruminaative or maladaptive guilt.
As predicted, all measures of guilt were highly correlated with the first three IGQ-67 subscales (O'Connor et al., 1997, 1999).

Other studies validated the underlying assumption that interpersonal guilt is based on human empathy, and indirectly, on altruism as a fundamental motivation. Results indicated that Survivor, Omnipotent and Separation Guilt are significantly correlated with Empathic Concern, Empathic Distress and Empathic Perspective-Taking, as measured by the Interpersonal Reactivity Index (IRI; Davis, 1980), demonstrating empirically that Survivor, Separation and Omnipotent Responsibility Guilt are empathy-associated emotions. We examined the correlations between various indexes of psychopathology, including depression, in order to test out the hypothesis that many clinical problems were associated with high levels of empathy-based guilt (O'Connor et al., 1997, 2002). Empathy, Survivor Guilt and Neuroticism, described above as a high risk factor for depression, are modelled in Figure 4.2.

In another study, we compared patients hospitalized for depression with a non-clinical sample in order to determine if the two samples differed significantly in levels of guilt, if they differed significantly in self- versus other-focused concerns, and if the clinical sample was higher than or equal to the non-clinical sample in subscales of empathy (O'Connor et al., 2002).

The results indicated that the depressed patients were equal to the non-clinical sample in Empathic Perspective-Taking and Concern and significantly higher than the non-clinical sample in Empathic Distress. These results demonstrate that

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**Figure 4.2.** Path analysis: the relationships of subscales of empathy, survivor guilt and neuroticism (as a marker of BDNF and high risk factor for depression). Several path models were tried, and Figure 4.2 was the best fitting model to our data. (Cumulative fit index = 0.95.) All path coefficients in the model are significant at 0.001
depressed patients do not differ from a normal sample in the ability to cognitively distinguish themselves from others. In order to compare self- and other-focused concerns in predicting depression in the depressed compared to non-depressed samples, we used instruments measuring worry about the self, including: Fear of Negative Evaluation (Brief-FNE; Leary, 1983); Fear of Envy Scale (FES; O’Connor et al., 2002), a measure of worrying that others will feel jealous about one’s success or happiness; the Social Comparison Scale (SCS; Allan & Gilbert, 1995), a measure of how people believe they rank in social status compared to others; and the Submissive Behaviour Scale (SBS; Allan & Gilbert, 1997), a measure of how submissive people believe themselves to be. Multiple regression analyses supported the hypothesis that, in depressed patients, other-focused concerns outweighed self-focused concerns in predicting depression, whereas in the non-clinical sample, both self- and other-focused concerns significantly predicted depression.

In other studies, interpersonal guilt significantly correlated with depression, assessed by a variety of measures: Beck Depression Inventory, Center for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977), and subscales of the Brief Symptom Inventory, as well as indirect indicators of depression such as automatic thoughts, pessimistic explanatory style and Neuroticism. Still other studies conducted in our laboratory found significant associations between guilt and other psychological problems, for example jealousy (Webster et al., 1997), addiction (Meehan et al., 1996), obsessive compulsive disorder (OCD), perfectionism and pessimism.

In a recent experiment investigating survivor guilt in non-depressed college students, subjects read a story designed to induce survivor guilt in the story’s hero, as the hero did better in a college class than did a second character in the story. The conditions varied only by the relationship between the main and the secondary characters; in one condition the hero surpassed his brother, in the second he surpassed a best friend, and in the third he surpassed a person with whom he had no personal relationship and who he never expected to see again. Subjects then wrote narratives about what they believed the hero would feel, think and do, under those conditions. Senior clinicians rated the narratives using a scale assessing levels of guilt and found it significantly lower in the stranger condition when compared to the family or friend conditions. We also found that scores on the IGQ-67 subscales predicted levels of guilt in the narratives written by each subject, indicating that IGQ-67 subscales reliably predict levels of guilt in behavioural indices.

In another recent study, we found that Generalized Anxious Temperament (GAT; Akiskal, 1998) correlated significantly with interpersonal guilt and with depression. According to Akiskal (1998), the GAT measures a personality type or style, typified by worries about self and others, that serves a protective function for the family and larger social group, and is therefore, from an evolutionary perspective, fitness enhancing at the level of the group.
Empathy and depression

The significant correlation between Survivor Guilt and depression has held up across cultures including Japan, Sweden and Germany, and across different cultural groups within the United States such as Filipino, Latin, African, Japanese, Middle Eastern and Chinese Americans. Figure 4.3 illustrates the correlations of the means of Survivor Guilt with the means of Depression across cultures.

In a study of 621 subjects, we examined the associations between guilt and self-reported acts of altruism. We found both Survivor and Omnipotent Guilt predictive of acts of altruism to family, friends and strangers. Comparing five cultures, significant differences were found in levels of altruism to family, with Asian, Middle Eastern and Hispanic subjects scoring significantly higher than European American subjects. In a study still underway with data collected from an internet sample, we are comparing Asian Americans to European Americans on guilt, empathy and altruism; with 348 subjects thus far, we have found no differences in major variables.

In another line of research related to depression, we conceptualize problematic temperament differences as reflecting low dopamine and/or low serotonin levels, instead of specific diagnoses, although we used diagnoses to validate a new measure, the Neurotransmitter Attributes Questionnaire (NAQ; O'Connor et al.,

![Figure 4.3](image)

Figure 4.3. The correlations of the means of survivor guilt with the means of depression across cultures. (O'Connor, L. E. Survivor Guilt Across Cultures. University of Michigan, 2004)
2005). The NAQ consists of 51 items derived from questions used by psychiatrists in evaluating new patients for psychopharmacological treatment. Two clinicians knowledgeable about the effects of medications on particular symptoms placed each item in the Low-Dopamine and/or Low-Serotonin subscale. An internet study of 700 subjects from the general population resulted in several significant findings, related to guilt and depression, as well as to Neuroticism and Generalized Anxious Temperament, or 'Altruistic Anxiety'. We found both dopamine and serotonin significantly correlated with depression in both men and women, and each remained significant in a multiple regression predicting depression. We also found that Neuroticism significantly associated with guilt as well as both neurotransmitter when looked at alone; however, in a multiple regression, low serotonin remained significant while dopamine lost significance. Guilt-proneness was found to be associated significantly with both neurotransmitters.

4.6 Other empirical studies: empathy and depression

Findings in social and clinical psychology have supported the link between empathy and depression, connected to gender differences in empathy as well as depression. It is well established that females are at greater risk for depression, with rates at least three times that of males at all ages beyond puberty (Rosenfield et al., 2000). In some studies females were also found higher than males in empathic concern and empathic distress (Bush et al., 2000; Eisenberg, 2000).

Other studies found links between empathy, depression and stressful life conditions in various populations such as disabled children, adults with medical problems, people in helping professions, medical interns and others. Multiple studies demonstrate that individuals who are sensitive to distress in others are at risk for depression, anxiety and other symptoms of psychological distress (Griens et al., 2002; Shicerman & Turner, 2001), especially if they are female (Bandura et al., 2003). Few studies failed to find an association between empathy and depression. For example, a cross cultural study of Iranian and American students found positive correlations between depression and empathic distress, but a negative relationship between depression and empathic concern (Ghorbani et al., 2003).

Studies of adult samples have routinely found chronic guilt correlated with depressive symptoms (Jones & Kugler, 1993; O'Connor et al., 2002; Quiles & Bybee, 1987). Situation-specific guilt in response to specific transgressions has been linked to empathic concern and other prosocial affects (Joorimana, 2004; Quiles & Bybee, 1997; Tangney et al., 1992). Situational guilt, assessed by measures designed to capture realistic, adaptive guilt, fails to significantly correlate with depressive symptoms as these measures do not include the dispositional, maladaptive and ruminative guilt common to both depression and anxiety disorders.
Empathy and depression

There remains a paradox in the findings on depression, empathy and guilt. On the one hand, empathy has been empirically linked to prosocial behaviour, moral maturity and emotional regulation, even among very young children. On the other hand, children who are high in empathy and other characteristics of maturity have higher rates of depressive symptoms in later years. In a longitudinal study of families conducted by Cowan and Cowan, it was found that survivor guilt, assessed in parents and adolescents followed since the children were about 4 years old, was significantly predicted for the adolescent girls by a behavioural test of ToM, administered at age 4 (L. Rasco, personal communication, 2004). It was considered unusually mature when these girls scored high on the test of ToM, and yet these findings suggest high ToM at a young age leads to problematic guilt-proneness. This adds another paradox to the already paradoxical findings that the empathic responses to others that hold our social groups together and enable cooperative group living are indirectly linked to the current epidemic of depression.

4.7 Evolutionary theories: empathy and depression

Over the past four decades sociobiologists, evolutionary psychologists and psychiatrists have attempted to fit depression into the adaptationist programme, explaining depression as a functional adaptation (Nesse, 2000). Price (1967) observed that symptoms found in psychiatric patients were similar to those seen in people who had lost status, making a connection between mental disorders and a dominance hierarchy. Price's dominance hierarchy theory of depression evolved into what is known as the ranking of depression, or 'an involuntary submission' in a losing situation, in order to avoid further futile conflicts, and to preserve one's self-interests by withdrawal from battle. Gilbert (1992) discussed depression as a method of signalling social group members that the depressed person is defeated, withdrawn and therefore no longer willing or able to fight. Gilbert and Allan (1994) gathered empirical evidence supporting this theory, examining social comparison and other self-focused variables related to social ranking, submission and depression. Our findings support both the concern for others theory of depression, and the social-ranking theory in a normal sample with minor symptoms. Hagen (2003) proposed that depressives use their illness as a means of social bargaining, with the intention of gaining resources.

Mismatch theory provides another evolutionary model of depression. While not suggesting that depression is adaptive, it explains it as the result of adaptations that were functional in the Era of Evolutionary Adaptation (EEA); that is, in the conditions in which our species evolved, but which have become maladaptive in present-day conditions. Some suggest that gender-specific attributes, adaptive for childrearing in a hunter-gatherer society, have become dysfunctional in the
context of birth control and high technology, in which women commonly work outside of the home. Another example is seen in our current problem with obesity. The archaeological record suggests food supplies in the EEA were often variable, sometimes plentiful and sometimes scarce. Our species developed biological adaptations to ensure survival and reproduction when food was scarce, as in famines. To meet this condition we developed an energy-conserving metabolism with a capacity to store energy in fat cells, along with dietary tastes creating cravings for fat, sugar and salt, often scarce in the EEA. These adaptations, under modern conditions, have resulted in obesity, heart disease and other chronic modern illnesses. The link between rates of depression and the rise in obesity and dieting may have a functional connection. Deliberate food restriction is now found among young children; some begin dieting by age 6, establishing a life-long eating disorder, associated in numerous studies with depression.

The lifestyle of hunter-gatherers required large amounts of physical activity, along with living in close and stable social groups (Cosmides & Tooby, 1992). Exercise increases levels of BDNF, while lower levels are implicated in depression, and exercise has been found to be an effective antidepressant (Russo-Neustadt et al., 2001). Lacking in natural opportunities for exercise, the contemporary lifestyle may be inherently harmful to genetically vulnerable people who respond with low BDNF and fall victim to depression. Studies of the hunter-gatherer groups remaining suggest that, in the distant past, people were social and cooperative by necessity. As small, slow moving mammoths, we were 'Man the hunted' instead of 'Man the hunter'. Frey to numerous predators who viewed us as a source of protein, our developing brain and predilection for cooperative social living were our only defence. Privacy and ownership had not yet appeared. Singing, dancing, gossiping, hunting, gathering and child care were conducted in protective cooperative groups, conducive to intimate social interactions, and provided the level of physical and social activity required to maintain adequate BDNF and the limbic system regulation that prevent depression (Levits et al., 2000; McGuire & Troisi, 1987; Raleigh & McGuire, 1986).

4.8 The evolution of altruism and evolved mechanisms at work in depression

While theories of the adaptive function of depression abound, they have been limited to adaptation at the level of the individual or the 'selfish gene', and have neglected to consider fitness at the level of the group. Two explanations of altruism, kin selection (inclusive fitness theory) (Hamilton, 1964) and reciprocal altruism (Trivers, 1971), have dominated, despite their inability to account for altruism extended toward strangers. Both theories focus on within-group competition. Costly display, another theory explaining the evolution of altruism with
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individual fitness as the goal, proposes that altruistic acts serve to signal social group members that the altruist holds a surplus of resources and is a desirable mate in competition for reproductive partners.

Tooby and Cosmides (1996) described the ‘the Banker’s Paradox’ theory, to explain altruism extended to friends, as well as to make sense of the evolution of our drive for recognition, or ‘individual uniqueness’. Altruism extended to friends is analogous to a banker loaning money to a person in need at the moment, who is expected to pay it back later when resources may be scarce for others. They theorized that having unique skills makes the pay back more likely, and this contributed to the evolution of individuality, as well as to altruism between friends. Here too, kindness to strangers is neglected.

The insistence on viewing altruism as a deception, covering hidden selfishness and individual fitness while denying the existence of primary altruism, has been part of a four-decade-long bias against group selection and multilevel selection theory, which only now is finally lifting (Borrello, 2005). Although Darwin accepted group selection as a factor in the evolution of altruism and cooperation in humans and other species, the theory was rejected, beginning in the 1960s. Ignoring the ‘science war’ going on in evolutionary theory, group selection was kept alive by a small group of evolutionary scientists and theorists (Sober & Wilson, 1998; Wilson, 1975) through the gene-centric era, sometimes referred to ‘ultra-Darwinism’. The recent reemergence of multilevel selection theory is joined and supported by the theory of gene-culture coevolution (Richerson & Boyd, 2004), in which cultural evolution, occurring more rapidly than genetically based evolution, provides the context for the evolution of group-focused cultural traditions that, in turn, affect genetics, as genetics affect culture. As a mark of the end of the selfish gene era and the resurrection of group selection, at the 2005 meeting of the Human Behavior and Evolution Society, E. O. Wilson, the father of sociobiology, stated that group selection is the most important factor in the evolution of cooperation and altruism, including in our species. The results of our research on empathy, guilt and depression, and the presence of survivor guilt across cultures have also been best explained in terms of mechanisms operating at the level of the group and, ultimately, a function of group selection (O’Connor et al., 2000).

Early human groups were relatively small, surviving by their ‘wits’ while prey to multiple predators, and engaged in intense warfare between groups, while developing the variations in customs known as culture, including traditions allowing groups to distinguish themselves from others. Cultural evolution is thus also a factor in competition between groups and was important in the evolution of altruism and cooperation. Prosocial emotions such as guilt, along with other emotional capacities that are in varied ways and degrees influenced by culture,
serve as proximate motivators for altruism. Transmission of social norms limits within-group competition and promotes within-group leveling, leading to forms of social organization and ideologies such as monogamy and social sanctions against large income differentials (Gintis et al., 2003).

Empirical studies of behaviour in economic games such as the 'Public Good', 'Ultimatum' and 'Prisoners' Dilemma', in vivo as well as in simulated computer experiments, demonstrate that groups with more altruists/cooperators do better in competition with groups who have fewer cooperators. Players are motivated to cooperate and will punish 'defectors', despite the personal cost. Several studies have demonstrated that guilt functions to increase cooperation in games. Guilt may function in two ways: first, it may serve as an internal warning signal, letting the person know that he or she is violating a social norm and is at risk of being punished. Second, guilt also serves as an internal signal letting the person know that he or she must take action to help someone else. This second function of guilt is based on the empathic response that begins with witnessing another's distress and, through the mirror neuron system, feeling it as one's own. This transforms into empathic concern, at which point the person feels compelled to help and, if failing, this becomes empathic distress, with guilt unresolved and often chronic. Therefore, guilt is the connector between empathy and the moral system, like a bell that goes off when action is needed, it is our form of alarm cry, telling us we must help a conspecific, and this becomes both an affective and a moral directive. Guilt may not always be reliable, as when it is exaggerated and unrealistic in depression. However, imperfect our signal to act, it motivates the non-conscious moral judgements that help hold us together.

In a recent study we conducted in collaboration with Wilson (unpublished data, 2004) the Survivor Guilt subscale on the IGQ-67 significantly predicted cooperative behaviour in the Public Goods game. Across cultures, cooperation and fair play are expected, even when groups are temporary (Fehr & Fischbacher, 2003); thus both fear of being punished and empathic guilt are emotional capacities providing proximate motivation for automatic moral judgements, and then for cooperative and altruistic behaviour, while the ultimate cause is group level fitness.

While altruistic punishment infers a cost to the punisher as well as to the punished, a recent study demonstrated the punisher also receives a reward for punishing; as when cocaine or nicotine are administered, the punisher is rewarded by the activation of the caudate nucleus. Subjects who wished to punish maximally received an additional reward by activation of the thalamus and the dorsal striatum, associated with rewards gained from goal-directed activities (de Quervain et al., 2004). Gintis et al. (2005) suggest that successful groups tend to have strong altruistic reciprocators who have received either altruistic rewards or altruistic punishments.
In order to distinguish the cooperator from the defector in a social group, people need to have a quick and implicit method of detecting cheaters (Cosmides & Tooby, 1992). ‘Cheater detection’, an evolved capacity forming what is referred to as a ‘module’ in our cognitive apparatus, enables people to detect and limit the invasion of cheaters in a community. Cheater detection and punishment of ‘cheaters’ (or altruistic punishment) are evolved capacities that blend into the area of empathy and moral judgement connected by guilt, that specifically become dysfunctional in mood disorders. People suffering from depression often exhibit hyper-moralistic standards and hyper-scrupulousy applied to others, but even more fiercely to themselves. In depression, cheater detection turns inward, unrealistic guilt signals the person of their so-called immoral intentions or actions, and altruistic punishment turns upon the self, providing one explanation for the self-destructive behaviours often seen in the clinic. Though arising with the evolution of cooperation, in depression these mechanisms, through the connecting emotion of guilt, have become dysfunctional and may sometimes render depressives victims of self-inflicted bodily injury.

The capacity to detect cheaters underlies survivor guilt. Cheater detection requires the ability to quantify and put value on something that a person gains, and then to determine if the value is deserved. Survivor guilt requires the capacity to detect cheaters, but instead of looking for cheating in others, the cheater to be detected is the self. People suffering from depression are looking at both others and themselves with suspicion, often believing whatever they have was obtained by cheating, and that it is more than they deserve. Depressives, burdened by moralistic standards, are harsh evaluators of both themselves and others. The self-punishment meted out by depressives is a common if disturbing symptom; while thinking ‘I deserve this’, they may engage in altruistic punishment turned upon the self. Just as altruistic punishers experience a neuronally based reward from punishing defectors, despite material costs, depressed patients report a sense of relief upon inflicting self-punishment. Patients who are ‘cutters’, describe relief from tension after cutting and depressives with suicidal ideation may describe the relief they felt when on the verge of attempting a suicidal action.

It is difficult to rationalize depression as adaptive from the point of view of the individual or even the group. However, from the perspective of an even higher level of organization, for example the species or whole eco-systems, depression may function to limit the growth of local populations, and to restrain depletion of worldwide resources, benefiting many species. In the most developed nations, increasing populations have settled in urban centres, resulting in local overcrowding. At the same time, rates of depression and rates of consumption of energy and other resources have been rising, while birth rates have been dropping for the past century. This situation resembles that of non-human animals living in
overcrowded conditions, where they have been known to display socially aberrant
behaviours, dropping rates of reproduction, rising rates of viral epidemics,
destruction of their environments and, in some extreme cases, species extinction.
In the ordinary time frame, the cost of depression is vast. However, at a higher
level, evolution may be in action, with the rise in depression and dropping birth
rates serving to limit the consumption of nations who are over-utilizing natural
resources. The hidden benefit to rising rates of depression may go to multiple
groups of people worldwide, as well as to numerous other species when looking
from a long, evolutionary time frame.

4.9 Empathy, depression, and current neuroscience

As advances in neuroscience, including brain imaging, are applied to the study of
depression and empathy, we are likely to also further study guilt. Serving as the
mediator between depression and empathy, guilt is the moral emotion, based on
empathic responses and our need for closely connected and stable social groups,
manifested in our pain at witnessing pain in others, and our need to help those in
distress. We are now able to compare normal people feeling empathic concern, or
making morality-based decisions in experimental conditions, to brain activity in
the sociopath, or autistic individuals with deficits in the capacity to feel empathy
for others. Specific functional differences between depressed and non-depressed
samples are compared through functional magnetic resonance imaging (fMRI),
positron emission tomography (PET), single photon emission computed tomo-
graphy (SPECT), magnetoencephalography (MEG) and other forms of imaging.
In molecular biology genetic variables are also now being identified, including
variations that appear correlated with vulnerabilities to depression, or expressed
only when the vulnerable person is in adverse conditions. Moral reasoning is
recognized as a function of non-conscious social emotions, and particularly
guilt, driving decisions before there is awareness. The affective neural basis for
social judgements, moral decisions and the detection of cheaters, all of which are
connected by guilt, and mechanisms underlying empathy, compassion and altru-
ism as well as depression are now under the gaze of brain imaging (Greene et al.,
2001; Greene & Haidt, 2002; Moll et al., 2002a, 2002b).

Moll et al. (2002b) reported on an fMRI study in which subjects were presented
with pictures representing six conditions: moral pictures portraying charged
unpleasant scenes; pleasant scenes without a moral meaning; unpleasant scenes
including an implicit moral connotation; neutral pictures with people and with
landscapes; and scrambled images. When subjects viewed the moral in comparison
to the non-morally tinged unpleasant pictures, findings demonstrated significantly
increased activation in paralimbic structures also noted in fMRI studies of guilt.
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In functional brain studies of depressed patients compared to normal samples, Lange and Irlé (2004) found abnormal activation in limbic structures, including enlarged amygdalae in patients with recent major depression. Other studies of depressed patients find unusual activation of the amygdala, abnormally under-active in psychopathy, characterized by the absence of normal moral decision-making and guilt. Pagani et al. (2004) observed increased cerebral blood flow in the anterior temporal lobe of depressed patients, also a focus of activity in guilt. Abnormal limbic and paralimbic system activity has been observed with major depression, which returns to normal with successful antidepressant treatment, indicating that some portion of the pathophysiology of depression is connected to overactivity in limbic and paralimbic structures, affecting moral decision-making and guilt (Shin et al., 2000).

Neumeister et al. (2004) reported that patients with a history of depression demonstrated chronic overactivity in the brain circuit central to emotion regulation. Hyperactivity was observed even when the patients were in remission, suggesting that dysfunctions in emotion regulation highlighted in this study may be genetic. Between depressive episodes, hyperactivity in the emotion regulation circuit included these same limbic and paralimbic structures associated with morality and empathy. The associations between empathy, guilt and depression were established using older research methods; however, brain imaging technology provides a detailed picture of both normal and abnormal activity in limbic and paralimbic structures and connected cortices that form the neural network of the social brain, the home of empathy and guilt. Imaging research allows us to begin to understand the mechanism by which the results of empathy become dysfunctional, when interpreted through the lens of chronic and unrealistic guilt found in depressive illness.

4.10 Conclusions

The review of research in this chapter brings together evidence from multiple areas in psychology and neuroscience demonstrating the connection between empathy, morality, guilt and depression. The neuroscience of empathy describes a complex network, beginning in limbic structures and leading to automatic moral decision-making, that appears also focused in the paralimbic system, associated with executive control and planning. Brain regions associated with the empathy system are also involved in other mental disorders characterized by a high proneness to empathy in combination with an overly active and even harsh moral system, such as obsessive compulsive disorder and addiction. Against the background of this network of inter-connected structures, we are able to place our empirical findings, connecting guilt and depression across cultures. Empirical studies of the social
brain, in its normal and abnormal states, are paving the way for a more patient-friendly and positive perspective on human motivation, and therefore also on depressives. In most studies, depressives are found to have similar empathic responses to non-clinical samples, with depressives differing only in that they are more prone to feeling greater guilt leading to greater distress upon witnessing distress felt by others.

The early work of developmental scientists has been confirmed by numerous studies demonstrating the connection between empathy and depression, connected by guilt which, in those vulnerable, tends to transform empathic concern into empathic distress. Paradoxically, guilt serves as a proximate motivation for altruistic actions. While playing economic games in experimental conditions, it is found that people usually expect cooperative behaviour from others, and behave cooperatively themselves. Those who express feeling guilty after failing to follow the social norm of cooperative behaviour in the first round of a game tend to behave more altruistically in the next round, compared to those who express no feelings of guilt at their failure to follow the norm of cooperation.

A shift in focus from a self- to other-centred view of depression is likely to encourage more positive attitudes in treatment providers, and thus to improve treatments as well as modes of prevention. The connection between depression and empathy is found in guilt – the moral emotion, the signal that makes us so uneasy when witnessing unfairness and inequity. The rapid rise in depression suggests that our current lifestyle may be less than ideal for the social brain; assuming the human brain evolved for life in cooperative and empathic social units, overcoming depression may require the development of social environments better suited to the nature of our species.

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