Does Mood Induction Elicit Emotion Recognition Biases? An Empirical Study with Implications for Depression Research

Steven Harding
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ACSENT Laboratory
Department of Psychology
University of Cape Town

Supervisor: Susan Malcolm-Smith

Compulsory Declaration: This work has not been previously submitted in whole, or in part, for the award of any degree. It is my own work. Each significant contribution to, and quotation in, this dissertation from the work, or works, of other people has been attributed, cited, and referenced.

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Date: 16 May 2015

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For my my family, who got me through.

Cheneal, 'bah'.

Susan, for your patient supervision despite everything.

Everyone at 22seven, Thank you so very much. A special mention to:

Lynda, this would never have reached completion without your kind support.

Christo, I am forever grateful for you calling me back for that chat.

Kenny, the existence of this thesis is proof that hops are therapeutic.

"Thank *goodness*" - Daniel C. Dennett

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Abstract

Depression is a highly prevalent, debilitating, and sometimes-fatal mental illness. Typically, its treatment approaches are conceptualised as a dichotomy between psychological and pharmaceutical. However, a new model, in line with cogent philosophical reasoning and recent empirical evidence, integrates these approaches. The *cognitive neuropsychological* model places affective processing biases as central to depression aetiology and treatment—in both biological psychiatry and cognitive psychology. One affective bias, emotion recognition, is central to the tenets of this model, which, unlike some cognitive theories, places improved affective biases as temporally prior to improved mood, and as the underlying mechanism of antidepressant action. To test this account of emotion recognition bias, 103 undergraduate students participants underwent negative, positive, and neutral mood induction in a between-groups design to assess whether mood-congruent emotion recognition biases would emerge in a multimodal (facial, vocal, musical) emotion recognition battery, while controlling for depression symptoms and assessing maladaptive cognitive schemas. Few significant emotion recognition biases resulted, but significant negative correlations between negative schemas and overall facial and musical accuracy emerged, even when controlling for depression—lending some support to the cognitive neuropsychological model's premise of a bilateral relationship between schemas and emotion recognition, both of which may play a substantial role in the etiology of depression.

Major depression is a highly prevalent, severely debilitating mental disorder that, in many cases, is fatal. More than a normal sad reaction to a distressing situation, it is a complex disorder of multiple aetiologies that, in addition to characteristic low mood or lack of interest in previously enjoyable activities (a requirement of either being necessary for diagnosis) affects sleep patterns, eating habits, executive functioning, and thoughts about oneself—to a degree that compromises everyday functioning (see Appendix A for full diagnostic criteria).

At least 10% of South Africans will experience a depressive episode at some point in their lives (Myer et al., 2008; Richards, 2011); the latest edition of the Diagnostics and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013) puts this figure at 7% for the United States population *per year*. Clearly, estimates vary (Ferrari et al., 2013), but undisputed is the fact that depression is an extremely common disorder—enough so for the World Health Organisation to list it as the leading cause of disability worldwide (World Health Organisation, 2012). In South Africa, similarly to other diseases, depression is most prevalent in those living in poverty (Lund et al., 2010) or with HIV (Pappin, Wouters, & Booysen, 2012; Simbayi et al., 2007), two groups which also receive the least access to care (Lund & Flisher, 2006).

In purely economic terms, depression places an enormous burden on countries, primarily through losses in work productivity. The United States alone is estimated to have lost $83 billion in the year 2000 to depression (Birnbaum et al., 2003); the cost in Europe in 2004 was estimated at an entire percentage of its combined gross domestic product : an astounding €118 billion (Sobocki, Jonsson, Angst, & Rehnberg, 2006).

In more human terms, depression is the cause of approximately 90% of successful suicide attempts, which cause around 800 000 deaths annually worldwide (Rihmer, 2001). Unsuccessful attempts are far more common, often causing permanent physiological damage (Farmer, 1988) , and suicidal ideation is common in those with depression (Nock et al., 2008). Even in those without this feature, depression greatly affects the quality of life of both sufferers (Angermeyer, Holzinger, Matschinger, & Stengler-Wenzke, 2002) and their families (Fadden, Bebbington, & Kuipers, 1987).

Thus, there is good reason for the considerable, sustained research effort surrounding depression (as one indication, a PsychINFO title search for 'depression' returns over 50 000 results), a large portion of which is ultimately aimed towards refining treatments for the disorder. The current thesis shares this aim.

Treatment Approaches

Although the full research into depression aetiology is complex and extensive (see, e.g., Beck & Alford, 2009; Hölzel, Härter, Reese, & Kriston, 2011; Sullivan, Neale, & Kendler, 2000, for reviews more extensive than possible here) the reality faced by patients and practitioners from the perspective of depression *treatment* is somewhat simpler. It is also from the examination of these approaches, and the theories that underpin them, that a novel neuropsychological question arises.

The most commonly-recommended treatment for depression is of antidepressants or psychotherapy, or a combination thereof (Cuijpers, van Straten, Warmerdam, & Andersson, 2009; Duval, Lebowitz, & Macher, 2011). This treatment approach is in line with most treatment guidelines (Davidson, 2010; Gelenberg, 2010), including those in South Africa (of the South African Society of Psychiatrists; Grobler, 2013). (The exceptions to this are in mild depression, where a minority recommend light exercise, and severe or treatment-resistant depression that may require electro-convulsive therapy)

Psychopharmacology. Approximately one in ten people over age 12 in the United States is currently prescribed an antidepressant (Pratt, Brody, & Gu, 2011). Although antidepressants as a drug class are not new, their use increased exponentially after the licensing of the first selective serotonin reuptake inhibitor (SSRI) fluoxetine (original trade name: Prozac) in 1987 (although initial testing of the compound began in 1972; see Wong, Perry, & Bymaster, 2005, for a detailed, fascinating account of this research). As one indication: in primary care alone, antidepressant use increased over 300% between 1989 and 2000 in the United States (Pirraglia, Stafford, & Singer, 2003). Similar trends are evident internationally, for example: in Australia, the increase in use between 1990 and 1998 was 160% (McManus et al., 2000).

Earlier antidepressants, discovered in the 1950s, target monoamines more broadly: Monoamine oxidase inhibitors reduce the breakdown of dopamine, epinephrine, melatonin, norepinephrine, phenethylamine, and serotonin. Tricyclic antidepressants primarily increase concentrations of serotonin and norepinephrine, but also agonise and antagonise several other receptors. It is this relative non-specificity that contributes to the severe side-effect profile and interactions of these drugs (López-Muñoz & Alamo, 2009).

The monoamine hypothesis. The dominant theory behind antidepressant action is, unsurprisingly, that depression is caused by a low level of monoamines, and antidepressants ease depression symptoms by increasing levels of these neurotransmitters (Hinz, Stein, & Uncini, 2012; Hirschfeld, 2006). This hypothesis began as the similar catecholamine

hypothesis (Bunney, 1982; Schildkraut, 1965), which was expanded upon when research showed serotonin particularly (which is not a catecholamine) to be instrumental in depression treatment.

This hypothesis, also referred to as the *chemical imbalance* hypothesis, is widely propagated (Lacasse & Leo, 2005), for at least three overlapping reasons. First, this explanation ties into the broader shift in psychiatry towards biological models of mental illness (Barondes, 1990), and their reification (Hyman, 2010), both of which lend themselves to biological (pharmaceutical) treatment. Second, it is clear that the monoamine hypothesis is both in the interests of, and actively promoted by, some pharmaceutical companies. The clearest example of this is in direct-to-consumer advertising in the United States, where some have explicitly advanced the term and explanation "chemical imbalance" (see, e.g., the 2003 Zoloft television advertisement: http://youtu.be/hu0ONl8aSGU). Creating the picture of a simple process with a straightforward, singular remedy is a sensible marketing strategy for those who are selling it (Lacasse & Leo, 2005; Leo & Lacasse, 2007). (This is in no way meant as an ethical judgement of "Big Pharma".) Third, the hypothesis allows for a reduction in the stigma associated with mental illness (but perhaps at the cost of worsened perceived prognosis by patients; Deacon & Baird, 2009). Thus, explanations citing a chemical imbalance frequently appear in public information leaflets (e.g., the depression brochure of the South African Depression and Anxiety Group; SADAG, n.d.) and this explanation (and treatment approach) is preferred by some patients (France, Lysaker, & Robinson, 2007).

Despite its widespread reach, the monoamine hypothesis, in its simplest form (that depression is caused by low levels of monoamines) is largely untenable. From the observation that increased monoamine concentrations alleviate depressive symptoms, it does not follow that low monoamines are the cause of these symptoms (Hindmarch, 2001). Although similar reasoning does hold true in some cases (e.g., vitamin deficiencies), additional evidence is needed before drawing causation. Consider, for instance, the role of low-dose opioids in effective headache treatment: increased opioid concentration often results in headache relief—but it would be absurd to conclude from this that headaches are caused by an 'opioid imbalance'. Indeed, empirically, some research finds that there is no correlation between measures of monoamines and depression in non-medicated individuals (Hayward, Goodwin, Cowen, & Harmer, 2005; Hinz et al., 2012; Leyton et al., 1997). Further, the monoamine hypothesis fails to account for the delay of therapeutic response in antidepressant treatment—monoamine levels increase hours after the first dose, but reduction in depression

symptoms only follow weeks later (Harmer, Goodwin, & Cowen, 2009). An alternative to the monoamine hypothesis is discussed later.

Psychotherapy. Many variants of psychotherapy exist (Norcross, Koocher, & Garofalo, 2006) that, contrary to earlier claims (Wampold et al., 1997) are likely *not* empirically equivalent (Budd & Hughes, 2009). More importantly, not all are logically equivalent, and pseudoscience abounds in this area (Beyerstein, 2001; Lilienfeld, Jay Lynn, & Lohr, 2003; Lilienfeld, 2010). For example: Even if *some* of Freud's principles now appear to have elements of neuroscientific support (Turnbull & Solms, 2007), this cannot retrospectively validate (see Clifford, 1877/1999) a method that was never based on scientific principles (Lattey, 1969; Webster, 2005). More plainly: These theories never constituted knowledge, as without evidence they did not meet the necessary (yet perhaps not sufficient; Gettier, 1963) requirements of being *justified*, true beliefs at the time (Dawson, 1981).

In fact, the framework of interest here, Beck's cognitive theory (Beck, 2005), emerged when an empirical investigation into an assumption of psychoanalysis proved false. Specifically: Psychoanalysis suggested that depression is associated with self-directed hostility in patients (Clewell, 2004; Freud, trans. 2005); however, Beck found more hostility in the dream content of *non-depressed* individuals than patients. Instead, what emerged in depressed patients' dreams were consistent themes of loss and abandonment—themes that were also present in their waking thoughts. Commonly, the conscious thoughts and self-appraisals of his patients contained negatively-biased distortions about themselves, their environment, and their future (Beck, 1961; but for a comprehensive retrospective see Beck & Alford, 2009). In finding that correcting these negative (and often factually incorrect) thoughts could facilitate remission of depression in as few as 10 sessions (Beck, 1967), Beck's iteration of cognitive therapy emerged. Although several evidence-based cognitive theories and therapies exist—it would be wrong not to mention the earlier, isolated, work of Albert Ellis and his Rational Emotive Therapy (Ellis, 1957; Ellis, 1980)—the work of Beck is particularly appealing because of his continued interest in emerging interdisciplinary evidence (Beck, 2008) and work with schemas, discussed later.

Combined with elements of behaviour therapy, which (as its name suggests) applies behavioural principles to drive therapeutic response (for an overview see Grossberg, 1964), cognitive *behavioural* therapy (CBT) is demonstrably effective in the treatment of depression and other psychiatric disorders (Butler, Chapman, Forman, & Beck, 2006; Hofmann, Asnaani, Vonk, Sawyer, & Fang, 2012). Partially because of its short time to therapeutic response compared to other psychotherapies (Churchill et al., 2002), and its suitability to computer-

based online administration (Andersson & Cuijpers, 2009; Richards & Richardson, 2012; which is often employed in the United Kingdom, see www.beatingtheblues.co.uk), is often the first-recommended psychotherapy. As one example: The UK National Institute for Health and Care Excellence (NICE) guidelines recommend CBT as the treatment of choice for mild to moderate depression (and in combination with antidepressant therapy for severe depression; National Institute for Health and Clinical Excellence, 2009).

Cognitive theory and schemas. Expanding on the basic premise of cognitive theory—that incorrect beliefs are related to depression symptoms—Beck applied the concept of *schemas* (or *schemata*) in the context of depression. Although the concept of schemas was not new, with its formulation originating with Kant and later adopted by Piaget, among others (see Dahlin, 2001, for a critical historical overview), Beck uniquely theorised how these relatively-persistent frameworks for interpreting information about oneself and the environment would influence mood. Thus, *negative* schemas would lead to a corresponding negative interpretation of stimuli—facilitating depression (Beck & Alford, 2009).

These schemas can be expressed in statements, such as "I am a failure", which may be latent until a relevant event (e.g., a poor grade on an exam) leads to its activation, in turn activating a more global negative processing biases (Beck, 2008).As for how negative schemas originate, Beck posits that negative life events, particularly in childhood, establish these interpretive frameworks—which may be reinforced with repeated activation by stressful events (Beck, 2005). This explanation, presented in Figure 1 (overleaf), provides a model in which the high correlation between childhood abuse and later depression can fit (Cukor & McGinn, 2006). Some variants of cognitive therapy seek to uncover and target maladaptive schemas directly; perhaps the most prominent of these being the work by Young and colleagues (e.g., Kellogg & Young, 2006; Young, Klosko, & Weishaar, 2003).

As with pharmacological treatments, patients may prefer talk therapy for specific reasons (Chilvers, Dewey, Fielding, & Gretton, 2001), commonly including an aversion to (or misunderstanding of) antidepressants, or the belief that psychotherapy will resolve the 'true' cause of the disorder—which is conceptualised in psychological rather than biological terms (Dwight-Johnson, Sherbourne, Liao, & Wells, 2000; van Schaik et al., 2004). Unsurprisingly, a mismatch between patient beliefs and preferences with the provided treatment leads to poorer adherence (Kwan, Dimidjian, & Rizvi, 2010; Swoiskin, 2001a).

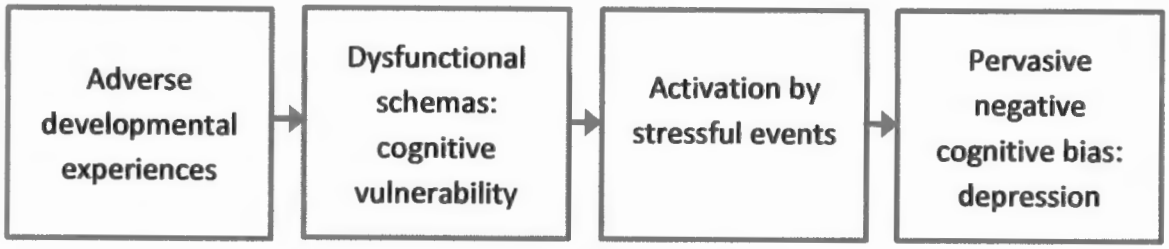

Figure 1. A developmental model of depression showing the vulnerability diathesis-stress model of schema activation. Adapted from "The Evolution of the Cognitive Model of Depression and its Neurobiological Correlates" by A. T. Beck, 2008, *American Journal of Psychiatry, 165,* p. 969-977.

Dualism and a false dichotomy. Thus, for many (patients and health practitioners alike) a dichotomy often exists in depression treatment and conceptualisation. This thinking was explicit in the first edition of the DSM (American Psychiatric Association, 1952), which differentiated between *organic* and *reactive* depression. The DSM-II (American Psychiatric Association, 1968) continued this divide, to a lesser extent, with the psychoanalytic terms *neurotic* and *psychotic* depression—but with a similar distinction in meaning. Theory held that so-called organic depression necessitated pharmacological treatment, whereas reactive depression was more responsive to psychotherapy (Parker, 2000). This distinction ceased from the third edition of the manual (American Psychiatric Association, 1980), in line with its supposedly atheoretical, or theoretically-agnostic, approach to the aetiology of listed disorders. (However, whether these subsequent editions are truly atheoretical—or if such a position is even possible—is debatable; Follette & Houts, 1996; Rogler, 1997)

Undeniably, then, various social forces play a role in the conceptualisation of mental illness (Bentall, 2005; Horwitz & Wakefield, 2007). The full nuances of the changes to depression as a diagnostic category are too detailed to adequately convey here, but the reader is encouraged to consult McPherson and Armstrong's (2006) excellent study of the diagnostic labelling of depression over time for greater context. Besides space constraints, this is excluded because, unfortunately, such great external influence is not easily changed. However, faulty logic is far easier to expose.

Another prominent reason for the above-mentioned dichotomy stems from an underlying acceptance, or even propagation, of a specific philosophy of mind, Cartesian dualism, which posits the mind and body as two discrete entities (Brenner, Roder, & Tschacher, 2006). Despite being both logically (Searle, 2008) and empirically (Panksepp, 2005) flawed, dualism persists both with the majority of the general public, and a sizeable

portion of health professionals (Demertzi et al., 2009). Its acceptance is problematic for patient care in broader medicine (Leder, 1984; Switankowsky, 2000), but especially so for psychology and psychiatry (Gabbard, 2000), where commercial interests may have had undue influence on dualism's persistence: As alluded to above, some in the pharmaceutical industry have emphasised exclusively biological interpretations of disease (Mehta, 2011), and some branches of psychotherapy continue to argue that exclusively cognitive explanations are superior (Purcell, 2008; Swoiskin, 2001a, 2001b).

Although there is certainly no consensus within the discipline of philosophy of mind, one particular alternative to dualism fits available neurological evidence congruently: *dual-aspect monism*. That is, what we refer to as *mind* and *brain* are ultimately referents to an identical entity; the former experienced subjectively, and the latter observed objectively (Diaz, 2000; Solms & Turnbull, 2011). Simply put: the question of whether depression is a malady of mind *or* brain (body) is ultimately nonsensical one—the answer is simultaneously *both.* As such, we should fully expect that depression or, in fact, any other subjective experience, correlates with observable brain changes, as these changes, rather than causal evidence for the experience, *are* the exact thing that the individual is feeling—observed objectively (Hardcastle, 1992).

Not merely a philosophical aside, this fundamental concept allows for a far more nuanced view of depression aetiology and treatment.

The cognitive neuropsychological model. As seen above, antidepressants do not effect clinical change linearly through an increase in monoamines—but they *are* effective in depression treatment (Fournier, Derubeis, Hollon, Shelton, & Fawcett, 2010; Nierenberg et al., 2008, but see Turner et al., 2008 on publication bias and Vöhringer & Ghaemi, 2011 on effect sizes.) How, then, does this change occur? Some authors have pointed to neurogenesis (Eyre & Baune, 2012; Willner, Scheel-Krüger, & Belzung, 2013), especially hippocampal repair (Hanson, Owens, & Nemeroff, 2011; Santarelli et al., 2003). This line of research is promising and fascinating, but a new model integrates multiples lines of cognitive and biological evidence in a congruent way that provides novel, testable, hypotheses relevant to neuropsychology.

Although mood-related changes in depression symptoms are not evident for several days following antidepressant treatment, subtle changes in cognitive biases are evident after even the first dose—even in non-depressed individuals. For example, a 20mg dose of citalopram (an SSRI) reduced amygdala response to fearful facial expressions only three hours after administration (Murphy, Norbury, O'Sullivan, Cowen, & Harmer, 2009) in non-

depressed volunteers. Similarly, a single 20mg dose of citalopram influenced the electrophysiological response (event-related potentials) of facial emotion decoding for positive emotions, showing that this dose created a perceptual bias towards the positive stimuli (Kerestes et al., 2009). In depressed patients, citalopram showed to increase recognition of happiness and surprise in two weeks—and the extent to which this occurred predicted positive treatment outcomes at six weeks (Tranter et al., 2009). In the other direction, acute tryptophan depletion (which, being a precursor of serotonin, leads to markedly reduced serotonin levels) results in the resurfacing of cognitive biases—including impaired recognition of happy facial expression—in remitted depressed individuals, but not in never-depressed controls (Hayward et al., 2005). Thus, a simple linear relationship between monoamine levels and mood seems unlikely.

Below, Figure 2 shows a comparison of the commonly-accepted view of antidepressant action versus the pathway proposed by the cognitive neuropsychological model.

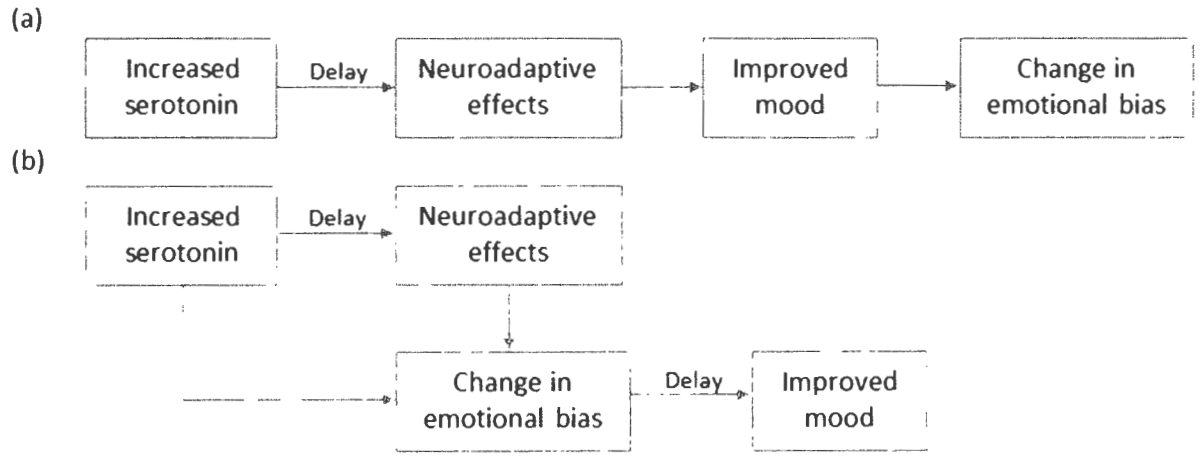

Figure 2. Comparison of the accepted view of mechanism underlying the delay in antidepressant action (a), and the pathway proposed by the cognitive neuropsychological model (b). Adapted from "Why do Antidepressants Take so Long to Work? A Cognitive Neuropsychological Model of Antidepressant Drug Action," by C. Harmer, G. Woodwin, and P. Cowen, 2009, *The British Journal of Psychiatry, 195,* p. 103.

Importantly, the cognitive neuropsychological model places changes in emotional bias as integral (and temporally prior) to improved mood, whereas other accounts place changes in emotional bias as a consequence of improved mood, which itself follows directly from downstream neuroadaptive effects. An extended version of the cognitive neuropsychological

model is shown in Figure 3 below. Here, uniquely, is a formulation of how biological and cognitive influences interact at different levels of processing.

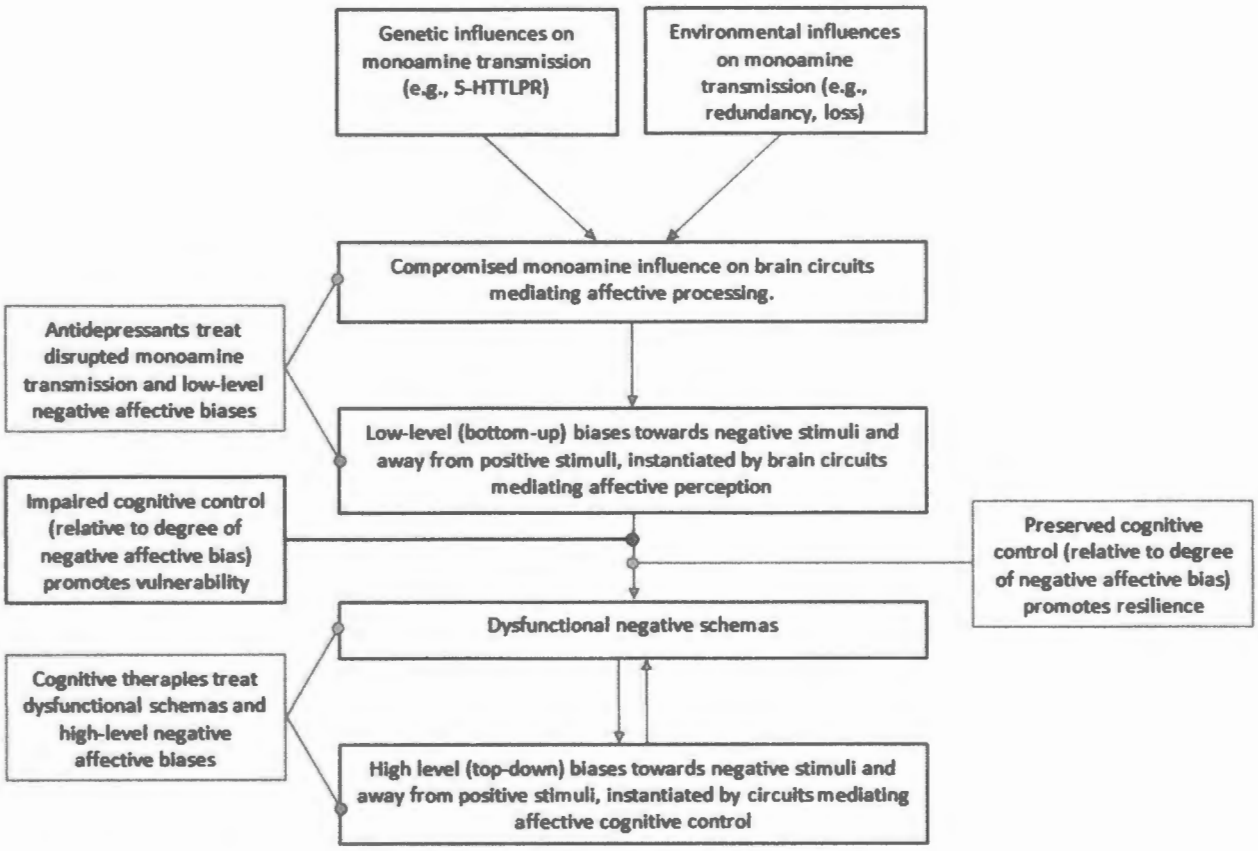

Figure 3. The extended cognitive neuropsychological model of depression. Red boxes represent factors contributing to the onset and progression of depression symptoms; green boxes represent factors in depression treatment. 5-HTTLPR: serotonin transporter-linked polymorphic region. Adapted from "Cognitive mechanisms of treatment in depression" by J. Rosier, R. Elliot, and B. Shakian, 2012, *Neuropsychopharmacology, 37,* p.119.

Uniquely, the cognitive neuropsychological model provides a framework for affective biases being central in depression onset and treatment—in both talk therapy *and* pharmacological approaches. In the latter instance, this model contends that the time delay seen antidepressants is in part because of the need for patients to first interact with the world while holding their new positively-valenced processing biases (Harmer et al., 2009).

Further evidence for this position comes from research into relapse rates across treatment approaches: After cessation of either treatment, psychotherapy typically shows lower rates of relapse over time (Hollon, 2005). Part of the reason for this is likely that reversal of negative low-level biases with antidepressant treatment persists only for as long as medication is continued; conversely, improvement of negative schemas with cognitive

therapy often persists after treatment (Nierenberg, 2003). Also more understandable by this view is the consistent finding (Blackburn, Bishop, Glen, Whalley, & Christie, 1981; Keller et al., 2000; Melvin et al., 2006) that a combination of psychotherapy and antidepressant treatment is often more effective than either treatment alone. By the cognitive neuropsychological model, this is explained by psychotherapy being more effective at changing schemas (top-down) when automatic processing biases are being influenced (bottom-up) by antidepressant action.

Thus, rather than two competing frameworks for understanding depression aetiology and treatment, the cognitive neuropsychological model provides a consistent explanation of both psychological and pharmaceutical approaches to depression—as is, a priori, expected from broader philosophy of mind—and one which places neuropsychology in a privileged place for depression research and treatment.

Cognitive Biases in Depression

What, then, is the extent of cognitive bias and deficit in depression? Broadly, maladaptive schemas, and the DSM-5's criterion of diminished attention (stated more precisely as" diminished ability to think or concentrate, or indecisiveness, nearly every day"; American Psychiatric Association, 2013) are already mentioned above—yet they fail to convey fully the extent of deficit (and the corresponding research base) in the disorder.

It can be useful to separate general deficits in depression from those more-specific to emotional content—although these are not always mutually exclusive. Take, for example, the example of executive dysfunction: Executive functioning is a broad term for high level cognitive control processes, which are frequently impaired in depressed individuals (Biringer et al., 2005; Snyder, 2012). However, it is possible that these impairments are (at least partly) the result of rumination—the compulsive focus of attention towards negative thoughts (Levens, Muhtadie, & Gotlib, 2009; Philippot & Brutoux, 2008; Watkins & Brown, 2002). Being, primarily, a disorder of maladaptive mood, this lack of clear distinction should perhaps be no surprise—yet other general deficits (lacking emotional content) also exist. Perhaps the most comprehensive review of these is in a recent meta-analysis (Rock, Roiser, Riedel, & Blackwell, 2014) that, for consistency, included only studies that employed the Cambridge Neuropsychological Test Automated Battery (CANTAB; see Sahakian & Owen, 1992). In sum (and in line with other research, e.g., Weiland-Fiedler et al., 2004), unmedicated depressed patients showed impairments on working memory, set-shifting, associative learning, pattern recognition memory, spatial recognition memory, and rapid

visual information processing (a measure of attention). However, some (e.g., Gotlib & Joormann, 2010) show that at least some of these observed effects are negated when tasks are administered in a more structured fashion, eliminating the possibility of rumination to influence task performance.

Where emotional content is involved, a general picture emerges of increased attention and preoccupation towards negative stimuli (Hamilton & Gotlib, 2008; Levens & Gotlib, 2010). Specifically, in free recall, depressed participants show fewer recollections of positive autobiographical memories (K. D. Young et al., 2012), instead recalling more general negative categories of events (Williams et al., 2007). Similarly, depressed individuals show a tendency to interpret ambiguous stimuli in a negative light (Hamilton & Gotlib, 2008). Similarly, in eye-tracking studies, depressed participants, although not more likely to orient towards negative stimuli, took longer to disengage from negative stimuli once focused thereupon (Baert, De Raedt, Schacht, & Koster, 2010; Caseras, Garner, Bradley, & Mogg, 2007). One specific subset of cognitive bias, though, is of particular interest to the predictions of the cognitive neuropsychological model.

Emotion recognition biases. Unlike more general deficits such as in attention and memory, emotion recognition biases represent a practical, interpersonal level of cognitive distortion—which is also closely linked to cognitive theory.

Facial emotion recognition biases. Unsurprisingly, the vast majority of emotion recognition research (in depression and otherwise) has relied on visual stimuli (Song, You, Li, & Chen, 2008). Faces are highly salient stimuli—the result of the evolution of specialised neural circuits dedicated to their processing (e.g., the fusiform face area; Kanwisher, McDermott, & Chun, 1997)—and provide a convenient means of reading the mental state of others (R. T. Schultz, 2001). In line with this evolved nature, a substantial research effort, dating back to Charles Darwin (1872/1904) shows uniformity in fundamental emotion displays not only across disparate cultures—but also species. The landmark work of Ekman (e.g, Ekman & Friesen, 1971; Ekman, 1992) showed that six *basic emotions* (anger, disgust, fear, happiness, sadness, and surprise) are universally recognisable in healthy humans—even those in culturally-sheltered environments, such as Papua New Guinea (Ekman, Sorenson, & Friesen, 1969).

A simplistic view of the biases seen in depression, supported by some evidence—and in line with other negative biases in the disorder, is that depressed individuals show decreased accuracy in identifying happy faces (Milders, Bell, Platt, Serrano, & Runcie, 2010), increased accuracy in identifying sad faces (Leppänen, Milders, Bell, Terriere, & Hietanen, 2004), and

a bias towards interpreting neutral faces as sad (Gray et al., 2006), as well as judging negative stimuli as more intense and positive stimuli as less so (Liverant, Brown, Barlow, & Roemer, 2008; Yoon, Joormann, & Gotlib, 2009). However, not all research supports this straightforward account.

Findings are contradictory regarding the identification of specific emotions. In negative stimuli, there is some evidence that depressed individuals are more accurate at identifying sad faces than nondepressed controls (Gollan, McCloskey, Hoxha, & Coccaro, 2010; Leppänen et al., 2004; Milders et al., 2010). However, others find that depressed individuals are *worse* than controls at identifying sad faces (Csukly, Czobor, Szily, Takács, & Simon, 2009).

For positive stimuli, depressed individuals make more errors in identifying happy faces than controls (Csukly et al., 2011; Milders et al., 2010; Surguladze et al., 2004) but in at least one case (Surguladze et al., 2004) depressed individuals made relatively more errors in identifying *sad* faces—contrary to what a general negative bias would predict. These findings lend some support to the suggestion that depression may carry a non-specific deficit in emotion recognition, where depressed individuals have relative difficulty in accurately identifying *all* emotions (Bourke, Douglas, & Porter, 2010). However, other research (Gollan, Pane, McCloskey, & Coccaro, 2008; Leppänen et al., 2004) has found no deficit in recognition accuracy in depressed participants relative to controls.

Studies using neutral stimuli, however, tend to find more consistent results, with depressed participants more likely to interpret neutral faces as sad than controls (Gollan et al., 2008; Leppänen et al., 2004)

Happy, sad, and neutral facial expressions are, of course, not the full range of facial emotion expression—though far fewer studies have directly addressed the recognition of other emotions in depression. For example, one study found no difference in fear recognition accuracy in depressed patients (Kan, Mimura, Kamijima, & Kawamura, 2004). Similarly, other studies have found no specific bias towards angry faces (Gotlib, Krasnoperova, Yue, & Joormann, 2004; Mogg, Millar, & Bradley, 2000).

Limitations of facial recognition studies. Cognitive theory predicts that there should exist a negatively-skewed bias in facial emotion recognition in depressed participants—yet, as seen above, existing research is ambiguous. There are several reasons why this may be the case, and why facial stimuli alone are insufficient, including: ceiling effects, inconsistent stimuli across studies, the effects of antidepressants, and over-arching global deficits.

Precisely because facial expressions are such salient stimuli (Fasel & Luettin, 2003), it is perhaps unsurprising that studies using stereotypical, unambiguous stimuli would encounter a ceiling effect (Hoffmann, Kessler, Eppel, Rukavina, & Traue, 2010). For this reason, many studies use more subtle stimuli—typically digitally-morphed photos that combine neutral expressions with stereotypical displays to create a range of emotional intensities. A common example of this is morphed versions of Ekman's Pictures of Facial Affect (POFA; Ekman & Friesen, 1971; discussed later). The Facial Expressions of Emotion: Stimuli and Test (FEEST; A. Young, Perret, Calder, Sprengelmeyer, & Ekman, 2002) morphs the POFA not with neutral expressions, but rather with *other emotions*—creating a so-called 'emotion hexagon'. The immediately-apparent trouble with this approach is that, objectively, the resulting images do not represent *any* particular emotion, and completely lack ecological validity. Similarly, some other studies employ computer-generated faces (Papesh & Goldinger, 2009) or very basic stick-figure line drawings (Hale, Jansen, Bouhuys, & van den Hoofdakker, 1998; Hale, 1998). Thus, studies employing such vastly different stimuli are not directly comparable.

Further, as outlined above in the cognitive neuropsychological hypothesis, antidepressant treatment alters emotion perception in depressed individuals—likely aiding to correct any pre-existing emotion recognition biases. However, very few of the above-mentioned studies attempt to control for this effect (or, indeed, even acknowledge it). Consequently, a lack of observed emotion recognition bias may instead be a reflection of effective antidepressant action in some studies (Harmer, Cowen, & Goodwin, 2011).

Because emotion recognition is a process reliant on other processes, some of the deficits seen may be due to underlying impairments unrelated to negatively-oriented cognitive biases. As mentioned above, impaired executive dysfunction is one such process—but, again, it is possible that the executive impairments seen are due to excessive occupation with negative stimuli (Philippot & Brutoux, 2008). Specific to facial (visual) stimuli, however, is the finding that gaze behaviour may explain at least some of the deficits seen in emotion recognition in depression. Negative mood states, common in depression, is associated with detailed processing of separate visual elements (local processing), and happy mood with the integration of elements (global processing; Gasper & Clore, 2002; Kimchi & Palmer, 1982). Where faces are concerned, this equates to a greater focus on the eyes and nose in local processing, and a focus on all facial features in global processing. Because mouth movement (i.e., smiling) is more essential for recognising happiness, the local processing associated with sad mood could lead to diminished identification of happy faces

by virtue of primary perception alone—a hypothesis with some empirical support from a mood-induction eye-tracking study with non-depressed participants (Schmid, Schmid Mast, Bombari, Mast, & Lobmaier, 2011).

Vocal emotion recognition biases. Studies employing vocal emotion recognition tasks are able to circumvent the possible confound of gaze behaviour found in facial recognition tasks.

Similarly to facial emotion studies, studies vary—with some finding a general deficit in vocal emotion recognition (Péron et al., 2011; Uekermann, Abdel-hamid, Lehmkämper, Vollmoeller, & Daum, 2008), and others finding depressed participants to show a negative bias only for neutral stimuli (Kan et al., 2004), and judging negative emotions as more intense than positive ones (Naranjo et al., 2011) relative to controls.

The literature here, however, is comparably much scarcer than facial emotion research, and features highly disparate stimuli—owing to the greater complexity of vocal emotion: Whereas facial emotion displays comprise of facial configuration only, vocal emotion contains both tone of voice (prosody) and its semantic content in words. In general, two options exist for vocal stimuli: creating stimuli with no semantic content (in the form of monosyllabic outbursts, or nonsense words or phrases), or using language with semantic content.

The inclusion of semantic content can, arguably, result in a very different research question than by testing prosody judgement only, especially if there is incongruence between the spoken words and their emotional intonation. Indeed, a study that used this paradigm (Uekermann et al., 2008) concluded that impaired executive functions likely caused the observed effect—which is unsurprising when considering the task difficulty of, for instance, identifying prosody while ignoring incongruent semantic content. (In fact, this paradigm bears similarities to the structure of a common measure of executive function, the Stroop test; Stroop, 1935). Further discussion and motivation for semantically-empty tasks follows in *Methods*.

Musical emotion recognition biases. Vocal expressions are not the only auditory emotional stimuli; music is both a highly expressive means of communicating emotion (Juslin & Laukka, 2003; Juslin, 2000) that is recognised universally (Fritz et al., 2009) and, more recently, forms the basis of a standardised emotion recognition stimulus set (Vieillard et al., 2008; described in more detail in *Methods*). Unlike vocal and facial stimuli, these stimuli are unique in that they are far less interpersonal in nature—thus potentially making them less prone to cognitive biases.

Contrary to what traditional cognitive theory would expect (because it is relatively lower-level, not involving maladaptive beliefs about people in which the depressed are in contact) however, similar biases to those seen in facial and vocal stimuli are also present in musical stimuli: Depressed individuals are less accurate at identifying happy and peaceful (neutral) excerpts (Naranjo et al., 2011), rate sad and fearful clips as more intense, and happy and peaceful excerpts as less intense than controls (Naranjo et al., 2011; Punkanen, Eerola, & Erkkilä, 2011).

What do Emotion Recognition Biases Mean?

Biased emotion recognition exists in depression that, at least by some studies, aligns with predictions from cognitive theory—with depressed individuals showing a negatively-skewed bias towards stimuli. Some evidence (Csukly et al., 2011) empirically links bias severity with cognitive schemas—but the effect sizes in this study are small and, as just mentioned, the existence of biases in impersonal (musical) stimuli does not align consistently with aspects of cognitive theory that posits attentional biases as high-level, interpretive biases. Complicating the current view even further, though, is the recent finding that emotion recognition biases can be elicited in non-depressed individuals via mood-induction.

Incidental mood and emotion recognition. It is somewhat surprising that the finding that incidental mood influences emotion recognition is a recent one (Lee, Ng, Tang, & Chan, 2008), given that mood-induction techniques have been used for some time in depressed samples to elicit various depression symptoms (Gross & Person, 1998). To date, only two published studies directly aimed at this question exist—and both have shortcomings.

The first study of this kind (Lee et al., 2008) assessed the facial emotion recognition abilities of 47 non-depressed participants following either happy, sad, or neutral mood induction. Participants in the sad mood-induction showed a negative bias towards ambiguous stimuli compared with the other groups. However, the small sample size limited other analytical possibilities, and the stimuli used were not ideal: photos unique to their research team were morphed across emotions instead of with a neutral condition.

A more-recent study (Schmid & Schmid Mast, 2010) improved on the above study by increasing the sample size to 93. Only sad and happy faces were used, selected from the POFA, and were morphed with neutral faces to create varying emotion intensities. Besides these improvements, though, the method was not optimal. Particularly: supposedly as part of the mood-induction procedure, mood-congruent music was played *during* the experimental condition. Rather than, or in addition to, inducing mood, it is likely that this music served as a

contextual clue for interpreting ambiguous faces. In addition, no intensity rating scale was provided, and participants simply had to identify the mood in the faces (happy or sad). Especially since only two conditions were present, this method does not allow for a nuanced view of the recognition abilities of the participants. Lastly, participants were not screened for (and therefore not balanced on) depression symptoms.

The need for a better method. Perhaps the most obvious limitation of the above two studies is that they were only conducted in the visual domain. As mentioned above, facial emotion recognition may be mediated influenced by gaze behaviour, which is in turn susceptible to the influence of transient mood. Thus, the biases observed in induced mood in normal participants may not hold for other modalities. The addition of a vocal emotion recognition task would answer this question. The addition of the final domain of emotion recognition, music, would allow for statistical comparisons across all three modalities—something that is only available in a minority of cases in the depression literature, and a first using normal participants.

Aims and Hypotheses

Plainly stated, the main aim here is to determine whether the types of emotion recognition biases seen in depression can be elicited in non-depressed individuals via a mood-induction paradigm. The cognitive neuropsychological model and the traditional cognitive model place emotion recognition biases at different temporal points; assessing the malleability of these biases by mood alone would assist in determining which causal framework is more likely, and perhaps aid depression treatment research.

Specifically, my hypotheses are:

- Contrary to what some cognitive theory predicts (Csukly et al., 2011), emotion recognition biases will emerge in non-depressed individuals across three modalities, when subjected to a mood-induction procedure.
- The above-mentioned biases will be mood-congruent; sad mood induction will elicit negative biases, and happy mood induction will elicit positive biases.
- Although biases will be present in the absence of negative schemas, where schemas are present, they will be associated with negative biases—in the *Neutral* induction condition (because this condition is the only one not confounded by mood induction that can trigger low-lying schemas).

Method

Design and Setting

The experiment was an independent measures design across three groups, namely: positive, negative, and neutral mood induction. Although a repeated-measures design would have been statistically superior in theory, in practice the high time commitment required of participants in such a design would have led to an unworkably high dropout rate. Instead, participants were matched as carefully as possible on depression scores, gender, and race to control for between-participant variability. Groups were compared on emotion recognition accuracy and intensity ratings.

All research was conducted in the Psychology Department at the University of Cape Town.

Participants

The final sample comprised 103 participants, all psychology undergraduates at the University of Cape Town. Table 1 shows relevant participant demographics.

Table 1

Participant Demographics

	Group				
Variable	Positive ($n = 35$)	Negative ($n = 33$)	Neutral ($n = 35$)	$F(2,100)/\chi^2$	p
Age	20.00 (1.55)	19.36 (1.31)	19.80 (1.56)	1.61	.22
BDI-II	7.02 (5.01)	7.27 (4.11)	7.57 (4.70)	.12	.88
Female	31	28	31	.20	.90
Male	4	5	4	.15	.92
Black	8	8	9	.08	.96
Coloured	8	8	8	.00	1.00
White	19	17	18	.11	.95

Note: Means and standard deviations are provided for Age and BDI-II score; frequencies are provided for other demographics.

Recruitment. Participants were recruited via the Student Research Participation Programme (SRPP) of the Psychology Department at the University of Cape Town.

Notices of the study appeared on the administration website of the SRPP website during April to November 2013. Any psychology student aged between 18-30, of any gender, not taking antidepressant medication was invited to fill in the online screening form, which consisted of a computerised version of the Beck Depression Inventory, Second Edition (BDI-II; Beck, Steer, Ball, & Ranieri, 1996), as well as a basic demographic questionnaire, asking participants their age, race, and gender.

In total, 799 unique responses were collected via the online form. Potential participants could complete the form at any point throughout the data collection period of the study. Although this was not ideal for matching purposes, doing so was unavoidable due to fluctuating participant availability and time constraints.

All eligible participants were added to a microsite in which the study's administration took place. Available assessment times appeared on this site, and all members of the site had the option to sign up for a time-slot, although there were inevitably more members of the group than available times. This excess of participants was unavoidable as participant availability did not always match researcher availability. Additionally, many of those eligible for the study instead chose to complete other studies available at the university. Participants were awarded SRPP points (non-graded course credits) for their efforts.

Materials and Apparatus

Depression measure. The BDI-II (Beck, Steer, Ball, & Ranieri, 1996) served as the sole measure of depression in both screening and experimental phases of the study. The scale is based on the Diagnostic and Statistial Manual of Mental Disorders, Fourth Edition (DSM-IV; American Psychiatric Association, 1994) criteria for depression. The introduction of the DSM-5 does not, a priori, affect the validity of this scale, because neither the core symptoms of major depressive disorder, nor the duration necessary for diagnosis thereof, have changed with the revision. In line with the diagnostic criteria, the scale focuses on emotional and behavioural changes in the two weeks preceding administration. The total score ranges from 0 to 63, with established cut-off scores for different severity levels: 0-13 indicates minimal depression, 14-19 mild depression, 20-28 moderate depression, and above 29 signifies severe depression. Of course, situational factors may affect scores on this measure, but arguable not to such an extent to compromise its usefulness in this study.

A recent, extensive review (Wang & Gorenstein, 2013) of the psychometric properties of the BDI-II shows its internal consistency to average 0.9, and the test-retest reliability to range from 0.73 to 0.96, with good sensitivity and specificity for detecting depression. Most

relevant here, these psychometric properties hold true even in diverse university samples samples (Al-Musawi, 2001; Carmody, 2005), and is often used in South African studies (e.g., Kagee, Nel, & Saal, 2014; Pillay & Sargent, 1999).

Anxiety measure. The Beck Anxiety Inventory (BAI; Beck & Steer, 1993) served as a measure of anxiety during the experimental protocol.

An anxiety measure was included in the study primarily as a control; because depression and anxiety co-exist so commonly, and anxiety also has an effect on emotion recognition, it was necessary to ensure that anxiety levels, independent of depression levels, were not influencing emotion recognition in the sample. The BAI differentiates anxiety from depression more effectively than many other anxiety measures (Enns, Cox, Parker, & Guertin, 1998).

The scale consists of 21 items, requiring respondents to report on a scale of 0 to 3 the extent to which various state anxiety symptoms have caused distress over the preceding month. As with the BDI-II, the total score ranges from 0 to 63, although different cut-offs exist here: 0-7 signifies minimal anxiety; 815, mild anxiety; 16-25, moderate anxiety; and above 26 suggests severe anxiety.

Although bearing his name, the BAI admittedly does not have as strong a psychometric profile or, indeed, widespread use boasted by the BDI-II. However, the BAI is generally a psychometrically-sound measure. It shows good construct validity, converging with other measures of anxiety, between r= 0.47 and r=0.81 (see Julian, 2014). Although the scale shows substantial correlation with the BDI-II (Wang & Gorenstein, 2013), the use of both the BDI-II and the BAI in this study allows for the impact of this limitation to be lowered—particularly since the scales serves only as a control here. The BAI has shown good psychometric properties in diverse university samples (Al-Musawi, 2001; Creamer, Foran, & Bell, 1995), and is often used in South Africa (e.g., Le Roux & Kemp, 2009; Nel & Kagee, 2013).

Schema measure. The short form of the Young Schema Questionnaire (YSQ-S3; J. E. Young, 2005) measured the extent of negative schemas in participants. Although initially developed in conjunction with Young's schema therapy, a variant of cognitive therapy, the questionnaire has come to be regarded as a reliable and useful measure of maladaptive schemas in both psychopathological and healthy individuals . The most recent iteration is available in short and long forms, containing 75 and 205 items respectively, although the former is used much more commonly in research . The short form contains 18 schemas and the long form 19, with the short form simply containing the items of the long form that load

most highly on those schemas (J. E. Young et al., 2003). As in other research, the short form was preferred here simply because of its relative ease of administration, since time was limited during the experiment.

The psychometric properties of the YSQ short-form are similar to the long form. Additionally, the short form shows good internal consistency and discriminative validity (Oei & Baranoff, 2007). The short form has been used extensively in cross-cultural settings, with, for example, Turkish and Finnish examples existing (Saariaho, Saariaho, Karila, & Joukamaa, 2009; Soygüt, Karaosmanoğlu, & Cakir, 2009). At least one study has directly measured the psychometric properties of the English version in a culturally-diverse sample, finding similar properties in the responses of Australian and Korean respondents (Oei & Baranoff, 2007). Unfortunately, specific details of the psychometric properties of the current version of the scale are currently unavailable. Similarly, the psychometric properties specific to the local population are unknown, although the scale has been used previously in South African research (e.g., Lochner et al., 2005).

Mood induction. Participants were induced to positive or negative mood states through an autobiographical writing task combined with mood-inducing background music. Specifically, participants in these two conditions were asked to write down the details of their happiest or saddest life experiences, while music shown to elicit positive or negative mood played via headphones. Participants wrote on a single blank piece of paper, and were informed before beginning that this page would remain anonymous. Pen and paper was preferred over electronic input in this experiment only because an informal pilot of the study (not reported here) revealed a considerable and limiting variation of typing speed of participants. The effects of written autobiographical tasks on mood have long been known (e.g., Baker & Guttfreund, 1993) and allow for a simple, ethical means of mood induction. Similarly, there is a strong and long-standing empirical foundation for the effect of music on mood (e.g., Pignatiello, Camp, & Rasar, 1986). The combination of these two techniques has been used successfully in many studies (Gerrards-Hesse, Spies, & Hesse, 1994; Petra Claudia Schmid & Schmid Mast, 2010). The choice of music used was based on previous validation (Petra C. Schmid et al., 2011) and included Eine Kleine Nachtmusik by Mozart for the happy condition, and Adagio for Strings, by Samuel Barber, for the sad condition. Those in the neutral condition were asked to recall their typical evening routine while listening to a piece of classical music considered soothing—Neptune the Mystic, by Gustav Holst.

Mood measure. A visual-analogue scale was used to measure the effectiveness of the mood induction. Specifically, a 12cm printed line with a dot on each end, the left-most point

labelled 'extremely sad' and the right-most point 'extremely happy' allowed participants to indicate, by placing a mark along this line, how they felt after the mood induction. This technique has been used successfully in other research (Petra Claudia Schmid & Schmid Mast, 2010) and provides a quick and simple indication of mood.

Other scales, particularly the Positive and Negative Affect Schedule (Watson, Clark, & Tellegen, 1988), which has been validated and used in many studies (Karim, Weisz, & Rehman, 2011; Thompson, 2007), could have been used here, but were excluded for two reasons. First, although a short-form of the PANAS exists (Thompson, 2007), its administration is still lengthier than a visual analogue scale. More importantly, though, the items on this scale presuppose a nuanced English vocabulary, which is not necessarily the case in the present culturally-diverse sample.

Emotion recognition tasks.

Facial emotion recognition. Images from the Pictures of Facial Affect series (POFA; Ekman & Friesen, 1971) measured facial emotion recognition. The set as a whole consists of 110 black-and-white photographs of 13 actors producing prototypical expressions of the basic emotions. For this study, sets of expressions of happiness, sadness, anger, and fear from 3 male and 3 female actors was used. To increase task difficulty, these photos were morphed with neutral expressions at three different rates: 25%, 50%, and 75%. This configuration yields a set of 72 stimuli. No expressions at full intensity were included to avoid a likely ceiling effect (Milders et al., 2010). An equal number of male and female faces was needed because of the known variability in recognition accuracy dependent on the gender of actors in the stimuli (Erwin, Gur, & Gur, 1992).

Additionally, there are two validity considerations regarding the usage of the POFA here. First, all the actors in the photos are white, which has the risk of causing race-based biases in responses: Individuals typically recognise the emotions of members of their own race or culture more easily (Elfenbein & Ambady, 2002; Elfenbein, Beaupré, Lévesque, & Hess, 2007; but see Papesh & Goldinger, 2009), and the current sample is racially diverse. To control for this possible effect, participants were matched as closely as possible on race. It is, however, worth noting that South Africa is an exceptionally diverse nation, wherein individuals are frequently exposed to many races—and recognition biases are negatively correlated with multicultural exposure (Beaupré & Hess, 2006; Elfenbein & Ambady, 2002). Second, as mentioned above, the actors in the POFA look outdated. More than a trivial aesthetic complaint, this fact may draw participants' attention to irrelevant details, such as an unusual haircut (Papesh & Goldinger, 2009). To avoid this potential confound, all non-facial

features (e.g., hair and ears) in the images were removed. This is a common technique when using the POFA (Naranjo et al., 2011).

With the considerable setbacks of the POFA listed above (and later in *Discussion*), the reader may be wondering at this point why, then, another stimulus set was not used instead. The simple answer to this query would be that the existing research corpus almost dictates it; the POFA series is iconic in the literature, and employing it here allowed for the results to be compared with existing research. Many modern batteries (e.g., the Emotion Recognition Index; Scherer & Scherer, 2011) make use of the POFA, and newer facial emotion databases are primarily created for facial recognition software development, and are thus inappropriate for clinical assessment. Overall, the POFA remain dominant in emotion recognition literature (D. Martin, Slessor, Allen, Phillips, & Darling, 2012) and their use here is justified despite their shortcomings.

Vocal emotion recognition. A subset of 64 stimuli from the Geneva Vocal Emotion Expression Stimulus set (GVEES; Scherer, Banse, & Wallbott, 2001) measured the vocal emotion recognition of participants. These stimuli consist of recordings of short, emotionally-tinged yet semantically-empty sentences spoken by two male and two female actors. Happiness, sadness, anger, and fear-laden sentences were used in the experiment, although the set also contains stimuli for disgust (omitted in this study for brevity).

The technique employed in creating the stimuli provides greater credibility to the set: Although many emotion recognition stimuli (including the POFA) simply ask the certain actors to convey a certain emotion, in creating the GVEES actors were asked to envisage specific scenarios. That is, rather than instructed to "sound sad", for example, actors were asked to recall or imagine the death of a loved one. Further, the provided scenarios were based on a previous study (Scherer, 1986) of the perceived emotional content and intensity of various life events . In doing this, the research team contends, interpretation of the emotion states is not left up to the actors, and a more consistent response was obtained during the recording.

To ensure that the stimuli gauge only participants' interpretation of the tone of the voices, the GVEES contain nonsensical sentences. These were created from seven-syllable sequences, comprised of at least one syllable each of six European languages (Danish, English, French, German, Italian, and Spanish). From several sequences, two were chosen by a group of vocal communication experts as being best with regards to articulation, language neutrality, and structure. These were: "Hat sundig pron you venzy," and "Fee got laish jonkill gosterr". Although the sentences contain European linguistic features, and English speakers

commonly judge the sentences to be related to German, the stimuli show similar recognition patterns across disparate language groups (Scherer, Banse, & Wallbott, 2001). Further, even though the home language of the current sample is varied, all participants were attending an English-medium university, thus having a relatively high level of exposure to this language.

The reasons for using these stimuli over other available options mirrors the rationale researchers used when creating this set. Specifically, some stimuli (e.g., Juslin & Laukka, 2001) use semantically-valid sentences, such as "It is eleven o'clock". Here, participants must deal with the emotional prosody of the spoken sentences as well as the semantic content—a situation made more complex by the fact that subtle differences in linguistic stress can change the meaning of the sentences. In attempting to solve this problem, some other stimuli (e.g., Belin, Fillion-Bilodeau, & Gosselin, 2008) use affective 'bursts' or single syllables of speech. Although this avoids the confound of semantic content in spoken sentences, the resulting stimuli bear little resemblance to actual speech or common expressive outbursts. Further, each recording is less than a second, thus not allowing a similar amount of information to be interpreted as would be in regular spoken interactions (Pell & Kotz, 2011). The GVEES avoids all of the above-mentioned limitations, allowing researchers to focus on participants' interpretation of spoken prosody in stimuli that mimic natural speech.

Music emotion recognition. Validated pieces of piano music (Vieillard et al., 2008) served as the basis of the musical emotion recognition task. The set consists of 56 short (~10s) clips of a single piano, based on the Western tonal system and comprising four emotion categories: happiness, sadness, threat, and peacefulness. (The category of 'threat' corresponds with fear, and 'peacefulness' can be considered a neutral category.)

The recordings themselves are computer-generated via the Musical Instrument Digital Interface (MIDI) standard, in a piano timbre. Because the recognition of emotion in musical pieces is a relatively novel area of research (as discussed in the above literature review), no norms for these stimuli currently exist besides the initial validation study. Though the stimuli are novel, there is a thorough research base surrounding the affective qualities of the Western tonal system on which the pieces are based (Balkwill & Thompson, 1999; Janata et al., 2002).

Of course, as its name suggests, the Western tonal system is neither universal nor the only structure on which music can be based and interpreted. For the current sample, however, the widespread use of this tonal system in music familiar to those at the university serves as justification of cultural validity of these stimuli in the present study. Put simply: It is extremely unlikely that any of the participants, being at university level in a highly

Westernised country, were sufficiently unfamiliar with this tonal system to have a bearing on their performance on this task.

Insight measures. To assess participants' insight into their own emotion recognition abilities, as well as whether they were aware of the hypothesis in question, four items questioned:

- "How good do you think you are at reading the emotions of others?"
- "What percentage of emotions do you think you identified correctly?"
- "How motivated were you to identify the emotions accurately?"

All of these questions, except one asking for a percentage, required a response ranging from 1 to 5.

Stimulus presentation software. OpenSesame (Mathôt, Schreij, & Theeuwes, 2012)is an open-source Python-based graphical experiment builder—essentially an alternative to proprietary offerings such as *E-Prime*. In this study, using this software allowed non-restrictive running of the experiment on various university and private computers without any concerns over commercial licensing.

Procedure

After recruitment, the study essentially consisted of two equal sessions that participants were allowed to complete on different occasions—although, with most participants wanting to finish their obligations as quickly as possible, only one participant opted for this arrangement (with all others preferring a single, longer, session.)

The first portion of the experimental session consisted of the depression, anxiety, and schema measures outlined above. After reading and signing an informed consent form, participants sat at a computer on which the BDI-II, BAI, and YSQ-S3 were formatted electronically, on OpenSesame. For each scale, items were displayed individually, with possible responses displayed on-screen and input via the keyboard. The approximate time taken for this portion ranged between approximately 10 and 30 minutes, depending on the speed of participants' responses.

After completing the above scales, participants were allowed a short break if necessary; both to ensure that they were not fatigued during the next portion, and because any interruptions at a later stage would be detrimental to data collection. This second half of the experiment consisted of the mood induction and emotion recognition tasks.

For the mood induction, participants put on a pair of headphones, through which the music matching their experimental condition was already playing. Having been provided with a pen and a blank sheet of paper, they followed the instruction of describing either their saddest or happiest memory, or their normal evening routine. After 10 minutes, participants ceased writing and rated their mood on the visual analogue scale.

The emotion recognition battery followed. Participants were seated at a computer workstation, with the monitor placed approximately at eye level, and followed on-screen instructions. For each emotion recognition subtest, a practice item preceded the actual tests. Participants first identified each emotion depicted, followed by a Likert-type rating scale of each emotion's intensity ranging from 1-7.

Upon completing the emotion recognition battery, participants completed a second visual analogue scale to assess mood persistence. Following this, they completed the insight questions on the computer. This second half of the procedure (including the emotion battery) generally took 60 minutes to complete.

Following the whole experiment, participants were thanked for their participation, and received a pamphlet containing information of local mental health organisations. Every participant was informed that all participants received this form, and it was in no way a result of the responses given in the experiment. Indeed, because the sample was selected precisely because of their low depression scores on the BDI-II, it was unlikely from the outset that these participants would require referrals.

Ethical Considerations

This study followed the ethical guidelines of the Health Professions Council of South Africa and the University of Cape Town, and was granted approval by the ethical review board of the Psychology Department of the University of Cape Town. Several elements of the aforementioned guidelines are worth highlighting here.

Participants were included in the study out of their own free will, and provided informed consent (see Appendix B) before any data was collected. To this end, the nature of the study, especially its potential harms and benefits, was disclosed to participants before any data was collected. Although students are encouraged to gain SRPP points for course requirements, students are able to choose from a variety of on-going studies, or may opt to complete a minor assignment if they are not comfortable with research participation. After consenting to the research, participants were still free to withdraw their participation at any point.

Confidentiality is essential in all research, and especially so in studies that collect such personal information as depression scores. Among the most sensitive data in this experiment was that gathered from the online screening form that, by necessity, contains potential participants' names. This form was hosted in a very secure manner, using a cloud storage service (drive.google.com) that utilises 2-step verification (requiring both a password and a unique code) for access. Once online recruitment had closed, a copy of the form was stored on an AES-256 encrypted flash drive, and all identifying information was removed from the online version.

For data collected during the experimental procedure, participants were assigned a code to use in place of their names on all their responses. Only one handwritten form, kept securely by the researcher (and only for SRPP administrative requirements), linked participants' names to these codes; all data recorded digitally was completely anonymous.

It is important that the experiment cause no harm. Two areas of concern were present in this experiment from the outset. First, the screening procedures had the potential to reveal clinically-relevant depression or anxiety scores in those who completed it. To this end, contact details of mental health resources (Appendix C) appeared on exit of the questionnaire for all students who accessed the form (including those who were not eligible; either those who clicked "decline" to the conditions outlined at the beginning, or who were not the correct age or on antidepressant medication). The organisations listed included the UCT Student Wellness Counselling Service, LifeLine, and the South African Depression and Anxiety Group. These were chosen for their low cost (or free) services that are available to all students regardless of financial circumstances. Second, those in the negative mood-induction procedure were asked to recall a sad event. Had any participant become overly distressed by this, the session would have been terminated and the participant reassured, and offered the option of a same-day appointment with counselling services on campus. However, no participants were visibly distressed by the mood induction.

Results

Overview of Statistical Analyses Used

In line with the hypotheses detailed earlier, the majority of the analyses below are devoted to assessing the difference between groups on emotion recognition accuracy and ratings *per emotion*. The approach taken here favours multiple univariate analyses, though it may initially appear (and would in other circumstances be appropriate) that multivariate analyses should precede univariate analyses. Because the hypotheses concern the between-group difference per each outcome variable (emotion), the linear composite of these variables (as assessed by multivariate analyses) is of little interest. Further, the rationale of first running a multivariate test simply to 'protect' against Type-I error in subsequent analyses is questionable—especially in the case of ANOVAs following a MANOVA (Bray & Maxwell, 1982). Analysing each emotion independently allowed for the more-frequent use of parametric tests, as often the combined data would violate assumptions necessary for MANOVAs, yet individual variables met the assumptions for ANOVAs. In turn, this allowed for effect sizes to be calculated more often, which are arguably of greater importance than statistical significance alone (Hojat & Xu, 2004), and offset the increased risk of Type-I error created by this approach. (For a more-thorough defence of the multiple univariate analysis approach, see Huberty & Morris, 1989).

Other hypotheses rest on the relationship between emotion recognition performance and the extent of maladaptive schemas. Here, arguably, the best approach (allowing sufficient insight without an excessive number of analyses) is the use of partial correlations—comparing experimental performance with schema levels while controlling for depression symptoms. Because the YSQ-S3 total score is a nonspecific indication of schemas, a factor analysis precedes the correlation analyses.

A note on data from scales. There exists considerable, long-standing debate around the appropriate treatment of data derived from Likert and Likert-type items (e.g., Armstrong, 1981; Norman, 2010). Specifically, because these items are ordinal, some argue that their inclusion in parametric tests is unsuitable. However, it is reasonable in some cases to acknowledge that, though individual items are indeed ordinal, *total* scale scores, such as the total BDI-II score, can be treated as interval data. This rationale is applied to the BDI-II, BAI, and YSQ-S3 scores here, following the example of many other analyses (e.g., de Klerk, Steyn, du Plessis, & Botha, 2004), and (when the relevant assumptions support doing so) the mean emotion intensity ratings.

The case of visual-analogue scales is less straightforward. Some (e.g.,Kersten, Küçükdeveci, & Tennant, 2012) argue that, although these single item measures employ a continuous scale (opposed to numbered responses), their data should nonetheless always be considered ordinal. Others (e.g., Harms-Ringdahl, 2012) contend that parametric analyses can be appropriate—if the data meet the requisite assumptions. This question is particularly important here because, as reported later, each of these methods suggest a different interpretation of the measure of mood induction persistence. As such, results of both types of analyses appear below for these measures.

Summary. Thus, in brief: For comparisons of means across groups, one-way ANOVA was the first choice—provided the assumptions required for the validity of this test were upheld. To this end, all data were analysed using boxplots, means plots, Shapiro-Wilk's test of normality, and Levene's test of homogeneity of variances; violations of any assumptions are reported for each test. Because of the specific nature of the hypotheses, planned contrasts followed any significant ANOVA. Where only the homogeneity of variances assumption is violated, Welch's F is reported. When necessary, the Kruskal-Wallis H test was used in place of, or in addition to, an ANOVA. All non-parametric pairwise analyses used Dunn's test with a Bonferroni correction, with adjusted *p* values reported.

Group Similarity

First, it is necessary to ascertain whether the three groups of participants were similar in depression, anxiety, and schema measures—because these factors are known to influence emotion recognition. To this end, one-way ANOVA's were run on BDI-II, BAI, and YSQ-S3 totals across the three participant groups, as well as the four factors of the YSQ-S3 derived from principal components analysis (discussed later).

As seen in the table 2 (overleaf), the groups showed no significant variation in depression, anxiety, or schema levels. Although, as could be expected a priori (because participants were selected based on low depression levels) the data used in the above ANOVAs were not normally distributed, Levene's test of homogeneity of variances was not significant for each case.

Table 2

Psychopathology Levels Across Groups: ANOVA Results

	Group				
Measure	Negative *M (SD)*	Neutral *M (SD)*	Positive *M (SD)*	$F(2,100)$	p
BDI-II	7.27 (4.11)	7.57 (4.70)	7.03 (5.01)	.12	.88
BAI	15.03 (8.22)	12.66 (8.30)	12.57 (10.81)	.77	.47
YSQ-S3					
Total	216.88 (50.62)	210.77 (47.71)	209.17 (58.87)	.20	.82
Factor 1	107.45 (28.69)	102.00 (28.64)	103.91 (37.44)	.25	.77
Factor 2	89.91 (27.91)	85.74 (25.01)	81.57 (27.50)	.82	.44
Factor 3	46.00 (12.15)	47.28 (12.08)	46.00 (14.97)	.11	.89
Factor 4	43.82 (10.87)	42.65 (11.34)	43.26 (10.87)	.09	.91

Mood Induction

The next important consideration to assess was whether the mood induction across the three groups was successful. Both parametric and non-parametric analyses revealed a significant overall difference between groups on the mood measures; however, the non-parametric analysis did not find any significant differences between those in the *Neutral* and *Positive* induction groups. Although planned comparisons on the ANOVA *did* reveal a significant difference between the mean ratings of these groups, the effect size was considerably lower than the other comparisons. Thus, although there was a large, significant difference in mood rating between those in the *Negative* induction and other groups, the difference in mood rating between the *Positive* and *Neutral* groups was considerably smaller, and of questionable statistical and practical significance. Full analyses are below.

Parametric analysis. The data met the assumption of normality, and no outliers were present; however, variances were not homogenous. The overall ANOVA was significant, Welch's $F(2, 65.49) = 42.3$, $p < .001$, $\omega^2 = .43$. Post-hoc analyses revealed that those in the *Negative* induction group ($M = 4.26$, $SD = 1.94$) rated their mood significantly lower than both those in the *Neutral* induction group ($M = 7.04$, $SD = 2.00$), $t(65.95) = 5.82$, $p < .001$, $r^2 = .34$, and those in the *Positive* induction group ($M = 7.93$, $SD = 1.24$), $t(53.75) = 9.24$, $p < .001$, $r^2 = .61$. Those in the *Positive* induction group had significantly higher mood ratings than those in the *Neutral* induction group, $t(56.61) = 2.23$, $p < .05$, $r^2 = .08$.

Non-paramatric analysis. A Kruskal-Wallis H test found a significant difference between groups, $H(2) = 44.01$, $p < .001$. Pairwise comparisons revealed that, although significant differences existed between the *Negative* (mean rank = 24.58) and *Neutral* (mean

rank = 58.66) induction groups, $p < .001$, and the *Negative* and *Positive* (mean rank = 71.20) groups, $p < .001$, there was no significant difference between the *Neutral* and *Positive* induction groups on mood rating, $p = .237$.

Emotion Recognition Tasks

For easier readability, and because the three modalities are not directly equivalent, the results from the emotion recognition tasks are presented per modality. Accuracy, rating intensity, and misattribution data are considered.

Vocal stimuli.

Vocal emotion recognition accuracy.

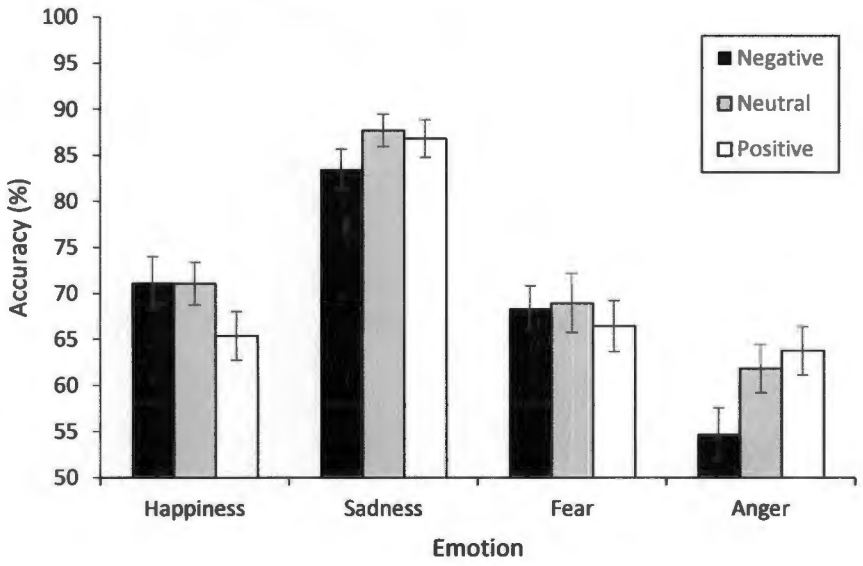

Figure 4. Vocal emotion recognition accuracy per mood induction group. Error bars represent standard errors.

Of all vocal emotion accuracy scores (Figure x), groups differed only on the *Anger* stimuli, $F(2,100) = 3.09$, $p < .05$, $\omega^2 = .01$. Planned contrasts showed a significant difference between the *Negative* ($M = 54.73$, $SD = 16.01$) and *Positive* ($M = 63.75$, $SD = 15.59$) groups on this emotion, $t(100) = 2.37$, $p < .05$, two tailed, $r^2 = .05$, as well as a significant difference between the combination of the *Positive* and *Neutral* groups and the *Negative* group, $t(100) = 2.43$, $p < .05$, two tailed, $r^2 = .05$.

The data for the *Anger* stimuli upheld all assumptions for ANOVA, but data for the three other simuli were not normally distributed. One outlier emerged in the *Happiness* stimuli, and four in the *Sadness* stimuli. Because Levene's test of homogeneity of variances

was nonsignificant for all groups, and nonparametric analyses (not reported here) also proved non-significant, ANOVA results are presented here—primarily because they have the benefit of effect size estimations, although the presence of outliers should nonetheless be considered while interpreting these. The results were: *Happiness,* $F(2, 100) = 1.68, p = .19, \omega^2 = .01$; *Sadness,* $F(2, 100) = 1.19, p = .31, \omega^2 < .01$; *Fear,* $F(2, 100) = .22, p = .81, \omega^2 = .02$.

Vocal emotion intensity ratings.

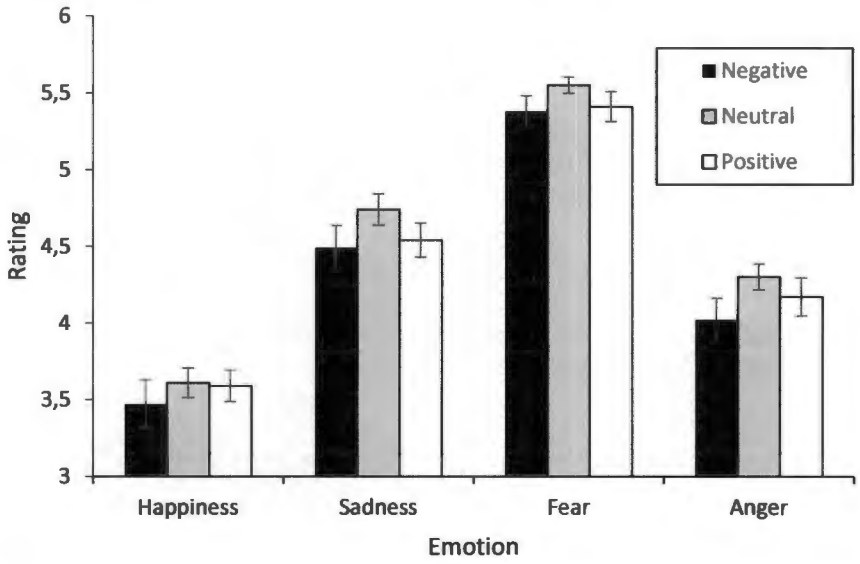

Figure 5. Vocal emotion ratings per mood induction group. Error bars represent standard errors.

Groups did not differ significantly on mean vocal emotion ratings (Figure X). Mean ratings on *Happiness* and *Sadness* stimuli were not normally distributed, several outliers existed on all emotion ratings except *Fear,* and all ratings except *Sadness* violated the assumption of homogeneity of variances—making non-parametric analyses necessary here.

Four Kruskal-Wallis tests found no significant difference between groups on each emotion rating: *Happiness*, $H(2) = 1.02, p = .60$; *Sadness*, $H(2) = 1.95, p = .37$; *Fear,* $H(2) = 3.01, p = .22$; *Anger*, $H(2) = 2.55, p = .27$.

Vocal emotion misattributions. Groups significantly differed in incorrectly identifying *Anger* stimuli as *Fear*, $H(2) = 11.23, p < .05$. Pairwise comparisons revealed this to be due to those in the *Negative* induction making this misattribution more often (mean rank

= 62.79) than those in the *Neutral* induction group (mean rank = 40.13), adjusted $p < .05$. No other significant differences in misattribution emerged.

Musical stimuli.

Musical emotion recognition accuracy.

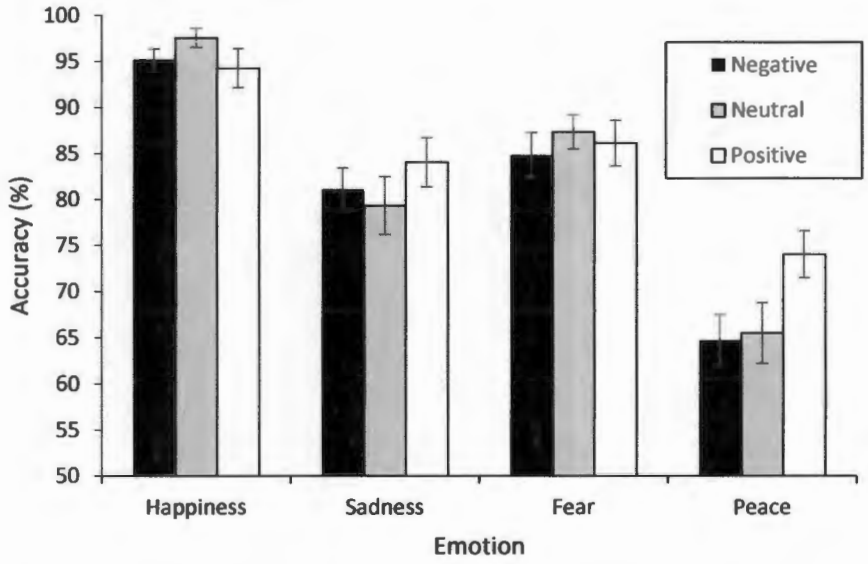

Figure 6. Musical emotion recognition accuracy per mood induction group. Error bars represent standard errors.

Among the musical accuracy scores (Figure X), only the *Peace* accuracy scores were significantly different, $F(2, 100) = 3.24$, $p < .04$. Those in the *Positive* induction group correctly identified more stimuli ($M = 74.08$, $SD = 15.11$) in this emotion group than those in both the *Negative* ($M = 64.71$, $SD = 15.87$) and *Neutral* ($M = 65.51$, $SD = 19.48$) groups, $t(100) = 2.54$, $p < .05$, two-tailed.

The assumptions of normality and homogeneity of variance were upheld for the *Peace* accuracy scores. Two outliers appeared on this group, both in the *Negative* induction, yet did not substantially influence the analysis—as both were on opposite extremes, with their mean (64.28%) similar to the overall mean (64.72%) for this induction group on *Peace* accuracy. Because all other emotion scores were not normally distributed, contained several outliers, and *Happiness* and *Sadness* scores violated the homogeneity of variances assumption, Kruskal-Wallis tests were used for these three stimuli groups. Results were: *Happiness,* $H(2) = 3.99$, $p = .14$; *Sadness*, $H(2) = 1.59$, $p = .45$; *Fear,* $H(2) = .61$, $p = .73$.

Musical emotion misattributions. Groups differed in the number of *Sadness* stimuli misidentified as *Happiness*, $H(2) = 13.52$, $p < .001$. Those in the *Neutral* induction (mean rank = 61.44) made this misattribution significantly more often than those in the *Negative* induction (mean rank = 45.00), $p < .001$, and those in the *Positive* induction (mean rank = 49.16), $p < .05$.

Groups also significantly differed in the misattribution of *Peace* stimuli as *Sadness*, $H(2) = 6.64$, $p < .05$, with those in the *Negative* induction group (mean rank = 60.61) making this misattribution more often than those in the *Positive* induction group (mean rank = 42.37), $p < .05$. No other significant differences in misattribution emerged.

Musical emotion intensity ratings.

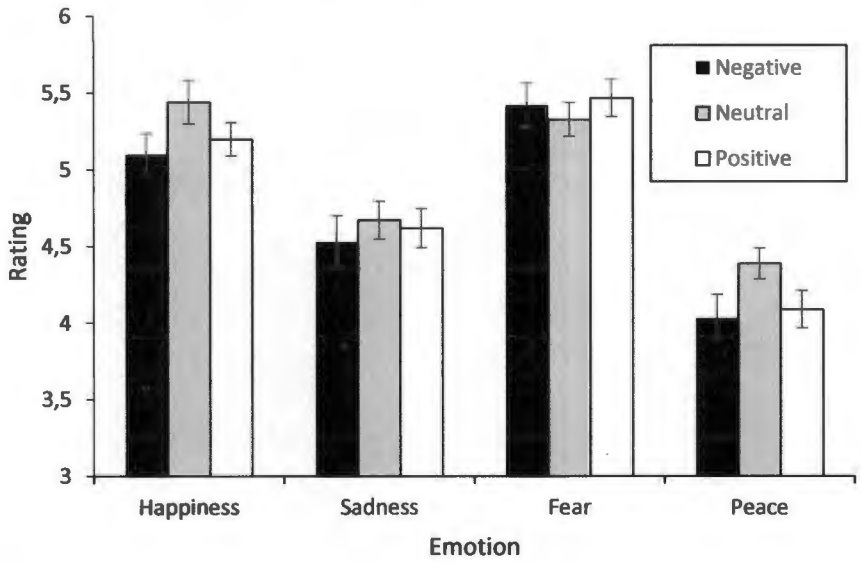

Figure 7. Musical emotion ratings per mood induction group. Error bars represent standard errors.

Groups did not differ significantly on mean musical emotion ratings (shown in Figure X).

For all groups, the assumption of normality was upheld. Three outliers each emerged on the *Sadness* and *Peace* ratings. The mean of two of the outliers on the *Sadness* rating, in the *Neutral* induction group, was similar to the overall mean (4.57 and 4.67, respectively), leaving only one outlier in the *Negative* induction group, rating value = 2. All rating means except *Peace* upheld the homogeneity of variances assumption. Following this, ANOVAs were run on all rating groups except *Peace*. The results were: *Happiness,* $F(2, 100) = 1.83$, p

= .17, ω^2 = .02; *Sadness,* $F(2, 100) = .26$, $p = .77$, $\omega^2 = .01$; *Fear*, $F(2, 100) = .33$, $p = .72$, ω^2 = .01; *Peace*, $H(2) = 4.95$, $p = .08$.

Facial emotion.

Overall facial emotion recognition accuracy.

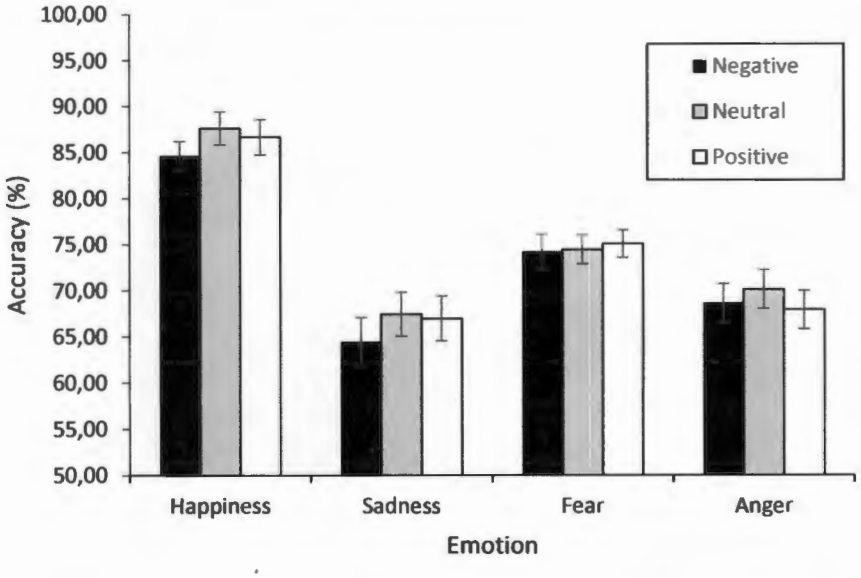

Figure 8. Overall facial recognition accuracy per mood induction group. Error bars represent standard errors

Groups did not differ significantly on mean facial emotion recognition accuracy (shown in Figure X).

Although the assumption of normality was violated for every emotion and an average of two outliers existed in each stimulus group, homogeneity of variances was upheld. ANOVA results were as follows: *Happiness*, $F(2, 100) = .72$, $p = .49$, $\omega^2 < .01$; *Sadness,* $F(2, 100) = .409$, $p = .67$, $\omega^2 = .01$; *Fear,* $F(2, 100) = .07$, $p = .93$, $\omega^2 = .02$; *Anger*, $F(2, 100) = .30$, $p = .74$, $\omega^2 = .01$.

Facial emotion misattributions. Groups showed no significant differences in facial emotion misattributions, $F(22, 182) = 1.04$, $p = .41$.

Overall facial emotion intensity ratings.

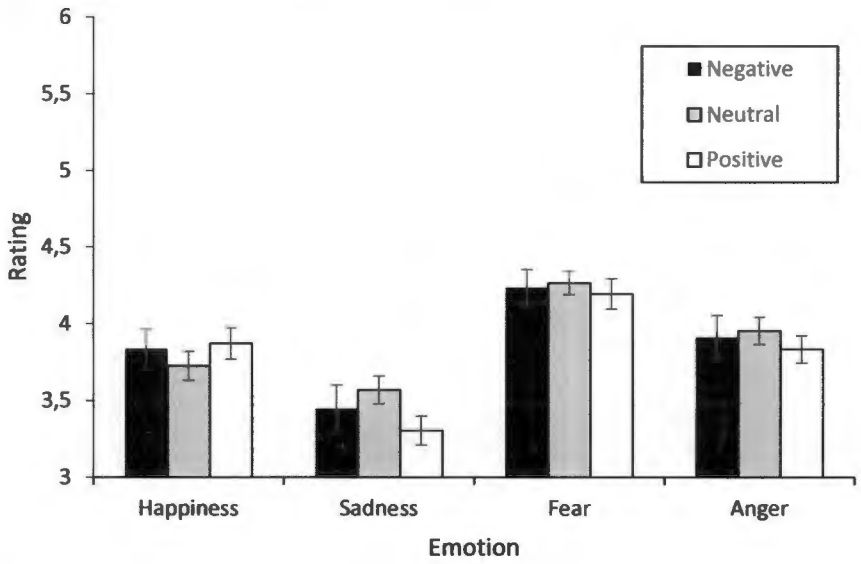

Figure 9. Overall facial emotion ratings per mood induction group. Error bars represent standard errors.

Groups did not differ on mean facial emotion ratings, $F(8,196) = .88$, $p = .53$.

The *Happiness* and *Fear* rating means met the assumptions of normality, had no outliers, and showed homogeneity of variances, and ANOVAs were run on these two emotion groups. However, the *Sadness* rating means failed tests of normality, and both it and the *Anger* ratings had several outliers, and failed Levene's test of homogeneity of variances, making non-parametric testing more appropriate.

Results were: *Happiness*, $F(2, 100) = .51$, $p = .59$, $\omega^2 < .01$; *Sadness*, $H(2) = 4.40$, $p = .11$; *Fear*, $F(2, 100) = .14$, $p = .87$, $\omega^2 < .01$; *Anger*, $H(2) = .47$, $p = .79$.

Mood Persistence

Following the emotion tasks, it is necessary to assess whether the induced mood had persisted throughout the experiment. Because this question is particularly important to the interpretation of the above data, several analyses follow.

The first of these, as with the initial mood rating, is an assessment of the difference between groups on their reported mood at the end of the emotion recognition procedure. Had the mood induction persisted, one would expect a significant difference to remain between the groups. As detailed below, this was only the case in the parametric analysis after removing four outliers, and only a small effect emerged, due to differences between the

Negative and *Neutral* groups. Again, a linear contrast rather than an omnibus ANOVA is warranted here

A second useful comparison is the difference in the two mood ratings (pre- and post-experiment) per induction group. To this end, the results of paired-samples *t*-tests also appear below. This analysis showed a significant improvement in reported mood of the *Negative* induction group, but no significant difference on the other two groups.

Parametric analysis. The mood persistence data passed tests of normality and homogeneity of variance, although one outlier each was present in the *Negative* and *Neutral* induction groups, and two in the *Positive* groups (of values 12, 2, 3.5, and 3, respectively). No significant difference emerged between mean group ratings, $F(2, 100) = 1.136$, $p = .325$, $\omega^2 < .01$.

Because some of the outliers here were extreme and, unlike outliers in previous analyses, were due to a single rating per participant (thus carrying greater likelihood of error per participant), the ANOVA was run again with outliers removed, which found a a significant result, $F(2, 96) = 3.542$, $p < .05$, $\omega^2 = .04$. Planned contrasts showed a significant difference between those in the *Negative* induction ($M = 6.63$, $SD = 1.60$) and those in the *Neutral* induction ($M = 7.61$, $SD = 1.69$), $t(98) = 2.57$, $p < .05$, $r^2 = .05$.

Non-parametric analysis. A Kruskal-Wallis H test, including the outliers in the mood persistence dataset, found no significant differences between groups, $H(2) = 3.27$, $p = .18$.

Paired-samples t-tests. Table 3 shows the results from paired-sampled *t*-tests across pre- and post-experimental mood induction measures. The difference scores of all groups met the assumptions of normality, and only the *Negative* induction group had outliers, which were removed for an additional analysis included in the table. As seen below, there was no significant change in reported mood in the *Neutral* and *Positive* groups before and after the emotion recognition tasks. However, a significant change towards more-positive mood is seen in the *Negative* induction group—which remains even after removing the two outliers.

Table 3

Change in Mood Before and After Experiment: Paired-Samples t-Test Results

	Pre-task	Post-task	Pre-Post				
Induction	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	95% CI	*t*(df)	*p*	r^2
Negative	4.59 (2.60)	6.80 (1.83)	-2.21 (2.95)	[-3.25, -1.15]	-4.29 (32)	< .05	.36
Neg*	4.54 (2.41)	6.81 (1.71)	-2.26 (2.32)	[-3.11, -1.41]	-5.43 (30)	< .05	.59
Neutral	7.05 (2.00)	7.45 (1.92)	-0.41 (1.57)	[-0.94, 0.13]	-1.53 (34)	.13	.06
Positive	7.79 (1.51)	7.14 (1.52)	0.66 (2.21)	[-0.10, 1.41]	1.76 (34)	.09	.08

YSQ-S3 Dimension Reduction

Table 4

Factor Loadings for Principal Component Factor Analysis with Varimax Rotation of YSQ-S3 Subscales

Subscale	Factor			
	F1	F2	F3	F4
Emotional Deprivation	.150	**.770**	-.060	.126
Abandonment	**.534**	.318	.191	.335
Mistrust	**.433**	**.493**	**.472**	.169
Social Isolation	.144	**.786**	.261	.151
Defectiveness	**.404**	**.700**	.121	.063
Failure to Achieve	.396	**.522**	.092	.167
Practical Incompetence	**.510**	**.430**	.159	.114
Vulnerability to Harm	**.723**	.310	.158	.144
Enmeshment	**.808**	.057	.061	-.078
Subjugation	**.696**	.369	.032	.153
Self-Sacrifice	.333	.002	**.714**	.098
Emotional Inhibition	.312	**.665**	.378	-.012
Unrelenting Standards	-.009	.198	**.863**	.127
Entitlement/Superiority	-.046	-.037	.311	**.822**
Insufficient Self Control	.147	**.466**	-.263	**.592**
Recognition-Seeking	.326	.213	.142	**.743**
Pessimism/Worry	**.635**	.302	.337	.214
Self-Punitiveness	**.411**	.179	.389	.123

Note: Factor loadings > .40 are in boldface.

Because the YSQ-S3 is composed of several subscales, use of only the overall schema score results in a loss of detail provided by variance on the individual subscales. One solution to this would be to run multivariate analyses using each of the 18 subscales—but this is problematic not only because each subscale is comprised of the sum of only 5 items (leading

to lower statistical power), but also because correlation analyses become overly complex with so many subscales. Following the example of Csukly et al. (2011), principal components analysis (PCA) is used here as a compromise. Because the long form of the YSQ (which includes an additional schema) was used in the abovementioned paper, the use of the YSQ short-form in following the factors identified is not directly equivalent. This proved empirically true in the analysis described below (c.f., Csukly et al., 2011, p. 133), which identified a different four-factor solution.

The YSQ-S3 data were suitable for PCA analysis. All variables had at least one correlation coefficient greater than 0.3, the overall Kaiser-Meyer-Olkin (KMO) measure was 0.84 (with all individual KMO measures greater than 0.5), and Bartlett's Test of Sphericity was statistically significant ($p < .001$).

Four components with eigenvalues greater than one emerged, explaining 41.2%, 8.6%, 7.7%, and 6.1% of variance; explaining 63.7% of the total variance. (These values are similar to those found by Csukly et al., 2011, which were: 49.8%, 9.1%, 7.1%, and 6.5%) Visual inspection of the scree plot supported a four-factor model, and this model met the inclusion criterion of > 0.4 factor loading per item. Table 4 shows component loadings of the rotated solution.

Schemas and Emotion Recognition in Neutral Group

Here, significant partial correlations between YSQ-S3 factors and emotion recognition performance are reported (controlling for BDI-II scores). Because bivariate outliers are particularly damaging to the validity of correlation analyses, scatterplots were analysed before each analysis, as were stem-and leaf plots for each variable. However, because of the very large number of analyses that would be needed to check the scatterplots for each factor, only the YSQ-S3 total score was compared with each variable beforehand, with the significant individual factors compared via scatterplot afterwards if significant results emerged—and only reported if outliers emerge at this point. This is defensible as the YSQ-S3 factors are, obviously, derived from the YSQ-S3 itself, and the most likely cause of outliers (greatly skewed overall schema levels, or participant data entry response bias) will likely carry over to individual factors. As mentioned before, only the *Neutral* induction group is considered here, as they are not subject to possible mood induction activation effects on latent schemas and emotion affective biases. All significant results below are at the $p < .05$ level, one-tailed, unless reported otherwise. A more-general analysis of overall schema levels and recognition accuracy follows the detailed analyses of individual emotions.

Vocal emotion recognition. When removing outliers, no significant partial correlations emerged between YSQ-S3 and emotion accuracy scores. Sadness intensity ratings significantly correlated with F4 (r = .37) even after removing two outliers. Similarly, a significant partial correlation between fear intensity ratings and F4 (r = .40) emerged after removing two outliers.

Facial emotion recognition. Only fear accuracy was significantly correlated to two YSQ-S3 factors, even after removing one outlier: *F2* (r = -.32), and *F3* (r = -.35).

On intensity ratings, *F4* showed significant correlations with all emotion groups: Happiness (r = .37), sadness, (r =.32), fear (r = .29), and (after removing three outliers) anger (r = .32).

Musical emotion recognition. No musical accuracy scores partially correlated with YSQ-S3 factors and, as seen elsewhere, several outliers (and a likely ceiling effect) was present for the *happy* emotion accuracy responses.

Intensity ratings of sadness correlated with *F4* (r = .33) after removing two outliers. Fear ratings correlated with *F1* (r = -.37) and *F2* (r = -.30).

Overall schema levels and emotion recognition. The above analyses allow an investigation into the effect of certain schemas on particular emotions across domains. However, it is also worth investigating whether overall emotion accuracy scores (all emotions combined) are related to schema levels in the *Neutral* induction group.

No overall correlation emerged between schema levels and vocal emotion recognition accuracy when controlling for depression, however, a significant correlation did emerge between BDI-II scores and overall vocal accuracy (r = -.31).

For both facial and music recognition accuracy, no significant partial correlation emerged for overall schemas and recognition accuracy, nor did significant zero-order correlations emerge between accuracy and BDI-II scores.

Global Schema levels and Emotion Recognition

For reasons explained elsewhere, one should be wary of combining and correlating data from all three experimental conditions (this will be expanded upon more in *Discussion*). However, when this dataset is considered as a whole, significant associations emerge between emotion recognition accuracy and schema levels, but (in some cases) *not* depression scores.

Overall facial emotion recognition, when controlling for BDI-II scores, is significantly correlated with YSQ-S3 scores (r = -.30, p < .001) as well as its factors: *F1* (r = -.24, p < .001), *F2* (r = -.20), *F3* (r = -.24), and *F4* (r = -.31). However, BDI-II is not significantly correlated with overall facial recognition accuracy (r = -.14, p = .08)

Similarly, a significant partial correlation for overall music accuracy and schema levels (r = -.17, p < .05) as well as *F2* (r = -.18, p < .05), but not for BDI-II scores and music accuracy (r = -.14, p = .08).

The exception to this, however, is overall vocal emotion recognition accuracy scores, where no significant correlation is seen between schema levels and recognition accuracy (r = -.01, p = .46), although an association emerges between BDI-II scores and this accuracy (r = -.19, p < .05).

Emotion Recognition Insight

Group differences. Groups did not differ on their percentage estimate of overall accuracy, $F(2,95) = .05$, $p = .95$, or estimate of motivation, Welch's $F(2,64) = 2.75$, $p = .07$, although there was a significant difference between groups on self-reported general skill at recognising emotions, $F(2, 100) = 4.05$, $p < .05$. In this last case, post-hoc analyses revealed the difference to be with those in the *Positive* induction ($M = 3.69$, $SD = .58$) significantly rating themselves as more accurate than those in the *Negative* induction ($M = 3.27$, $SD = .63$), $p < .05$.

Insight. More importantly than group differences, though, is whether participants showed insight into their performance. Because of no significant difference between groups on both percentage estimates and overall recognition accuracy (and because analyses with individual groups, not reported here, yielded similar results), these are considered together here. The below analyses are one-tailed partial correlations, controlling for induction group membership. Two outliers were removed from the *Percentage Estimate* variable, three from overall facial recognition scores, and one each from overall vocal and musical scores.

Estimated task performance was unrelated to overall facial emotion accuracy (r = .004, p = .47). Overall vocal emotion recognition, however, correlated with participants' estimates (r = .25, p < .05), as did musical emotion recognition scores (r = .18, p < .05).

A subtly different question, participants rated how skilled they are in day-to-day recognition of emotions. Although one significant correlation did emerge, between participants' self-judgements of this skill and their performance on the music tasks (r = .17, p < .05), the responses here, including the scale of participants' motivation are not suitable for this type of correlational analysis, as will be discussed later.

Discussion

A substantial, perhaps overwhelming, amount of data appears above; the main findings, however, are somewhat simpler. Contrary to hypotheses, very few significant differences between emotion recognition accuracy and intensity emerged between groups—many of which were not in the predicted mood-congruent direction.

More specifically: Those in the *Negative* induction group were significantly worse at identifying angry voices, and were more likely than those in the *Neutral* condition to misidentify angry voices as fearful. In musical emotion: Those in the *Positive* induction were significantly more accurate at identifying peaceful clips than other groups, the *Neutral* group more often attributed happiness to sad clips than both other groups, and more often attributed peace to sad clips than those in the *Positive* group—though the mood-congruent bias hypothesis predicts that this trend should exist in the opposite direction. In the facial emotion tasks, however, no significant differences emerged.

Regarding schemas levels of those in the *Neutral* condition (the only group in which this was assessed, as to eliminate the confound of mood induction) significant positive correlations emerged between ratings of sadness and fear in voices and the *F4* YSQ-S3 factor. For faces, this group showed a negative correlation between fear accuracy and *F2* and *F3* factors, and the *F4* factor showed a significant positive correlation with the intensity ratings of all four measured emotion. In music clips, the *Neutral* group showed a positive correlation between intensity ratings of sad clips and *F4*, and negative correlations between ratings of fear stimuli and *F1* and *F2* factors. When comparing overall accuracy per modality (that is, the total number of correctly-identified stimuli per facial, vocal, and musical stimuli), no significant correlation emerged in the vocal domain (although a significant negative correlation emerged here with accuracy and BDI-II scores), or in the facial and musical domains when compared individually with overall YSQ-S3 scores. BDI-II scores were controlled for in all the preceding analyses.

When comparing the schema levels of all groups with the overall emotion recognition accuracy per domain, the YSQ-S3 (and its four factors) showed a significant negative correlation with overall facial recognition (and with uncontrolled BDI-II scores); a significant negative relationship also emerged with this complete sample and music accuracy scores. However, no significant relationship between schemas and overall emotion vocal emotion accuracy emerged—although BDI-II scores showed a correlation with vocal recognition accuracy here.

On measures of insight, those in the *Positive* induction group self-reported their task performance as significantly higher than those in the *Negative* induction group—but this was not an accurate judgement when tested empirically, with all groups showing similar task performance. In testing correlations, only vocal and musical emotion stimuli accuracy scores showed significant correlations with self-judgements of task performance.

As such, the hypothesis that mood induction altering emotion recognition biases has not found considerable support in this study. However, there are *many* other factors which should be considered along with these results—not the least of which being the effectiveness of the emotion recognition task employed, and the experimental stimuli used. A systematic critical discussion of the observed results follows.

Validity of Sample

Although long-standing debate surrounds the validity of using university students in social science research (e.g., Cunningham et al., 1974; Gordon et al., 1986), with statistical analyses showing significant differences between students and the larger population on many experimental variables (Peterson, 2001), the use of students in this study is defensible. First, there is strong existing empirical evidence on the universality of both emotion recognition and depressive symptoms (see *Literature Review*). Second, the student population at this particular university is diverse, and is not subject to the charge of being entirely *WEIRD* (westernised, educated, industrialised, rich and democratic; see Henrich, Heine, & Norenzayan, 2010), a common criticism of homogenous psychology research samples (e.g., Arnett, 2008). Third, the present study was conceptualised as a preliminary investigation only, with further data collection, including participants from the general population, envisioned as part of future research. However, it is nonetheless possible that being limited to a narrow population group did have *some* effect on the results—particularly (as discussed later) since the lack of substantial incentives may have had an effect on participant motivation to fully engage with the experiment.

Mood Induction and Persistence

Central to a research question concerning the effect of induced mood is, simply, whether the mood induction procedure had the desired effect on participants—and (if so) whether this mood change persisted throughout the course of the experiment. The answer to the first of these questions is at least partly true, as seen in the above analyses (although there was only a small difference between those in the *Neutral* and *Positive* induction groups). The

answer to the second question, however, is slightly less desirable—as it was clear that those in the *Negative* condition showed a significantly more-positive mood by the end of the experimental procedure.

Of course, a mood-induction technique that does not maintain the desired mood for the length of the experiment is not an optimal one. However, the choice of mood induction was a difficult one, and two principle criteria guided this decision here: cross-cultural applicability, and ethics. First, because of the cross-cultural sample, it was necessary to use a method that would induce mood as equally as possible among participants. The commonly-used method of validated film excerpts (Coan & Allen, 2007) may have allowed for a simpler induction technique, it is likely that this method would have shown consistent mood change across participants. Not only do these excepts have varying effects on those who have not seen the full films from which they are drawn versus those who have (allowing these viewers deeper empathy for the characters), but the clips themselves are deeply embedded in Western culture, with some content seeming inappropriate for more socially-conservative individuals. (For a specific example: A commonly-used film excerpt intended to induce happiness is clip which depicts a female pretending to orgasm—not only is this unlikely to induce *happiness* per se, let alone a lasting happy mood, but some may find this appropriate; Sato, Noguchi, & Yoshikawa, 2007).

The second consideration, ethics, eliminated another possible procedure for mood induction: situational manipulation. This method involves altering the mood of participants by placing them in an emotive situation, and usually involves deception. For instance, a dummy task, such as a test with a fixed outcome of high or low results, could lift or lower participants' mood, respectively (Coan & Allen, 2007). This possibility was excluded not only to limit the unnecessary use of deception, but also because it was possible that the disappointment of negative feedback may hinder the motivation of those in the sad mood induction in completing the remaining tasks. Thus, the mood induction technique seemed the most effective ethical technique for the sample involved.

Despite pilot testing (not reported here), however, the mood induction procedure did not perform as well as hoped—with both little difference seen among the *Positive* and *Neutral* participants, and with the apparent improvement of mood of those in the *Negative* induction throughout the time-course of the experiment. In the first instance, this is at least partly due to the varied level of motivation of participants towards the experiment—something not unique to this study, using university students (Peterson, 2001), and with some participants visibly making minimal effort to complete the autobiographical writing task. In

the latter case, again, pilot testing suggested that the length of the experimental procedure would not exceed 60 minutes—yet actual data collection often exceeded 90 minutes per participant. More extensive and representative pilot testing, admittedly, was necessary.

Ideally, local (culturally-sensitive and relative) mood induction techniques should be investigated for use in future research. Further, the practice of requiring reluctant students to participate in studies for course credit (particularly when there is little choice among available studies) is problematic—especially when it is known that participants often feel used in these situations (Oldfather, 1995; D. P. Schultz, 1969)

Emotion Recognition Task Performance

Tasks used. Despite what previous research suggested (Lee, Ng, Tang, & Chan, 2008; Petra Claudia Schmid & Schmid Mast, 2010), only a few significant differences between groups on both emotion recognition accuracy and intensity ratings—and even where significant results emerged, the effect sizes were marginal. (Admittedly, it is also necessary to acknowledge that, by definition, a null-hypothesis approach using a p value of $< .05$ would, on average, imply that 1 in 20 significant results are actually type I errors.)

There are several (overlapping) possible reasons for this lack of expected bias in emotion recognition—each with varying levels of empirical and *a priori* support. The first of these, in line with the above discussion of the mood induction and persistence, is that the mood induction procedure simply was not effective enough to induce as strong as mood as necessary to elicit possible effects. Emotion elicitation, which has a relatively brief time-course, is a complex practise (Hickey & Davis, 2003); sustained mood induction is even more difficult (M. Martin, 1990).

Second, some of the emotion recognition tasks used were perhaps not suitable (for this or, frankly, any modern research). Specifically, the POFA proved sub-optimal. Their inclusion here was based on the fact that they are among the most widely-used set of emotion recognition stimuli, and normative data exist for various populations (Trevisani, Johnson, & Carver, 2008). However, its continued popularity perhaps exists primarily because of previous dominance in the research, and not out of intrinsic value as a research tool. The images themselves are dated (evidenced most notably in the appearance of the actors), provided in low resolution, of varying exposure, and, in a few items, out of focus. Further, the set is essentially incomplete, with a fear photo missing for one of the actors. This frank assessment is necessary because very few studies mention these limitations—which are

especially relevant given the $175 fee for the use of the stimuli. Here, again, the creation of locally-validated (non-proprietary) stimuli would be optimal.

Specific to facial emotion stimuli: Although stimuli such as the POFA, which are often employed by digitally morphing neutral expressions with stereotypical emotional displays, are superior to stimuli sets that morph disparate emotion types (often of opposite valence; see A. Young et al., 2002), the POFA (and other and other stimuli which might morph neutral and stereotypical displays) are also subject to this criticism of low ecological validity—albeit to a lesser extent. Because subtle emotion displays involve a discrete set of muscles relative to full displays (Wehrle, Kaiser, Schmidt, & Scherer, 2000), an artificial mix of two images does *not* produce an ecologically-valid display of emotion. Regrettably, also, despite using morphed images (for a variety of intensities) in this study, a ceiling effect was apparent in the POFA clips of 75% intensity. (This may partly explain why the tasks in the facial modality showed the least significant results.)

Similarly, although good existing research provided sound rationale for the use of the musical clips (Fritz et al., 2009), some participants seemed amused by their inclusion—at times seeming variously visibly amused, perplexed, and bored.

Computer literacy. Another relevant (and partly unforeseen) aspect of data collection was the wide range of computer literacy skills seen in participants. Although some variation in time taken to respond to items presented on the computer is expected, it was clear with some participants that they were not very familiar with the use of a computer—and even the limited set of keyboard responses required may have influenced the time taken. Although response latency of items was compromised in other ways (see below), the additional time to complete the entire battery could have conceivably influenced the persistence of the mood induction.

Disparity with other studies. Some of the above-mentioned limitations are also present in published studies that found significant results (e.g., Petra Claudia Schmid & Schmid Mast, 2010), yet why is there a disparity? Some of the above-mentioned factors are relevant. Additionally, I must concede that the experimental design employed here may have been too ambitious—placing a large cognitive burden on participants. Not only were three modalities included (with accuracy *and* intensity ratings), but most participants elected to complete the psychometric scales during the same session as the experimental protocol, likely only contributing to fatigue and boredom. Additionally, the lack of flexibility with undergraduate scheduling (both by hour and date—particularly relative to stressful life events, such as exams) may have had undue influence on the results. Not only does affect

fluctuate daily in non-depressed individuals (Brabant & Toiviainen, 2014; Hasler, Mehl, Bootzin, & Vazire, 2008), a common feature of depression (Gordijn, Hoofdakker, & Reinink, 1994) but also sleep deprivation (common during high-stress times among university students) significantly impairs emotion recognition (van der Helm, Gujar, & Walker, 2010). Inability to control for these factors may likely have hampered results, and future studies should set out more narrowly-defined recruitment times and criteria.

Task Performance Insight

Participants' judgements of their task performance correlated significantly with vocal and musical recognition accuracy—perhaps lending more support to the growing picture that the facial recognition tasks (the morphs thereof, or their culturally bound and outdated nature) were not the best choice for this study.

In-depth analyses were not possible on the other insight questions (regarding participants' judgements of their overall emotion recognition skills, and their motivation throughout the task), admittedly because of the poor choice of a 5-item Likert-like scale for these two questions—greater variability (perhaps in the form of a visual-analogue scale) would have allowed for more nuanced analyses. However, the result remains that those in the *Positive* induction group rated themselves as significantly better at everyday emotion recognition than those in the *Negative* group. It is tempting to consider this as a case of depressive realism (where those in a positive mood have optimistically-inflated worldviews; (Moore & Fresco, 2012), although there lack of difference between groups, and the uncertainty over the efficacy of the mood induction procedure makes this an appealing but unfounded conclusion (especially since this question was at the end of the experimental procedure—when induced mood would have presumably begun to wear off).

Task Response Times

Another unfortunate result was the realisation that data for response times per each emotion item were not reliable, due to the use of a variety of old computers and (the worst culprit) *thin client* terminals (which possess minimal processing power—but instead rely on a server, which can cause unacceptable latency if incorrectly configured or overloaded). Because response time to emotional stimuli is a topic of focus within depression , future studies should ensure that the experimental equipment is capable of delivering reliable timing data.

Conclusion

This study began from a broad background of depression theory and research, with the dual aim of highlighting a relatively-new novel approach to depression aetiology and treatment that aligns well with (arguably) the best knowledge and philosophical thinking today concerning the mind-brain and its disorders. From this, specifically, arose the question of how a particular subset of automatic processing biases—emotion recognition—relates to depression. More specifically: Is it possible to elicit similar negatively-biased emotion recognition seem in depression by a mood-induction procedure? Similarly: Is it also possible to produce positively-biased emotion recognition biases? More than a curiosity, a definite "no" to either of these answers would contribute considerably to current treatment theory—if it is not possible to alter these biases by mood alone, it become more clear that more than a *mere* mood disorder, depression is inherently linked to maladaptive reasoning and automatic biases. Such a finding would also help explain why cessation of antidepressant treatment frequently leads to depressive relapse. Going one step further, it may even suggest that any treatment that does not address negative schemas or automatic cognitive biases will *always* lead to relapse unless such treatment is maintained indefinitely. This is no small question.

Did the current study answer that question? Unfortunately, no—not entirely, and not for any one reason. Limiting factors included a sub-optimal mood-induction procedure, a relatively small sample size, emotional stimuli not validated for the local context, and an often unmotivated research sample. However, future studies can surely build on these mistakes—including measures such as a matched currently-depressed group for a more accurate comparison of response to mood by depressed versus non-depressed individuals.

One noteworthy finding, though, is that negative schemas, even when controlling for depression, are significantly correlated to some aspects of emotion recognition. This finding adds further weight to the suggestion that maladaptive schemas are a useful screening tool for those at risk for depression, to more-accurately monitor treatment outcomes, and a more objective and sensitive research tool. Further, this finding (though modest) is in line with predictions of the cognitive neuropsychological model.

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Appendix A

DSM-5 Diagnostic Criteria for Major Depressive Disorder (American Psychiatric Association, 2013).

A. Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure.
    - **Note:** Do not include symptoms that are clearly attributable to another medical condition.
    1. Depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g., feels sad, empty, hopeless) or observation made by others (e.g., appears tearful). (**Note:** In children and adolescents, can be irritable mood.)
    2. Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation).
    3. Significant weight loss when not dieting or weight gain (e.g., a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day. (**Note:** In children, consider failure to make expected weight gain.)
    4. Insomnia or hypersomnia nearly every day.
    5. Psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down).
    6. Fatigue or loss of energy nearly every day.
    7. Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick).
    8. Diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others).
    9. Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.

B. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

C. The episode is not attributable to the physiological effects of a substance or to another medical condition.

Note: Criteria A–C represent a major depressive episode.

Note: Responses to a significant loss (e.g., bereavement, financial ruin, losses from a natural disaster, a serious medical illness or disability) may include the feelings of intense sadness, rumination about the loss, insomnia, poor appetite, and weight loss noted in Criterion A, which may resemble a depressive episode. Although such symptoms may be understandable or considered appropriate to the loss, the presence of a major depressive episode in addition to the normal response to a significant loss should also be carefully considered. This decision inevitably requires the exercise of clinical judgment based on the individual's history and the cultural norms for the expression of distress in the context of loss.

Appendix B

Informed Consent Form

UNIVERSITY OF CAPE TOWN DEPARTMENT OF PSYCHOLOGY

Does Transient Mood Produce Emotion Recognition Biases? An Experimental Study

1. Invitation and Purpose

Hi there! You're invited to take part in a research study that will examine differences in emotion recognition between people. I'm a Masters in Neuropsychology student, and this study will form part of my degree requirement.

2. Procedures

If you agree to participate in this study, I will ask to see you once in person, during which time you will be asked to judge the emotional content of some faces, voices, and musical clips. At the beginning of each session, I will ask you to remember and write down a particular emotional event in your life, but what you write will remain completely anonymous.

The session should take no longer than an hour.

3. Risks, Discomforts & Inconveniences

There are no realistic risks in this study. Recalling a negative memory may be a bit upsetting for you, but if you become overly distressed by this, we can stop the experiment immediately. If you are extremely distressed, qualified clinical psychologists are available on campus. Just to be sure, everyone will be given contact numbers of mental health resources after the experiment.

You might also become a bit tired during the testing and, if this happens, you are welcome to take a break at any point.

4. Benefits

This study includes a screening measure for various psychological problems, including depression. Although I am not qualified to provide formal diagnoses, like a clinical

psychologist or medical doctor would, the screening measure will be able to determine whether you should consider psychological treatment.
Participation in this experiment will also give you some insight into psychological research, as well as a few novel emotion recognition stimuli. Once the experiment is over, I will be happy to answer any questions you have or discuss the research in more depth.

5. Alternatives (Other Options)

You do not have to participate in this study—it is completely voluntary. If you decide to participate, you may stop at any point (although you will only receive the full amount of compensation if you complete the entire experiment).

6. Privacy and Confidentiality

Your personal information will be kept very safe in this study. In fact, your name will not be on any information that you provide, and I will keep all hard-copy data in a locked drawer, and all electronic data on an encrypted flash drive.
The information collected from this research might be published, but there will be no way of linking this data to you—especially when it is combined with all the other participants'.

7. Compensation

You will receive 2 SRPP credits upon the successful completion of the study.

8. Questions

If you have questions, concerns, or complaints about the study, please contact Steven Harding ([REDACTED])

9. Signatures

{Participant's name}_____ has been informed of the nature and purpose of the procedures described above including any risks involved in its performance. He or she has been given time to ask any questions and these questions have been answered to the best of the researcher's ability. A signed copy of this consent form will be made available to the subject.

Investigator's Signature Date

I have been informed about this research study and understand its purpose, possible benefits, risks, and discomforts. I agree to take part in this research as a participant. I know that I am free to withdraw this consent and quit this project at any time, and that doing so will not cause me any penalty or loss of benefits that I would otherwise be entitled to enjoy.

Participant's Signature Date

Appendix C

Mental healthcare provider contact details.

Useful Contacts

UCT Student Wellness Service

021 650 1017

28 Rhodes Ave

Mowbray 7700

Registered Psychologists at a nominal fee for students

Lifeline Western Cape

086 132 2322

24/7 Telephonic counselling services

South African Depression and Anxiety Group (SADAG)

www.sadag.org

011 262 6396

Online resources, and telephonic counselling services