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Exploring Compassion: 
A meta-analysis of the association between self-compassion and psychopathology

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Abstract

Compassion has emerged as an important construct in studies of mental health and psychological therapy. Although an increasing number of studies have explored relationships between compassion and different facets of psychopathology there has as yet been no systematic review or synthesis of the empirical literature. We conducted a systematic search of the literature on compassion and mental health. We identified 20 samples from 14 eligible studies. All studies used the Neff Self Compassion Scale (Neff 2003, a). We employed meta-analysis to explore associations between self-compassion and psychopathology using random effects analyses of Fisher’s Z correcting for attenuation arising from scale reliability. We found a large effect size for the relationship between compassion and psychopathology of \( r = -0.54 \) (95%CI = -0.57 to -0.51; \( Z = -3.42; p < .0001 \)). Heterogeneity was significant in the analysis. There was no evidence of significant publication bias. Compassion is an important explanatory variable in understanding mental health and resilience. Future work is needed to develop the evidence base for compassion in psychopathology, and explore correlates of compassion and psychopathology.

Keywords: Compassion, mental health, depression, anxiety, stress, meta-analysis
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1. Introduction

The ‘third wave’ of cognitive behavioural therapies, such as Acceptance and Commitment Therapy (ACT, Hayes, Strosahl and Wilson, 1999), Mindfulness Based Cognitive Therapy (MBCT, Segal, Williams & Teasdale, 2002), Dialectical Behavioural Therapy (DBT, Linehan, 1993) and Compassion Focussed Therapy (CFT, Gilbert, 2005, 2010) give greater prominence to positive affect in the therapeutic process than existing cognitive behavioural therapy schools. They also share a focus on ameliorating psychological distress through changing the person’s relationship with their problems. An important aspect of this is the development of a non-judgmental or compassionate attitude. Mindfulness, loving kindness and compassion based interventions show considerable promise in alleviating emotional distress and promoting well-being, reflected in decreased negative affect and increased positive affect (Öst, 2008; Keng, Smoski & Robins, 2011; Hofmann, Sawyer, Witt & Oh, 2010). Cultivation of a compassionate attitude towards oneself and one’s own difficulties may be an underlying mechanism in mindfulness-based interventions (Kuyken et al., 2010).

1.1. Conceptualisations of compassion

There has also been increasing interest in the relationship between compassion and mental health outcomes. The field is undergoing rapid expansion and the construct of compassion itself can be understood from a number of different perspectives. For example, Goetz, Keltner & Simon-Thomas (2010) defined compassion as “a distinct affective experience whose primary function is to facilitate
cooperation and protection of the weak and those who suffer” (p.351). This distinctive affective state arises from witnessing another’s suffering and that motivates a subsequent desire to help. They locate compassion within a broader family of compassion-related states including sympathy, empathy and pity. These states share a focus upon amelioration of the suffering of others, but differ in terms of their cognitive and behavioural components (Keltner & Lerner, 2010). In this model, compassion is thought to constitute an evolutionarily advantageous trait evolved as part of a caregiving response to vulnerable offspring leading to the preferential selection of compassionate individuals in mating. Correspondingly, compassion emerged as a desirable trait in cooperative relations between non-kin. In this sense, Goetz and colleagues (2010) link the evolution of compassion with the development of positive reputations - i.e. if you get a reputation for being kind-hearted this is good for your survival. This can be viewed as a transactional model placing the appraisal of costs, benefits and motivation at the centre.

Buddhist approaches focus on attentional sensitivity to suffering and a commitment to relieve it. In the Buddhist traditions intentionality and motivation are central and compassion is not seen as an emotion as such. Buddhist traditions co-locate compassion within a system of motivational constructs including loving-kindness, sympathetic joy and equanimity (Buddhaghosa, 1975; Hofmann, Grossman & Hinton, 2011). In this conceptualisation compassion can be understood as an attention and intention towards alleviating interpersonal distress (The Dalai Lama, 2001).

Neff (2003a, b) focuses on self-compassion as a healthy attitude and relationship with oneself. Neff (2003b) defines self-compassion as “being touched by and open to one’s own suffering, not avoiding or disconnecting from it, generating the desire to alleviate one’s suffering and to heal oneself with kindness. Self-compassion also involves offering
nonjudgmental understanding to one’s pain, inadequacies and failures, so that one’s experience is seen as part of the larger human experience.” (Page 87). Neff has argued that self compassion should be associated with increased wellbeing as reflected in lower depression, lower anxiety and greater satisfaction with life.

Gilbert (2010) conceptualises compassion in evolutionary terms, focussing on the interplay between threat, motivational and soothing systems which have choreographed neurophysiological substrates (Depue & Morrone-Strupinsky, 2005; Liotti & Gilbert, 2011). The threat-based system is designed for the detection of threat and the engagement of survival mechanisms to protect individuals against danger. The threat system is closely linked to negative emotions including anger, fear, disgust and shame. There are two positive affect systems including drive and soothing systems. The drive system is linked to motivation and reward based systems linked to evolutionary necessities of food, sexual opportunities, alliances, nest sites and territories. Finally the soothing system is linked to the mammalian evolution of attachment system. It is a social mentality that becomes focused by intention and motivation to alleviate distress in others, recruiting key attributes for attentional sensitivity, sympathy, distress tolerance, empathy and non-judgement. In this model compassion has its roots in the capacity for mammals to co-operate and engage in kinship caring, and the formation of attachment bonds (Bowlby, 1973; Gilbert, 2005; Hrdy, 2009). Therefore, compassion is understood as an evolved motivational system designed to regulate negative affect through attuning to the feelings of self and others, and expressing and communicating feelings of warmth and safeness (e.g. Gilbert, 1989; Spikins, Rutherford & Needham, 2010).

1.2. Empirical status of compassion in mental health
Theoretical models of compassion emphasise different aspects including compassionate appraisals (Goetz et al., 2010), self-compassion and healthy self to self relating (Neff, 2003a,b), compassionate attention and intentionality (Dalai Lama, 2001) and social mentalities which recruit compassionate qualities to attune to and alleviate distress in others (Gilbert, 2010). These differences in conceptualisation suggest subtle but important differences in measurement constructs. However, all models predict that compassion would be associated with improved wellbeing and reduced emotional distress. Although there have been several narrative overviews of compassion in mental health (e.g. Neff, 2003b; Gilbert, 2005, 2010) there has not been a systematic review of the association between compassion and psychopathology. Therefore the current review sought to review the literature on the measurement of compassion in psychopathology. In particular, we wished to estimate the strength of association between compassion and common psychopathology, namely depression, anxiety and stress. We also sought to evaluate the effect of potential demographic moderator variables (e.g. sampling, gender) on the relationship between compassion and psychopathology.

2. Method

2.1. Literature search

Relevant studies were initially identified by searching the following databases: EMBASE (1996 – 2011 Week 16), Ovid MEDLINE(R) (1966 – April, Week 3, 2011), and PsycINFO (January 1960 – April, Week 3, 2011). The following search terms were used as keyword or heading searches, using a three component strategy, as follows: Component 1: {COMPASSION or SELF-COMPASSION or SELF COMPASSION or COMPASSIONATE}; Component 2: {PSYCHOPATHOLOGY or DISORDER or SYMPTOMS}; Component 3: {DEPRESSION or ANXIETY or
PSYCHOSIS]. Limits were then implemented to further refine the scope and ensure quality: databases were de-duplicated; searches limited to peer-reviewed articles; searches limited to human studies; searches limited to adult studies. Studies were eligible for inclusion if they included a clearly defined self-report or interview-based measure of compassion, and a validated self-report or interviewer rated measure of psychopathology was used (e.g. measure of depression, anxiety, general symptoms). Studies were excluded if they did not include a validated or standardised measure of compassion, were published in a language other than English and were not published in a peer-reviewed publication, e.g. conference abstracts, book chapters, dissertations.

Reference lists of all relevant articles were screened by the first author and reviewed by the second author to ensure no studies were overlooked. In addition, Google Scholar was used to search for peer-reviewed, in press studies of involving compassion available online but not yet indexed on databases. Where there was disagreement regarding the suitability of a study for inclusion, eligibility was resolved by review of the full article by both authors. To test reliability of the review process 12% of the articles in the final data set were reviewed by an independent second reviewer, with 100% agreement on inclusion.

2.2. Selection of studies

The initial search strategy generated 728 articles. After review of abstracts, 37 potentially fulfilled the eligibility criteria. After close scrutiny of the text of these publications, a further 21 papers were excluded for the following reasons: no association with clinical measures reported (n=2), case series (n=1), no association with compassion measure reported (n=15), no compassion measure used (n=1), literature review (n=1), qualitative study (n=1). Furthermore, a paper reporting
associations between PTSD symptoms and compassion (Thompson & Waltz, 2008) was excluded as effect size data were reported for PTSD subscales only. Finally, one paper was excluded from the analysis as data were unavailable on total compassion scores (Mills et al., 2007). Therefore, 14 publications were eligible for review, representing twenty participant samples and generating 32 different effect sizes (15 for depressive symptoms; 12 for anxiety and 5 for stress). Included studies are listed in Table 1, subdivided according to clinical symptom. The majority of studies reported the relationship between compassion to continuous measures of symptoms, rather than to a diagnosis. As the current review sought to survey the range of research relating compassion to symptoms correlational and between-subjects studies were included.

**INSERT TABLE 1 APPROXIMATELY HERE**

2.3. Measurement of compassion

All studies used the Self Compassion Scale (SCS, Neff, 2003a). The SCS is a 26-item self-report measure of compassionate responding to oneself, with six subscales measuring three components of self-compassion (Neff, 2003a; 2003b). These components consist of opposing pairs – the ability to treat oneself with kindness (Self-Kindness) vs. critical self judgment (Self-Judgment); seeing one’s experiences as part of a common shared humanity (Common Humanity) vs. isolating one’s experiences (Self-Isolation); and finally being able to hold one’s thoughts in a balanced awareness (Mindfulness) vs. overidentifying with them (Over-Identification). As a self-report measure, the SCS acts a measure of beliefs and attitudes towards self-compassion, and thus does not measure motivational and
interpersonal aspects of compassion emphasized in some conceptualizations of compassion (e.g. Gilbert, 2010).

In order to maintain a parsimonious approach to the analysis we report data for SCS total score only. One study (Gilbert, McEwan, Matos & Rivis, 2011) created subscales for self-compassion and self-coldness by combining the self-kindness, common humanity and mindfulness; and self-judgment, isolation and over-identification subscales respectively. Therefore, in the current analysis we used the self-compassion sub-scale as a proxy for total score. Gilbert et al., (2011) were also the only study to compare measures of compassion, comparing the SCS with the Compassionate Love Scale (CLS; Sprecher & Fehr, 2005). They reported a significant correlation of $r=.31 \ (p<.01)$ for the association between self compassion and compassionate love for others in their student sample, but no significant correlation between self-compassion and compassionate love in their therapist sample ($r=.21, \ p=n.s.$). There was no correlation between self-coldness and compassionate love for others in either the student or therapist sample ($r=.00; \ p=n.s.; \ r=-.04; \ p=n.s.$).

2.4. Effect size coding

Effect sizes for correlations were directly reported ($r$ values). Effect sizes for categorical effects were calculated using Cohen’s $d$, then converted to $r$ values using the formula: $r = \sqrt{\frac{d^2}{4 + d^2}}$ where $d$ is the effect size $d$. Reliability coefficients (alpha) were recorded for the SCS and symptom measures in order to correct for measurement unreliability. Where a reliability coefficient for a measure was unavailable for a given study the reliability coefficient from the original psychometric properties of the measure was used.
2.5. Analytic procedure

A primary meta-analysis was conducted on all identified studies reporting an association between compassion and psychopathology. Weighted mean effect sizes, heterogeneity, sensitivity and subsequent moderator analyses were calculated using Comprehensive Meta-Analysis version 2.2.046 (Borenstein, Hedges, Higgins & Rothstein, 2005) and IBM SPSS Statistics Version 20 (SPSS, Chicago, Illinois), incorporating Hoffman’s (2012) meta-analysis macros and Lipsey & Wilson’s (2001) guidelines. Relevant equations for the analytic procedure are given within the text.

2.5.1 Publication bias

There is potential for over-inflation of mean effect sizes due to the tendency for non-significant findings to remain unpublished. Therefore, publication bias was assessed through visual analysis of funnel plots of sample size (standard error) against reported effect size (Fisher’s z). In the absence of publication bias the plot forms a symmetrical funnel shape with large samples clustering around the mean effect size and greater variability associated with smaller samples. Visual analysis of the current data set suggested symmetrical distribution of samples. Additionally, Orwin’s fail-safe N (FSN; Orwin, 1983) was calculated for the total data set to indicate the number of unpublished studies of non-significant effect size required to reduce the mean observed effect size to zero. Sensitivity of Orwin’s fail-safe N was calculated for a range of hypothesized mean effect sizes based on criterion effect sizes of $r_{\text{range}}=0.1-0.6$ for unpublished studies.

2.5.2. Independence of effect sizes

Effect sizes for the association between compassion and mental health derived from multiple measures of psychopathology (e.g. depression, anxiety and stress) were
reported in nine samples (Raes 2010; Gilbert et al., 2011 both samples; Van Dam, Sheppard, Forsyth & Earleywine., 2011; Costa & Pinto-Gouivea, 2011; Roemer, Lee, Salters-Pedneault, Erisman, Orsillo, & Mennin. 2009, 1st study; Neff, Kirkpatrick & Rude, 2007 2nd study; Neff 2003a, both studies). Reporting of multiple effect sizes reported from the same study violate the assumption of independence used in meta-analytic modeling. We took the following steps to address this issue. First, for samples reporting multiple dependent correlations we calculated an averaged within-sample effect size for each study, using Rosenthal and Rubin’s (1986) formula:

\[
\text{Combined}Z_r = \sum \frac{Z_r}{\sqrt{\rho m^2 + (1 - \rho)m}},
\]

where \(Z_r\) is the \(Z\) transformation of the measured correlation, \(\rho\) is the typical intercorrelation between the response measures, and \(m\) is the number of response measures contributing to the effect size. The primary meta-analysis was calculated using one effect size per study sample. Second, we conducted a series of sensitivity analyses by repeating the primary meta-analysis with the adjustment that studies with multiple comparisons only contributed one effect size derived from either the depression, anxiety or stress measure in each study. All other effect sizes were kept constant in each sensitivity analysis.

For studies reporting multiple independent samples within a paper, data were coded from each sample. One study (Gilbert et al., 2011) used two measures of compassion (SCS and CLS). As these measures were significantly but not highly correlated it was decided to retain SCS data only for the analyses.
Modeling followed Gentes & Ruscio’s (2011) protocol for correlational meta-analyses. To correct for standard error skew in correlational analyses effect sizes were transformed using Fisher’s Z transformation (Hedges & Olkine, 1985); defined as: \( ES_zr = .5 \log_r \left( \frac{1 + r}{1 - r} \right) \), where \( ES_zr \) is the Fisher’s Zr transformed correlation and \( r \) is the original effect size correlation. The Standard Error was calculated using the formula: \( SE_zr = \sqrt{\frac{1}{n - 3}} \). Effect sizes were weighted by their inverse variance using the formula: \( w_zr = n^{-3} \), where \( n \) is the number of participants contributing to the effect size. Effect sizes were converted back from Fisher’s z to r values for presentation in the results section, using the formula: \( r = \left( \frac{e^{2Zr} - 1}{e^{2Zr} + 1} \right) \), where \( e \) is the base of the natural logarithm and \( ESz \) is the Z transformed Effect Size. The standard error of the correlation was calculated using the formula: \( SE_zr = \left( 1 - r^2 \right) SE_zr \), where \( r \) is the reported correlation and \( ES_zr \) is the Z transformed effect size (Borenstein, Hedges and Rothstein, 2007).

Heterogeneity of effect sizes was assessed using the Q statistic. The Q statistic approximates a chi-square distribution, testing whether the distribution of effect sizes around the mean is significantly greater than expected from sampling error. In addition, the \( I^2 \) statistic \( (I^2 = 100\% \frac{Q - df}{Q}) \), where \( Q \) is the heterogeneity statistic and \( df \) the degrees of freedom) was calculated to provide an estimate of the total variance attributable to between-study variance; whereby 25%, 50% and 75% are considered to be cut-offs for low, medium and high heterogeneity (Higgins,
Thompson, Deeks & Altman, 2003). It is recommended that where between-study variance is anticipated to be substantial, random effects models (where between-study variance and within-study random sampling error are integrated into the model) are used (Borenstein, Hedges & Higgins, 2009). Given the heterogeneous sampling and measurement differences it was decided a-priori to use a random effects analysis.

2.5.4. Correction for attenuation

Corrected mean effect sizes were reported in order to correct for attenuation (measurement variation due to reliability differences in the measures used). This was accomplished using the following formula: \[ \rho = \frac{r}{\sqrt{(\alpha_{\text{symptom measure}})(\alpha_{\text{ecs})}}}, \] where \( \rho \) is the correlation corrected for attenuation, \( r \) is the reported correlational effect size and \( \alpha \) is the reliability coefficient for the measure (Lipsey & Wilson, 2001). Correspondingly, inverse variance weights were also calculated, using the formula: \[ w' = w\left((\alpha_{\text{symptom measure}})(\alpha_{\text{ecs}})\right), \] where \( w' \) is the corrected inverse variance weight, \( w \) is the uncorrected variance weight, and \( \alpha \)s are the respective reliability coefficients. Random effects analyses were then repeated using the corrected data.

2.5.5 Moderator Variables

We conducted a series of follow-up analyses on the data set to assess the impact of study characteristics as potential sources of heterogeneity. First we analyzed the effect of clinical status – either from a clinical or non-clinical population. This reflects findings suggesting clinical groups have difficulty in accessing compassion related thoughts and feelings (e.g. Gilbert, 2010; Pauley & McPherson, 2011). Second, we
conducted a further analysis for study population differentiating between student samples and non-student samples. Our rationale here was to assess the degree to which results from student samples, although useful for theory testing, may not generalize to the general population. Third, we chose to examine gender, expressed as percentage of sample that was female. This moderator took into account the higher reporting of mood and anxiety disorders in females (Kessler, McGonagle, Swartz, Blazer, & Nelson, 1993; Van de Velde, Bracke, Levecque & Meuleman, 2010). Finally, we included age as a moderator to assess possibility that levels of compassion have a differential effect according to age. Weighted ANOVAs were used as the post-hoc test for categorical moderator variables and weighted regression for continuous moderator variables. Both sets of post hoc test used random effects models estimated via the method of moments.

3. Results

3.1. Effect size reporting for association between compassion and psychopathology

Individual uncorrected effect sizes and standard error values for all studies included in the meta-analysis are displayed in Table 1. Summary statistics for the meta-analytic models are displayed in Table 2. The aggregate uncorrected random effects estimate for the relationship between compassion and psychopathology was $r = -0.54$ (95% CI = -0.57 to -0.51; $Z = -34.02$; $p < .0001$), meeting Cohen’s (1992) convention for a large effect size. The confidence interval excluded zero, indicating statistical significance for the observed association. This represents a large effect size in the direction of greater self-compassion associating with lower levels of symptomatology. When corrected for attenuation the aggregate effect size for the relationship between compassion and psychopathology remained of large magnitude, with $\rho = -0.61$ (95% CI = -0.64 to -0.57; $Z = -34.02$; $p < .0001$). The effect size
estimate for the total sample was significantly heterogeneous (Q = 185.56, p<.0001), with $I^2 = 89.6$ indicating 90% of the variance in effect size was attributable to between-study variance, suggesting substantial variability between the included studies.

3.2. Sensitivity analysis
Nine studies in the meta-analysis included multiple measures of symptoms (depression, anxiety and stress). Therefore a set of sensitivity analyses were conducted to evaluate whether different symptom measures impacted on the mean effect size for the total data set. Each study contributed one effect size to the analysis. As can be seen in Table 2, the mean effect sizes and 95% CI’s for these analyses gave comparable results to the main analysis.

3.3. Moderator analyses
As specified a-priori we investigated a set of categorical and continuous moderators. First, variability in effect sizes was not explained by clinical status (Q=1.66, df=1, p=.19), Second, variability in effect sizes was not explained population sampled (Q=2.79, df=1, p=.09). Results of the meta regression analyses indicate that gender (Q=2.42, df=1, p=.119) and age (Q=3.05, df=1, p=.08) were not significant predictors of effect size.

3.4. Publication bias
We also tested for publication bias in the total sample using Owin’s Fail Safe N procedure (1983). Following Naragon-Gainey’s (2010) methodology for correlational meta-analysis and adopting a criterion mean effect size of missing studies of $r=.10$ gives a FSN value of 128, indicating the number of studies reporting
negligible effect sizes correlations between compassion and symptoms required to reduce the observed effect size to zero magnitude. Adopting a highly conservative criterion mean effect size of missing studies of r=.60 gives a FSN value of 38 studies.

4. Discussion

The current meta-analysis sought to estimate the strength of relationship between compassion and common expressions of psychopathology, primarily depression, anxiety and stress. A systematic review of the literature identified that measurement of compassion was synonymous with measurement of self-compassion (Neff, 2003a, b). Consistent with our initial aim we were able to establish an aggregate effect sizes to derive a global estimate of the relationship between self compassion and mental health. Specifically, we observed a large effect size for the relationship between compassion psychopathology; demonstrating higher levels of compassion were associated with lower levels of mental health symptoms. The reported association provides empirical evidence for the relevance of theoretical models of compassion that emphasize the importance of self compassion for developing wellbeing, reducing depression and anxiety, and increasing resilience to stress (e.g. Gilbert, 2010; Hofmann et al, 2011; Feldman & Kuyken, 2011).

Although the meta-analysis provides robust evidence for the relationship between compassion and psychopathology, a notable number of studies in the data set reported associations for multiple symptoms. Our sensitivity analyses showed that replacing average estimates of effect size in these studies with a single effect for depression, anxiety or stress did not markedly alter the mean estimate and confidence intervals for the aggregate effect size. Given these studies violated assumptions of independence, via potential inter-correlation of symptom measures,
we did not analyse symptom as a potential moderator variable. However the similar aggregate effect size estimates suggests that the relationship between compassion and psychopathology is non-specific to a given set of symptoms.

The relationship between self-compassion and psychopathology was characterized by a high level of heterogeneity. However, follow-up analyses of clinical status, study population, age and gender did not identify significant moderators of the association between compassion and psychopathology. We therefore acknowledge that a proportion of between studies variance remains unaccounted for. However, the goal of the meta-analysis was to establish an evidence base for the validity of this relationship rather than identify all potential correlates of the relationship (Card, 2011). Therefore further research should seek to identify and explore additional possible moderators of the aggregate effect size.

Our analysis also highlights the need for further scrutiny of the measurement construct of compassion. In Neff’s model (2003a, b), a total score for self-compassion emerges from three overlapping components, arranged as positive-negative opposing pairs: self-kindness versus self-judgment, a sense of common humanity versus isolation, and mindfulness versus over-identification. Although the current meta-analysis identified associations between higher self-compassion and lower psychopathology, we cannot deconstruct this further to address whether this is due to high positive self-compassion, or instead low levels of self-judgment and self-isolation. Research suggests that inclusion of positive and negative components of compassion would be beneficial in identifying the ‘active’ components of self-compassion (van Dam et al., 2011; Gilbert et al, 2011).
We acknowledge a series of limitations with the identified studies and analysis, reflecting the relative infancy of evidence-based research in compassion. First, the ubiquity of the SCS (Neff 2003a) enables clear comparisons across data sets with a robust and reliable measure, but limits exploration of the data to self-compassion. As noted above most included studies reported the total scale score rather than specific subscales. Second, the literature is based wholly on self-reports. Neff et al., (2007) measured therapist ratings of compassionate responding, but in a non-standardized way, based on ‘intuitive judgment’. Therefore, it would benefit the development of a robust research literature if an interview-based rating scale could be developed. An analogous situation occurs in attachment research, where development of self-report measures (e.g. Experiences in Close Relationships Scale; Brennan, Clark & Shaver, 1998) and an interview based measure (Adult Attachment Interview, Main, Goldwyn & Hesse, 2002) has considerably enriched data on attachment in clinical samples (e.g. Steele & Steele, 2008; Shaver & Mikulincer, 2010).

Third, compassion has been measured with reference to specific goals, thus integrating consideration of motivation and intentionality to the measurement construct (Crocker and Canevallo, 2008; Crocker, Canevallo, Breines, & Flynn 2010). Compassionate goals have been shown to predict lower distress and greater interpersonal connectedness (Crocker et al., 2010). This approach to measuring compassion is also in keeping the Buddhist position of compassion as a motivational construct in itself (Hofmann et al, 2011).

Fourth, although gender was not a significant moderator in the meta-analysis we note that the gender distribution in the reported data is skewed, with female participants representing over 70% of the sample, thus limiting generalization to the general population. Only three papers identified in the review explicitly reported gender as a potential covariate, with men generally reporting higher self-compassion
scores than women (Neff 2003a; Neff, et al., 2005; Raes 2010), therefore the role of
gender as a covariate of compassion remains under-evaluated.

As a quantitative cross-sectional review, the current paper cannot inform
questions of causality between compassion and symptoms. Higher levels of
compassion may act as a buffer against mental health symptoms (Brown & Ryan,
2003), Equally, lower levels of psychopathology or distress may facilitate taking a
compassionate stance towards oneself. Longitudinal datasets on changes in
compassion over time, or indeed over treatment, are urgently required. Indeed, the
SCS facilitates the recruitment of relatively large samples, enabling the use of
mediational analyses and structural equation modeling for data analysis. In this vein,
Raque-Bogdan, Ericson, Jackson, Martin, & Bryan (2011) demonstrated a mediational
relationship between compassion, attachment and mental health, whereby self-
compassion mediated the relationship between attachment and mental health.

We note that we excluded several methodologically heterogeneous studies
that used compassion as process measure of change in psychological interventions
(Shapiro, Astin, Bishop & Cordova, 2005; Shapiro, Brown & Biegel, 2007; Shapiro,
Brown, Thoresen & Plante, 2011; Abercrombie, Zamora & Korn., 2007; Orzech,
Shapiro, Brown & McKay, 2009; Laithwaite et al., 2009; Schroevers & Brandsma,
2010; Lee & Bang, 2010; Rimes & Wingrove, 2011). The majority of treatment studies
reported significant change in self-compassion either over the course of treatment or
in treatment as against a control intervention. However, this does not resolve
whether changes in self-compassion were associated with symptomatic
improvement, or whether changes in self compassion merely represented associated
epiphenomena of treatment. Kuyken et al (2010) reported that self-compassion (and
mindfulness) mediated the effect of MBCT for depression, with increased self-
compassion during treatment significantly associated with lower depressive
symptoms at follow-up. This is consistent with Gilbert’s (2010) model that increased self-compassion acts as a protective psychological buffer against depressogenic stressors. That said, there is a degree of tautology inherent in the SCS as a measure of change in studies of mindfulness interventions, given it has a mindfulness subscale within it’s factor structure (see below for further discussion of the limitations of the SCS factor structure). Consequently, our review highlights the need for careful consideration of how self-compassion may act as a mechanism of change in therapeutic interventions, particularly those where mindfulness is an active component of the intervention.

In conclusion, this review provides the first survey of the literature on the relevance of compassion to psychopathology. It has established that the measurement of self-compassion, predominantly via the SCS (Neff, 2003a), provides robust, replicable findings liking increased self-compassion to lower levels of mental health symptoms. Conversely, lower levels of self-compassion were associated with higher levels of psychopathology. Future work will be invaluable in establishing the strength of these associations in clinical samples, in longitudinal studies, and in mechanism evaluation studies embedded in clinical trials of psychological therapies. This is particularly pertinent to therapies that promote acceptance and compassion as key aspects of the therapeutic process (Hayes, Strosahl and Wilson, 1999; Segal, Williams & Teasdale, 2002; Gilbert, 2005, 2010). There is also an important need to develop measures of compassion more closely aligned to specific variants of the self reported, intentional, attentional and behavioral components of compassion that have been proposed in the literature (Neff, 2003a,b; Gilbert, 2010; Goetz, Keltner & Simon-Thomas, 2010).
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<td>ZSDS</td>
<td>Students</td>
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<td>19.8 (N/R)</td>
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<td>Neff et al., 2008 Taiwan</td>
<td>164</td>
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<td>Students</td>
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<td>20.5 (N/R)</td>
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<td>-0.95</td>
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<tr>
<td>Gilbert et al., 2011</td>
<td>222</td>
<td>DASS</td>
<td>Students</td>
<td>Cross sectional</td>
<td>22.7 (7.07)</td>
<td>168/54</td>
<td>-0.27</td>
<td>-0.31</td>
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<td>Gilbert et al., 2011</td>
<td>59</td>
<td>DASS</td>
<td>Therapists</td>
<td>Cross sectional</td>
<td>39.52 (10.99)</td>
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<td>Students</td>
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<td>Students</td>
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<td>Ying, 2009</td>
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<td>CPID</td>
<td>Social work students</td>
<td>Cross sectional</td>
<td>28.12 (5.4)</td>
<td>58/7</td>
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<td>-0.34</td>
</tr>
<tr>
<td>Van Dam et al., 2011</td>
<td>504</td>
<td>BDI</td>
<td>Community Clinical Sample</td>
<td>Cross sectional</td>
<td>38.2 (11.1)</td>
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<td>DASS</td>
<td>Community Clinical Sample</td>
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<td>Students</td>
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<td>Students</td>
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<td>Measure</td>
<td>Group Type</td>
<td>Design</td>
<td>Mean (SD)</td>
<td>Effect Size</td>
<td>Notes</td>
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<td>Raes, 2010</td>
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<td>STAI-T</td>
<td>Students</td>
<td>Cross sectional</td>
<td>18.14 (1.25)</td>
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<td>Neff et al., 2007 Study i</td>
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<td>STAI-T</td>
<td>Students</td>
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<td>20.9 (1.5)</td>
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<td>Neff, et al., 2007 Study ii a)</td>
<td>40</td>
<td>STAI-T</td>
<td>Students</td>
<td>Cross sectional</td>
<td>21.05 (1.05)</td>
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<tr>
<td>Neff et al., 2005</td>
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<td>STAI-T</td>
<td>Students</td>
<td>Cross sectional</td>
<td>20.94 (2.03)</td>
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<td>Gilbert et al., 2011 a)</td>
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<td>Students</td>
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<td>168/54</td>
<td>-0.25</td>
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<td>Gilbert et al., 2011 a)</td>
<td>59</td>
<td>DASS</td>
<td>Therapists</td>
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<td>49/10</td>
<td>-0.09</td>
<td>0.13</td>
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<td>145/87</td>
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<td>Community Clinical sample</td>
<td>Cross sectional</td>
<td>38.2 (11.1)</td>
<td>396/108</td>
<td>-0.31</td>
<td>0.04</td>
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<td>Costa &amp; Pinto-Gouveia (2011) a)</td>
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<td>DASS</td>
<td>Community Clinical sample</td>
<td>Cross Sectional</td>
<td>60.81 (13.24)</td>
<td>82/21</td>
<td>-0.37</td>
<td>0.09</td>
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<td>Roemer et al., 2009; Study 1 a)</td>
<td>395</td>
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<td>Students</td>
<td>Cross Sectional</td>
<td>23.2 (N/R)</td>
<td>253/142</td>
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<td>0.04</td>
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<td>Roemer et al., 2009; Study 2 32 (16 in each group)</td>
<td>32</td>
<td>GAD-Q-IV</td>
<td>Clinical sample vs. non-clinical controls</td>
<td>Cross Sectional</td>
<td>GAD group: 32.75 (11.86)</td>
<td>22/10</td>
<td>-0.53</td>
<td>0.13</td>
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<td>Costa &amp; Pinto-Gouveia (2011) a)</td>
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<td>DASS</td>
<td>Community Clinical sample</td>
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<td>60.81 (13.24)</td>
<td>82/21</td>
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<td>Raque-Bogdan, et al., 2011</td>
<td>208</td>
<td>SF-12v2</td>
<td>Students</td>
<td>Cross sectional</td>
<td>20 (1.6)</td>
<td>153/44</td>
<td>-0.55</td>
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<td>Study</td>
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<td>Measure</td>
<td>Group</td>
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<td>Cases</td>
<td>Effect Size</td>
<td>SEr</td>
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<td>Gilbert et al., 2011 a)</td>
<td>222</td>
<td>DASS</td>
<td>Students</td>
<td>Cross sectional</td>
<td>22.7 (7.07)</td>
<td>168/54</td>
<td>-0.29</td>
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<td>Gilbert et al., 2011 a)</td>
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<td>DASS</td>
<td>Therapists</td>
<td>Cross sectional</td>
<td>39.52 (10.99)</td>
<td>49/10</td>
<td>-0.17</td>
<td>0.13</td>
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<tr>
<td>Costa &amp; Pinto-Gouivea (2011)</td>
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<td>DASS</td>
<td>Community Clinical Sample</td>
<td>Cross Sectional</td>
<td>60.81 (13.24)</td>
<td>82/21</td>
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<td>0.06</td>
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<tr>
<td>Birnie et al., 2010</td>
<td>51</td>
<td>SOSI</td>
<td>Community</td>
<td>Cohort treatment evaluation</td>
<td>47.4 (10.87)</td>
<td>35/16</td>
<td>-0.01</td>
<td>0.14</td>
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</tbody>
</table>

Notes: BDI = Beck Depression Inventory (Beck < Rush, Shaw & Emery, 1979); BDI-II = Beck Depression Inventory-II (Beck, Brown & Steer, 1996); ZSDS = Zung Self-Rating Depression Scale (Zung, 1965); DASS = Depression, Anxiety and Stress Scale (Lovibond & Lovibond, 1995); CPID = California Psychological Inventory for Depression (Jay & John, 2004); HRSD = Hamilton Rating Scale for Depression (Williams, 1988); STAI-T = State Trait Anxiety Inventory – Trait Version (Spielberger, 1983); BDI = Beck Anxiety Inventory (Beck & Steer, 1983); GAD = Generalized Anxiety Disorder; GAD-Q-IV = Generalized Anxiety Disorder Questionnaire-IV (Newman, Zuellig, Kachin, Constantino,Przeworski, Erickson, & Cashman-McGrath, 2002); SF-12v2 Mental Health Summary; Symptoms of Stress Inventory (Ware, Kosinski, & Keller, 1996); SOSI= Symptoms of Stress Inventory, Leckie & Thompson, 1979); RCT = Randomized Controlled Trial; N/R = Not Reported.. r = Uncorrected Effect size; SEr = Standard Error of r (uncorrected); a) denotes studies contributing multiple effect sizes to the overall model.
Table 2: Meta-analyses of association between compassion and psychopathology (random effects model, uncorrected and corrected for attenuation).

<table>
<thead>
<tr>
<th>Random Effects Model</th>
<th>k</th>
<th>N</th>
<th>Mean Effect Size r</th>
<th>95%CI</th>
<th>Z</th>
<th>ρ</th>
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<td>20</td>
<td>4007</td>
<td>-0.54</td>
<td>-0.57 to -0.51</td>
<td>-34.02***</td>
<td>-0.61</td>
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<tr>
<td>Sensitivity analyses</td>
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</tr>
<tr>
<td>All studies including depression effects b)</td>
<td>20</td>
<td>4007</td>
<td>-0.52</td>
<td>-0.55 to -0.49</td>
<td>-32.50***</td>
<td>-0.58</td>
</tr>
<tr>
<td>All studies including anxiety effects b)</td>
<td>20</td>
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<td>-0.51</td>
<td>-0.55 to -0.48</td>
<td>-32.36***</td>
<td>-0.58</td>
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<tr>
<td>All studies including stress effects b)</td>
<td>20</td>
<td>4007</td>
<td>-0.54</td>
<td>-0.57 to -0.51</td>
<td>-34.00***</td>
<td>-0.61</td>
</tr>
</tbody>
</table>

k=number of studies; n= total sample size; mean Effect size r= average uncorrected correlation; 95% CI = lower and upper limits of 95% confidence interval for uncorrected correlations; ρ= average correlation corrected for attenuation.; ***=p<.0001; a) Each study that contained multiple measures of symptoms contributed a single average effect size to the overall model; b) In these analyses only one effect size per study was used.