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Blame Recovery: Modeling the Effects Of Personality, Religious-spiritual Belief, and Gender On Blame Attributions and Psychological Wellbeing After A Failed Romantic Relationship

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Blame Recovery: Modeling The Effects Of Personality, Religious-spiritual Belief,

And Gender On Blame Attributions And Psychological Wellbeing

After A Failed Romantic Relationship

(Thesis Format: Monograph)

By

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ABSTRACT

Blame attribution (BA) is the complex cognitive-affective process through which individuals feel negative feelings of internalized self-blame/guilt or externalized other-blame by varying degrees. High BA is accompanied by distress, anxiety, depression and reduced health outcomes, while low BA indicates healthful release from negative affect and direction of attention toward past transgressions or negative events. Previous research has demonstrated a multitude of personality and individual difference associations with BA and psychological wellbeing (PWB) in cross-sectional samples, but little focus has been directed at determining if such traits affect changes in (i.e. recovery from) BA and PWB over time. The present study seeks to address this knowledge gap using a widespread blame context: romantic breakup. It was hypothesised that the personality traits of neuroticism (N) and extraversion (E), as well as the individual difference characteristics of trait emotional intelligence (EI), religious-spiritual belief (RSB), and gender would affect blame recovery and PWB change over time. A sample of 302 undergraduates completed measures of BA, PWB, E, EI, N, and RSB in two online sessions approximately 28 days apart. Hierarchical regression results indicate that only EI was influential, leading to increased PWB growth over time and greater reduction in self-blame/guilt. Modelled together, the traits did not explain BA or PWB change, despite various significant correlations with the three outcome variables at the single time point level.

Keywords: blame, personality, extraversion, neuroticism, emotional intelligence, religious-spiritual belief, gender, psychological wellbeing

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CHAPTER ONE: INTRODUCTION AND LITERATURE REVIEW

1. Introduction

A component of Heider's (1958) general *attribution theory*, blame attribution (BA; see Table 1 for a listing of all acronyms used in this paper) refers to the process by which individuals make assessments about causality in response to observed, affect-based negative outcomes perceived as failures (by the self or others, and including social transgressions; Harvey & Dasborough, 2006; Meier & Robinson, 2004; Fast & Tiedens, 2010). Individuals judge causality based on three main dimensions of attribution: locus of causality—what proportion of the cause is from internal/personal characteristics (internal blame attribution) and/or external/environmental factors (external blame attribution; eBA), stability (is the proposed cause stable over time or not), and control over outcome (Mclean, Strongman, & Neha, 2007). The absence of/inattention to both internal and external attributions signals a recovered state with null blame feelings experienced (Hart, Hanks, Bogner, Millis, & Esselman, 2007; Ferrari & Russell, 2001; Heider, 1958; Gudjonsson & Singh, 1989).

Research into how individuals assess blameworthiness has shown that characteristics of the failure itself are important (e.g. blame and blame-related guilt attributions generally increase with perceived failure severity, intent, and avoidability; Shell, Colvin, & Bruning, 1995; Gill & Andreychik, 2009; Lagnado & Channon, 2008; Bulman & Wortman, 1977; Malle & Knobe, 1997; Plaks, McNichols, & Fortune, 2009), but also that these failure characteristics are subordinate to individual differences in personality and cognitive style, which shape overarching processing and response patterns (Meier & Robinson, 2004; Plaks, McNichols, & Fortune, 2009; Fletcher,

Danilovics, Fernandez, Peterson, & Reeder, 1986). Likewise, a wealth of research has explored connections between personality and individual difference traits, feelings of blame, and recovery from other forms of hurt or injury (e.g. car or head injuries or bereavement; Yen & Siegler, 2003; Martin, Doster, Critelli, Purdum, Powers, Lambert, & Miranda, 2011; Stucke, 2003; Bolger, 1990; Fox & Leicht, 2005; Williams, 1990; Mahasneh, Al-Zoubi, & Batayeneh, 2013; Gulyn & Youssef, 2010; Mclean, Strongman, & Neha, 2007; Field, Diego, Pelaez, Deeds, & Delgado, 2009; Inbar, Pizarro, Gilovich, & Ariely, 2013; O'Connor, Berry, Inaba, Weiss, & Morrison, 1994; Hill et al., 2000). Yet, if and how personality and other individual difference traits influence *blame recovery* itself (that is, the rate of coping with feelings of internal and external blame such that they return to or approach the null/emotionally neutral state; Thompson et al., 2005) has yet to be considered. This is unfortunate, as blame recovery has very practical applications in the social tolerance and victim aid spheres, where blame is a major impediment to general health trajectories (Ferrari & Russell, 2001; Hart et al., 2007).

The present study seeks to begin exploration of individual differences in blame recovery by determining if the personality and individual difference traits of extraversion (E), neuroticism (N), trait emotional intelligence (EI), gender, and religious-spiritual belief (RSB; briefly defined here as the degree to which an individual intrinsically follows a transcendental, spiritual belief system—this concept will be discussed further in a later section of this chapter) significantly influence and predict rates of change in eBA, self-blame/guilt (G), and related psychological wellbeing (PWB) to indicate coping with blame.

1.1. Blame, recovery, and wellbeing

Attributions are central in the long-term management of affective and psychological reactions. This is especially so for blame attributions, as they are more personally meaningful and rooted in emotional response than simply cognition (Harvey & Dasborough, 2006). Blame is also crucial for motivating processing and handling of a complex or unnerving situation (Rosenthal & Schlesinger, 2002) when individuals seek explanations for painful, ambiguous, or negative events (Gulyn & Youssef, 2010). Self-blame in particular has been shown to be an important moderator for adjustment to trauma and stress for significant length of time (i.e. several years after the event; O'Neill & Kerig, 2000; Feiring & Cleland, 2007; Graham & Juvonen, 2002). Self-blame is even defined by some as a maladaptive affect-focused coping strategy that is positively related to physical and psychological distress, low self-esteem, depression, PTSD symptomologies, anxiety, and use of additional maladaptive strategies over time (Bussel & Naus, 2010; Najdowski & Ullman, 2009; Bulman & Wortman, 1977; Major & Hildebrandt, 1985; Harper, 2012; Mann & Cheng, 2012; Cacciatore, Frøen, & Killian, 2013; Branscombe et al., 2003; Ulman et al., 2007; Bolger, 1990). The creation of the initial distress level and the driving down of recovery rate is also credited to self-blame (such that PWB requires self-blame reduction; Koss & Figueredo, 2004; Najdowski & Ullman, 2009). Relinquishment of self-blame is thus considered a characteristic of general subjective psychological wellbeing (Feiring & Cleland, 2007; Harper, 2012).

Operationally, self-blame is often measured using one of its major component indicators: guilt (Wasserman, de Mamani, & Suro, 2012). Guilt is defined as the self-judgment of regret over an act or behaviour (Albertsen, O'Connor, & Berry, 2006;

O'Connor, Berry, Inaba, Weiss, & Morrison, 1994; Yang & Fan, 2008; Martinez-Pilkington, 2007) and is strongly related to internalization problems while also being completely unassociated with the blaming of others (Lutwak, Panish, & Ferrari, 2003). Guilt has been shown cross-culturally to impair health and PWB in the same fashion as other measurements of self-blame and internalized psychopathologies (e.g. depression, anxiety; Tilghman-Osborne et al., 2008; Langman & Chung, 2013; Inbar, Pizarro, Gilovich, & Ariely, 2013; Weinberg, 1994; Gulyn & Youssef, 2010; Bybee, Zigler, Berliner, & Merisca, 1996; Langman & Chung, 2013; O'Connor, Berry, Inaba, Weiss, & Morrison, 1994). However, some researchers suggest that guilt is more complicated: that it is more of a strong, positively associated proxy marker of self-blame (Tilghman-Osborne et al., 2008) that can bolster wellbeing via agency (Weinberg, 1995; Tangney et al. (1995) and seeking forgiveness (which is positively related to PWB and positive self-image; Van Dyke & Elias, 2008; Ysseldyk & Wohl, 2012). However, this claim has been met with mixed results (Gulyn & Youssef, 2010; Langman & Chung, 2013).

Comparatively, significantly less researcher attention has been given to understanding the relation between eBA and wellbeing. However, the few studies that have included eBA also show an inverse association with health and PWB (e.g. Harper, 2012; Tennen & Affleck, 1990; Cacciatore, Frøen, & Killian, 2013; Sholomskas, Steil, & Plummer, 1990; Glenn & Byers, E2009; Hart et al., 2007; Weinberg, 1994) such that depressive symptoms increase with externalizing blame level (Green, Moll, Deakin, Hulleman, & Zahn, 2012). Some findings do counter this trend in specific circumstances: specifically, individuals who feel superior beforehand do display a tendency to blame others (Gilbert & Miles, 2000). This disjoint could be explained by a hypothetical

tipping point, after which chronic eBA mingles with abnormal egocentrism. This would

be logical, as very strong eBA is often coincidental with self-oriented, low-guilt

psychopathologies such as narcissism and psychoticism (Batson, Gudjonsson, & Gray,

2010; Shine, 1997; Gudjonsson & Singh, 1989; Cima, Merckelbach, Butt, Kremer,

Knauer, & Schellbach-Matties, 2007; Stucke, 2003; Fox & Leicht, 2005; Dolan, 1995).

Moreover, individuals with high eBA tendency have been shown to be less forgetful of

failures and to have a reduced tolerance for possible negative evaluation by others

(greater concern with being judged; Hochreich, 1975). Thus, eBA can indicate increased

self-defensiveness and negative affect not conducive to wellbeing.

Hence, both self-blame/guilt and eBA appear to undermine general health.

Comparative examinations of internal and external blame effects on health and wellbeing

confirm this: regardless of perceived locus of causality, life satisfaction and recovery

from physical injury significantly increase over time when concern with blame (both self

and eBA) is low, or when blame overall is not attended to (Hart et al., 2007; Ferrari &

Russell, 2001). Therefore, blame recovery should be characterized in the present study

by reductions in both self-blame/guilt and eBA, and PWB level should grow with

recovery in eBA and self-blame/guilt.

1.2. Blame induction context: Dissolution of romantic relationships

A distinct boon of BA literature is the diversity of creative paradigms and

contexts drawn upon by researchers to examine blame, from simple perspective-taking

vignettes (Plaks, McNichols, & Fortune, 2009; Tennen & Affleck, 1990) to classroom

and workplace scenarios (such as blame for childhood obesity or work group project

failure; Iobst et al., 2009) to studies of prisoners and survivors of bereavement, abuse,

rape, and miscarriage (Weinberg, 1995; Shine, 1997; Stucke, 2003; Gudjonsson & Singh, 1989; Donovan, 2007; Koss & Figueredo, 2004; Rusinko, Bradley, & Miller, 2010; Filipas & Ullman, 2006; Babcock & DePrince, 2012). In terms of blame recovery, survivors of trauma are conceptually more likely to demonstrate trackable BA problems, given the intensely affective and severe nature of the event that caused feelings of blame and guilt (Feiring & Cleland, 2007). However, these contexts are pragmatically difficult to tap based on researcher access and ethical concerns.

With this in mind, the current study was designed to employ the dissolution of a romantic relationship (romantic breakup) as the context for blame and guilt induction. Romantic breakup is conceptually similar to bereavement when the relationship is considered intimate and committed by at least one partner and is also a commonly occurring experience within the general population (especially in young adults in postsecondary education; Gilbert & Sifers, 2011). Moreover, breakup is known to be a highly distressing and painful process undermining the PWB of both individuals involved (Fagundes, 2011; Eastwick, Finkel, Krishnamurti, & Loewenstein, 2008; Parkes, 1995). Specifically, breakup typically results in strong feelings of sadness, anger, blame (both self and other-oriented), guilt, insecurity, severe intrusive thoughts, and helplessness— sometimes to the point of suicide (Chung et al., 2002; Davis, Shaver, & Vernon, 2003; Kendler, Hettema, Butera, & Gardner, 2003; Field, Diego, Pelaez, Deeds, & Delgado, 2009).

When using breakup as a context, however, several critical components of the breakup must be controlled in order to properly track personality influences on blame recovery. These components include initial post-breakup distress (that leads to blame and

guilt duration of maladjustment), duration and/or intimacy/seriousness of a relationship, the amount of time since breakup, and whether a new partner has been found since (Chung et al., 2002; Field et al., 2009). Recovery is dramatically accelerated with the finding of a new partner, and when the previous (breakup) relationship is more casual in nature (Chung et al., 2002). As well, breakup distress naturally decreases with time, and measurement within the first two to three months following breakup has been suggested as best for observing high distress and recovery processes (Eastwick et al., 2008; Knox, Kusman, Kaluzny, & Cooper, 2000; Moller, Fouladi, McCarthy, & Hatch, 2003).

Additionally, evidence suggests that adjustment following breakup directly relates to locus of causality and who initiated the breakup (Field et al., 2009; Mclean, Strongman, & Neha, 2007; Collins & Clark, 1989). Individuals who did not initiate or agree to breakup are far less likely to externalize hurt by negatively evaluating their ex-partner, which increases the degree of depressive affect they experience over time (Fagundes, 2011).

1.3. Personality, blame, and wellbeing

1.3.1. Extraversion-introversion

Thus far, we have discussed how blame significantly relates to wellbeing and recovery in general and have proposed a context for blame recovery. However, researchers have stressed that personality and individual differences (e.g. cognitive style, Type D personality, trait resiliency, agreeableness) strongly influence how individuals respond to negative situations (Martin et al., 2011; Meier & Robinson, 2004; Martel et al., 2007; Strelan, 2007). For example, significant relations have been demonstrated between personality traits and blame levels, stress, general coping with stressful life

events, and coping strategy effectiveness (Connor-Smith & Flachsbart, 2007; Martin et al., 2011; Weizmann-Henelius, Sailas, Viemero, & Eronen, 2002; Miller, Handley, Markman, & Miller, 2010; Ferrari & Russell, 2001; Martel et al., 2007; Meier & Robinson, 2004; Watson, David, & Suls, 1999; Gulyn & Youssef, 2010; Zeidner & Saklofske, 1996). These personality-coping relations vary in consistency—based on the trait examined, regularity of coping measurement, stressors, and environment—and are typically stronger in younger samples (which are often more stressed; Connor-Smith & Flachsbart, 2007; Einstein & Lanning, 1998). Despite the variability, however, these relations provide a valuable precedent for inclusion of such traits as influential factors in BA changes (blame recovery) and subsequent wellbeing over time.

Evidence for extraversion-introversion, the first dimensional personality trait of interest to the present study, as a major explanatory variable in blame recovery is extensive. Under Gray's Biobehavioural Reinforcement Sensitivity Theory (a neurobiological extension of Eysenck's classic personality model), high E is characterized by low response inhibition, reduced stimuli sensitivity (including physical pain) and stress, and high activation with movement toward goals and defensive escape from punishment (Jackson, 2003; Aron & Aron, 1997). The low inhibition pattern is associated with positive emotions and positive emotion regulation over time, indicative of effective coping (Zeidner & Saklofske, 1996), while the more introverted high inhibition system pattern is related to negative affect and reduced self-forgiveness (Johnson, Kim, Giovannelli, & Cagle, 2010; Austin, Saklofske, & Egan, 2005; Williams, 1990; Fernandez-Berrocal & Extremera, 2006; Rusting, 1998). High E is also associated with higher general self-esteem and confidence/assertiveness, reduced reflection/rumination

and guilt, reduced strictness with the self, and greater interest in spontaneous/impulsive sensation-seeking and externalization of problems (Alujaa, García, & García, 2003; Aron & Aron, 1997; Chung et al., 2002; Edelmann & McCusker, 1986; Martel et al., 2007; Rusinko, Bradley, & Miller, 2010; Shine, 1997; Ysseldyk & Wohl, 2012)—all attributes that should contribute to reducing self-blame (predisposing high E individuals to greater self-forgiveness and possibly earlier wellbeing restoration and recovery of G feelings; Fernandez-Berrocal & Extremera, 2006). Narcissism, which has significant links to high E as well, further increases E proneness toward eBA (Stucke, 2003; Ong et al., 2011).

Conversely, introversion shows a reversed pattern of blame and wellbeing potential: introverted children have been shown to respond significantly worse to blame accusations, and perform cumulatively worse following repeated accusations relative to their more extraverted counterparts. In contrast, introverted children respond far better to praise, which cumulatively improves their future performance relative to their extraverted fellows (Thompson & Hunnicutt, 1944; Komazaki, 1956). Likewise, social inhibition—a classic characteristic of introversion—and introversion as a whole are associated with higher general affect sensitivity, stress response, breakup distress, negative affect, social embarrassability, shyness with the risk of internalization problems, and risk of suicidality/suicide ideation over time. These relations indicate possible impairment to self-blame recovery and wellbeing from trauma via sensitization to hurt, prolonged rumination, and negative affect tendencies (Marshall, Benjanyan, & Ferenezi, 2013; Martin et al. 2011; Aron & Aron, 1997; Brezo, Paris, & Turecki, 2006; Edelmann & McCusker, 1986; Yen & Siegler, 2003; Rowsell & Coplan, 2013). However, introversion

(lower E) via latent inhibition is also associated with reduced external judgmentalism (i.e. lower eBA; Gill & Andreychik, 2009), which should indicate reduced eBA.

Thus, E appears to promote a pattern of general forgiveness while minimizing self-blame/guilt and encouraging PWB, which should make it a promoting factor for fast recovery. Patterns of eBA recovery via E are less clear, however.

1.3.2. Neuroticism

Like its fellow core personality trait E, neuroticism (N) has shown considerable relations to blame and coping, though in the opposing manner. As with the introverted pole of E, N is a source of significant negative affectivity across time and situations (Martin et al., 2011; Rusting, 1998). Moreover, N is prone to focused ruminations on negative aspects of the self, others, and the world; negative mood regulation (Fernandez-Berrocal & Extremera, 2006; Rusting, 1998); positive mood suppression (Williams, 1990); and—like introversion—high sensitivity, embarassability, guilt, suicidality, and self-blame (Brezo, Paris, & Turecki, 2006; Aron & Aron, 1997; Edelmann & McCusker, 1986; Eysenck, Eysenck, & Barrett, 1985; Einstein & Lanning, 1998; McCann, 2010; Shine, 1997). Like E, N is also consistently linked to high eBA (Newberry & Shuker, 2011; Shine, 1997). N has even been found to be a valuable predictor of trauma symptoms in individuals within the present study's context of post-breakup affect (Chung et al., 2002; Marshall, Benjanyan, & Ferenezi, 2013). Thus, the patterns between mood and N indicated a general negative association with coping and the use of negative coping strategies (e.g. wishful thinking, emotion-focused coping, withdrawal/avoidance; Bolger, 1990; Connor-Smith & Flachsbart, 2007), which should result in reduced orientation toward problem-focused healing and PWB in the face of blame feelings.

1.4. Emotional intelligence, blame, and wellbeing

Moving beyond core personality, Salovey and Mayer's individual difference characteristic, emotional intelligence (EI) is likewise supported by growing evidence that it may be a strong positive influence on blame recovery. EI can be measured in two ways: as a stable trait that measures an individual's self-perceptions about their emotional intelligence or as a state-dependent ability measuring emotional intelligence behaviours (Petrides & Furnham, 2001; Keefer, Holden, & Parker, 2013). The present study will address and employ emotional intelligence in its trait form only, as this form is more closely linked to mental health outcomes (Schutte, Malouff, Thorsteinsson, Bhullar, & Rooke, 2007). The ability version is expected to share similar relations to blame and recovery, however.

Though definitions of EI vary across researchers, the underlying construct and measurement remains relatively unified, and comprises the following four components: emotion perception/awareness, emotion/mood regulation, emotion understanding, and emotion utilization to guide thoughts and actions (Ciarrochi, Chan, & Caputi, 2000; Austin, Saklofske, & Egan, 2005; Harvey & Dasborough, 2006; Fernandez-Berrocal & Extremera, 2006; Harvey & Dasborough, 2006). Consistently, EI has been shown to be positively related to heightened tolerance to stress, happiness and positive affect, optimism, life satisfaction, physical and mental health, and help-seeking behaviour when distressed (Austin, Saklofske, & Egan, 2005; Ciarrochi, Chan, & Caputi, 2000; Gupta & Kumar, 2010; Schutte et al., 2007; Tsaousis & Nikolaou, 2005). EI has also been negatively related to psychological distress, psychopathologies, loneliness, N, negative affect, mood reactivity to negative events, and depression (Austin, Saklofske, & Egan,

2005; Carvalho, Neto, & Mavroveli, 2010; Fernandez-Berrocal & Extremera, 2006; Salovey et al., 1995; Leweke, Leichsenring, Kruse, & Hermes, 2012; Lundh, Johnsson, Sundqvist, & Olsson, 2002; Picardi, Fagnani, Gigantesco, Toccaceli, Lega, & Stazi, 2011). Moreover, high EI results in a heightened tendency toward forgiveness and empathy (Lundh, Johnsson, Sundqvist, & Olsson, 2002; Fernandez-Berrocal & Extremera, 2006; Harvey & Dasborough, 2006), reduced long-lasting resentment and rumination (Carvalho, Neto, & Mavroveli, 2010; Einstein & Lanning, 1998; Edelmann & McCusker, 1986), and positive and proactive (nondestructive) coping strategies (Keefer, Holden, & Parker, 2013) that potentially drive recovery.

Together, these results indicate that EI possesses a protective function for mental wellbeing. However, some researchers caution EI's universal effectiveness: EI can sometimes result in N-like mood perturbation and prolonged empathic suffering if a negative event is particularly disturbing, rather than distressing (Fernandez-Berrocal & Extremera, 2006). Yet this issue is less concerning in the face of the finding that EI is positively linked to general self-efficacy—a critical requirement for initiating and maintaining general recovery processes (Najdowski & Ullman, 2009; Harvey & Dasborough, 2006; Gupta & Kumar, 2010), and augmentation of positive environmental factor effects on mood (Fernandez-Berrocal & Extremera, 2006) to help maximize healing factors. As a result, the perturbation factor of EI is likely an issue with very severe situations only. Therefore, as ameliorating harm/loss in recovery explicitly requires regulation of negative emotions and changing/management of the problem itself rather than negative coping (Mclean, Strongman, & Neha, 2007), EI's multiple regulatory, positive augmentation, and efficacy functions likely make it, in most

circumstances, a recovery promotion factor, bolstering PWB and reducing eBA and G (Bugay, Demir, & Delevi, 2012; Keefer, Holden, & Parker, 2013).

1.5. Religious-spiritual belief, blame, and wellbeing

Unlike E, N, and EI, research behind religious and spiritual beliefs' relation to blame and wellbeing has been both overlooked and riddled with contention and definitional challenges. The central terms involved have no clear or agreed upon definitions (Hodge, 2003; Hill et al., 2000), and many researchers unintentionally equate participation in organized religious activity with religious beliefs (Hodge, 2003; Behere, Da, Yadav, & Behere, 2013; Koenig, McCullough, & Larson, 2001), and subsequently proclaim religious belief and the modern concept of spirituality as wholly independent concepts (Estanek, 2006; Behere et al., 2013). In the absence of rational scientific consensus, we must turn to traditional dictionary definitions as a guide for inquiry. Thus, *religion* can be considered to be "the belief in and worship of a god or gods, or any system of belief and worship" (Religion, n.d. – *Cambridge English Dictionary*), which implies neither a mandate of institution nor an actual deity. Nature and human relationships are very capable of being worshipped, also, which blends into the realm modern spirituality—the next ambiguous, subjective term. Again by dictionary definition, *spirituality* is "the quality that involves deep feelings and beliefs of a religious nature, rather than the physical parts of life" (Spirituality, n.d.a - Cambridge English Dictionary) and "sensitivity or attachment to religious values" (Spirituality, n.d.b – *Encyclopedia Britannica*; Spirituality, n.d.c - *Merriam-Webster English Dictionary*). Interestingly, definitions of *religious* likewise reference back to spirituality as "relating to or manifesting faithful devotion to an acknowledged ultimate reality or deity" (Religious,

n.d.a – *Encyclopedia Britannica*; Religious, n.d.b - *Merriam-Webster English Dictionary*).

While debate about conceptual separations between these terms continue, it has been shown that the underlying, core principles and criteria of what is generally intended by *religious belief* and *spirituality* are identical (Friedman et al., 2010). These criteria include: the search for the sacred through self and others (and the feelings and behaviours arising from such a search), transcendence, use of religious belief as a guide in life, and belief in an encompassing power (Hill et al., 2000; Friedman et al., 2010). Additionally, most participant descriptions and interpretations of the terms "religious" and "spiritual" are often very similar (Pargament, 1997; Hill et al., 2000). Some spiritual-religious beliefs (e.g. destiny) have even been found to be constant across religious affiliation— including agnosticism and atheism (Mann, & Cheng, 2012; Norenzayan, & Lee, 2010; Albertsen, O'Connor, & Berry, 2006), negating the possibility of religious practice as the source of the belief.

This is not to say that religious belief and spirituality are entirely the same in all cases, but they are innately entwined such that religious belief frequently breeds spirituality, and vice versa to the point that the two act as a common, multi-dimensioned working construct. For example, separate religious belief measures and spirituality measures have even been shown to tap the same single underlying factor in factor analysis test development studies (e.g. Hodge, 2003), while others combine the two terms into one as in the present study (Leung et al., 2002).

It has been suggested that the major reason for the definitional splitting of the two concepts was the widespread cultural disillusionment with traditional churches in the

1960s and 1970s. At that time, "religion" and "religious" were considered outdated terms relating only to traditional churches, and were uncouth in the face of a growing secularist and scientific culture (Koenig, McCullough, & Larson, 2001; Hill et al., 2000). Thus, new social labels were necessary to disassociate people's beliefs from institutions, irreverent to the inter-dependent nature of the terms' definitions. Indeed, many scientists—including many psychologists who study religion—continue to admit dismissive or hostile attitudes toward religious beliefs, but claim they are spiritual (Hartog & Gow, 2005; Hill et al., 2000). Regardless of the fracture's cause, however, spirituality and religious belief will be used in the present study as a single, unified concept, religious-spiritual belief (RSB), to describe the common intrinsically-oriented core beliefs of the terms (participation in religious group activities, which is based on extrinsic, social perception motivations rather than intrinsic belief, is not represented in RSB; Behere, 2013; Van Deursen, Pope, Warner, 2012).

In the past decade, a rekindled interest in religious belief and spirituality research has begun producing interesting evidence of RSB's potential contribution to blame recovery. Traditionally, researchers often reported G and mental illness as byproducts of religion, implying belief resulted in reduced PWB (Albertsen, O'Connor, & Berry, 2006; Koenig & Larson, 2001). While some modern studies still report either mixed results (Maltby, 2005; Mann & Cheng, 2012) or the traditional finding that religion can augment initial distress, such as during major medical treatment or during states of mental instability (Bussel & Naus, 2010; Hill et al., 2000), the vast majority of studies (approximately 80%; Koenig & Larson, 2001) now show positive relations between intrinsic religious beliefs and positive mental status, health, self-efficacy (Hartog

& Gow, 2005; Maltby, 2005; Unruh, 2007). Moreover, theses studies indicated religious

belief coincides positively with positive mood and morale, resilience (Behere et al., 2013;

Faiver, O'Brien, & Ingersoll, 2000), endurance, and coping (Blanchard-Fields, Hertzog,

& Horhota, 2012; Van Dyke & Elias, 2008). Religious belief also appears to positively

influence attribution judgments specifically following romantic breakup (Blanchard-

Fields, Hertzog, & Horhota, 2012), and promotes empathy and forgiveness (Bugay &

Demir, & Delevi, 2012; Van Dyke & Elias, 2008), long-term post-traumatic growth

(Bussel & Naus, 2010), adaptive pain management (Unruh, 2007), and life satisfaction

(Koenig & Larson, 2001). Conversely, religious belief is negatively related to G

(Albertsen, O'Connor, & Berry, 2006; Martinez-Pilkington, 2007; Walinga, Corveleyn, &

van Saane, 2005), blaming of others (Van Deursen, Pope, Warner, 2012), rumination and

fixation on problems (Gervais & Norenzayan, 2012; Koenig & Larson, 2001), negative

coping strategies (e.g. drug and alcohol as stress relievers, avoidance, and emotion-based;

Hill et al., 2000; Krägeloh, Chai, Shepherd, & Billington, 2012; Roes & Ano, 2003),

depression and anxiety (Van Dyke & Elias, 2008), and affective disorders (Koenig &

Larson, 2001; Van Dyke & Elias, 2008).

  Similarly, spirituality research also indicates associations that imply pro-

recovery. Spirituality is positively related to forgiveness (Albertsen, O'Connor, & Berry,

2006), positive coping strategies (Unruh, 2007), and PWB (Langman & Chung, 2013),

and negative correlations with G, anxiety, and maladaptive coping strategy use (Mann &

Cheng, 2012). However, in some situations, spirituality does not always improve coping

and health: like N, it has positive relations to sensitivity (Albertsen, O'Connor, & Berry,

2006) and externalization of control that can undermine recovery by impairing self-

efficacy (Mann & Cheng, 2012; Norenzayan & Lee, 2010). Interestingly, extrinsic spiritual and religious affiliation (which indicates extrinsic practice only) has been found to correlate positively with guilt and blaming of others (Albertsen, O'Connor, & Berry, 2006; Van Deursen, Pope, Warner, 2012). However, institutionalized confession following a negative event does appear to improve forgiveness for self and others and reduces G (Martinez-Pilkington, 2007). While institutionalized participation is not a component of RSB (it does not indicates intrinsic belief), this finding provides a valuable understanding of religious behaviour's impact on recovery when internal belief is not necessarily present. Thus, intrinsic religious belief and spirituality (RSB), but not necessarily extrinsic religious behaviour, encourage and provide meaning, hope, and a sense of forgiveness, as well as reduced anxiety and trauma levels (Koenig & Larson, 2001; Van Dyke & Elias, 2008), which should positively influence recovery processes for BA and PWB.

1.6. Gender, blame, and wellbeing

Gender has been a defining factor in differential responding and wellbeing in psychological theory. Gender's impact on health outcomes has been a topic of concern from Gilligan's classic study of morality, which found that men are conditioned to think about morality and attributions in terms of absolute justice, rights, and rules while women are socialized to morality and ethics based on caring for individuals and concern for wellbeing (Grazzani Gavazzi, Ornaghi, & Antoniotti, 2011), to more recent investigations indicating women's higher sensitivity (Aron & Aron, 1997) and reduced tendency to forgive (Boon & Sulsky, 1997). Other findings on this issue show that women also possess higher levels of N and E (Brezo, Paris, & Turecki, 2006), which—as we have

seen—drive up self-blame and external blame, respectively, and impair recovery processes. Moreover, females show higher and prolonged distress (Field et al., 2009), more internalizing psychopathologies for failure (e.g. depression, anxiety), higher rates of suicide ideation, and increased self-blame following a negative event (Brezo, Paris, & Turecki, 2006; Chung et al., 2002; O'Connor et al., 1994; Madden, 1988). Conversely, men display externalized pathologies, such as anger and aggression, increasing the likelihood of eBA (Brezo, Paris, & Turecki, 2006; Fernandez-Berrocal & Extremera, 2006). Men are also less willing to administer self-punishment when they do feel G (Inbar et al., 2013) and feel more able to problem-solve and handle stress relative to women (Keefer, Holden, & Parker, 2013), indicating that men have a lower sensitivity to G and a greater sense of self-efficacy that can aid coping. However, it is important to note that the issue of gender and wellbeing is complicated by environment expectation: women must usually cope with higher levels of distress and negative affect to begin with, as they frequently suffer from additional outside blame being placed upon them for negative events—a problem that men usually do not experience (O'Connor et al., 1994). This means that women's recovery is further handicapped relative to men, so the initial level of negative affect post-breakup must be controlled before recovery rate distinctions can be evaluated.

Thus, men and women differ significantly in their response to negative events—including distress from romantic breakup (Chung et al., 2002). It appears that men display a pattern of reduced self-blame with higher eBA, but also a higher degree of perceived agency in coping. Relative to women, who have large degree of impediments

to self-blame reduction and coping, men likely have an easier time handling negative

affect after a blame event.

1.7. Rationale and hypotheses

        To date, research has identified patterns of coping and overall blame levels

associated with averse life events based on personality, belief, and gender, but how such

variables influence the rates of change from recovery in those negative feelings of blame

specifically has yet to be investigated. The present study will extend the knowledge of

blame beyond overall post-trauma correlations *with* a trait and into how those traits

impact the changing of blame itself over time. Specifically, this study seeks to answer 1)

how do the personality and individual difference characteristics of E, N, EI, and RSB

affect the rate of blame recovery and PWB growth (following an acute blame-induction

event: romantic breakup) individually and together? and 2) does gender influence

recovery rates such that women's blame feelings reduce, and their PWB grows, more

slowly over time relative to men?

        Five hypotheses were proposed: A) As high PWB is a state of health and

positive affect, it was expected that blame feelings such as eBA and G (as measured by

the RB-BAI) would negatively relate to PWB, and that changes in eBA and G over time

would similarly negatively relate to changes in PWB, signaling a recovery trend via

growth of PWB and reduction of blame feelings; B) Gender would predict recovery such

that being female would decrease G and eBA reduction rates and decrease PWB growth

rate relative to being male, after controlling for relationship closeness, time since

breakup, breakup initiator, whether a new relationship had been entered since the breakup

and during study, initial breakup distress and blame and PWB levels, and time between

study measurement sessions; C) E, EI, and RSB would predict recovery such that they increase the rates of self-blame/G and eBA reduction, and also the rate of PWB growth, over time post-breakup (i.e. E, EI, and RSB would be negative predictors of G and eBA reduction over time, and positive predictors of PWB growth), controlling for the same variables listed in B; D) N would predict recovery such that it decreases the rates of G and eBA recovery and decreases the rate of PWB growth (i.e. N would be a positive predictor of G and eBA reduction, and a negative predictor of PWB growth), after controlling for the same variables as in B and C; E) That E, EI, N, RSB, and gender would provide a meaningful model for predicting blame recovery.

CHAPTER TWO: METHOD

2. Method

2.1. Participants

A sample of 378 undergraduate students (256 females, 119 males, 34 unspecified) ranging from 17 to 43 years of age was recruited for this study. Recruitment occurred via two methods: 1) online recruitment via the Department of Psychology's subject pool and 2) on-campus advertising via posters and instructor-authorized in-class presentations. Participants were compensated, depending on how they were recruited, with either one research credit per session towards completion of an introductory psychology course (the subject pool participants) or entry into a draw to win monetary prizes (all other participants). Prior to analyses, 59 cases were excluded (see Table 2) due to nonparticipation (consent and visiting of the study without completion of any measures; n = 44) and inattention to study (taking longer than 2 h to complete one or both sessions of this two-part study; n = 15). An additional 17 were excluded as outliers following Tukey's modified outlier labeling rule (using the recommended stable g factor value of 2.20; the original value of 1.50 is known to provide incorrect upper and lower boundaries for outlier cutoff up to half the time; Hoaglin, Iglewicz, Tukey, 1986). Therefore, final sample size was N = 302 (approximately 202 females, 100 males[1]), also aged 17-43 years (*M* = 19.31, *SD* = 2.43), who were primarily from the social science, science, and health science faculties (see Table 3).

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[1] Approximations for descriptive purposes; actual post-MI pooled estimates are 202.01 females, 99.99 males

2.2. Measures

A short relationship demographic survey was employed to gather information on important characteristics of the romantic breakup in question. The survey was comprised of both Likert scale items, such as "How serious was this relationship?" and "How important do you feel having a romantic partner is?" as well as categorical items such as "How long ago did the relationship end? 0-4 weeks/4-8 weeks/8-12 weeks" and "Have you been in a romantic relationship since the breakup in question?" See Appendix A for the full survey.

2.2.1. Blame (eBA and G)

External blame attribution and guilt were measured using the *Romantic Breakup Blame Attribution Inventory* (RB-BAI). The 14-item RB-BAI is an adaptation by the author of the *Revised Gudjonsson Blame Attribution Inventory* (GBAI-R; Gudjonsson & Singh, 1989) targeted at non-criminal populations using minimal item language changes. The original GBAI-R assesses three state (time-dependent) domains of blame attribution: eBA, mental element (that is, mental instability—a special, psychopathology-driven variant of eBA that was not found to be stable following adaptation to non-criminal populations; Tohver, Smith, & Saklofske, 2013), and G. Test-retest reliabilities between eBA and G in the GBAI-R are reported as .60 to .62 (Cima et al., 2007), indicating both subscales are amenable to measuring state attribution changes over time. In both the GBAI-R and RB-BAI, the eBA subscale (8 items) measures attribution to external factors as defined in Heider's (1958) attribution theory, and includes statements such as "I should not blame myself [for the act/for the relationship ending]" and "The relationship

ending was beyond my control." Conversely, G attribution (6 items in the RB-BAI) is a measure of remorse and self-blame for the act in question, and includes assertions such as, "I can't forgive myself for the relationship ending" and "I think constantly about the breakup." Items are rated on a 1 (*Strongly Disagree*) to 5 (*Strongly Agree*) Likert scale. Scores on the G subscale range from 6 to 30 and scores on the eBA subscale range from 8-40, with higher values indicating greater respective eBA and G. Before summing each subscale of the RB-BAI, items 2 ("I do not deserve to be blame for the relationship ending."), 3 ("The relationship ending was beyond my control", and 7 ("I should not blame myself for the relationship ending.") are reverse coded.

As the GBAI-R's mental element (ME) subscale was intended to explain special externalizing attributions based on psychopathologies which are high in criminals but not nearly as large of a factor in the general population (Raine, 1993), the ME subscale was removed during adaptation. G and eBA subscales were also trimmed during scale adaptation and development (from 18 items for G in the GBAI-R and 15 for eBA) based on the presence of an unexpected fourth factor that could not be consistently explained in a sample of 374 students (Tohver & Saklofske, 2013a). Preliminary findings support acceptable to good reliability of both the eBA (α = .79) and G (α = .87) subscales with all CFA factor loadings >.60 (Tohver & Saklofske, 2013b).

To determine whether blame patterns observed are likely specific to romantic-breakup context (and to the RB-BAI measure), an additional non-contextual blame measure—the 32-item *Characterological-Behavioural Self-blame Scale* (CBS; Janoff-Bulman, 1979; a seminal measure of self-blame for a negative event) was used to determine RB-BAI subscales' validity. This scale asks about blame and negative affect

directed at the self relative to the environment, other people, and chance. Respondents

answer questions about how much they feel they would blame themselves, others, etc. if

placed in four different hypothetical situations (romantic breakup being one of them).

Examples of questions include "How much do you blame yourself for the kind of person

you are?" and "How much do you think you deserve what happened?" Items are

answered on scale of 1 (*Not at all*) to 6 (*Completely*). After reverse coding for

externalized blame items within each scenario, scores across the four scenarios are

summed together. Final scores range between 36 and 216, with higher scores indicating

higher self-blame. Previous testing of this measure within the context of romantic

breakup using a sample of 265 university students found the CBS had good internal

consistency (α = .85; Tohver & Saklofske, 2013b).

To determine the effectiveness of the RB-BAI to measure changes from blame

recovery, general forgiveness was collected using the 18-item Heartland Forgiveness

Scale (HFS; Thompson & Synder, 2003). HFS and change scores in the RB-BAI

subscales should be negatively related if the RB-BAI captures blame recovery via

forgiveness's neutralizing influence. Items include statements such as "I hold grudges

against myself for negative things I've done." and "I continue to be hard on other who

have hurt me" that are rated on a Likert scale from 1 (*Almost always false of me*) to 7

(*Almost always true of me*). Psychometric evaluations of this measure indicate it

possesses adequate to good internal and test-retest reliability ranging from α = .80 to .87

(Thompson & Synder, 2003; Bugay, Demir, & Delevi, 2012; Tohver & Saklofske, 2013).

2.2.2. Psychological Wellbeing (PWB)

Subjective PWB was measured using the Flourishing Scale (FS; Diener et al., 2010), an 8-item instrument containing statements such as "I am competent and capable in the activities that are important to me" and "I am a good person and live a good life". The scale taps a variety of important PWB facets, including self-esteem, purpose in life, social relationship quality, perceived competency, and optimism (Silva & Caetano, 2013) to measure general wellbeing. Respondents indicate to what degree they agree with each statement by writing a number along the scale of 1 (*Strongly Disagree*) to 7 (*Strongly Agree*) beside each item. Scores are summed, and can range from 8 to 56, with higher values indicating greater levels of subjective PWB. All items are positively phrased.

The FS has demonstrated strong single-factor structure (Silva & Caetano, 2013; factor loadings between .60 and .76) with acceptable to good reliability ($\alpha = .73$ to .87; Diener et al., 2010; Silva & Caetano, 2013) and a decent temporal stability of .71 (Diener et al., 2010) for measuring state PWB. The scale also positively associates to other measures of PWB ($r = .43$ to $r = .73$; Diener et al., 2010; Silva & Caetano, 2013).

2.2.3. Personality (E and N)

The short form of the *Revised Eysenck Personality Questionnaire* (EPQ-R-SF; Eysenck, Eysenck, & Barrett, 1985) was used to measure E and N. The EPQ-R-SF is a widely-known and frequently-used 48-item measure with four subscales: E, N, Psychoticism (P; hard-mindedness), and Lying (social desirability). Respondents agree or disagree with item statements such as "Are your feelings easily hurt?" and "Do you like plenty of bustle and excitement around you?" by circling either YES (1) or NO (0). For the present study, only the E and N subscales were used (12 items per subscale), as both P and Lying frequently have low internal consistency ($\alpha = .44$ to .77 for L and $\alpha = $

.43 to .68 for P; Eysenck, Eysenck, & Barrett, 1985; Tohver & Saklofske, 2013;

Alexopoulos & Kalaitzidis, 2004). Comparatively, E and N possess acceptable to good

consistency (α = .78 to .88 and α = .71 to .87, respectively; Eysenck, Eysenck, & Barrett,

1985; Tohver & Saklofske, 2013; Alexopoulos & Kalaitzidis, 2004). Test-retest

reliabilities have been discussed rarely in the literature, but have been reported as .69 for

E and .84 for N by some researchers (Alexopoulos & Kalaitzidis, 2004). Concurrent

validity with other E and N scales have found positive results in a multitude of studies,

ranging from .59 to .95 for E and .62 to .95 for N (Alexopoulos & Kalaitzidis, 2004).

Items 14 and 20 are reverse coded, then E and N subscale items are respectively summed

to generate overall E and N scores, which range from 0 to 12.

2.2.4. Emotional Intelligence (EI)

Petrides and Furnham's (2006) 30-item *Trait Emotional Intelligence*

Questionnaire - Short Form (TEIQue-SF) was used to measure global trait EI via 15

main facets of the EI construct (the scale does not provide scores on the individual's

facets, however). Items such as "I usually find it difficult to regulate my emotions" and

"I'm usually able to influence the way other people feel" are scored on a Likert scale

from 1 (*Completely disagree*) to 7 (*Completely agree*). After reverse coding items 2, 4, 5,

7, 8, 10, 12, 13, 14, 16, 18, 22, 25, 26, and 28, items are summed into a final score, with

higher sum scores indicating higher global EI. Analyses of TEIQue-SF psychometrics

indicate the scale has a good to excellent internal consistency (α = .81 to .89; Cooper &

Petrides, 2010; Tohver & Saklofske, 2013; Deniz, Özer, & Isik, 2013).

2.2.5. Religious-spiritual Belief (RSB)

To measure RSB, Hodge's (2003) adaptation of Allport and Ross' (1967) *Intrinsic Religion Scale*—the *Intrinsic Spirituality Scale* (ISS)—was used. The 6-item Likert measure taps the primary construct used in the major traditional scale of intrinsic religion (that is, religious belief; Hodge, 2003) but was also designed to include modern spirituality. After the adaptation, both religious belief and spirituality mapped as a single strong construct on this scale via CFA (Hodge, 2003), making it ideal to measure the combination of the two in RSB. Items such as "In terms of the questions I have about life, my spirituality answers" and "Growing spirituality is…" are answered on a scale of 1 (No importance; e.g. *No questions/Not at all* important) to 11 (High importance; e.g. *Most of my questions/More important than anything else in my life*." Items are summed for a total score between 6 and 66). With a mean item validity coefficient of 1.74 times the error, the ISS is a reasonably valid measure of RSB. Likewise, mean reliability and internal consistency values are good to excellent (α = .80 and α = .96, respectively; (Hodge, 2003). These high values, as well as high average inter-item correlation (r = .65, split-half reliabilities (α = .91), and > .70 standardized loadings on the proposed single factor structure have been more recently replicated (Gough, Wilks, & Prattini, 2010).

2.3. Procedure

Following recruitment, participants were directed via URL to Part 1 of the online study. During session 1, participants were given the option to provide an email address if they wished so that Part 2 of the study could be forwarded to them four weeks after Part 1. After 28 days, participants were electronically sent Part 2, where they were asked to complete all measures a second time, with the exception of the romantic breakup demographics survey (which was not presented again). However, participants were asked

at the start of Part 2 if they had entered a new romantic relationship during the time since

Part 1 of the study. Following completion of each session of the study, participants were

debriefed. Approval to perform this study was granted by the University of Western

Ontario's Research Ethic's Board.

CHAPTER THREE: RESULTS

3. Results

3.1. Data analytic strategy

Prior to screening and analyses, 13.34% of data points were missing (see Figure 1). Listwise deletion was employed for preliminary screening while hypotheses were conducted on data after monotone regression-based multiple imputation (MI; Enders, 2010; IBM, 2013; Kline, 2011; Baraldi & Enders, 2010; Schafer, 1999). Traditionally, researchers have considered 5-10 imputations to be sufficient for moderate missing data, but modern examinations now recommend a variety of minimum cutoffs for the number of imputations, from 1 imputation per percent of missing data (von Hippel, 2009) to a minimum of 20 imputations at all times (Little, 2013). Following Little's recommendation, 20 imputations were computed in the present study to counteract solution and variance instability resulting from the moderate percentage of missing data (von Hippel, 2009; Kenward & Carpenter, 2007). All procedures were conducted in SPSS v. 22.

Pre-MI estimates of univariate kurtosis (KU) and univariate skew (SU) were calculated to determine univariate normality in the dataset. It is suggested that these values should not have an absolute value greater than 1.00 or 2.00 (depending on the desired stringency a priori) for normality to be implied (Kline, 2011). The current study set the cutoff at |2.00|. Age and the number of days between when participants completed Part 1 and started Part 2 of the study (Interval) were found to be positively skewed and kurtotic (see Table 3). As well, the ISS scale and how serious the relationship had been (HS) as measured during Part 1 were found to be negatively skewed and positively

kurtotic. While transformations such as Box-Cox (Box & Cox, 1964), Tukey's gh (He & Raghunathan, 2006), or general power transforms are often cited as mandatory for all parametric tests (including MI), they are generally ill-advised unless absolutely necessary (Kline, 2011; Osborne & Waters, 2002). It has recently been demonstrated that transformations create more error against the parametric imputation model in MI than merely entering the skewed or kurtotic variables (i.e. transformed data produce greater disruption and eroding of genuine relationships between variables and create more severe and frequent outliers during imputation than untransformed data; Graham, 2009). Additionally, after MI, normality is not required for any predictor or covariate; MR's normality assumption is particular to criterion variables and their residuals only (Berry & Feldman, 1985). Therefore, skew and kurtosis is not a significant concern in the dataset.

Multicollinearity was evaluated before MI by first checking univariate relations such that no zero-order correlation between variables of interest should be greater than |.90|, followed by evaluation of multivariate relations by computing variables' variance inflation factor (i.e., VIF). A variable with a VIF > 10 is considered redundant against other predictive variables within a model and should be removed (Kline, 2011).

As recommended by Bodner (2008), dummy code variables representing categorical control variables for subsequent hierarchical multiple regression (hMR) analyses were created prior to MI to prevent biasing in estimation that would impede hMR interpretation. The categorical variables that were dummy coded were: time since the breakup (3 levels: 0-4 weeks/4-8 weeks/8-12 weeks), initiator of the breakup (4 levels: Me/My ex-partner/Both/Other), entry into a new relationship since the breakup (asked during Part 1 of the study; 3 levels: Yes/No/Uncertain), and entry into a new

relationship since Part 1 of this study (asked during Part 2 of the study; 3 levels: Yes/No/Uncertain). The resulting dummy variables were: 0-4 weeks since breakup vs. not (HLA1), 4-8 weeks since breakup vs. not (HLA2), participant initiated breakup vs. not (WI1), participant's ex-partner initiated breakup vs. not (WI2), both initiated breakup vs. not (WI3), new relationship since breakup vs. not (RelB1), no new relationship since breakup vs. not (RelB2), new relationship since Part 1 (RelP1), and no new relationship since Part 1 (RelP2)

Following MI, relations between PWB, eBA, and G were examined to determine a general pattern of recovery. A series of 2-step hMR analyses were then conducted to test the individual difference factor hypotheses of whether each of the 5 predictors independently explain change over time in PWB (ΔPWB), G (ΔG), and eBA (ΔeBA) after controlling for breakup demographic variables, initial distress level, initial BA and PWB levels, and time between the two study sessions. Time 1 values for the 5 individual difference characteristics were used in the models, as EI, E, N, G, and RSB are conceptualized to be stable traits under the current measurement tools (Time 2 values were reserved for checking measures' test-retest reliability). Last, the individual difference traits were combined within a single model to determine the relative impact between them, if any, in explaining changes in ΔPWB), ΔG, and ΔeBA. This stepped analysis allows greater control over understanding the nature of the coefficient biasing that occurs with each added predictor variable in the MR equation, which would not be possible with simple simultaneous entry of all five factors without considering each one in isolation first (Osborne & Waters, 2002).

Pooling of *p* values for omnibus tests (*F* and R significances) was conducted using Ruschendorf's (1982) elegant scaled averaging method, which has been recently corroborated by Vovk (2012). While there are several families of methods for pooling probabilities, (see Won, Morris, & Elston, 2009; Kost & McDermott, 2002; Wardrop, 2011; Birnbaum, 1954), these methods are predominantly limited by their ability to only approximate pooled values while forcing assumptions about component *p*'s independence. However, Ruschendorf proved that averaging a vector of *p* values and then scaling the average by a factor of 2 always returns an optimal and bona fide probability value (that can legitimately be greater than 1; when this occurs, rounding down to 1 is the general rule for presentation, though it not necessary; see Theorem 1 of Ruschendorf, 1982, or Vovka, 2012 for a simpler explanation) without requiring any assumption other than that the vector of values averaged are values between absolute 0 and 1. The downside to this method is that it is conservative, erring on the side of high pooled *p* with a somewhat higher chance of Type II error. However, this conservatism is shared among most of the popular pooling methods (Won, Morris, Elston, 2009).

Though there is no agreed upon rule for required sample size in MR—guidelines vary from small equations (50 + 8 × number of predictors; Tabachnick & Fidell, 2007) to ratios (e.g. 15 to 40 subjects per predictor; Cohen & Cohen, 1983) to minimums of 400 (see Pedhazur, 1997), the present study sought a 15:1 minimum ratio. Preliminary screening retained a high enough sample size for a 16.78:1 ratio for the most demanding models used.

Change (Δ) scores for PWB, G, and eBA were computed as Time 2 score – Time 1 score to track recovery, meaning that smaller ΔG and ΔeBA values represent greater

recovery between time points. Conversely, larger ΔPWB values represent greater

recovery rate relative to small ΔPWB values. Modeling of continuous variables in MR

was performed using standardized versions of each variable. It should be noted that the

standardized difference scoring is only one of several valid strategies for examining

scores across two time points; various alternative methods, such as residual score

differences or separate time point analysis, are also justifiable options.

3.2. Preliminary Analysis

      Subject pool participants comprised the large majority of the sample (79%). A

detailed summary of demographical information of the sample is provided in Table 3.

 Generally, participants felt that having a romantic partner was somewhat important (*M* =

5.32, *SD* = 1.11; see Appendix A for the demographic survey), and had been in an

average of 1.84 serious romantic relationships in the past (*SD* = 1.05). Participants

reported their previous relationship had, on average, been quite serious and intimate (*M* =

5.48, *SD* = 1.17). Most individuals (49.93%) had broken up from their last serious

romantic relationship 8-12 weeks prior to beginning the study; 23.96% had broken up

less than 4 weeks prior. Over 80% of individuals had not entered a new relationship

since the breakup in question at the start of the study (though 6.94% of participants were

unsure about their current relationship status at study onset); approximately 64.37% of

participants had remained single for the duration of the study. Also, there was a

relatively close ratio of individuals who initiated the breakup themselves and those who

had mutually agreed to a breakup with their ex-partner (approximately 34.37% and

31.80% of the sample, respectively). Approximately 25% of participants reported that

their partner ended the relationship.   See Table 3 for a full proportional summary of demographics.

Means, standard deviations, alpha reliabilities, and SU and KU statistics for all continuous variables at both time points are presented in Table 4, and indicated no univariate multicollinearity and no skew or kurtosis of concern for MR analysis (i.e. no variables intended to be used as criterion variables had skew or kurtosis over the allowed cutoff). Overall, internal consistencies were good to excellent (.80-.95) for all scales except N and eBA ($\alpha = .78$ and $\alpha = .79$ at Time 1, respectively; $\alpha = .70$ and $\alpha = .77$ at Time 2, respectively). Correlations between all variables (including categorical dummy contrast variables to be entered in MR analyses) are present on Table 5 and indicate low risk of multicollinearity. Test-retest reliabilities of all scales were between .32 and .70 (see Table 6).

3.3. The RB-BAI versus non-contextual baselines

Contextual and forgiveness baseline testing of the RB-BAI showed mixed results. As expected, both the eBA and self-blame G subscales were positively related to one another at both Time 1 ($r(300) = .56$ $p < .001$) and Time 2 ($r(300) = .56, p < .001$), and were also positively related to the CSB at Time 1 ($r(300) = .22$, $p < .001$ and $r(300) = .32$, $p < .001$, respectively) and Time 2 ($r(300) = .24$, $p < .001$ and $r(300) = .32$, $p < .001$, respectively). However, the relations are small to moderate (Cohen, 1992), indicating some constructual divergence between the two tests. Interestingly, both subscales were not significantly different in the strength of their relation to CBS at either Time 1 or Time 2 (Steiger's $z = -2.18$, $p = 0.98$ and Steiger's $z = -1.57$, $p = 0.94$, respectively), despite CBS's design being intended to target self-blame in particular

(meaning the relation to G should be greater; Janoff-Bullman, 1979). As well, both RB-BAI scales showed the expected significant negative relations to the HFS at Time 1 (eBA: $r(300) = -.11$, $p = .048$, G: $r(301) = -.34$, $p < .001$) and Time 2 (eBA: $r(301) = -.21$, $p < .001$, G: $r(301) = -.41$, $p < .001$). G and eBA again displayed no significant difference in the strength of their relation to HFS (Time 1: $z = -4.73$, $p = 1$; Time 2: $z = -3.772$, $p = 1$). Therefore, the RB-BAI is related to the non-contextual baseline and forgiveness as intended, with high eBA and G coinciding with increased CBS blame scores, and with low eBA and G scores coinciding with increasing forgiveness and theoretical recovery. However, the small relation of the RB-BAI subscales to the CBS indicates that the RB-BAI blame concept is indeed specific and somewhat divergent. Therefore, results from the RB-BAI and interpretations of blame recovery using it may not hold outside the romantic breakup context.

3.4. Hypothesis A: eBA, G, and PWB association patterns

The hypothesis that wellbeing and its growth, indicative of recovery, would be negatively related to both eBA and G and their respective reduction rates was supported: moderately strong negative associations between PWB and eBA ($r(300) = -.27$, $p < .001$), PWB and G ($r(300) = -.34$, $p < .001$), and PWB and non-contextual CBS blame ($r(300) = -.28$, $p < .001$) were observed at Time 1. The same pattern was replicated for PWB during Time 2 ($r(300) = -.28$, $p < .001$), $r(300) = -.38$, $p < .001$, and $r(300) = -.34$, $p < .001$, for eBA, G, and CBS, respectively). Additionally, as mentioned, G and eBA were positively (though far from redundantly) related to each other at both time points, however, G and eBA were also positively related across time points, though to a lessened extent (Time 1 eBA with Time 2 G: $r(300) = .41$, $p < .001$; Time 2 eBA with Time 1 G:

$r(300) = .37$, $p < .001$). Finally, growth in PWB over time coincided with mild reduction in G ($r(300) = -.14$, $p = .021$) over time. PWB change's negative relation with ΔeBA also closely approached significance ($r(300) = -.12$, $p < .051$).

3.5. Hypothesis B: Gender recovery patterns

Examination of initial correlational relations between gender, eBA, G, and the change variables were mostly unsupportive of the second hypothesis that gender influences blame recovery. Gender only correlated significantly with Time 1 eBA ($r(300) = -.17$, $p = .003$). Correlations between gender and PWB, eBA at Time 2, G, ΔPWB ($r(300) = 0$, $p = .95$), ΔeBA ($r(300) = .04$, $p = .51$), and ΔG ($r(300) = -.08$, $p = .23$) were nonsignificant (see Table 5), indicating that gender did not affect the G and PWB rates of recovery overall. However, the likelihood of a relation to eBA remains possible, as the association was very near the arbitrary significance cutoff. Interestingly, the direction was positive, such that being female (which was coded as 1) near-significantly related to increased reduction in eBA over time relative to males (who were coded as 0).

Further hMR analyses of gender on change recovery variables were conducted, controlling for the influence of relationship seriousness (HS), time since breakup (HLA1/2), breakup initiator (WI1/2/3), having entered a new relationship since (RelB1/2 and RelP1/2), initial BDS, Interval between when individuals completed the two parts of the study, and PWB at Time 1. Results from the hMR for ΔPWB corroborated the original correlations, indicating that gender did not influence ΔPWB after accounting for the control variables ($F(14, 286) = 1.97$, $p = 1$; $\beta = 0.03$, $p = .630$; see Table 7). Addition of gender also did not influence ΔeBA ($F(14, 286) = 1.93$, $p = .071$; $\beta = -0.08$, $p = .168$;

see Table 8) or ΔG after controlling for the abovementioned variables ($\beta = -0.09$, $p =$

$.140$; $F(14, 286) = 2.88$, $p = 003$; see Table 9). It should be noted that as the focus of

these analyses is the effect of gender after accounting for controls rather than model

evaluation, model significance (F) is not a major concern and the coefficients of variables

other than gender are not interpreted.

3.6. Hypothesis C: E, EI, and RSB recovery patterns

Initial results for E yielded mixed patterns; it was negatively related to G at

Time 2 ($r(300) = -.12$, $p = .047$) and positively related to PWB (Time 1: $r(300) = .45$, $p <$

$.001$; Time 2: $r(300) = .31$, $p < .001$). However, E was not significantly related to ΔPWB

($r(300) = -.02$, $p = .713$), eBA ($r(300) = -.07$, $p = .198$ and $r(300) = -.07$, $p = .235$, for

Time 1 and 2 eBA, respectively), ΔeBA ($r(300) = -.05$, $p = .438$), or ΔG ($r(300) = -.08$,

$p = .167$). Results from hMR analyses with the same control variables as those used for

the gender hMRs (thus, possessing the same Step 1s) showed that E did not significantly

influence ΔeBA after accounting for the controls ($F(14, 286) = 1.84$, $p = .179$; $\beta = 0.27$, p

$= .346$; see Table 10), ΔG ($F(14, 286) = 2.94$, $p = .002$; $\beta = -0.11$, $p = .082$; see Table

11), or ΔPWB ($F(14, 286) = 2.08$, $p = .034$; $\beta = 0.09$, $p = .198$; see Table 12).

Analysis of EI revealed a very promising pattern of influence: EI was, in line

with the expectation of pro-recovery from blame feelings, negatively related to Time 1

eBA ($r(300) = -.31$, $p < .001$), Time 1 G ($r(300) = -.42$, $p < .001$), Time 2 eBA ($r(300) =$

$-.23$, $p < .001$), and Time 2 G ($r(300) = -.35$, $p < .001$). Additionally in line with

expectation, EI was positively related to Time 1 PWB (Time 1 PWB ($r(300) = .68$, $p <$

$.001$) and Time 2 PWB ($r(300) = .51$, $p < .001$). However, EI was not initially related to

ΔPWB, ΔeBA or ΔG (see Table 5). Follow up hMRs found that, as expected, EI,

significantly and positively affected ΔPWB ($F(14, 286) = 2.87$, $p = .002$; β = .27, $p = $ .001; see Table 13), with higher EI coinciding with increased PWB growth, after accounting for control variables. EI also showed the expected pro-recovery negative influence on ΔG ($F(14, 286) = 3.11$, $p = .001$; β = -0.15, $p = .029$; see Table 14), such that higher EI coincided with significantly greater reductions over time in G. EI did not show any influence over ΔeBA ($F(14, 286) = 1.18$, $p = .104$; β = –0.05, $p = .445$; see Table 15), after accounting for the control variables.

Examination of RSB found that it was not initially correlated with eBA, G, PWB, or the recovery/change variables. Hierarchical regressions confirmed this: after controlling for the breakup variables, Interval, and respective initial blame and PWB values, RSB did not influence ΔeBA ($F(14, 286) = 1.77$, $p = .118$; β = –0.01, $p = .824$; see Table 16), ΔG ($F(14, 286) = 2.80$, $p = .003$; β = 0.07, $p = .239$; see Table 17), or ΔPWB ($F(14, 286) = 1.95$, $p = .056$; β = 0.01, $p = .869$; see Table 18).

3.7. Hypothesis D: N recovery patterns

Preliminary examination of N with eBA and G revealed that, in line with general recovery-inhibitive predictions, N was positively related to both Time 1 and Time 2 eBA ($r(300) = .12$, $p = .045$ and $r(300) = .14$, $p = .020$, respectively) and G (Time 1: $r(300) = $ .27, $p < .001$; Time 2: $r(300) = .18$, $p = .002$). Likewise, following the proposed pattern of reduced recovery, N was negatively related to Time 1 and 2 PWB ($r(300) = –.40$, $p < $ .001 and $r(300) = –.27$, $p < .001$, respectively). In terms of recovery rates, N was positively correlated to ΔG over time ($r(300) = .14$, $p = .021$) such that slower G reduction coincided with higher N. However, N did not significantly relate to ΔeBA ($r(300) = .03$, $p = .615$) or ΔPWB ($r(300) = –.01$, $p = .846$). Hierarchical MR further

showed that N did not affect PWB change over time ($F(14, 286) = 2.21$, $p = .023$; $\beta = -0.12$, $p = .080$; see Table 19), eBA change ($F(14, 286) = 1.84$, $p = .096$; $\beta = 0.06$, $p = .371$; see Table 20), or change in G ($F(14, 286) = 2.69$, $p = .007$; $\beta = 0.03$, $p = .597$; see Table 21) after accounting for HLA, BDS, WI, HS, RelB, RelP, Interval, and respective initial eBA, G, or PWB scores.

3.8. Hypothesis E: Individual difference trait model effectiveness

Simultaneous entry of N, E, EI, RSB, and gender into three models predicting ΔeBA, ΔG, and ΔPWB revealed that the five factors were not a predictive model of ΔeBA. Gender, RSB, E, N, and EI did not influence ΔeBA when not controlling for the breakup study variables ($F(18, 282) = 3.25$, $p = .017$; $\beta = -0.08$, $p = .171$; $\beta = 0$, $p = .996$; $\beta = 0.04$, $p = .575$; $\beta = 0.06$, $p = .425$; and $\beta = 0$, $p = .958$, respectively; see Table 22) or when controlling for them ($F(18, 282) = 1.61$, $p = .159$; $\beta = -0.20$, $p = .152$; $\beta = -0.01$, $p = .906$; $\beta = -0.03$, $p = .689$; $\beta = 0.06$, $p = .418$; and $\beta = 0$, $p = .988$, respectively). The same was observed for models of ΔG with ($F(18, 282) = 2.74$, $p = .001$; gender: $\beta = -0.07$, $p = .245$; RSB: $\beta = 0.08$, $p = .209$; E: $\beta = -0.05$, $p = .423$; N: $\beta = -0.03$, $p = .719$; and EI: $\beta = -.13$, $p = .117$; see Table 23) and without breakup controls ($F(18, 294) = 7.43$, $p < .001$; gender: $\beta = -0.07$, $p = .166$; RSB: $\beta = 0.07$, $p = .255$; E: $\beta = -0.05$, $p = .490$; N: $\beta = -0.01$, $p = .834$; and EI: $\beta = -0.13$, $p = .100$). However, EI was found to be of value in the combined model for ΔPWB both without breakup control variables ($F(6, 294) = 4.48$, $p < .001$; EI: $\beta = 0.24$, $p = .007$; gender: $\beta = 0.02$, $p = .767$; RSB: $\beta = 0.02$, $p = .726$; E: $\beta = 0.05$, $p = .475$; and N: $\beta = -0.03$, $p = .677$; see Table 24) and with them ($F(18, 282) = 2.30$, $p = .007$; EI: $\beta = 0.24$, $p = .007$; gender: $\beta = 0.03$, $p = .582$; RSB: $\beta = .02$, $p = .733$; E: $\beta = 0.03$, $p = .619$; and N: $\beta = -0.04$, $p = .569$).

Standardized residuals of all hMR models performed in this study met the assumption of residual normality via P-P residual plot inspection (see Figure 2 for a typical example from the analyses). Likewise, homoscedasticity was supported for all models as illustrated by fit line testing in SPSS (see Figure 3 for a standard example from the analyses).

CHAPTER FOUR: DISCUSSION

4. Discussion

The present study attempted to determine if the personality and individual difference characteristics of extraversion-introversion, neuroticism, trait emotional intelligence, and religious-spiritual belief influenced rates of recovery from feelings of blame and guilt toward health and psychological wellbeing. It was expected that E, EI, and RSB would be protective factors that encouraged greater reductions in G and eBA over time, and greater growth of PWB over time. Conversely, N and being female were expected to slow G and eBA reductions as well as PWB growth over a 1-month period of study.

Hypothesis A, which predicted a pattern of general wellbeing where G and eBA would be negatively related to PWB, and where growth in PWB would coincide with reductions in G and eBA, was generally supported. G and eBA were positively correlated with one another, and were negatively correlated to PWB at each time point. Reductions in G over time likewise coincided with mild growth in PWB from Time 1 to Time 2. Moreover, while the expected relation of reduction in eBA coinciding with PWB growth was not significant, it was very close ($p = .051$). Therefore, eBA and G appear to be orientations that generally reduce emotional stability and health. That said, however, Zeidner and Saklofske (1996) caution that adaptiveness of a coping strategy such as affective coping (which is often considered maladaptive, and which includes eBA and self-blame) depends on a variety of situational and vulnerability factors; what appears hurtful may possess a hidden protective function. However, the present study overall found support for recovery via reduction of G and eBA, and growth in PWB over time.

Conversely, results of this study did not support hypothesis B, which predicted that gender would relate to recovery such that being female would coincide with less PWB growth and less eBA and G reduction overt time relative to being male. There was no significant relation between gender and PWB, eBA, or G except locally at Time 1 eBA measurement. MR analyses confirmed this non-relation, showing that gender did not influence any of the recovery change variables after accounting for important breakup-context variables such as which partner initiated the breakup, how serious/intimate the relationship was, and how long ago the breakup occurred. However, the near-significant positive zero-order relation between gender (being female, which was coded as 1 whereas males were coded as 0) and eBA change is noteworthy. As females more frequently internalize their negative affect, reduction of externally focused affect would be expected to be more rapid for them relative to males, but this directionality suggests the opposite, indicating that being female nonsignificantly coincided with reduced eBA recovery rate. However, the non-effect of gender on eBA recovery after holding the set of situational breakup factors constant indicates that this relation— regardless of its unexpected directionality—would not likely be from gender itself. Additional situational factors—perhaps cognitive evaluation for the discounting of external causes—would be involved in any such eBA recovery pattern functioning alongside gender.

As with gender, hypothesis C, which stated that E and RSB would be recovery-driving factors that hastened G and eBA reduction and PWB growth, was also not supported. While basic correlations between some of the recovery variables at local time points within the study were observed for E (e.g. positive zero-order relations to PWB

and negative relations to G), E was not related to changes in eBA, G, or PWB over time before or after accounting for breakup context variables. As E is a core dimension of personality, which dictates more global responding styles, it is unreasonable to wholly discount it as a factor in recovery. Rather, a confound seems more likely. Specifically, length of time since the breakup remains a concern; the control variable used in the present study for this factor was a categorical variable that did not allow for variability within the category time frames of <4 weeks, 1 to 2 months, and 2 to 3 months. Only about 20% of participants had broken up between within a month of beginning the present study, when recovery rates and dispositional effects are strongest. Perhaps any significant influence on recovery and recovery rates from E had already occurred, thereby blocking the present study from identifying it. This late-comer possibility is supported by the high average PWB in the sample at Time 1 of the study ($M = 43.48$, $SD = 7.84$; Diener et al., 2010), indicating that the proposed 0 to 3 month window of study was not ideal, and possibly came too late for effect measurement in these individuals. Of course, 0-12 weeks is used often in breakup literature (Eastwick et al., 2008; Knox et al., 2000), but most physiological recovery trajectories demonstrate a key phase—called the *initial acute phase response*—directly following an event where a burst of responding occurs that sets the course for future recovery from very soon after the event (Clark, Beamer, Wynn, & Coull, 1998). As negative affect produces very physiological responses, it follows that blame recovery rates are likely most influenced very soon after breakup and then slow to asymptote or oscillate around an equilibrium after. Additionally, it is possible that the categorical HLA variables (intended to control for time since breakup)

were not sensitive enough to control for the interpersonal variation in recovery possible between, for instance, the first 4-8 weeks after breakup.

Conversely, results for EI as a pro-recovery factor in hypothesis C were mostly supported. Initial correlations with eBA, G, and PWB suggested that outside recovery, EI was a protective, anti-blame and pro-PWB factor. Additionally, after accounting for the breakup context variables, EI remained positively related to PWB growth such that high EI coincided with larger increases in PWB over time, and was negatively influential for G reduction such that high EI significantly explained greater reductions in G over time. Surprisingly, EI and eBA change were not related before or after breakup control variables were accounted for, indicating that EI within breakup contexts might be more internally directed (e.g. self-evaluation rather than other-evaluation). Therefore, for PWB and G, EI was pro-recovery. Perhaps eBA reduction was not related based on the sample: most participants had reported either themselves as the initiators of the breakup or a mutual breakup, where their would justifiably be reduced other-blame in general.

In terms of RSB, belief was found to not positively drive blame reduction or PWB growth rates as expected. In fact, RSB did not relate to PWB, eBA, or G at either time of measure. One important reason for this could be that RSB in the sample was moderate to low in value, such that beliefs were held relatively weakly and would have little impact on recovery processes. As beliefs are a purposeful, cognitive strategy for coping, individuals must actively seek out their belief systems and explain to themselves their feelings and the situation through that belief system lens before it can be helpful or hurtful; if belief is not strong enough, there is little motivation for an individual to draw on it as a coping strategy.

Findings for hypothesis D, which suggested that N would be a recovery-inhibiting factor, were also mixed. N displayed the expected patterns of negative effect on PWB and positive effect on G and eBA within and across the study's time points, and displayed a positive initial relation with G recovery. However, N did not affect the change rate following control of breakup variables, indicating —as was suggested for the near-relation between eBA and gender—that N was not the source of the initial effect. Moreover, N's independence from PWB and eBA change rates suggests the same dilemma as core E; perhaps N functions on recovery rates sooner in the recovery trajectories and was not captured in the scope of the present research.

Finally, no support was found for the evaluative value of the five characteristics as a unified model of predicting eBA, G, and PWB change rate scores under hypothesis E. None of the variables, save EI for predicting PWB change rate, were found to be predictive. Likewise, model fits were generally poor with little combined variance explained. Therefore, without further investigation these variables cannot be taken together as indicative of blame and wellbeing change rates after breakup.

Overall, the above results highlight an important issue of effect timeline. In the present study, the period of 0 to 3 months since breakup was based on frequent preceding use in the relationship literature (Eastwick et al., 2008; Knox, Kusman, Kaluzny, & Cooper, 2000; Moller, Fouladi, McCarthy, & Hatch, 2003). However, this timeline does not appear sufficient for capturing specific rates, as PWB was already very high for many participants, with very little room for improvement. Specifically, E and N are very well-developed as factors in recovery in coping literature (Fernandez-Berrocal & Extremera, 2006; Bolger, 1990; Connor-Smith & Flachsbart, 2007), and their nonsignificant

influence would be logical if they had already finished operating on recovery rates long before measurement. However, it is possible these are true findings and that E and N have been circumvented by motivational factors. For example, some researchers argue that the personality to coping style trend is malleable based on personal motivation and intent (Gallagher, 1996), which would obscure any E or N relation if enough of the sample was malleable (though this seems far less likely than the mismatched timeline explanation).

4.1. Limitations and directions for future research

The design of the present study anticipated the 0-3 month post-breakup recovery timeline used by prior relationship dissolution researchers. However, as this timeline appears inadequate in terms of already high PWB on entry, replication of factor effects on recovery rates using a shorter cutoff would be required (e.g. within the first month after breakup) before the present study's results can be considered valid.

Additionally, the present study did not account for self-selection of participants. Specifically, according to the *dual process model* (DPM; Caserta, Utz, Lund, Swenson, & de Vries, 2014) of coping from the bereavement literature, individuals oscillate between confronting and experiencing loss and restorative phases during coping, meaning that affect levels can look like a damped curve, with sinuous peaks and valleys, depending on the phase of coping. The present study may have only attracted individuals who were on a post-breakup peak, when negative feelings are limited or nonexistent (and when they would be more willing to spend time on a study). Thus, rates of recovery that fluctuate with time and cause observed effects and correlations over the recovery period to fluctuate as well may not have been properly represented via linear regression.

Therefore, multi-wave longitudinal study, ideally employing variable measurement intervals that reduce the chance of sampling only during a particular phase, would be needed.

Future research would likewise benefit from cohort and attachment-orientation examination, as blame tendencies, distress sensitivity, and coping effectiveness are known to fluctuate with age (Iobst et al., 2009; Blanchard-Fields & Beatty, 2005; Grazzani Gavazzi, Ornaghi, & Antoniotti, 2011; Douglas & Shaffer, 1971) and relationship attachment style (Downey & Feldman, 1996; Rowsell & Coplan, 2013; Marshall, Benjanyan, & Ferenezi, 2013; Sprecher, Felmlee, Metts, Fehr, & Vanni, 1998; Caron, LaFontaine, Bureau, Levesque, & Johnson, 2012). Exploration of the facets underlying TEI (the TEIQue does include a general wellbeing facet of EI; Cooper & Petrides, 2010; Petrides & Furnham, 2003) would also be prudent.

Finally, methodological considerations must be made for Type I inflation (in the present study, the number of models may inflate alpha), and the number of prediction variables. Simplification via full, simultaneous modeling is advisable in future examinations.

4.2. Concluding remarks

The present study did not support the hypotheses that E, RSB, gender, or N affected blame and psychological wellbeing rates of recovery after romantic breakup, though E, gender, and N showed influence on blame and wellbeing levels at single time points. Evidence for EI influencing self-blame guilt and wellbeing were supported, such that higher EI signalled greater reductions in guilt over time and greater growth in wellbeing over time, as well as relating significantly to single time point measures of

blame, guilt, and wellbeing. However, EI did not influence eBA reduction rate.

Moreover, the five factors did not form a cohesive and functional model for predicting

rates of change in blame and wellbeing. Refinement of the timeline for measuring

recovery after breakup and longitudinal cohort examination are suggested to clarify the

present results and to further understand the impact of personality and individual traits on

blame affect following a blame-inducing event.

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Table 1.

Table of Acronyms

BA	Blame attribution
eBA	External blame attribution
G	Guilt (a component of blame attribution)
PWB	Psychological wellbeing
ΔeBA	Change in eBA (Time 2 minus Time 1)
ΔG	Change in G (Time 2 minus Time 1)
ΔPWB	Change in PWB (Time 2 minus Time 1)
E	Extraversion
N	Neutoricism
TEI	Trait emotional intelligence
HS	Seriousness of relationship
BDS	Breakup Distress Scale
CBS	Characterological-Behavioural Self-Blame Scale
HFS	Heartland Forgiveness Scale
RSB	Religious-spiritual belief
RB-BAI	Romantic Breakup Blame Attribution Inventory
HLA	How long since breakup (dummy coded variable)
WI	Who Initiated breakup (dummy coded variable)
RelB (1 and 2)	New relationship since breakup at Time 1 of study (dummy coded variables; Y/N/Unsure)
RelP (1 and 2)	New relationship since Time 1 of study (dummy coded variables; Y/N/Unsure)
Interval	Time between Time 1 and Time 2 of study

Table 2.

Data Screening: Breakdown of Case Exclusions and Missing Categorical Data Imputations

Screening Criteria	Cases (n)
Excluded	
Inattention (>2 h to complete either survey session)	15
No data	44
Reported no serious past relationship[1]	3
Post-MI Outliers (Outlier Labeling Rule)	17
Imputed missing categorical information	
No gender reported[1]	3
No time since breakup (T1RelS) reported[1]	3
No breakup initiator entered[1]	4
No new relationship status since breakup reported[1]	4
No new relationship status since Time 1 (T2NRel) reported[2]	82

[1] *Time session 1 of study,* [2] *Time session 2 of study (approximately 4 weeks after Time 1)*

Table 3.
Categorical Demographic Proportions (Pooled; Post-MI)

	%		%
Gender		How long since breakup (HLA)	
Female	66.89	0-4 weeks	23.96
Male	33.11	4-8 weeks	26.11
Faculty		8-12 weeks	49.93
Arts & Humanities	5.63	Initiator of breakup/who initiated (WI)	
Music	0.66	Participant	34.37
Education	0.33	Participant's ex-partner	25.40
Engineering	4.64	Both	31.80
Graduate/Post-doc	0.33	Other	8.43
Health Sciences	14.24	Relationship since breakup (Time 1; RelB)	
Information/Media Studies	5.63	Yes	10.48
Law	0.33	No	82.58
Business	0.66	Uncertain	6.94
Medicine/Dentistry	0.66	New Relationship since study Part 1 (Time 2; RelP)	
Science	19.21	Yes	≈ 22.28 [Q]
Social Science	46.69	No	≈ 64.37 [Q]
Other	0.99	Uncertain	≈ 13.34 [Q]

N = 319, [Q] Rounded approximations intended for basic description only; RelP returned non-integer values during MI (it was the only categorical variable in the table above that did).

Table 4.
Descriptive Statistics (Pooled)

Variables	Mean	SD	α	SU	KU
1. Age	19.31	2.43		-1.23	33.97
2. HS	5.48	1.17		-0.08	2.14
3. BDS [1]	38.36	10.78	.91	0.27	-0.47
4. BDS [2]	32.39	8.99	.89	-0.24	0.16
5. CBS [1]	53.85	11.23	.75	-0.20	0.93
6. CBS [2]	52.67	9.65	.87	0.27	1.18
7. HFS [1]	80.12	14.25	.82	0.55	0.21
8. HFS [2]	82.48	12.04	.81	-1.76	1.12
9. RSB [1]	27.52	15.26	.95	0.17	-0.93
10. RSB [2]	35.85	4.55	.94	.20	-0.34
11. E [1]	8.65	3.34	.86	-0.76	-0.53
12. E [2]	9.80	3.58	.83	-0.62	-0.18
13. N [1]	6.41	3.18	.78	0.01	-0.78
14. N [2]	8.04	3.93	.81	0.04	-0.83
15. TEI [1]	141.71	23.57	.88	0.02	-0.67
16. TEI [2]	142.14	20.43	.88	0.04	0.67
17. eBA [1]	21.59	5.80	.79	0.17	0.10
18. eBA [2]	20.29	5.33	.77	0.22	0.81
19. G [1]	16.14	5.32	.84	0.44	-0.47
20. G [2]	14.06	4.69	.83	-0.48	0.08
21. PWB [1]	43.48	7.84	.90	-0.81	-0.64
22. PWB [2]	43.62	7.01	.87	.99	1.25
23. Interval [n]	29.71	4.24		0.08	11.99
24. ΔeBA	-0.89	3.51		-0.29	0.72
25. ΔG	-1.89	3.56		-0.08	0.86
26. ΔPWB	-0.12	4.70		-1.23	1.28

HS = relationship seriousness before breakup, [1] = Time 1, [2] = Time 2, Δ scores = Time 2 minus Time 1 scores, [n] Number of days between Time 1 and Time 2 measurement sessions.

Table 5.
Correlation Matrix

		Age	1	2	3	4	5	6	7	8
1.	Gender	.04								
2.	HS	.08	.11							
3.	BDS [1]	.04	0	.19***						
4.	BDS [2]	-.01	-.09	0	.50***					
5.	CBS [1]	-.12*	-.04	.03	.34***	.21***				
6.	CBS [2]	-.05	-.05	-.06	.07	.31***	.32***			
7.	HFS [1]	.08	.02	0	-.35***	-.22***	-.30***	-.21***		
8.	HFS [2]	.10	.09	.01	-.22***	-.41***	-.30***	-.34***	.56***	
9.	RSB [1]	.10	0	-.06	.01	.09	.05	.05	-.06	-.10
10.	RSB [2]	.11*	.04	-.13*	.01	.13*	0	.07	-.01	-.10
11.	E [1]	-.11	.13*	.04	-.05	-.04	-.18**	-.16**	.33***	.15**
12.	E [2]	-.17**	.07	.01	.04	-.11	-.11	-.21***	.20***	.16**
13.	N [1]	-.033	.12*	.01	.32***	.15*	.32***	.23***	-.46***	-.30***
14.	N [2]	-.063	-.042	0	.16*	.29***	.22***	.33***	-.36***	-.48***
15.	TEI [1]	.10	.08	.05	-.28***	-.25***	-.35***	-.29***	.57***	.41***
16.	TEI [2]	.05	.07	.04	-.21***	-.36***	-.30***	-.35***	.45***	.58***
17.	eBA [1]	-.05	-.17**	.02	.13*	.21***	.22***	.16**	-.11*	-.16**
18.	eBA [2]	-.04	-.12	-.04	.08	.26***	.21***	.24***	-.10	-.21***
19.	G [1]	-.07	-.07	.03	.58***	.47***	.32***	.22***	-.34***	-.32***
20.	G [2]	-.04	-.08	-.06	.35***	.65***	.24***	.32***	-.25***	-.41***
21.	PWB [1]	.06	.09	.02	-.30***	-.24***	-.28***	-.24***	.42***	.29***
22.	PWB [2]	.03	.08	.06	-.15**	-.33***	-.23***	-.34***	.33***	.47***
23.	Interval [n]	-.02	-.04	-.06	-.05	-.08	-.04	-.08	.04	.02
24.	ΔeBA	-.03	-.04	-.01	-.03	.05	.05	.10	-.02	-.09
25.	ΔG	.01	-.08	-.07	-.15*	.15**	-.03	.07	.03	-.06
26.	ΔPWB	-.03	0	.04	.06	-.10	.01	-.14*	.10	.23***
27.	HLA1 [c]	-.04	-.14*	-.05	.11	.14*	0	.05	-.05	-.15*
28.	HLA2 [c]	-.06	.02	.02	-.04	-.06	-.01	-.10	-.01	.08
29.	WI1 [c]	-.07	.08	.08	-.20***	-.10	-.03	.05	.10	.09
30.	WI2 [c]	.06	-.08	-.09	.12*	.10	.02	-.05	-.09	-.15*
31.	WI3 [c]	0	-.01	.03	.01	.01	-.09	-.01	.05	.09
32.	RelB1 [c]	.03	.07	-.12*	-.03	-.07	.07	0	-.06	.02
33.	RelB2 [c]	-.06	-.05	.11	.01	.03	-.05	-.01	.11	.03
34.	RelP1 [c]	-.07	.04	-.05	-.01	-.16*	.06	-.05	0	.01
35.	RelP2 [c]	.10	-.04	.15*	0	.06	-.08	-.06	.07	.08

*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$, [c] Dummy coded contrast variables for time since breakup (HLA1/2), Breakup initiator (WI1/2/3), Relationships since breakup(Time 1; T1RelB1/2), and Relationship since end of Part 1 (RelP1/2)

	9	10	11	12	13	14	15	16	17
10. RSB [2]	.70***								
11. E [1]	.06	.09							
12. E [2]	.07	.09	-.68***						
13. N [1]	-.06	.01	-.30***	-.21***					
14. N [2]	.09	.12*	-.24***	-.23***	.56***				
15. TEI [1]	-.01	-.04	.43***	.29***	-.54***	-.44***			
16. TEI [2]	-.03	-.06	.30***	.37***	-.40***	-.57***	.66***		
17. eBA [1]	0	.03	-.07	-.04	.12*	.09	-.31***	-.23***	
18. eBA [2]	-.02	.02	-.07	-.09	.14*	.15**	-.23***	-.30***	.64***
19. G [1]	.03	.04	-.05	-.04	.27***	.21***	-.42***	-.31***	.56***
20. G [2]	.03	.07	-.12*	-.16**	.18**	.31***	-.35***	-.45***	.41***
21. PWB [1]	.08	0	.45***	.35***	-.40***	-.28***	.68***	.52***	-.27***
22. PWB [2]	.02	0	.31***	.41**	-.27***	-.40***	.51***	.71***	-.24***
23. Interval[n]	0	-.01	-.01	.01	.01	-.02	-.02	.05	-.08
24. ΔeBA	0	-.01	-.05	-.05	.03	.07	.02	-.08	-.22***
25. ΔG	.06	.05	-.08	-.12*	-.04	.10	.01	-.11	-.08
26. ΔPWB	-.01	-.04	-.02	.03	-.01	-.14*	0	.18**	0
27. HLA1[c]	.04	.07	.06	.09	.02	.06	-.07	-.10	.05
28. HLA2[c]	-.02	-.01	.09	.06	-.01	-.04	-.01	.04	-.04
29. WI1[c]	-.04	.07	.06	0	-.08	-.06	.03	.02	.09
30. WI2[c]	0	-.01	-.12*	-.13*	.07	.11	-.03	-.07	-.18**
31. WI3[c]	0	-.10	.09	.11	-.03	-.08	.05	.10	.02
32. RelB1[c]	.15*	.14*	-.03	-.02	.02	.04	-.05	-.03	-.08
33. RelB2[c]	-.14*	-.10	.06	.01	-.06	-.06	.12*	.07	.04
34. RelP1[c]	.12	.06	0	.05	.04	-.01	-.04	.02	-.09
35. RelP2[c]	-.12	-.12	0	-.01	-.06	-.03	.09	.06	.03

*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$, [c] Dummy coded contrast variables for time since breakup (HLA1/2), Breakup initiator (WI1/2/3), Relationships since breakup(Time 1; T1RelB1/2), and Relationship since end of Part 1 (RelP1/2)

	18	19	20	21	22	23	24	25	26
19. G [1]	.37***								
20. G [2]	.56***	.60***							
21. PWB [1]	-.21***	-.34***	-.29***						
22. PWB [2]	-.28***	-.26***	-.38***	.60***					
23. Interval[n]	-.08	-.11	-.09	.05	.01				
24. ΔeBA	.35***	-.08	.20***	.03	-.05	.01			
25. ΔG	.17**	-.31***	.34***	.02	-.09	.05	.39		
26. ΔPWB	-.09	.03	-.08	-.20***	.38***	-.06	-.11	-.14*	
27. HLA1[c]	.08	.16**	.15*	.09	.02	-.09	.05	-.02	-.03
28. HLA2[c]	-.16**	-.03	-.07	0	.06	.08	-.13*	.02	.07
29. WI1[c]	.13*	-.19***	-.11	.03	-.07	-.06	.01	.07	-.10
30. WI2[c]	-.14*	.07	.07	-.05	.01	.11	.02	.01	.02
31. WI3[c]	-.03	.06	0	.05	.10	-.05	-.01	-.04	.07
32. RelB1[c]	-.03	-.05	-.02	-.03	-.03	.12	.03	.01	-.07
33. RelB2[c]	-.04	-.02	-.04	.07	.07	-.09	-.06	0	.04
34. RelP1[c]	-.04	-.05	-.08	.01	.04	-.01	.10	-.05	.09
35. RelP2[c]	-.02	-.05	-.04	.03	.04	.03	-.10	.01	-.01

*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$, [c] Dummy coded contrast variables for time since breakup (HLA1/2), Breakup initiator (WI1/2/3), Relationships since breakup(Time 1; T1RelB1/2), and Relationship since end of Part 1 (RelP1/2)

	27	*28*	*29*	*30*	*31*	*32*	*33*	*34*
28. HLA2 [c]								
29. WI1 [c]	-.07	.05						
30. WI2 [c]	0	0	-.42***					
31. WI3 [c]	.07	-.06	-.49***	-.40***				
32. RelB1 [c]	.02	-.10	-.02	.08	-.09			
33. RelB2 [c]	-.10	.15**	-.03	0	.06	-.75***		
34. RelP1 [c]	-.02	-.05	0	.02	-.01	.28***	-.32***	
35. RelP2 [c]	-.09	.10	-.01	-.01	.02	-.30***	.37***	-.74***

*$p \le .05$, ** $p \le .01$, *** $p \le .001$, [c] Dummy coded contrast variables for time since breakup (HLA1/2), Breakup initiator (WI1/2/3), Relationships since breakup(Time 1; T1RelB1/2), and Relationship since end of Part 1 (RelP1/2)

Table 6.

Test-Retest Reliabilities

	r
BDS	.50***
CBS	.32***
HFS	.56***
RSB	.70***
N	.56***
E	.68***
TEI	.67***
eBA	.64***
G	.60***
PWB	.60***

*** *p < .001*

Table 7.
Analysis of ΔPWB by Gender after Breakup Control Variables

	R	R^2_{change}	B	SE	β	C_s^2	Part r	VIF
Step 1	.29	.09*						
Intercept			-0.25	0.38	0.02			
HLA1			0.05	0.15	0.08	.01	.02	1.21
HLA2			0.18	0.14	-0.09	.06	.07	1.15
WI1			-0.19	0.24	0	.12	-.05	3.51
WI2			0.01	0.24	0.04	.01	0	3.05
WI3			0.08	0.24	-0.09	.06	.02	3.37
RelB1			-0.28	0.31	-0.02	.05	-.05	2.37
RelB2			-0.05	0.26	0.19	.02	-.01	2.45
RelP1			0.55*	0.26	0.11	.09	.12	2.30
RelP2			0.28	0.22	0.02	0	.07	2.39
HS_z			0.04	0.07	-	.02	.04	1.11
BDS^1_z			-0.03	0.07	-	.04	-.03	1.24
$Interval_z$			-0.05	0.06	-	.04	-.05	1.06
PWB^1_z			-0.22***	0.06	-	.49	-.20	1.14
Step 2	.29	0						
Intercept			-.30	0.39	0.03			
HLA1			0.06	0.15	0.08	.01	.02	1.24
HLA2			0.18	0.14	-0.09	.06	.07	1.15
WI1			-0.19	0.24	0.01	.12	-.05	3.51
WI2			0.02	0.24	0.04	.01	.01	3.05
WI3			0.08	0.24	-0.09	.06	.02	3.37
RelB1			-0.28	0.31	-0.02	.05	-.06	2.38
RelB2			-0.05	0.26	0.19	.02	-.01	2.45
RelP1			0.56*	0.26	0.11	.09	.12	2.30
RelP2			0.28	0.22	0.03	0	.07	2.39
HS_z			0.04	0.07	-	.02	.04	1.12
BDS^1_z			-0.03	0.07	-	.04	-.03	1.24
$Interval_z$			-0.05	0.06	-	.04	-.05	1.06
PWB^1_z			-0.22***	0.06	-	.49	-.20	1.15
Gender			0.06	0.13	0.03	0	.03	1.07

* $p < .05$, *** p z Standardized variable, [1] Time 1, C_s^2 = Squared structural coefficient, indicating how much of model R^2 is explained by predictor in isolation from other predictor variables.
Step 1: $F(13, 287) = 2.09$, $p = .039$, Step 2: $F(14, 286) = 1.97$, $p = .051$

Table 8.
Analysis of ΔeBA by Gender after Breakup Control Variables

	R	R^2_{change}	B	SE	β	C_s^2	Part r	VIF
Step 1	.28	.08						
Intercept			0.22	0.36				
HLA1			0.03	0.15	0.01	.03	.01	1.18
HLA2			-0.29*	0.15	-0.13	.20	-.12	1.15
WI1			0.11	0.23	0.05	0	.03	3.51
WI2			0.02	0.24	0.01	0	.01	3.11
WI3			0.04	0.24	0.02	0	.01	3.37
RelB1			-0.17	0.30	-0.05	.02	-.03	2.37
RelB2			-0.12	0.24	-0.05	.05	-.03	2.43
RelP1			0.08	0.26	0.03	.11	.02	2.31
RelP2			-0.15	0.22	-0.06	.12	-.04	2.39
HS_z			0	0.06	-	0	0	1.11
BDS^1_z			0.01	0.07	-	.01	0	1.15
$Interval_z$			0.01	0.06	-	0	.01	1.06
eBA^1_z			-0.23***	0.06	-	.61	-.22	1.08
Step 2	.29	.01						
Intercept			0.36	0.38				
HLA1			0	0.15	0	.03	0	1.21
HLA2			-0.29*	0.15	-0.13	.19	-.12	1.15
WI1			0.11	0.23	0.05	0	.03	3.51
WI2			0	0.24	0	0	0	3.13
WI3			0.04	0.24	0.02	0	.01	3.37
RelB1			-0.15	0.30	-0.05	.01	-.03	2.38
RelB2			-0.12	0.24	-0.05	.04	-.03	2.43
RelP1			0.07	0.26	0.02	.11	.02	2.31
RelP2			-0.16	0.22	-0.06	.11	-.04	2.39
HS_z			0.01	0.06	-	0	.01	1.12
BDS^1_z			0.01	0.07	-	.01	.01	1.16
$Interval_z$			0.01	0.06	-	0	.01	1.06
eBA^1_z			-0.24***	0.06	-	.57	-.23	1.12
Gender			-0.18	0.13	-0.08	.02	-.08	1.09

* $p < .05$, *** $p < .001$, z Standardized variable, [1] Time 1, C_s^2 = Squared structural coefficient, indicating how much of model R^2 is explained by predictor in isolation from other predictor variables.
Step 1: $F(13, 287) = 1.90$, $p = .086$, Step 2: $F(14, 286) = 1.93$, $p = .071$

Table 9.
Analysis of ΔG by Gender after Breakup Control Variables

	R	R^2_{change}	B	SE	β	C_s^2	Part r	VIF
Step 1	.34	.11**						
Intercept			.16	.36				
HLA1			.06	.15	0.03	0	.03	1.19
HLA2			.06	.15	0.02	0	.02	1.15
WI1			.15	.23	0.07	.04	.04	3.56
WI2			.18	.23	0.08	0	.04	3.04
WI3			.10	.23	0.05	.01	.03	3.38
RelB1			-.06	.30	-0.02	0	-.01	2.38
RelB2			-.08	.24	-0.03	0	-.02	2.45
RelP1			-.40	.25	-0.13	.02	-.09	2.32
RelP2			-.24	.22	-0.09	0	-.06	2.41
HS_z			-.06	.06	-	.04	-.06	1.11
BDS^1_z			.06	.08	-	.21	.04	1.64
$Interval_z$			.01	.06	-	.02	.01	1.05
G^1_z			-.35***	.07	-	.82	-.27	1.61
Step 2	.35	.01						
Intercept			0.31	0.37	0.02			
HLA1			0.03	0.15	0.07	0	.01	1.21
HLA2			0.05	0.15	0.08	0	.02	1.15
WI1			0.16	0.23	0.04	.04	.04	3.56
WI2			0.16	0.23	-0.02	0	.04	3.05
WI3			0.10	0.23	-0.02	.01	.03	3.38
RelB1			-0.04	0.30	-0.15	0	-.01	2.39
RelB2			-0.08	0.24	-0.09	0	-.02	2.45
RelP1			-0.40	0.25	-0.02	.02	-.09	2.32
RelP2			-0.25	0.22	0.02	0	-.06	2.41
HS_z			-0.05	0.06	-	.04	-.05	1.12
BDS^1_z			0.06	0.08	-	.21	.05	1.65
$Interval_z$			0.01	0.06	-	.02	.01	1.06
G^1_z			-0.35***	0.07	-	.82	-.28	1.62
Gender			-0.20	0.13	-0.09	.05	-.09	1.06

** $p < .01$, *** $p < .001$, $_z$ Standardized variable, [1] Time 1, C_s^2 = Squared structural coefficient, indicating how much of model R^2 is explained by predictor in isolation from other predictor variables.

Step 1: $F_{(13, 287)} = 2.87$, $p = .004$, Step 2: $F_{(14, 286)} = 2.88$, $p = 003$.

Table 10.
Analysis of ΔeBA by E after Breakup Control Variables

	R	R^2_{change}	B	SE	β	C_s^2	Part r	VIF
			Step 1 identical to Step 1 in Table 7					
Step 2	.29	0	0.04	0.15				
Intercept			-0.27	0.15	-0.12	.03	.02	1.19
HLA1			0.12	0.23	0.05	.19	-.11	1.17
HLA2			0.02	0.24	0.01	0	.03	3.57
WI1			0.06	0.24	0.03	0	.01	3.10
WI2			-0.16	0.30	-0.07	0	.02	3.42
WI3			-0.10	0.24	-0.03	.01	-.03	2.37
RelB1			0.08	0.26	0.03	.04	-.02	2.45
RelB2			-0.15	0.22	-0.05	.11	.02	2.31
RelP1			0	0.06	0	.11	-.04	2.39
RelP2			0	0.07	-0.12	0	0	1.11
HS_z			0.01	0.06	-	.01	0	1.16
BDS^1_z			-0.23	0.06	-	0	.01	1.06
$Interval_z$			0.06	0.06	-	.57	-.22	1.09
eBA^1_z			0.04***	0.15	-	.03	.06	1.06
E^1_z			0.27	0.15	-	.03	-.02	1.19

*** $p < .001$, $_z$ Standardized variable, ^{1} Time 1, C_s^2 = Squared structural coefficient, indicating how much of model R^2 is explained by predictor in isolation from other predictor variables.

Step 1: $F(13, 287) = 1.90$, $p = .086$, Step 2: $F(14, 286) = 1.84$, $p = .179$.

Table 11.

Analysis of ΔG by E after Breakup Control Variables

	R	R^2_{change}	B	SE	β	C_s^2	Part r	VIF
Step 1 identical to Step 1 in Table 8								
Step 2	.35	.01						
Intercept			0.10	0.36				
HLA1			0.09	0.15	.04	0	.04	1.20
HLA2			0.09	0.15	.04	0	.04	1.17
WI1			0.18	0.23	.08	.04	.05	3.58
WI2			0.17	0.23	.08	0	.04	3.04
WI3			0.14	0.23	.06	.01	.03	3.40
RelB1			-0.04	0.30	-.01	0	-.01	2.38
RelB2			-0.05	0.24	-.02	0	-.01	2.46
RelP1			-0.40	0.25	-.13	.02	-.09	2.32
RelP2			-0.24	0.22	-.10	0	-.06	2.41
HS_z			-0.06	0.06	-	.04	-.05	1.11
BDS^1_z			0.05	0.08	-	.20	.04	1.65
$Interval_z$			0.01	0.06	-	.02	.01	1.05
G^1_z			-0.35***	0.07	-	.77	-.28	1.62
E^1_z			-0.11	0.06	-	.06	-.10	1.05

*** $p < .001$, $_z$ Standardized variable, [1] Time 1, C_s^2 = Squared structural coefficient, indicating how much of model R^2 is explained by predictor in isolation from other predictor variables.

Step 1: $F(13, 287) = 2.87$, $p = .004$, Step 2: $F(14, 286) = 2.94$, $p = .002$

Table 12.
Analysis of ΔPWB by E after Breakup Control Variables

	R	R^2_{change}	B	SE	β	C_s^2	Part r	VIF
			Step 1 identical to Step 1 in Table 6					
Step 2	.30	.01						
Intercept			-0.22	0.38				
HLA1			0.04	0.15	0.02	.01	.01	1.21
HLA2			0.15	0.14	0.07	.06	.06	1.17
WI1			-0.21	0.24	-0.10	.11	-.05	3.56
WI2			0.02	0.24	0.01	.01	.00	3.04
WI3			0.05	0.24	0.02	.06	.01	3.40
RelB1			-0.29	0.30	-0.09	.05	-.06	2.37
RelB2			-0.06	0.26	-0.02	.01	-.02	2.45
RelP1			0.56*	0.25	0.19	.08	.13	2.30
RelP2			0.28	0.22	0.11	0	.07	2.39
HS_z			0.04	0.06	-	.02	.04	1.11
BDS^1_z			-0.04	0.07	-	.04	-.04	1.28
$Interval_z$			-0.05	0.06	-	.04	-.05	1.05
PWB^1_z			-0.26***	0.07	-	.46	-.21	1.45
E^1_z			0.09	0.07	-	.01	.08	1.32

* $p < .05$, *** $p < .001$, $_z$ Standardized variable, [1] Time 1, C_s^2 = Squared structural coefficient, indicating how much of model R^2 is explained by predictor in isolation from other predictor variables.
Step 1: $F(13, 287) = 2.09$, $p = .039$, Step 2: $F(14, 286) = 2.08$, $p = .034$

Table 13.
Analysis of ΔPWB by EI after Breakup Control Variables

	R	R^2_{change}	B	SE	β	C_s^2	Part r	VIF
Step 1 identical to Step 1 in Table 6								
Step 2	.35	.04**						
Intercept			-0.17	0.37				
HLA1			0.13	0.15	0.09	.01	.05	1.24
HLA2			0.22	0.14	-0.10	.04	.09	1.16
WI1			-0.23	0.24	-0.02	.08	-.06	3.54
WI2			-0.04	0.24	0.01	0	-.01	3.05
WI3			0.03	0.23	-0.15	.04	.01	3.39
RelB1			-0.31	0.30	-0.03	.04	-.06	2.37
RelB2			-0.11	0.25	0.21	.01	-.03	2.47
RelP1			0.56*	0.25	0.09	.06	.13	2.30
RelP2			0.26	0.22	0.01	0	.07	2.39
HS_z			0.03	0.06	-	.01	.03	1.12
BDS^1_z			-0.01	0.07	-	.03	-.01	1.27
$Interval_z$			-0.03	0.06	-	.03	-.03	1.06
PWB^1_z			-0.40***	0.09	-	.34	-.28	2.08
TEI^1_z			0.27***	0.08	-	0	.19	2.05

* $p < .05$, ** $p < .01$, *** $p < .001$, $_z$ Standardized variable, ^{1} Time 1, C_s^2 = Squared structural coefficient, indicating how much of model R^2 is explained by predictor in isolation from other predictor variables.
Step 1: $F(13, 287) = 2.09$, $p = .039$, Step 2: $F(14, 286) = 2.87$, $p = .002$

Table 14.

Analysis of ΔG by EI after Breakup Control Variables

	R	R^2_{change}	B	SE	β	C_s^2	Part r	VIF
Step 1 identical to Step 1 in Table 8								
Step 2	.36	.02						
Intercept			0.10	0.36				
HLA1			0.06	0.15	.03	0	.03	1.19
HLA2			0.04	0.15	.02	0	.02	1.16
WI1			0.16	0.23	.07	.03	.04	3.56
WI2			0.20	0.23	.08	0	.05	3.05
WI3			0.13	0.23	.06	.01	.03	3.39
RelB1			-0.04	0.30	-.01	0	-.01	2.38
RelB2			-0.02	0.24	-.01	0	-.01	2.47
RelP1			-0.40	0.25	-.14	.02	-.09	2.32
RelP2			-0.24	0.22	-.09	0	-.06	2.41
HS_z			-0.05	0.06	-	.03	-.05	1.12
BDS^1_z			0.05	0.08	-	.19	.04	1.65
$Interval_z$			0	0.06	-	.02	0	1.06
G^1_z			-0.40***	0.08	-	.73	-.30	1.82
TEI^1_z			-0.15*	0.07	-	0	-.13	1.28

$* p < .05$, $*** p < .001$, $_z$ Standardized variable, ^{1} Time 1, C_s^2 = Squared structural coefficient, indicating how much of model R^2 is explained by predictor in isolation from other predictor variables.

Step 1: $F(13, 287) = 2.87$, $p = .004$, Step 2: $F(14, 286) = 3.11$, $p = .001$

Table 15.
Analysis of ΔeBA by EI after Breakup Control Variables

	R	R^2_{change}	B	SE	β	C_s^2	Part r	VIF
			Step 1 identical to Step 1 in Table 7					
Step 2	.28	0						
Intercept			0.19	0.36				
HLA1			0.02	0.15	.01	.03	.01	1.18
HLA2			-0.30*	0.15	-.13	.20	-.12	1.15
WI1			0.11	0.23	.05	0	.03	3.52
WI2			0.02	0.24	.01	0	.01	3.11
WI3			0.05	0.24	.02	0	.01	3.39
RelB1			-0.16	0.30	-.05	.02	-.03	2.37
RelB2			-0.10	0.25	-.04	.05	-.02	2.47
RelP1			0.08	0.26	.03	.11	.02	2.31
RelP2			-0.14	0.23	-.06	.12	-.04	2.39
HS_z			0	0.06	-	0	.00	1.10
BDS^1_z			-0.01	0.07	-	.01	-.01	1.22
$Interval_z$			0.01	0.06	-	0	.01	1.05
eBA^1_z			-0.24***	0.07	-	.61	-.22	1.18
TEI^1_z			-0.05	0.07	-	.01	-.05	1.23

* $p < .05$, *** $p < .001$, $_z$ Standardized variable, [1] Time 1, C_s^2 = Squared structural coefficient, indicating how much of model R^2 is explained by predictor in isolation from other predictor variables.
Step 1: $F(13, 287) = 1.90$, $p = .086$, Step 2: $F(14, 286) = 1.18$, $p = .104$

Table 16.
Analysis of ΔeBA by RSB after Breakup Control Variables

	R	R^2_{change}	B	SE	β	C_s^2	Part r	VIF
Step 1 identical to Step 1 in Table 7								
Step 2	.28	0						
Intercept			0.22	0.36				
HLA1			0.03	0.15	0.01	.03	.01	1.18
HLA2			-0.29*	0.15	-0.13	.20	-.12	1.15
WI1			0.10	0.23	0.05	0	.03	3.54
WI2			0.02	0.24	0.01	0	0	3.12
WI3			0.04	0.24	0.02	0	.01	3.39
RelB1			-0.16	0.30	-0.05	.02	-.03	2.38
RelB2			-0.12	0.24	-0.04	.05	-.03	2.44
RelP1			0.08	0.26	0.03	.11	.02	2.32
RelP2			-0.15	0.23	-0.06	.12	-.04	2.39
HS_z			0	0.06	-	0	0	1.10
BDS^1_z			0.01	0.07	-	.01	0	1.14
$Interval_z$			0.01	0.06	-	0	.01	1.05
eBA^1_z			-0.23***	0.06	-	.61	-.22	1.08
RSB^1_z			-0.01	0.06	-	0	-.01	1.04

* $p < .05$, *** $p < .001$, $_z$ Standardized variable, [1] Time 1, C_s^2 = Squared structural coefficient, indicating how much of model R^2 is explained by predictor in isolation from other predictor variables.
Step 1: $F(13, 287) = 1.90$, $p = .086$, Step 2: $F(14, 286) = 1.77$, $p = .118$

Table 17.

Analysis of ΔG by RSB after Breakup Control Variables

	R	R^2_{change}	B	SE	β	C_s^2	Part r	VIF
Step 1 identical to Step 1 in Table 8								
Step 2	.35	.01						
Intercept			0.13	0.36				
HLA1			0.06	0.15	0.03	0	.02	1.19
HLA2			0.05	0.15	0.02	0	.02	1.15
WI1			0.17	0.23	0.08	.04	.04	3.56
WI2			0.20	0.23	0.08	0	.05	3.07
WI3			0.12	0.23	0.06	.01	.03	3.38
RelB1			-0.08	0.30	-0.03	0	-.02	2.39
RelB2			-0.07	0.24	-0.02	0	-.02	2.45
RelP1			-0.41	0.25	-0.14	.02	-.09	2.32
RelP2			-0.23	0.22	-0.09	0	-.06	2.41
HS_z			006	0.06	-	.04	-.05	1.10
BDS^1_z			0.06	0.08	-	.20	.04	1.61
$Interval_z$			0.01	0.06	-	.02	.01	1.05
G^1_z			-0.35***	0.07	-	.77	-.28	1.59
RSB^1_z			0.07	0.06	-	.03	.07	1.04

*** $p < .001$, $_z$ Standardized variable, [1] Time 1, C_s^2 = Squared structural coefficient, indicating how much of model R^2 is explained by predictor in isolation from other predictor variables.

Step 1: $F(13, 287) = 2.87$, $p = .004$, Step 2: $F(14, 286) = 2.80$, $p = .003$

Table 18.
Analysis of ΔPWB by RSB after Breakup Control Variables

	R	R^2_{change}	B	SE	β	C_s^2	Part r	VIF
Step 1 identical to Step 1 in Table 6								
Step 2	.29	0						
Intercept			-0.25	0.38				
HLA1			0.05	0.15	.02	.01	.02	1.21
HLA2			0.18	0.14	.08	.06	.07	1.15
WI1			-0.19	0.24	-.09	.12	-.05	3.54
WI2			0.02	0.24	.01	.01	0	3.07
WI3			0.08	0.24	.04	.06	.02	3.38
RelB1			-0.28	0.31	-.08	.05	-.06	2.37
RelB2			-0.05	0.26	-.02	.02	-.01	2.46
RelP1			0.55	0.26	.19	.09	.12	2.31
RelP2			0.28	0.22	.11	0	.07	2.39
HS_z			0.04	0.07	-	.02	.04	1.11
BDS^1_z			-0.03	0.07	-	.04	-.03	1.25
$Interval_z$			-0.05	0.06	-	.04	-.05	1.04
PWB^1_z			-0.22***	0.06	-	.49	-.20	1.16
RSB^1_z			0.01	0.06	-	0	.01	1.05

*** $p < .001$, $_z$ Standardized variable, [1] Time 1, C_s^2 = Squared structural coefficient, indicating how much of model R^2 is explained by predictor in isolation from other predictor variables.
Step 1: $F(13, 287) = 2.09$, $p = .039$, Step 2: $F(14, 286) = 1.95$, $p = .056$

Table 19.
Analysis of ΔPWB by N after Breakup Control Variables

	R	R^2_{change}	B	SE	β	C_s^2	Part r	VIF
Step 1 identical to Step 1 in Table 6								
Step 2	.31	.01						
Intercept			-0.23	0.37				
HLA1			0.05	0.15	0.02	.01	.02	1.20
HLA2			0.18	0.14	0.08	.05	.07	1.15
WI1			-0.20	0.24	-0.09	.10	-.05	3.54
WI2			0.01	0.24	0.01	.01	0	3.04
WI3			0.07	0.24	0.03	.05	.02	3.38
RelB1			-0.29	0.30	-0.09	.05	-.06	2.37
RelB2			-0.07	0.26	-0.02	.01	-.02	2.46
RelP1			0.56*	0.25	0.19	.08	.13	2.30
RelP2			0.27	0.22	0.11	0	.07	2.39
HS_z			0.04	0.06	-	.02	.04	1.11
BDS^1_z			-0.01	0.07	-	.04	-.01	1.32
$Interval_z$			-0.05	0.06	-	.04	-.04	1.05
PWB^1_z			-0.26***	0.07	-	.43	-.23	1.29
N^1_z			-.12	.07	-	0	.10	1.27

* $p < .05$, *** $p < .001$, $_z$ Standardized variable, [1] Time 1, C_s^2 = Squared structural coefficient, indicating how much of model R^2 is explained by predictor in isolation from other predictor variables.
Step 1: $F_{(13, 287)} = 2.09$, $p = .039$, Step 2: $F_{(14, 286)} = 2.21$, $p = .023$

Table 20.
Analysis of ΔeBA by N after Breakup Control Variables

	R	R^2_{change}	B	SE	β	C_s^2	Part r	VIF
Step 1 identical to Step 1 in Table 7								
Step 2	.29	0						
Intercept			0.20	0.36				
HLA1			0.03	0.15	0.01	.03	.01	1.18
HLA2			-0.29*	0.15	-0.13	.19	-.12	1.15
WI1			0.11	0.23	0.05	0	.03	3.52
WI2			0.02	0.24	0.01	0	.01	3.11
WI3			0.05	0.24	0.02	0	.01	3.38
RelB1			-0.16	0.30	-0.05	.01	-.03	2.37
RelB2			-0.10	0.24	-0.04	.04	-.03	2.45
RelP1			0.08	0.26	0.03	.11	.02	2.31
RelP2			-0.14	0.22	-0.06	.11	-.04	2.39
HS_z			0	0.06	-	0	0	1.10
BDS^1_z			-0.01	0.07	-	.01	-.01	1.25
$Interval_z$			0.01	0.06	-	0	.01	1.05
eBA^1_z			-0.23***	0.06	-	.57	-.22	1.09
N^1_z			.06	.07	-	.01	.06	1.14

* $p < .05$, *** $p < .001$, $_z$ Standardized variable, [1] Time 1, C_s^2 = Squared structural coefficient, indicating how much of model R^2 is explained by predictor in isolation from other predictor variables.
Step 1: $F(13, 287) = 1.90$, $p = .086$, Step 2: $F(14, 286) = 1.84$, $p = .096$

Table 21.
Analysis of ΔG by N after Breakup Control Variables

	R	R^2_{change}	B	SE	β	C_s^2	Part r	VIF
Step 1 identical to Step 1 in Table 8								
Step 2	.34	0						
Intercept			0.14	.36				
HLA1			0.07	.15	0.03	0	.03	1.19
HLA2			0.06	.15	0.02	0	.02	1.15
WI1			0.15	.23	0.07	.04	.04	3.54
WI2			0.18	.23	0.08	0	.04	3.05
WI3			0.11	.23	0.05	.01	.03	3.37
RelB1			-0.06	.30	-0.02	0	-.01	2.38
RelB2			-0.07	.24	-0.03	0	-.02	2.46
RelP1			-0.40	.25	-0.13	.02	-.09	2.32
RelP2			-0.24	.22	-0.09	0	-.06	2.41
HS_z			-0.06	.06	-	.04	-.05	1.10
BDS^1_z			0.05	.08	-	.21	.04	1.69
$Interval_z$			0.01	0.06	-	.02	.01	1.05
G^1_z			-0.35***	0.08	-	.82	-.28	1.61
N^1_z			0.03	0.06	-	.02	-.03	1.15

*** $p < .001$, $_z$ Standardized variable, [1] Time 1, C_s^2 = Squared structural coefficient, indicating how much of model R^2 is explained by predictor in isolation from other predictor variables.
Step 1: $F(13, 287) = 2.87$, $p = .004$, Step 2: $F(14, 286) = 2.69$, $p = .007$

Table 22.
Analysis of ΔeBA by E, EI, N, RSB, and Gender without (A) and with Breakup Control Variables (B)

Model A	R	R^2_{change}	B	SE	β	C_s^2	Part r	VIF
Step 1	.22	.05***						
Intercept			$-4.863e^{-17}$	0.06				
eBA^1_z			-0.22	0.06	-	.99	-.22	1.00
Step 2	.25	.01						
Intercept			0.12	0.10				
eBA^1_z			-0.24***	0.06	=	.77	-0.23	1.14
Gender			-0.18	0.13	-0.08	.03	-0.08	1.09
RSB^1_z			0	0.06	=	0	0	1.01
E^1_z			0.04	0.07	=	.04	0.03	1.27
N^1_z			-0.06	0.07	=	.02	-0.05	1.51
EI^1_z			0	0.08	=	.01	0	1.77

Model B	R	R^2_{change}	B	SE	β	C_s^2	Part r	VIF

Step 1 identical to Step 1 in Table 7

Model B	R	R^2_{change}	B	SE	β	C_s^2	Part r	VIF
Step 2	.30	.01						
Intercept			0.33	0.38				
HLA1			0.01	0.15	0	.02	0	1.23
HLA2			-0.29	0.15	-0.13	.18	-.12	1.19
WI1			0.13	0.24	0.06	0	.03	3.56
WI2			-0.01	0.24	0	0	0	3.13
WI3			0.05	0.24	0.02	0	.01	3.42
RelB1			-0.13	0.30	-0.04	.01	-.03	2.39
RelB2			-0.10	0.25	-0.04	.04	-.02	2.47
RelP1			0.07	0.26	0.02	.10	.02	2.32
RelP2			-0.16	0.23	-0.06	.11	-.04	2.40
HS_z			0.01	0.06	-	0	.01	1.12
BDS^1_z			0-.01	0.07	-	.01	-.01	1.28
$Interval_z$			0.01	0.06	-	.00	.01	1.05
eBA^1_z			-0.25***	0.07	-	.53	-.23	1.23
Gender			-0.20	0.14	-	.02	-.09	1.16
RSB^1_z			-0.01	0.06	-	0	-.01	1.05
E^1_z			-0.03	0.07	-	.02	.02	1.35
N^1_z			0.06	0.08	-	.01	-.05	1.60
EI^1_z			0	0.08	-	.01	0	1.86

*** $p < .001$, z Standardized variable, [1] Time 1, C_s^2 = Squared structural coefficient, indicating how much of model R^2 is explained by predictor in isolation from other predictor variables.
Model A: Step 1: $F(1, 299) = 15.21$, $p < .001$, Step 2: $F(6, 294) = 3.25$, $p = .017$
Model B: Step 1: $F(13, 287) = 1.90$, $p = .086$, Step 2: $F(18, 282) = 1.61$, $p = .159$

Table 23.

MR Analysis of ΔG explained by E, EI, N, RSB, and Gender without (A) and with Breakup Control Variables (B)

Model A	R	R^2_{change}	B	SE	β	C_s^2	Part r	VIF
Step 1	.31	.09***						
Intercept			$5.355e^{-17}$	0.05				
G^1_z			-0.31	0.06	-	.99	-.31	1.00
Step 2	.36	.03						
Intercept			0.12	0.10				
G^1_z			-0.37	0.06	-	0.74	-.33	1.27
Gender			-0.18	0.13	-0.08	0.04	-.08	1.07
RSB^1_z			0.07	0.06	-	0.02	.07	1.01
E^1_z			-0.05	0.07	-	0.06	.04	1.30
N^1_z			-0.01	0.07	-	0.01	.01	1.52
EI^1_z			-0.13	0.08	-	0	-.10	1.88

Model B	R	R^2_{change}	B	SE	β	C_s^2	Part r	VIF

Step 1 identical to Step 1 in Table 8

	R	R^2_{change}	B	SE	β	C_s^2	Part r	VIF
Step 2	.39	.03						
Intercept			0.18	0.38				
HLA1			0.05	0.15	0.02	0	.02	1.24
HLA2			0.06	0.15	0.03	0	.02	1.18
WI1			0.20	0.23	0.09	.03	.05	3.58
WI2			0.20	0.23	0.09	0	.05	3.08
WI3			0.16	0.23	0.07	.01	.04	3.41
RelB1			-0.04	0.30	-0.01	0	-.01	2.40
RelB2			-0.01	0.24	0	0	0	2.48
RelP1			-0.42	0.25	-0.14	.01	-.09	2.33
RelP2			-0.25	0.22	-0.10	0	-.06	2.41
HS_z			-0.04	0.06	-	.03	-.04	1.12
BDS^1_z			0.06	0.08	-	.16	.04	1.69
$Interval_z$			0	0.06	-	.02	0	1.06
G^1_z			-0.40***	0.08	-	.62	-.30	1.84
Gender			-0.16	0.14	-0.07	.04	-.07	1.13
RSB^1_z			0.08	0.06	-	.02	.08	1.05
E^1_z			-0.06	0.07	-	.05	.05	1.37
N^1_z			-0.03	0.07	-	.01	.02	1.60
EI^1_z			-0.13	0.08	-	0	-.09	1.95

*** $p < .001$, $_z$ Standardized variable, [1] Time 1, C_s^2 = Squared structural coefficient, indicating how much of model R^2 is explained by predictor in isolation from other predictor variables.

Model A: Step 1: $F(1, 299) = 31.87$, $p < .001$; Step 2: $F(18, 294) = 7.43$, $p < .001$

Model B: Step 1: $F(13, 282) = 2.87$, $p = .004$; Step 2: $F(18, 282) = 2.74$, $p = .001$

Table 24.
MR Analysis of ΔPWB explained by E, EI, N, RSB, and Gender without (A) and with Breakup Control Variables (B)

Model A	R	R^2_{change}	B	SE	β	C_s^2	Part r	VIF
Step 1	.20	.04**						
Intercept			$-1.024e^{-16}$	0.06				
PWB^1_z			-0.20	0.06	-	0	-.20	1.00
Step 2	.29	.04						
Intercept			-0.03	0.10				
PWB^1_z			-0.40***	0.08	-	.50	-.28	2.02
Gender			0.04	0.13	0.02	0	.02	1.06
RSB^1_z			0.02	0.06	-	0	.02	1.02
E^1_z			0.05	0.07	-	.01	-.04	1.34
N^1_z			-0.03	0.07	-	0	.02	1.51
EI^1_z			0.24**	0.09	-	0	.15	2.32

Model B	R	R^2_{change}	B	SE	β	C_s^2	Part r	VIF
			Step 1 identical to Step 1 in Table 6					
Step 2	.36	.04						
Intercept			-0.23	0.38				
HLA1			0.13	0.15	0.06	.01	.05	1.29
HLA2			0.21	0.14	0.09	.04	.08	1.19
WI1			-0.23	0.24	-0.11	.08	-.06	3.57
WI2			-0.02	0.24	-0.01	0	-.01	3.08
WI3			0.02	0.24	0.01	.04	.01	3.42
RelB1			-0.33	0.30	-0.10	.03	-.07	2.39
RelB2			-0.11	0.25	-0.04	.01	-.03	2.47
RelP1			0.56*	0.25	0.19	.06	.13	2.31
RelP2			0.27	0.22	0.11	0	.07	2.40
HS_z			0.03	0.06	-	.01	.03	1.12
BDS^1_z			-0.01	0.07	-	.03	-.01	1.34
$Interval_z$			-0.03	0.06	-	.03	-.03	1.05
PWB^1_z			-0.42***	0.09	-	.32	-.28	2.22
Gender			0.07	0.13	0.03	0	.03	1.13
RSB^1_z			0.02	0.06	-	0	.02	1.06
E^1_z			0.03	0.07	-	0	-.03	1.43
N^1_z			-0.04	0.07	-	0	.03	1.60
EI^1_z			0.25**	0.09	-	0	.16	2.45

* $p < .05$, ** $p < .01$, *** $p < .001$, $_z$ Standardized variable, [1] Time 1, C_s^2 = Squared structural coefficient, indicating how much of model R^2 is explained by predictor in isolation from other predictor variables.

Model A: Step 1: $F(1, 299) = 13.26$, $p = .001$; Step 2: $F(18, 294) = 4.48$, $p < .001$

Model B: Step 1: $F(13, 287) = 2.09$, $p = .039$; Step 2: $F(18, 282) = 2.30$, $p = .007$

Figure 1.

Missing value analysis breakdown of frequency and proportion of missing values in original dataset. Variables indicates the number of variables that have missing data, Cases indicates the number of participants with missing values, and Values indicates how much data across the entirety of the dataset is missing.
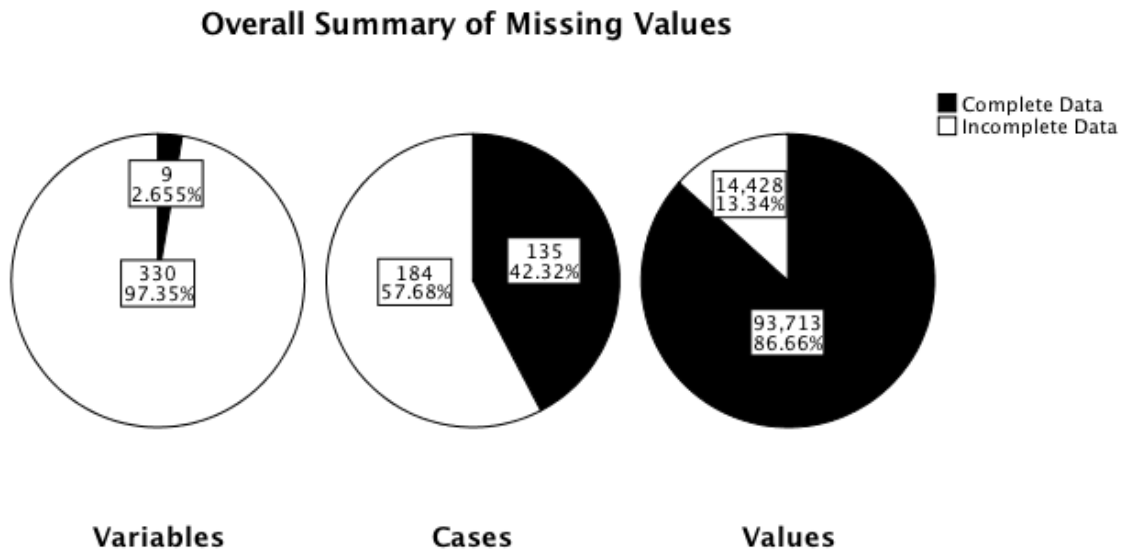
Figure 2.

A typical example from analysis of inspection of residual normality assumption for MR analysis for one imputation in a model with criterion ΔeBA. A close fit between residuals and the normal fit line indicates the assumption of residual normality is reasonably upheld.

Normal P–P Plot of Regression Standardized Residual
Dependent Variable: Zscore(Delta_eBA)

Imputation Number: 1
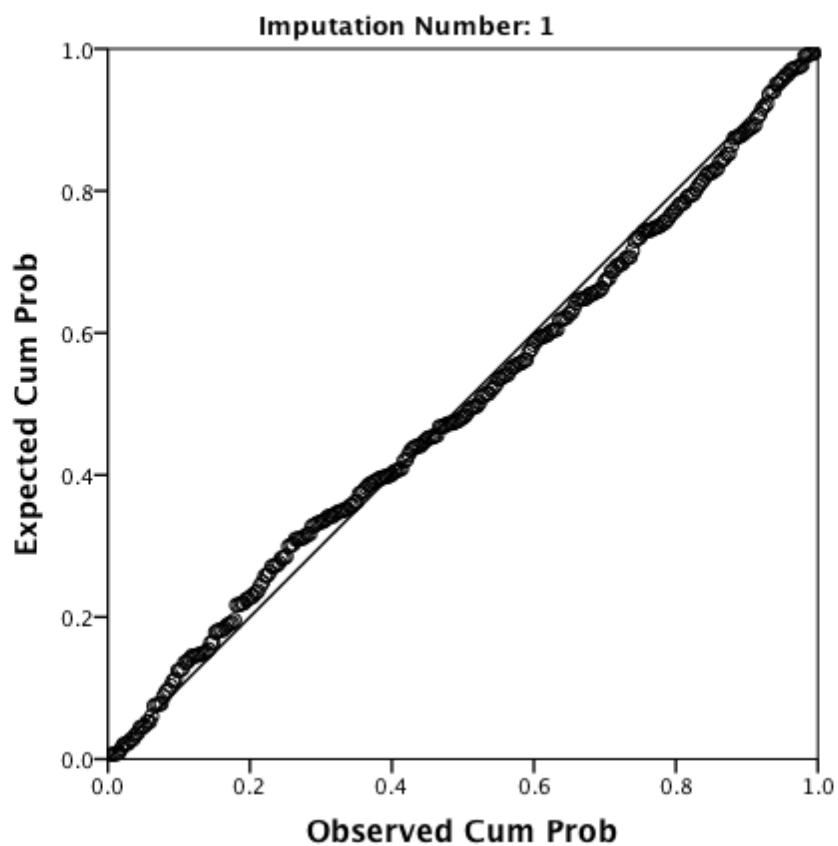
Figure 3.

Typical example from analysis of a test of homoscedasticity for a single imputation set within a model with criterion ΔG. A horizontal fit line indicates the residuals are homoscedastic across predicted criterion values.

Scatterplot
Dependent Variable: Zscore(Delta_G)
Imputation Number: 19

R^2 Linear = -6.661E-16

$y=-5.42E-17+-5.74E-16*x$

Regression Standardized Residual

Regression Standardized Predicted Value
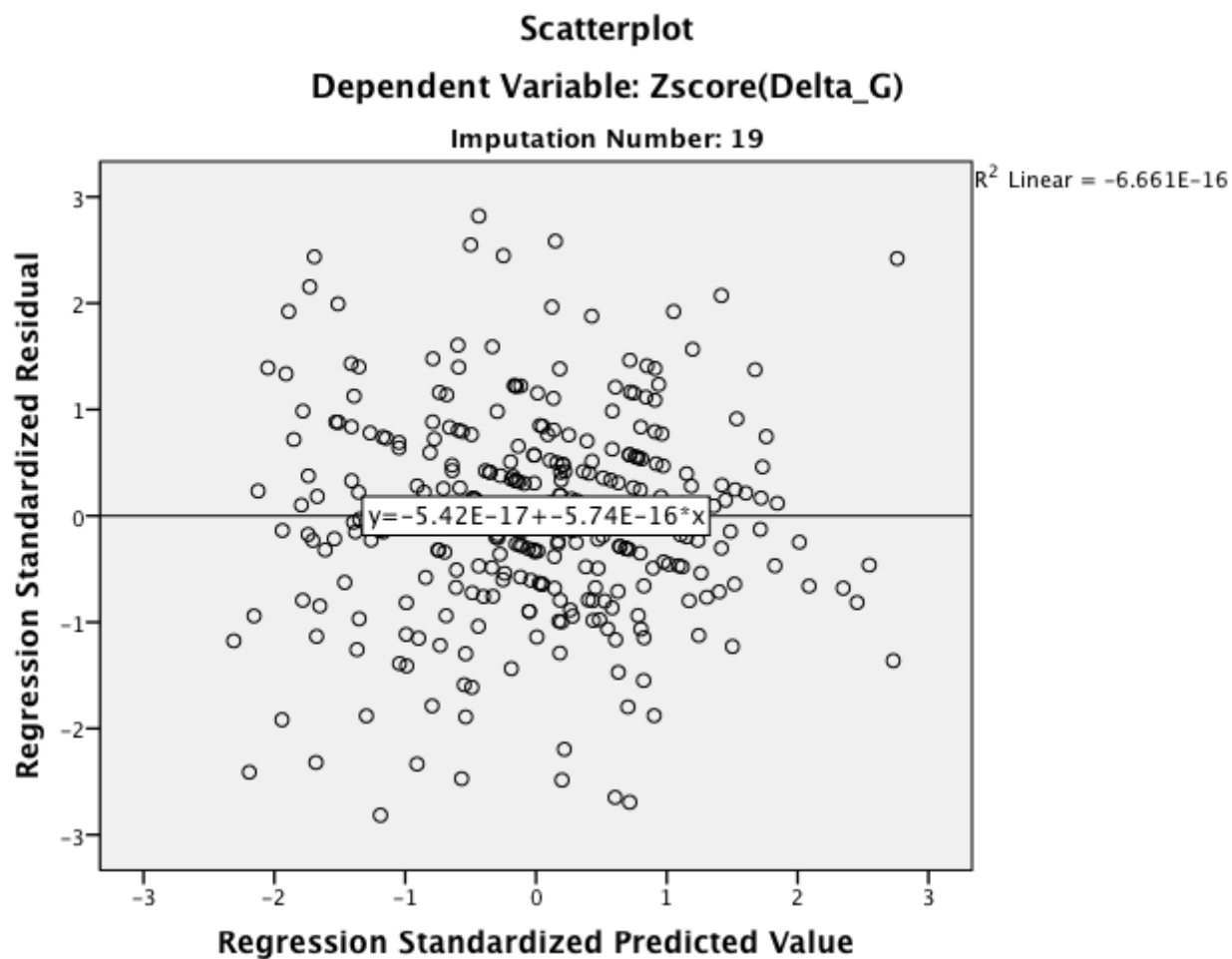
APPENDIX A

Demographics

1. Age (years): _____

2. Gender: _____

4. How many serious romantic relationships have you been in? _____

5. How important do you feel having a romantic partner is?

Not at all Important   Very
                                        Important
    1 . . . . 2 . . . . 3 . . . . 4 . . . . 5 . . . . 6 7

The following section focuses on the romantic relationship that ended 3 or less months ago.

6. How long ago did the relationship end?	< 1 month	4-8 weeks	8-12 weeks	12+ weeks

7. Based on the above scale, how serious was this relationship (please circle a value along the scale)?

Very Casual   Very
                                        Serious
    1 . . . . 2 . . . . 3 . . . . 4 . . . . 5 . . . . 6 7

8. Who initiated ending the relationship (please select one)?

Me	My partner	Both my partner and I	Neither my partner nor I	Uncertain

9. Have you been in another romantic relationship since the breakup in question?	Yes	No	Uncertain

Curriculum Vitae

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PUBLICATIONS AND CONFERENCES

➤ Tohver, G. C. & Smith, M. M. (June, 2014). Forgive or Forget? Perfectionistic Concerns, Propensity for Forgiveness, and the mediating role of Emotional Intelligence. Paper presented at the Canadian Psychological Association's 75th Annual Convention, Vancouver, British Columbia.

➤ Tohver, G. C. & Saklofske, D. H. (June, 2014). Rebound: Re-modeling personality, belief, and gender effects on blame recovery and psychological well-being after romantic breakup. Paper presented at the Canadian Psychological Association's 75th Annual Convention, Vancouver, British Columbia.

➤ Tohver, G. C. (May 2014). *Profiling Blame Recovery*. Roundtable lead at the Trent-Western Psychology Research Forum.

➤ Austin, E. J., Saklofske, D. H., **Smith, M. M.,** & Tohver, G. (in press). Associations of the Managing the Emotions of Others (MEOS) scale with personality, the Dark Triad, and trait EI. *Personality and Individual Differences*.

➤ Tohver, G. & Saklofske, D. H. (2013). Pointing the finger: Introversion-extraversion, emotional intelligence, and neuroticism relations to judgments of transgressor blameworthiness. Paper (to be) presented at the International Society for the Study of Individual Differences Biannual Conference, Barcelona, Spain.

➤ Tohver, G., Veselka, L., & Vernon, P. A. (2013) Conditions of Blame: Does Intelligence and Excuse Complexity Affect Blame Attributions? Paper (to be) presented at the Canadian Psychological Association's 74th Annual Convention, Quebec City, Quebec.

➤ Tohver, G. & Saklofske, D. H. (2013). Blame recovery: Modeling the effects of personality, age, and gender on blame attributions and psychological well-being after a failed romantic relationship. Paper (to be) presented at the Canadian Psychological Association's 74th Annual Convention, Quebec City, Quebec.

EXPERIENCE & PROFESSIONAL WORKSHOPS

THESIS CO-SUPERVISOR   September 2013 – May 2014
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CROSS-CULTURAL COMMUNICATION WORKSHOP   November 2013
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HUMAN RESEARCH ETHICS COMMITTEE   October 2013 – May 2014
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S.O.G.S. PSYCHOLOGY COUNCILOR   2012 - 2013
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GRADUATE STUDENT CONFERENCE ON TEACHING   September 2012
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RESEARCH ASSISTANT   2010 - 2012
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ACHIEVEMENTS

- ➢ Ontario Graduate Scholarship, 2013 - 2015 — $30000 ($15000 pending for Sept. 2014 – Aug. 2015)
- ➢ University of Western Ontario, Angela Armitt Award, 2012
- ➢ Social Sciences and Humanities Research Council of Canada Scholarship, 2012 — $17500
- ➢ University of Western Ontario, Faculty Association Scholarship, 2011 -- $900
- ➢ Queen Elizabeth II Scholarship, 2006 – 2010 -- $13076
- ➢ University of Western Ontario, Social Science Dean's Honour List, 2007-2012