

Wagner, Ernest H., Hoelterhoff, Mark and Chung, Man Cheung (2017) Posttraumatic stress disorder following asthma attack: the role of agency beliefs in mediating psychiatric morbidity. *Journal of Mental Health*, 26 (4). pp. 342-350.

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## Posttraumatic Stress Disorder following asthma attack: The role of agency beliefs in mediating psychiatric morbidity

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Manuscript total word count not including abstract, tables, or references:

**3983 words (excluding table insertion instructions), or 3999 for whole document**

## Abstract

### Background

The link between serious illness and subsequent Posttraumatic stress disorder (PTSD) and psychiatric comorbidity has been established. In populations with asthma, however, few studies have investigated this link, or what psychological mechanisms mediate it. Healthcare guidance for chronic conditions, and PTSD literature, highlight ‘agency beliefs’ as a direction for investigation.

### Aims

To determine the prevalence of PTSD following asthma attack, and investigate whether agency beliefs mediate PTSD and comorbid psychiatric symptoms in this population.

### Method

We recruited 110 adults with asthma from online peer support forums. Participants completed the Asthma Symptom Checklist, PTSD Checklist, GHQ-28, General Self-Efficacy scale, and Multidimensional Health Locus of Control scale.

### Results

20% of our sample met criteria for PTSD. Regression results indicated that higher asthma severity significantly predicted PTSD and psychiatric co-morbidity. Lower self-efficacy significantly predicted PTSD symptoms while controlling for asthma severity, however Locus of Control (LoC) did not improve the model further. Self-efficacy, but not LoC, significantly partially mediated the effect of asthma severity on PTSD severity and psychiatric co-morbidity.

### Conclusions

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PTSD and other psychiatric symptoms in asthma populations are mediated in part by self-efficacy. Safeguarding and improving self-efficacy in this population is an important area for future research and intervention.

### **Declaration of interest**

The authors have no conflict of interest to declare.

Key words: PTSD, psychiatric co-morbidity, Self-efficacy, Locus of Control, chronic illness, agentic beliefs

## 1. Introduction

The link between Posttraumatic stress disorder (PTSD) and chronic or life-threatening medical illness has been established, with PTSD prevalence ranging up to 64% in these populations (Tedstone and Tarrier, 2003; Wu et al., 2005; Griffiths et al., 2007). In contrast, the general population has a lifetime prevalence of 1-12% (Stein et al., 1997; Bronner et al., 2009). Further understanding of what psychological mechanisms might explain PTSD following serious illness is important for intervention and/or prevention. Furthermore, addressing the mental health of those with chronic health conditions can increase wellbeing and quality of life, whilst reducing physical symptoms and overall healthcare expenditure (Naylor et al., 2012).

One such illness with increased likelihood of PTSD, is Asthma - a chronic disease affecting 235 million people worldwide (WHO, 2013). The experience of acute asthma symptoms can create feelings of helplessness and uncertainty (Trollvik and Severinsson, 2004), and anxiety and fear (Cooper et al., 2007). Yet few studies have specifically investigated PTSD in conjunction with asthma. One study reports PTSD prevalence in adults with asthma to be 3% for full PTSD, and 44% for partial PTSD (Chung et al., 2012), while another suggests 20% meet criteria for full PTSD (Kean et al., 2006).

In addition to PTSD, asthma is associated with increased risk of psychiatric disorders such as depression and anxiety disorders (Goodwin et al., 2004), and panic disorder (J.M. Feldman et al., 2009). The mechanism of association between asthma and psychiatric

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symptoms is likely complex, and may be explained by a variety of factors (Goodwin et al., 2004).

There is a bidirectional relationship between psychiatric functioning and asthma (Chida et al., 2008) whereby stress-induced immune system dysfunction can increase vulnerability to asthma (Alonso et al., 2014), meanwhile severe asthma can lead to psychiatric symptoms in later life (Goodwin et al., 2013). Thus, there is both an effect of psychosocial stress on health, while poor health can negatively impact psychological functioning. As the latter is the current focus, we constrain our interest to factors which might explain this relationship.

Asthma's impact on psychological functioning might be related to alexithymia (Chung & Wall, 2013), decreased feelings of control (Adams et al., 2004), and coping and social support (Lind et al., 2015). However, few studies investigate psychological outcomes, and instead investigate the impact of psychological functioning on health outcomes in asthma (Yorke et al., 2006). Still, preliminary evidence points toward wellbeing and quality of life benefits from a range of psychological interventions for chronic health conditions. These include cognitive and behavioural approaches, relaxation, bio-feedback, and psycho-education (Smith & Jones, 2015). However, the three systematic reviews in the past decade failed to find firm evidence for any one intervention due to small studies and suboptimal methods (see Smith and Jones, 2015).

In contrast, systematic reviews strongly support the health and quality of life benefits of supported self-care and self-management for asthma in routine healthcare. (Pinnock, 2015; Taylor et al., 2014). A central aspect of self-management in chronic illness is 'perceived control'. Decreased perceived control is common in people with asthma and is associated

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with health outcomes (Adams et al., 2004), and both Marks et al. (2005) and Taylor et al. (2014) suggest an important aim of self-management of chronic illness is to enhance the agency/control construct ‘self-efficacy’.

Perceived control is a heterogeneous concept with many different names and related concepts (Skinner, 1996). We will use the term ‘agency beliefs’ since we believe it to be an umbrella term which encompasses the idea of self-efficacy in Bandura’s (2006) theory of human agency, while still including other terms such as Locus of Control.

We will investigate established constructs related to agency beliefs, namely self-efficacy and Locus of Control (LoC), which have been linked to psychological outcome following trauma (Arnstein et al., 1999; Benight et al., 1999; Maciejewski, 2000; Benight and Bandura, 2004). That is, people’s beliefs about whether they have an important role to play in their own perception, experience, and response to adverse events, has been shown to explain the relationship between trauma and subsequent PTSD and psychiatric co-morbidity following trauma.

Self-efficacy, which stems from social-cognitive theory (Benight and Bandura, 2004), is a person’s belief in their own agentic ability to adequately manage and control events that impact their life (Bandura, 1977, 1982, 1989, 1993). Self-efficacy is suggested to influence cognitive processes such as emotion and stress, motivation, goal setting, and decision making, which in turn affect mental and physical health (Bandura, 1994).

Multi-dimensional Health Locus of control (LoC) is a similar, yet theoretically distinct concept, which describes a person’s belief about whether a health outcome is contingent on his or her own behaviour, or on external causes such as chance or powerful-

others (Rotter, 1975, 1990). Founded in social learning theory, health-LoC describes a learned orientation of belief about the cause of a person's health status (Wallston et al., 1994).

Previous studies found that high self-efficacy and internal LoC are related to resilience in both mental and physical health outcomes (Strickland, 1978; Strecher et al., 1986; Weiss and Larsen, 1990; Bandura, 1993; Wu et al., 2004; Shelley and Pakenham, 2004; Zajacova et al., 2005; Marks et al., 2005; Cross et al., 2006; Leontopoulou, 2006; Cohen et al., 2008; Waldron et al., 2010; Craig et al., 2011; Oliveira et al., 2012). Specifically in regard to PTSD, self-efficacy and LoC are significantly associated with individual differences in pathology and posttraumatic recovery (Benight and Bandura, 2004; Heinrichs et al., 2005; Chung et al., 2006, 2007, 2012; Jones et al., 2006; Mak et al., 2010).

Accordingly, this study aims to further understand the relationship between asthma and subsequent PTSD and psychiatric co-morbidity, by investigating constructs related to agency beliefs. We propose that the relationship between asthma and psychiatric outcome could be partially explained by a reduction in a personal sense of meaningful control over important life events.

We will determine the prevalence of PTSD in our population, whether there is a relationship between asthma severity, and outcomes of PTSD and psychiatric co-morbidity, and whether self-efficacy or locus of control will mediate this association. We hypothesise that self-efficacy, and LoC, will partially explain the relationship between asthma symptoms and PTSD and co-morbid psychiatric symptoms following asthma attack.

## 2. Methods

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### *Participants*

110 people who had experienced asthma attack completed the study. Participants were recruited through online asthma support forums, social networks, and email, according to three main inclusion criteria: a) must be 18 years or older, b) have a formal diagnosis of asthma, and c) have had an asthma attack or serious asthma episode which can be recalled to memory. Potential participants were directed to a website created for the study, which included study information, and an informed consent page where an ‘I agree’ button was clicked to proceed. 433 unique visitors viewed the study’s webpage, and 153 gave consented; 43 questionnaires were removed due to missing data for one or more entire measure, resulting in 110 complete responses.

### *Measures*

The Asthma Symptom Checklist (ASC; Kinsman et al., 1973) is a self-report measure of the subjective frequency of asthma symptoms on a five-point scale from ‘never’ to ‘always’. We used the 36 item version, which has been thoroughly validated (Lemaigre et al., 2005; Cooper et al., 2007; De Peuter et al., 2007; Deshmukh et al., 2008; Ritz et al., 2008; Jonathan M Feldman et al., 2009; Meuret and Ritz, 2010; Chung et al., 2012), and demonstrated good reliability in the current sample for all five subscales including hyperventilation ( $\alpha=.82$ ), bronchoconstriction ( $\alpha=.88$ ), irritability ( $\alpha=.87$ ), panic/fear ( $\alpha=.94$ ), and fatigue ( $\alpha=.94$ ).

The PTSD Checklist (PCL; Blanchard, 1996; Ruggiero et al., 2003) is a 17-item self-report measure which assesses symptom clusters of posttraumatic stress in line with the DSM-IV (current at time of use). In relation to their asthma attack, participants rate symptoms on a five-point scale from ‘not at all’ to ‘extremely’. The PCL is a widely-used and

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well validated screening and diagnostic measure of PTSD (Smith et al., 1999; Walker et al., 2002; Grubaugh et al., 2007), with good reliability in the current sample for all three subscales including re-experiencing ( $\alpha=.83$ ), avoidance ( $\alpha=.85$ ), and hyperarousal ( $\alpha=.87$ ).

The General Health Questionnaire (GHQ-28; Goldberg and Hillier, 1979) is a 28-item self-report questionnaire which measures psychiatric co-morbidity on a four-point scale. The GHQ scoring method for screening (0-0-1-1) was used for the current study. Overall the GHQ-28 is a well validated and widely used measure of general health (Banks and Clegg, 1980; Banks, 1983; Bridges and Goldberg, 1986; Lobo et al., 1986; Goldberg et al., 1997; Makowska and Merecz, 2002; Gibbons et al., 2004; Willmott et al., 2004, 2008), and showed good reliability in our sample for the four subscales, somatic symptoms ( $\alpha=.80$ ), anxiety ( $\alpha=.83$ ), social dysfunction ( $\alpha=.83$ ), and severe depression ( $\alpha=.82$ ).

The General Self-efficacy scale (Schwarzer et al., 1999) (GSE) consists of ten items, has been validated cross-culturally and for internet use (Schwarzer et al., 1999; Scholz et al., 2002; Luszczynska et al., 2005a, 2005b), and has very good internal consistency ( $\alpha=.94$ ) in the current sample.

The Multidimensional Health Locus of Control (Wallston et al., 1978) (LOC) is an 18-item measure, which has been validated (Winefield, 1982; Kuwahara et al., 2004; Wallston, 2005a, 2005b), and in our sample has good reliability for Powerful-others-LoC ( $\alpha=.78$ ), and moderate for the Internal-LoC ( $\alpha=.64$ ) and Chance-LoC ( $\alpha=.66$ ) scales.

## *Analysis*

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Of the data for 110 participants in the analysis, only 1% of data was missing, which was addressed using Estimation Maximisation (EM).

Our power calculation assumes analysis by multiple regression. With a given sample size of 110 and alpha set at 0.05, the study will have a power of 0.95 (critical  $t=1.65$ ). This was selected as the medium effect ( $f^2=0.10$ ) that would be important to detect, in the sense that any smaller effect would not be clinically or substantively significant. It is also assumed that this effect size is reasonable, in the sense that an effect of this magnitude could be anticipated in this field of research.

Due to the variety of sources recruited from, we tested homogeneity of sample demographics for all measures. We then calculated PTSD prevalence, and compared differences in all variables of interest between the PTSD and no-PTSD groups.

A correlation matrix was used to initially investigate relationships between variables. Following this, we computed regression models to find whether self-efficacy or LoC would explain variance in either PTSD severity or co-morbid psychiatric symptoms, after controlling for asthma symptom severity.

PROCESS, a modelling tool for mediation and moderation analysis, was used to explore the mediation effects (Hayes, 2013). PROCESS is a computational macro combining the functions of statistical tools from INDIRECT, SOBEL, MODPROBE, MODMED, RSQUARE and MBESS. Bias-corrected bootstrapping is used to generate confidence intervals (Preacher & Hayes, 2008) which address the problem of power resulting from the asymmetric and non-normal sampling distributions of an indirect effect (MacKinnon et al, 2004). The bootstrapping sampling ( $n=1000$ ) distributions of the indirect effects are

produced by selecting a sample of cases from the complete data set and calculating the indirect effects in the re-samples. Point estimates and confidence intervals (95%) are estimated for the indirect effects. When zero is not contained in the confidence interval, point estimates of indirect effects are then considered to be significant.

### 3. Results

#### *Homogeneity of sample*

Analyses were performed to find differences according to gender or country group (see Table 1). For gender, the Levenes test indicated homogeneity of variance for all measures. Independent samples t-tests revealed no significant differences between genders on any of the five scales. Between the three country groups, the Levenes test confirmed homogeneity of variance on all measures. The between-group NOVA was only significant for internal LoC ( $F(2, 107) = 5.71, p < .01$ ). Tukey Post hoc comparison revealed that Internal-LoC for the 'U.S.&Canada' was significantly higher than the 'UK' ( $MD=2.78, p < .01$ ). However there were no significant differences between either Europe&other and UK, or Europe&other and U.S.&Canada groups.

#### *Prevalence of Posttraumatic Stress Disorder*

As shown in Table 1, 20.0% of participants met criteria for full PTSD using PCL scoring (Smith et al., 1999; Walker et al., 2002; Grubaugh et al., 2007). The remaining 80% may have up to two symptoms, but did not meet diagnostic criteria for PTSD.

(insert Table 1 and 2)

Comparing PTSD and no-PTSD groups (Table 2), the Levenes test indicated homogeneity of variance for asthma severity, while psychiatric co-morbidity was marginally significant ( $p=.050$ ), so equal variance was not assumed. Independent samples t-tests indicated that the PTSD group had significantly higher asthma severity scores ( $M=127.92$ ,  $SD= 21.31$ ) than the no-PTSD group( $M=91.46$ ,  $SD = 23.84$ ), and also significantly higher scores of psychiatric co-morbidity in the PTSD group( $M=12.01$ ,  $SD=6.74$ ), than the no-PTSD group( $M=4.51$ ,  $SD=5.05$ ). This suggests that those with PTSD also tend to report worse asthma symptoms and mental health problems than those without PTSD.

Comparing PTSD and no-PTSD groups (Table 2) in regard to self-efficacy and LOC, Levenes tests confirmed homogeneity of variance for all scales. Independent samples t-tests indicated that the PTSD group had significantly lower self-efficacy ( $M=26.45$ ,  $SD=6.24$ ) compared to no-PTSD ( $M=31.59$ ,  $SD=5.26$ ), and significantly higher PHLC in the PTSD group( $M=19.30$ ,  $SD=6.23$ ) than the no PTSD group( $M=15.76$ ,  $SD=5.34$ ). However, no significant difference was found between groups for IHLC, or CHLC measures.

(Insert Table 3)

A two-tailed Pearson's correlation (Table 3) revealed significant positive relationships between asthma severity and GHQ scores ( $r=.49$ ,  $p<.001$ ), and PTSD severity ( $r=.59$ ,  $p<.001$ ), indicating that increased severity of asthma symptoms are related to increased psychological morbidity, and increased severity of PTSD. GHQ scores and PTSD severity were also significantly positively related ( $r=.57$ ,  $p<.001$ ), indicating that greater psychiatric co-morbidity is related to increased PTSD severity.

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The correlation matrix (Table 3) also showed that self-efficacy was significantly negatively related to PTSD severity ( $r = -.36, p < .001$ ), asthma severity ( $r = -.30, p < .01$ ), and psychiatric co-morbidity ( $r = -.41, p < .001$ ), indicating that lower levels of self-efficacy are associated with higher PTSD and asthma severity, and greater psychiatric co-morbidity. For locus of control, powerful-others was positively related to PTSD severity ( $r = .29, p < .01$ ), and internal was negatively related to psychiatric co-morbidity ( $r = -.22, p < .05$ ), indicating that an orientation of belief that others are in control is associated with greater severity of PTSD symptoms, while an internal sense of control is inversely related to psychiatric co-morbidity.

### *Regression analyses of locus of control and self-efficacy*

We computed hierarchical regression models (Tables 4 & 5) to investigate psychiatric outcomes, as predicted by asthma severity (model 1), self-efficacy (model 2), and LoC (model 3).

Turning first to PTSD following asthma attack (Table 4), model 1 explained a significant proportion of the variance [ $F(1,108)=57.31, p < 0.0001, f^2=0.51$ ]. It explained 34% ( $R^2_{adj}=0.341$ ) of the variance. After controlling for asthma symptom severity, model 2 significantly improved the prediction of PTSD [ $F(1,107)=6.14, p < 0.05, R^2_{adj}=0.371, f^2=0.61$ ]. The inclusion of self-efficacy resulted in an additional 3.5% ( $\Delta R^2 = 0.035$ ) of the variance being accounted for. The overall model 3 did not improve the prediction [ $F(3,104)=2.48, ns, R^2_{adj}=0.396, f^2=0.65$ ]. Correlation coefficients showed that asthma severity and self-efficacy consistently predicted PTSD.

(Insert Table 4)

In terms of psychiatric co-morbidity (Table 5), model 1 explained a significant proportion of the variance [ $F(1,108)=33.71, p<0.0001, R^2_{adj}=0.231, f^2=0.31$ ] with 23% of the variance being accounted for. After controlling for asthma symptom severity, model 2 significantly improved the prediction [ $F(1,107)=11.92, p<0.001, R^2_{adj}=0.301, f^2=0.45$ ]. The inclusion of self-efficacy yielded an additional 8% ( $\Delta R^2 =0.076$ ) of the variance being accounted for. Model 3 did not improve the prediction [ $F(3,104)=0.52, \text{ns}, R^2_{adj}=0.292, f^2=0.48$ ]. Correlation coefficients showed that asthma symptom severity and self-efficacy also consistently predicted the outcome.

(Insert Table 5)

We carried out mediation analyses to examine whether self-efficacy would mediate the relationship between asthma symptom severity and PTSD or psychiatric co-morbidity. We suggest a mediation model to explain how asthma severity is related to PTSD severity and psychiatric symptom severity: a higher frequency of asthma symptoms is likely to have a negative impact on self-efficacy, thus creating vulnerability to negative psychiatric outcomes. Tables 6 and 7 show the total, direct and indirect effects of X on Y. The results confirmed the mediation effects. Looking at the model summaries, asthma symptom severity was negatively correlated ( $r=-0.06$ ) with self-efficacy which in turn was negatively correlated with PTSD ( $r=-0.45$ ; Table 6) and psychiatric co-morbidity ( $r=-0.30$ ; Table 7). Visualisations of the paths can be seen in Figures 1 and 2.

(Insert Table 6 and 7, and Figures 1 and 2)

#### 4. Discussion

In our sample of people who experienced asthma-attack, 20% met criteria for PTSD, equal to the prevalence found in one previous study on adolescents (Kean et al., 2006), but much greater than the 3% found in a more recent study on adults (Chung et al., 2012).

Consistent with previous research on chronic or life-threatening illness (Tedstone and Tarrier, 2003; Wu et al., 2005; Griffiths et al., 2007), our findings suggest people who experience asthma-attack are at greater risk of PTSD than those in the general population.

Those with PTSD tended to experience asthma symptoms with greater frequency, and also had increased likelihood of co-morbid psychiatric symptoms such as depression and anxiety. This is consistent with previous findings of increased risk of psychiatric comorbidity for those with PTSD (Davidson et al., 1991), and for those with asthma (Goodwin et al., 2004).

We also investigated whether agency beliefs, Locus of Control and self-efficacy, would relate to PTSD following asthma attack. Previous studies have suggested that those with PTSD more often have external locus of control, and have lower levels of self-efficacy (Shelley and Pakenham, 2004; Wu et al., 2004; Mak et al., 2010). Self-efficacy in the current sample was significantly lower for the PTSD group than for those without PTSD, which confirmed the expected result. In contrast to expectations, powerful-others-LoC was greater

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for the PTSD group; however, internal- and chance-LoC showed no differences between groups.

Additionally, relationships between agency beliefs and health outcomes were tested. Self-efficacy was significantly negatively correlated with asthma severity, PTSD severity, and psychiatric co-morbidity. In our sample, lower self-efficacy is related to greater frequency of asthma and PTSD symptoms, and greater likelihood of psychiatric co-morbid symptoms. Again, LoC did not relate with health outcomes as expected (see Wu et al., 2004). Powerful-others-LoC was positively related to PTSD severity, and internal-LoC was inversely related to psychiatric co-morbidity, but no other correlations were found for LoC; this is partly in line with expectations, but lacks congruence, suggesting further investigation is required.

Regression models confirmed that self-efficacy negatively predicted both PTSD and psychiatric co-morbidity, even after controlling for asthma severity. So, those with higher self-efficacy were less likely to report PTSD or other psychiatric symptoms, even after accounting for their asthma symptoms. LoC scales, however, were not significant predictors of either PTSD or psychiatric co-morbid symptoms after asthma symptoms and self-efficacy were controlled for.

Finally, we aimed to investigate whether agency beliefs might mediate the relationship between asthma severity and health outcomes including PTSD severity and psychiatric co-morbidity. Specifically, we hypothesised that self-efficacy, and LoC, would partially explain the relationship between asthma symptoms and PTSD and co-morbid psychiatric symptoms following asthma attack.

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We did not go on to investigate the mediating effect of LoC since there was no evidence of a relation between LoC and psychiatric outcome in the regression models. As such, in this sample, LoC did not explain the presence PTSD or psychiatric co-morbid symptoms following asthma attack.

In contrast, self-efficacy was found to partially mediate the relationship between asthma severity, and both PTSD severity, and psychiatric co-morbid symptoms. Thus, the hypothesis that individual differences in self-efficacy explain the relationship between asthma and subsequent PTSD and psychiatric co-morbidity was confirmed.

Our results support previous findings that self-efficacy mediates mental health outcomes (Maciejewski, 2000). More specifically, self-efficacy is an important mediator between trauma and posttraumatic stress (Benight et al., 1999; Benight and Bandura, 2004), and between chronic illness and psychological co-morbidity (Arnstein et al., 1999; Wu et al., 2004). Likewise, our study suggests that people with little confidence in their ability to cope with and manage their asthma (low self-efficacy) are at increased risk of developing serious mental health problems such as depression, anxiety, or PTSD. On the other hand, high self-efficacy is protective against these adverse outcomes. Moreover, previous research has shown that self-efficacy can be improved, thus bolstering capacity for resilience, and reducing the likelihood of negative mental health outcomes (Benight and Bandura, 2004).

Since self-efficacy was confirmed as a mediator, and locus of control was not, suggests they are different. Indeed, while locus of control is primarily concerned with ascribing causation to outcomes, self-efficacy is a belief about intentional personal ability. Internal control simply refers to the belief that outcomes have been caused by the person, rather than by an external influence. If the person has low self-efficacy, and internal control,

they may feel responsible for outcomes, but not have confidence in their ability to perform better (Bandura, 1977). In contrast, high self-efficacy explicitly assumes that a person has agency, both the ability and the motivation, to make good decisions and act on them accordingly (Bandura, 1982). The difference is that locus is a broad generalisation of causation. Whereas self-efficacy describes a person's judgement about whether their success or failure is due to effort or ability (see Bandura, 1994).

Self-efficacy is a meaningful focal point for practical intervention within the context of asthma, PTSD and co-morbid disorders. It is a central, dynamic factor which is vulnerable to negative experiences, but can also be supported – all the while mediating psychiatric outcomes (Bandura, 1994; Benight and Bandura, 2004). Self-efficacy can be safeguarded, cultivated, and built-up. According to Bandura (1994), there are four main ways to enhance self-efficacy: Mastery experiences which result in positive reinforcement, vicarious experience of positive coping, social support, and adjusting cognitive appraisal. These might be practically achieved through health education, support groups, and Cognitive Behavioral Therapy (CBT). Biofeedback therapy and psycho-education are also likely to be beneficial, through empowering experiences of effective coping, stress management, and relaxation (Pope et al., 2014; Smith et al., 2007), which may lead to increased self-efficacy (and reduced psychiatric morbidity). Systematic reviews on psychological interventions for asthma found that these methods may be effective, but were inconclusive due to small sample sizes and differing outcome measures (see Smith & Jones, 2015; Yorke et al., 2007). Future studies on interventions should more explicitly incorporate efficacy building and longitudinally measure changes in self-efficacy to further support the importance of self-efficacy for people with

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asthma, PTSD, and co-morbid disorders (Battersby et al., 2010; Marks et al., 2005; Taylor et al., 2014).

Although our study offers valuable insights, there are methodological issues and limitations. As a cross-sectional design, causation cannot be determined; this study cannot in itself say that increased symptoms lead to low self-efficacy, and subsequent psychiatric symptoms; though the data are congruent with this model. Future research should investigate further using longitudinal or experimental designs (Benight et al., 1999). Additionally, the sample size was not sufficient to explore differences in culture, age, and other demographic variables.

## Conclusion

PTSD and psychiatric co-morbidity are more prevalent following asthma attack than in the general population. One in five people experiencing asthma attack met criteria for PTSD. We found that those with higher frequency of asthma symptoms, were more likely to have PTSD, and report more psychiatric symptoms. This adds to the consensus in literature that mental health outcomes in complex health conditions, such as asthma, require additional awareness and investigation. In particular we suggest that a high priority is to find which factors mediate negative psychiatric outcomes following asthma attack.

We investigated whether agency beliefs would explain the link between asthma severity and subsequent PTSD and psychiatric co-morbidity. Self-efficacy was a partial mediator, while LoC was non-significant. Self-efficacy was negatively associated with asthma severity, PTSD, and psychiatric co-morbidity. This evidence supports the notion that agency beliefs play an important part in mental health outcomes for people with asthma.

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Building self-efficacy can be achieved through education, positive reinforcement, social support, and CBT, and should be incorporated more explicitly into existing interventions. In sum, an important focus for future research and intervention is to reduce the risk of psychiatric symptoms following asthma attack through safeguarding and enhancing individuals' self-efficacy.

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## Appendices

Table 1

*Descriptive statistics of total PTSD symptom scores for each PTSD group*

Posttraumatic Stress Disorder following asthma attack

		N	%	Mean	S.D.	Min	Max
<b>Age</b>		110	100	14.82	36.76	18	73
<b>Gender</b>	Male	26	23.6				
	Female	84	76.4				
<b>Country</b>	UK	62	56.4				
<b>Group</b>	U.S. &						
	Canada	36	32.7				
	Europe &						
	Other	12	10.9				
<b>PTSD</b>	No-PTSD	88	80	27.19	7.41	17	
<b>symptoms</b>	Full PTSD	22	20	54.24	8.95	42	79
	Total	110	100	32.60	13.32	17	79
<b>Time since asthma attack</b>	No-PTSD			112.72	141.32	0	756
	Full PTSD			42.14	50.82	1	180
	Total			98.60	131.31	0	756
<b>(months)</b>							

Table 2

*Means and standard deviation of asthma severity, psychiatric co-morbidity, self-efficacy, and LoC scales for each PTSD group*

	Asthma	Psychiatric	Self-	Health locus of control					
				severity	co-morbidity	efficacy	Powerful others	Internal	Chance
<b>No-PTSD</b>	91.46	4.51	31.59	15.76	23.87	17.61			
	(23.84)	(5.05)	(5.26)	(5.34)	(4.38)	(4.97)			
<b>Full</b>	127.92	12.01	26.45	19.30	23.18	18.23			
<b>PTSD</b>	(21.31)	(6.74)	(6.24)	(6.23)	(5.27)	(4.62)			

## Posttraumatic Stress Disorder following asthma attack

	98.75	6.01	30.56	16.47	23.73	17.74
<b>Total</b>						
	(27.49)	(6.18)	(5.82)	(5.68)	(4.56)	(4.89)

Table 3

*Correlations between HLOC scales, self-efficacy, and health outcomes*

	HLOC			Self-efficacy	PTSD severity	Asthma severity	Psychiatric co-morbidity
	Powerful others	Internal	Chance				
<b>HLOC</b>							
<b>Powerful others</b>	-						
<b>Internal</b>	.08	-					
<b>Chance</b>	.40***	-.21*	-				
<b>Self-efficacy</b>	-.11	.30**	-.06	-			
<b>PTSD severity</b>	.29**	-.01	.12	-.36***	-		
<b>Asthma severity</b>	.19	-.12	.09	-.30**	.59***	-	
<b>Psychiatric co-morbidity</b>	.09	-.22*	.11	-.41***	.57***	.49***	-

\*p<.05, \*\*p<.01, \*\*\*p<.001

Table 4:

*Regression models predicting PTSD severity*

		b	Std. Error	Beta	t value	Sig.
<b>Model 1</b>	Asthma Severity	.285	.038	.589	7.571	.000***
<b>Model 2</b>	Asthma Severity	.257	.039	.529	6.645	.000***
	Self-efficacy	-.452	.182	-.198	-2.479	.015*
<b>Model 3</b>	Asthma Severity	.245	.038	.505	6.375	.000***

### Posttraumatic Stress Disorder following asthma attack

Self-efficacy	-.504	.187	-.220	-2.690	.008**
Powerful Others LoC	.364	.199	.155	1.830	.070
Internal LoC	.332	.240	.114	1.386	.169
Chance LoC	.060	.231	.022	.259	.796

\* p<.05, \*\* p<.01, \*\*\* p<.001

Table 5:

### *Regression models predicting Psychiatric Co-morbidity*

		<i>b</i>	<i>Std. Error</i>	<i>Beta</i>	<i>t value</i>	<i>Sig.</i>
<b>Model 1</b>	Asthma Severity	.110	.019	.488	5.806	.000***
<b>Model 2</b>	Asthma Severity	.090	.019	.401	4.773	.000***
	Self-efficacy	-.308	.089	-.290	-3.453	.001**
<b>Model 3</b>	Asthma Severity	.090	.019	.399	4.653	.000***
	Self-efficacy	-.285	.094	-.269	-3.030	.003**
	Powerful Others LoC	-.034	.100	-.031	-.337	.737
	Internal LoC	-.099	.120	-.073	-.827	.410
	Chance LoC	.076	.116	.060	.658	.512

\* p<.05, \*\* p<.01, \*\*\* p<.001

Table 6:

### *Total, direct, and indirect effects of asthma severity on PTSD severity mediated by self-efficacy*

	Effect	SE	Boot	t-value	p	LLCI		Boot	Boot
						SE	ULCI		
Total	.2853	.0377		7.5707	.0000***	.2106	.3600		
<b>effect of X</b>									
on Y									

Posttraumatic Stress Disorder following asthma attack

Direct	.2565	.0386	6.6453	.0000***	.1800	.3330		
effect of X								
on Y								
Indirect	.0288		.0154				.0046	.0669
effect of X								
on Y (Self-efficacy)								

\* p<.05, \*\* p<.01, \*\*\* p<.001

Table 7:

*Total, direct, and indirect effects of asthma severity on psychiatric symptoms mediated by self-efficacy*

	Effect	SE	Boot SE	t-value	p	LLCI	ULCI	Boot LLCI	Boot ULCI
<hr/>									
Total	.1096	.0189		5.8061	.	.0722	.1470		
effect of X									
on Y					*				
Direct	.0900	.0189		4.7727	.	.0526	.1274		
effect of X									
on Y					*				
Indirect	.0196		.0089					.0060	.0411
effect of X									
on Y (Self-efficacy)									
<hr/>									

\* p<.05, \*\* p<.01, \*\*\* p<.001

## Posttraumatic Stress Disorder following asthma attack

Figure 1

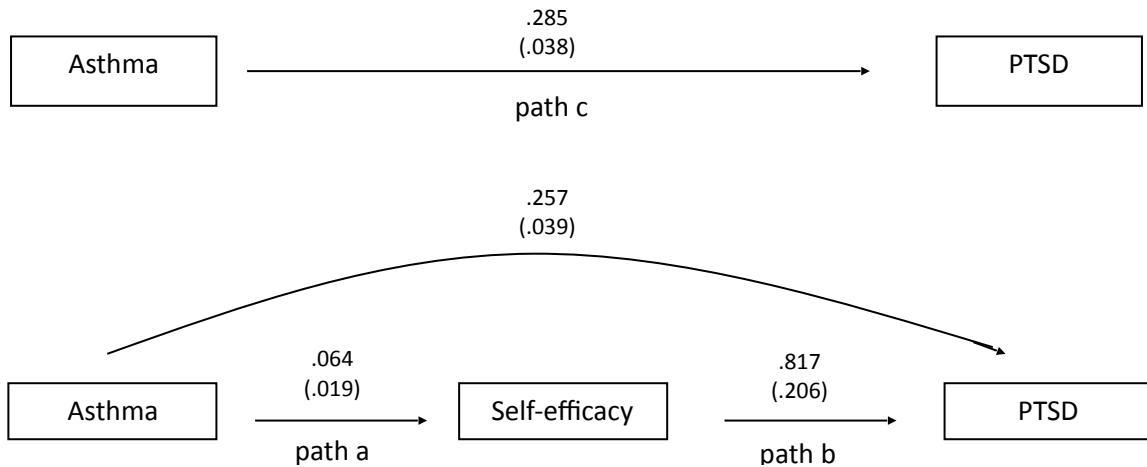


Figure 2

