Psychological Medicine, 2004, **34**, 1465–1474. © 2004 Cambridge University Press
DOI: 10.1017/S0033291704002739 Printed in the United Kingdom

Asthma and depressive and anxiety disorders among young persons in the community

RENEE D. GOODWIN*, DAVID M. FERGUSSON AND L. JOHN HORWOOD

Department of Epidemiology, Mailman School of Public Health, Columbia University, 1051 Riverside Drive, Unit 43, New York, NY 10032, USA; Christchurch Health and Development Study, Department of Psychological Medicine, Christchurch School of Medicine and Health Sciences, Christchurch, New Zealand

ABSTRACT

Background. The objectives of the study were to examine linkages between asthma and depressive and anxiety disorders in a birth cohort of over 1000 young persons studied to the age of 21 years. Specifically, the study aimed to ascertain the extent to which associations between asthma and depressive and anxiety disorders could be explained by non-observed fixed confounding factors.

Method. Asthma and depressive and anxiety disorders were measured prospectively over the course of a 21-year longitudinal study. Fixed effects logistic regression models were used to determine the relationship between asthma and depressive and anxiety disorders, adjusting for potentially confounding factors.

Results. Asthma in adolescence and young adulthood was associated with increased likelihood of major depression (OR 1·7, 95% CI 1·3–2·3), panic attacks (OR 1·9, 95% CI 1·3–2·8), and any anxiety disorder (OR 1·6, 95% CI 1·2–2·2). Associations between asthma and depressive and anxiety disorders were adjusted for confounding factors using a fixed effects regression model which showed that, after control for fixed confounding factors, asthma was no longer significantly related to major depression (OR 1·1), panic attacks (OR 1·1), or any anxiety disorder (OR 1·2). Additional *post hoc* analyses suggested that exposure to childhood adversity or unexamined familial factors may account for some of the co-morbidity of asthma and depressive and anxiety disorders.

Conclusions. These results confirm and extend previous findings by documenting elevated rates of depressive and anxiety disorders among young adults with asthma, compared with their counterparts without asthma, in the community. The weight of the evidence from this study suggests that associations between asthma and depressive and anxiety symptoms may reflect effects of common factors associated with both asthma and depressive and anxiety disorders, rather than a direct causal link. Future research is needed to identify the specific factors underlying these associations.

INTRODUCTION

Asthma is the most common chronic medical condition and a leading health problem, both in prevalence (Sterk *et al*. 1998; Cunningham *et al*. 1999; Asher *et al*. 2000; Weiss *et al*. 2000), associated social and physical morbidity (Weil *et al*. 1999), and societal costs (Sterk *et al*. 1998) in the United States and worldwide (Sterk *et al*. 1998; Weiss *et al*. 2000; Mannino *et al*. 2002). In recent years, a growing number of studies has also investigated the relationship between asthma and depressive and anxiety disorders (Yellowlees *et al*. 1987; Bussing *et al*. 1996; Perna *et al*. 1997; Carr, 1998; Koltek *et al*. 1998; Brown *et al*. 2000; Vila *et al*. 2000; Afari *et al*. 2001; Goethe *et al*. 2001; McQuaid *et al*. 2001; Swadi, 2001; Nascimento *et al*. 2002; Ortega *et al*. 2002; Slattery *et al*. 2002; Weisberg *et al*. 2002; Goodwin & Eaton, 2003). These studies

* Address for correspondence: Dr R. Goodwin, Department of Epidemiology, Mailman School of Public Health, Columbia University, 1051 Riverside Drive, Unit 43, New York, NY 10032, USA. (Email: rdg66@columbia.edu)

have used two general approaches. First, numerous studies have noted elevated rates of anxiety symptoms including panic attacks, panic symptoms, overanxious disorder and separation anxiety disorder in clinical samples of pediatric and adult asthma patients (Yellowlees *et al.* 1987; Bussing *et al.* 1996; Perna *et al.* 1997; Carr, 1998; Koltek *et al.* 1998; Brown *et al.* 2000; Vila *et al.* 2000; Afari *et al.* 2001; Goethe *et al.* 2001; McQuaid *et al.* 2001; Swadi, 2001; Nascimento *et al.* 2002; Slattery *et al.* 2002; Goodwin & Eaton, 2003). Also, studies of psychiatric samples have noted elevated rates of asthma amongst those with anxiety disorders (Koltek *et al.* 1998). For instance, Koltek *et al.* (1998) found an excess of asthma among adolescent psychiatric in-patients with post-traumatic stress disorder (PTSD). More recently, Weisberg *et al.* (2002) found an elevated prevalence of asthma in primary-care patients with PTSD, compared to those without.

Secondly, despite numerous reports from clinical samples, a considerably smaller number of epidemiological studies have examined the linkages between asthma and psychiatric diagnoses (Ortega *et al.* 2002). In one study of 1285 young people in a cross-sectional community-based sample, Ortega and colleagues (2002) found an association between asthma and increased prevalence of social phobia, separation anxiety disorder and overanxious disorder, but not depression, among 9- to 17-year-olds. A more recent study examined the relationship between asthma and panic attacks among adults in the community (Goodwin & Eaton, 2003) and found evidence of significantly increased risk of panic attacks amongst those with asthma compared to those without asthma.

While existing evidence suggests statistically significant linkages between asthma and increased risk of depressive and anxiety disorders in clinical settings, relatively little is known about this relationship in the general population and even less is known about the mechanisms that might lead to such associations. Against this background, this paper reports on the relationship between asthma and depressive and anxious symptomatology in a birth cohort studied to the age of 21 years. The specific aims of this study were: (1) to examine the associations between asthma and depressive and anxiety disorders (e.g. major depression, anxiety disorders, panic attacks) over the period of ages 16–21 years; (2) to adjust the relationship between asthma and depressive and anxiety disorders for potentially confounding factors using a fixed effects logistic regression model. (Fixed effects regression makes it possible to adjust associations between asthma and depressive and anxiety disorders for non-observed common factors that exert a fixed effect on both outcomes; see Method section for a more detailed description.)

METHOD

Participants

The data described in this report were gathered during the course of the Christchurch Health and Development Study (CHDS). The CHDS is a longitudinal study of an unselected birth cohort of 1265 children (635 males, 630 females) born in the Christchurch, New Zealand, urban region in mid-1977. This cohort has now been studied at birth, 4 months, 1 year and annual intervals to age 16 years, and again at ages 18 and 21 years. The present analysis is based on a sample of 1053 individuals for whom information on asthma and depressive and anxiety disorders was available at age 18 or 21 years. This sample represented 83% of the original birth cohort. However, since not all participants were assessed at both ages the observed sample numbers vary between age 18 ($n=1025$) and age 21 ($n=1011$). The following measures were used in the analysis.

Asthma

At ages 18 and 21 years, participants were questioned about their experience of asthma since the preceding interview. Specifically, all subjects were first asked whether they had ever been diagnosed with asthma by a doctor. Respondents who reported such a diagnosis were then questioned further about the occurrence of asthma symptoms since the previous assessment, their current frequency of symptoms, frequency of medication use, the number of medical consultations for asthma and the frequency of asthmatic attacks in the preceding year. For the purposes of the present analysis, a dichotomous definition of asthma was used in which the individual was classified as having asthma in a given time period (ages 16–18,

18–21 years) if the subject reported: (*a*) ever being diagnosed with asthma by a doctor and (*b*) experiencing any asthma symptoms during the period. Overall, 27% of the sample reported a diagnosis of asthma by age 21 years, with 17·9% reporting asthmatic symptoms in the period 16–18 years and 20·1% in the period 18–21 years.

In addition, the data were used to define a series of alternative measures of asthma. These included: (*a*) the number of medical consultations for asthma in the preceding 12 months; (*b*) the number of asthmatic attacks in the preceding 12 months, where an attack was defined as any episode of asthma that required medical intervention or an increased dose of regular medication to bring under control; (*c*) frequency of medication for asthma at ages 18 and 21 years, defined on a 4-point scale (most days; some days; now and again; never); and (*d*) current frequency of asthma symptoms (including daytime wheeze, night-time wheeze, daytime cough and night-time cough) at ages 18 and 21 years, coded on a 4-point scale (most days; some days; now and again; never).

Depressive and anxiety disorders

At ages 18 and 21 years, respondents were questioned about their experience of depressive and anxiety symptoms since the previous assessment, using an interview based on the Composite International Diagnostic Interview (CIDI; WHO, 1993). All interviews were conducted using trained lay interviewers. This information was used to assess the following measures of depressive and anxiety disorders over the periods 16–18 and 18–21 years.

Major depression

Items from the CIDI were used to assess DSM-IV (APA, 1994) symptom criteria for major depression and associated impairment. At age 18 years subjects were asked to report on symptoms occurring during the intervals 16–17 and 17–18 years, and at age 21 for the intervals 18–20 and 20–21 years. Subjects were classified as having major depression if they met DSM-IV criteria for a major depression episode at any time during the interval. Overall, 22·1% of the entire sample was classified as having major depression during the period from 16–18 years, and 23·5% from 18–21 years.

Anxiety disorders

Items from the CIDI were used to assess DSM-IV symptom criteria for a range of anxiety disorders in each assessment period. Anxiety disorders assessed included generalized anxiety disorder, social phobia, specific phobia and agoraphobia. A total of 17·1% of the whole sample was classified as meeting DSM-IV diagnostic criteria for at least one of these disorders during the period 16–18 years, and 12·9% from 18–21 years.

Panic attack

At each assessment, sample members were also questioned about their experience of panic attacks and related symptomatology. In particular, subjects were asked on how many occasions, if any, they 'had a spell or attack in which you all of a sudden felt frightened, anxious or uneasy in a situation where most people would not be anxious or afraid', since the previous assessment. After an affirmative response to this inquiry, additional questions about the circumstances and the setting of the panic attack were used to gather information needed to determine the clinical significance of the panic attack (e.g. to eliminate those that did not include panic symptoms or were in response to real life-threatening situations). These assessments showed that 6·8% of the entire sample reported at least one panic attack in the period 16–18 years and 8·1% over the interval from 18–21 years.

Exposure to childhood adversity

An index of childhood adversity was constructed in the following way. First, a range of adverse childhood and family risk factors were selected from the database of the study. These variables were chosen on the basis of prior knowledge of the variables in the database that had consistently been shown to be related to psychosocial outcomes in adolescence/young adulthood. The measures were selected to span a number of domains of social and family functioning including: family socio-economic disadvantage, family instability and conflict, child abuse exposure and parental adjustment problems. Within each domain the selected measures were then used to create a series of dichotomous indicators of disadvantage. The measures

https://doi.org/10.1017/S0033291704002739 Published online by Cambridge University Press

included in the adversity index and the criteria for each indicator are described below.

Measures of socio-economic adversity

(*a*) Family socio-economic status at the time of the survey child's birth was assessed using the Elley–Irving (Elley & Irving, 1976) scale of socio-economic status for New Zealand. This scale classifies individuals into six classes on the basis of paternal occupation. For the purpose of the present analysis, sample members were classified as being of low socio-economic status if they were classified into levels 5 or 6 on the Elley–Irving scale (semi-skilled or unskilled occupations). (*b*) Parental education: both maternal and paternal education levels were assessed at the time of the survey child's birth using a three-level classification system reflecting the highest level of educational attainment (no formal qualifications; high school qualifications; tertiary qualifications). Sample members were classified as coming from an educationally disadvantaged background if neither parent had obtained formal educational qualifications. (*c*) Standard of living: at each assessment from ages 1–12 years, interviewer ratings of the family's standard of living were obtained using a 5-point scale that ranged from 'obviously affluent' to 'obviously poor/very poor'. For the purposes of the present analysis sample members were classified as experiencing low living standards if the family had been rated on three or more occasions as having below average (poor or very poor) living standards.

Measures of parental change and conflict

Comprehensive data on family placement and changes of parents were collected at annual intervals from birth to age 16 years. This information was used to construct two measures of family stability over the period 0–16 years. (*a*) Single-parent family: this measure was based on whether the child had ever spent time in a single-parent family before age 16 either as a result of entering a single-parent family at birth, or as a result of parental separation/divorce. (*b*) Changes of parents: an overall measure of family instability was constructed on the basis of a count of the number of changes of parents experienced by the child before age 16 years as a result of parental separation/divorce, remarriage, reconciliation and related changes. Sample members were classified as experiencing high family instability if they had experienced three or more changes of parents by age 16 years. Information on family instability was supplemented by a further measure of parental conflict. (*c*) Inter-parental violence: at age 18 years, sample members were questioned using items from the Conflict Tactics Scale (Straus, 1979) to assess the extent to which they had witnessed incidents of physical violence or serious threats of physical violence between their parents during childhood (prior to age 16). For the purposes of the present analysis sample members were classified as having experienced inter-parental violence if they reported witnessing any episode of physical violence between their parents during childhood.

Measures of child abuse exposure

At ages 18 and 21 years, cohort members were questioned concerning their experience of child abuse prior to age 16 years. (*a*) Parental use of physical punishment: young people were asked to describe their parents' use of physical punishment on a 5-point scale ranging from 'parent never used physical punishment' to 'parent treated me in a harsh and abusive way' (Fergusson & Lynskey, 1997). The questioning was conducted separately for the mother and father. For the purposes of the present analysis the young person was defined as having been exposed to physical abuse if he or she reported at either 18 or 21 years that either parent had used physical punishment too often or too severely or had treated the respondent in a harsh and abusive manner during childhood. (*b*) Childhood sexual abuse: young people were also questioned at 18 and 21 years concerning their experience of sexual abuse in childhood ranging in severity from episodes of non-contact abuse to various forms of sexual penetration (Fergusson *et al.* 1996). For the purposes of the present analysis the young person was classified as having experienced sexual abuse if she/he reported at either 18 or 21 years any episode of sexual abuse involving physical contact with the perpetrator.

Measures of parental adjustment

(*a*) Parental alcohol problems: when sample members were aged 15 years, parents were questioned whether there was a history of

Table 1. *Rates (%) of major depression, panic attack, and anxiety disorder by presence/absence of asthma in the CHDS cohort ages 16–18 and 18–21 years*

	16–18 years			18–21 years		
Measure	No asthma ($n=842$)	Asthma ($n=183$)	OR (95% CI)	No asthma ($n=808$)	Asthma ($n=203$)	OR (95% CI)
Major depression	19·5	33·9	2·1 (1·5–3·0)	21·7	31·0	1·6 (1·2–2·3)
Panic attack	5·7	12·0	2·3 (1·3–3·8)	7·2	11·8	1·7 (1·05–2·9)
Any anxiety disorder	15·8	23·0	1·6 (1·1–2·3)	11·3	19·2	1·9 (1·2–2·8)

alcohol problems for any parent. (*b*) Parental criminality: also at age 15, information was obtained from parents on whether any parent had a history of criminal offending. (*c*) Parental illicit drug use: when sample members were aged 11 years, information was obtained from parents concerning their history of illicit drug use.

The dichotomous indicator measures described above were then combined into a single composite measure by summing the indicators to create a point-scale reflecting the degree of exposure to childhood adversity (Fergusson & Horwood, 2003). The use of a points-score approach such as this is common in epidemiological contexts where there is a need for a convenient means of data reduction to produce an overall scale of adversity. The resulting scale ranged from a minimum of 0 to a maximum of 10, with a mean of 1·02 and a standard deviation of 0·79. The scale has been shown in a previous analysis to exhibit strong dose–response associations with a range of psychosocial adjustment problems in adolescence/young adulthood, including depression and anxiety disorders (Fergusson & Horwood, 2003). The reliability of the scale, assessed using coefficient alpha, was 0·69.

Statistical methods

Bivariate associations between asthma and depressive and anxiety disorders were computed at ages 16–18 and 18–21 (Table 1). For each time period the odds ratios between asthma and depressive and anxiety disorders were estimated. The unadjusted associations between asthma and depressive and anxiety disorders (Table 2) were calculated using a generalized estimating equation model (Zeger & Liang, 1986) to estimate an averaged association across the two time periods. The model fitted was:

$$\text{logit}(Yit) = B_0 + B_1 Ait,$$

where logit (Yit) was the log odds of the depressive and anxiety disorder (major depression, panic attack, anxiety disorder) for subject i in period t, and Ait was the corresponding measure of asthma. From this model, the population-averaged estimate of the odds ratio between Yit and Ait is given by e^{B_1}, where e is the base of natural logarithms.

Table 2. *Estimated odds ratios (95% CI) between asthma and measures of major depression, panic attack, and anxiety disorder before and after adjustment for confounding fixed factors*

	Unadjusted			Adjusted		
Measure	OR	(95% CI)	p	OR	(95% CI)	p
Major depression	1·7	(1·3–2·3)	<0·001	1·1	(0·5–2·1)	>0·80
Panic attack	1·9	(1·3–2·8)	<0·001	1·1	(0·4–3·0)	>0·80
Any anxiety disorder	1·6	(1·2–2·2)	<0·005	1·2	(0·5–2·9)	>0·60

The data were then analyzed using a fixed effects logistic regression model to estimate the association between asthma and depressive and anxiety disorders taking into account non-observed fixed effects (see Table 2). The model fitted was:

$$\text{logit}(Yit) = \alpha i + B_1 Ait,$$

where logit (Yit) was the log odds of depressive and anxiety disorder for subject i in period t; Ait was the corresponding measure of asthma; and αi was an individual-specific intercept term reflecting non-observed fixed factors that

https://doi.org/10.1017/S0033291704002739 Published online by Cambridge University Press

influenced the log odds of depressive and anxiety disorder for subject *i*. This model estimates the effects of asthma on the log odds of depressive and anxiety disorder after adjustment for the non-observed confounding factors represented by the intercept term αi. An account of the estimation of such models has been given by Hamerle & Ronning (1995). In the present study, all regression models were fitted using STATA 6 (StataCorp, 1999). In addition, all models fitted included a binary indicator measure of the period of measurement (16–18, 18–21 years) to allow for changes in the base rate of depressive and anxiety disorders with age.

To examine the effects of sample losses on the representativeness of the sample, the 1053 subjects included in the analysis were compared to the remaining 212 subjects on a series of measures of socio-demographic characteristics assessed at the point of birth. These comparisons suggested that there were slight tendencies for the obtained sample to under-represent children from socially disadvantaged families characterized by low parental education, low socio-economic status or single parenthood. To address this issue, the data-weighting methods described by Carlin *et al.* (1999) were used to examine the possible implications of selection effects arising from the pattern of missing data. These analyses produced essentially identical results to those reported here, suggesting that the conclusions of this study were unlikely to have been influenced by selection bias.

RESULTS

Bivariate associations

Table 1 shows the associations between asthma at ages 16 to 18 and 18 to 21 years and rates of depressive and anxiety disorders at these ages. Each association is tested for statistical significance using the χ^2 test and the associations between asthma and depressive and anxiety disorders are measured by the odds ratio. The table shows the presence of relatively small but nonetheless pervasive associations between asthma and depressive and anxiety disorders in adolescence and young adulthood.

Adjustment for fixed confounding factors

To examine the extent to which the associations between asthma and depressive and anxiety disorders could be explained by common factors that exerted fixed effects on these measures, fixed effects regression models were fitted to the data summarized in Table 1. In these models, the rate of each disorder in a given year was modeled as a function of: (*a*) asthma in the year; (*b*) non-observed fixed confounding factors (see Method section). The results of these analyses are summarized in Table 2, which shows estimates of the associations between asthma and depressive and anxiety disorders after adjustment for fixed effects. For comparative purposes the table shows the unadjusted estimates derived from a generalized estimating equation model which includes only the measure of disorder as a predictor of asthma. The table shows that, in all cases: (*a*) before adjustment for fixed effects the associations between asthma and the odds of disorder were statistically significant ($p < 0{\cdot}01$); (*b*) after adjustment for fixed effects all associations became small and statistically non-significant. These results strongly suggest that the associations between asthma and disorder reflected the presence of common confounding factors.

Exposure to childhood adversity as a common risk factor

The results in Table 2 suggest that following adjustment for non-observed fixed covariate factors, asthma and depressive and anxiety disorders were unrelated. A limitation of this analysis is that it does not identify the fixed factors that explain the associations. To address this issue, an exploratory analysis was undertaken to locate observed covariate factors that may explain the associations between asthma and depressive and anxiety disorders. This analysis suggested that one common factor that linked both sets of outcomes was the individual's accumulative exposure to childhood adversity (see Method section). Table 3 shows the sample classified according to their accumulative adversity scores ranging from those with no exposure to adversity to those with high adversity. For each group, the table reports rates of asthma and depressive and anxiety disorders over the period from ages 16–21 years. The table shows that increasing exposure to childhood adversity was associated with increasing risks of asthma, depression, anxiety disorder and panic attacks. These results clearly suggest that

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Table 3. *Rates (%) of major depression, panic attack, any anxiety disorder and asthma (16–21 years) by childhood adversity score (0–16 years) among CHDS cohort members*

	Childhood adversity score					
Measure	0 ($n=260$)	1–2 ($n=413$)	3–4 ($n=170$)	5–6 ($n=103$)	7+ ($n=46$)	p†
Major depression	21·5	31·8	41·2	44·7	45·2	<0·0001
Panic attack	8·1	11·2	14·7	19·4	28·3	<0·0001
Any anxiety disorder	14·6	23·1	23·5	33·0	56·5	<0·0001
Asthma	20·4	22·3	32·4	27·2	28·3	<0·05

† Based on χ^2 tests of independence.

exposure to childhood adversity was one common factor that could explain linkages between asthma and depressive and anxiety disorders.

To examine the contribution of childhood adversity to the co-morbidities between asthma and depressive and anxiety disorders the data were re-analyzed using generalized estimating equation models in which the measures of depressive and anxiety disorders were dependent variables and asthma and childhood adversity were the predictor variables. This analysis showed that when due allowance was made for exposure to childhood adversity, the associations between asthma and depressive and anxiety disorders were reduced: asthma/depression (OR 1·5, 95% CI 1·2–2·0, $p<0·01$); asthma/anxiety disorders (OR 1·3, 95% CI 0·98–1·9, $p=0·06$); asthma/panic attacks (OR 1·5, 95% CI 1·0–2·3, $p<0·05$). Nonetheless, two of the three associations between asthma and depressive and anxiety disorders remained statistically significant following statistical control for childhood adversity, suggesting the presence of other fixed factors related to asthma and depressive and anxiety disorders.

Sensitivity analysis

Model specification

The analysis above assumes that depressive and anxiety disorders were dependent variables whose risk was influenced by asthma. To explore the sensitivity of the results to this model specification, the model was reversed by assuming that asthma was a dependent variable that was influenced by depressive and anxiety disorders. As might be expected, this alternative model specification also showed that when due allowance was made for non-observed fixed effects, asthma and depressive and anxiety disorders were unrelated.

The assessment of asthma

The results in Table 1 use a simple measure that records the presence or absence of asthma in a particular time period. It could be suggested that use of more refined measures of asthma could produce differing results. To explore this issue the data were re-analyzed using a range of measures of asthma including: frequency of medication use at ages 18 and 21 years; frequency of asthma symptoms at ages 18 and 21; doctor visits for asthma in the past year at ages 18 and 21; and number of asthma attacks in the past year at ages 18 and 21 (see Method section). All analyses yielded very similar conclusions: (*a*) prior to adjustment for fixed effects those with asthma had elevated rates of depressive and anxiety disorders; (*b*) after adjustment for fixed effects asthma was unrelated to risks of depressive and anxiety disorders.

DISCUSSION

In this study we have used data gathered over the course of a 21-year longitudinal study of a birth cohort of New Zealand-born young people to examine the linkages between asthma and depressive and anxiety disorders. These data are consistent with and extend previous findings by providing initial evidence of an association between asthma and significantly elevated levels of anxiety and depressive disorders, which has not previously been investigated in a representative community sample of young adults. Young people who reported asthma had odds of depressive and anxiety disorders that were

https://doi.org/10.1017/S0033291704002739 Published online by Cambridge University Press

between 1·6 and 2·3 times higher than young people who did not report asthma. These findings are consistent with at least two explanations of the linkages between asthma and depressive and anxiety disorders. First, these associations may reflect cause-and-effect associations in which (*a*) asthma may provoke depression or anxiety disorders or (*b*) depressive and anxiety disorders may provoke asthma. Alternatively, the associations may reflect the presence of common confounding factors that are correlated with development of both asthma and depressive or anxiety disorders.

In this paper two approaches have been used to control for the effects of such common confounding. In the first approach, fixed effects regression was used. This approach makes it possible to control the associations between asthma and depression/anxiety disorder for *non-observed* confounding factors, subject to the assumption that these confounders exert a common fixed effect on the outcomes. This analysis showed that controlling for non-observed fixed factors adequately explained the associations. After adjusting for fixed effects, the odds ratios between asthma and disorder became small and non-significant (ORs 1·1–1·2). These results strongly suggest that the associations between asthma and depressive/anxiety disorders are non-causal and reflect common factors that are correlated with both outcomes.

Yet, a limitation of the fixed effects regression model is that it does not provide an indication of the factors involved in these common causal processes. To examine this issue, regression models that included observed covariate factors were also fitted to the data.

These analyses showed that the associations between asthma and depressive/anxiety disorders could be only partially explained by observed factors. In particular, adjustment for exposure to childhood adversity reduced the odds ratios between asthma and disorder only slightly from 1·6–1·9 to approximately 1·3–1·5. These results suggest the presence of other non-observed factors whose effects were captured by the fixed effects regression model but not by the observed measure of childhood adversity. Such factors could include common genetic, social and individual processes that increase susceptibility to asthma and depressive/anxiety disorders.

Finally, it is also conceivable that the associations between asthma and depressive/anxiety disorders are artefactual and due to biases in the reporting of asthma symptoms. For instance, higher levels of asthma symptom reporting, and somatic symptom over-reporting in general, by persons with depressive and anxiety disorders is common. Therefore, use of self-report data could result in higher rates of asthma in those with disorder. Another possibility is that the link between asthma and mental disorders may be explained by, or related to, other phenomenological factors relating to asthma attacks and attack context. For instance, a recent study by Ortega *et al.* (2003) suggested that the association between asthma and psychiatric disorder appeared to be specific to having experienced an asthma attack, rather than to other criteria such as having an asthma diagnosis or being hospitalized with asthma. Moreover, Greaves *et al.* (2002) have explored the role of 'attack context', including both the recency of attack and the historical frequency of attacks in affecting psychological status. Findings suggest that patients with recent asthma attacks had significantly higher panic fear and lower control confidence in terms of asthma than those with recent stable asthma. Thus, recent attack context seems to influence psychological status. Future studies are needed that can examine the role of sequencing and recency of asthma attacks, as well as diagnostic status, in examining linkages between asthma and depressive and anxiety disorders.

The present study has a number of limitations that should be borne in mind. Data on asthma and depressive and anxiety disorders has been gathered over relatively wide time-intervals (16–18, 18–21 years). There are two liabilities of this approach. First, the width of measurement periods precludes detailed examination of the timing of the onsets and offsets of asthma and depressive and anxiety disorders. Secondly, the wide time periods may mean that the measures of depressive and anxiety disorders and asthma are subject to reporting and recall errors. However, there is little reason to believe that recall errors would lead to a spurious correlation between asthma and depressive and anxiety disorders and it is more likely that errors of recall would lead to an attenuation of the associations (Rothman & Greenland, 1998).

The prevalence rates of asthma in this birth cohort are consistent with other national reports in New Zealand (Asher *et al*. 2000; Holt & Pearce, 2000), which are higher than in less-developed countries but are thought generalizable to most westernized countries, including the United States. Finally, the use of lay interviewers to obtain self-report information on asthma limits the reliability of the data on asthma diagnosis, as there were no clinician-evaluation diagnostic data or physiological data available.

Despite these limitations the present study confirms previous research that has found linkages between asthma and depressive and anxiety disorders and extends this association to young adults in the community. The results also strongly suggest that this association arises because of common or correlated factors that are associated with increased vulnerabilities to asthma and depressive and anxiety disorders. The present analysis suggests that one factor that may contribute to this co-morbidity may be exposure to childhood adversity. It may be of interest to examine the potential role of common genetic factors in explaining the association, using a genetically informative research design.

ACKNOWLEDGEMENTS

Research into the Christchurch Health and Development Study cohort has been supported by grants from the Health Research Council of New Zealand, the National Child Health Research Foundation, the Canterbury Medical Research Foundation and the New Zealand Lottery Grants Board. Work on this project was supported in part by NIMH grant no. MH 64736.

DECLARATION OF INTEREST

None.

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