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Journal of Psychosomatic Research 55 (2003) 179–187

Journal of Psychosomatic Research

Relationship between abdominal pain subgroups in the community and psychiatric diagnosis and personality
A birth cohort study

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Received 18 January 2001; accepted 15 October 2002

Abstract

Introduction: It is unclear if there is a causal link between psychiatric disorders and unexplained chronic gastrointestinal (GI) symptomatology. The role of personality is also in dispute. We aimed to assess the association of these factors with functional GI symptoms in a birth cohort study. **Methods:** The Dunedin birth cohort is well characterised and has been followed-up prospectively to age 26 ($n = 980$). Measured were upper and lower GI symptoms over the prior year at age 26 using a validated questionnaire, psychiatric diagnoses at ages 18 and 21 by standardised interview applying DSM-III-R criteria, and personality at age 18 using the Multidimensional Personality Questionnaire (MPQ). Natural symptom groupings were identified using factor analysis and *k*-means clustering. The association of these clusters and psychiatric diagnoses or personality was assessed by logistic regression. **Results:** The *k*-means analysis produced a six-cluster solution, which was made up of a health group, and five ''disease'' clusters defined by higher than average scores on a single symptom. A diagnosis of depression at age 18 *or* 21 years was associated with increases in the odds of 1.69 (95% CI: 1.27–2.25) for all GI, of 2.16 (95% CI: 1.12–4.16) for dysmotility and of 2.07 (95% CI: 1.13–3.80) for constipation, but not with the other clusters. Similar results were observed with respect to anxiety disorders for the odds of GI overall (OR = 1.42, 95% CI: 1.01–1.99) and constipation (OR = 2.11, 95% CI: 1.17–3.79). The personality subscales were not strongly linked; membership of ''any'' diseased cluster was associated with a reduced odds of being in the fourth quartile for the well-being scale (OR = 0.64, 95% CI: 0.46–0.88) but increased odds of being in the fourth quartile for the social potency scale (OR = 1.64, 95% CI: 1.18–2.28). **Conclusions:** In a young adult community sample, unexplained GI symptoms appear to be linked to psychiatric disorders but personality differences were minimal.

Keywords: Functional disorders; Gastrointestinal symptoms; Personality; Psychiatric disorder

Introduction

Chronic unexplained gastrointestinal (GI) symptoms are now recognised to be highly prevalent in the general population but the pathogenesis of these symptoms remains in dispute. Selected personality characteristics [1–9] as well as psychological distress [10–15] have been linked to irritable bowel syndrome (IBS), which is one of the more widely recognised symptom complexes, characterised by abdominal pain or discomfort and disturbed defaecation linked to the pain. For example, outpatient and volunteer studies have reported that subjects with IBS tend to be significantly more neurotic and anxious than controls, applying standardised instruments such as the Eysenck Personality Inventory (EPI) [1,3], Spielberger Trait Anxiety Inventory [4–7] or the Minnesota Multiphasic Personality Inventory [7–9]. However, it remains unclear whether such personality scores are causally linked to IBS or other painful type GI symptoms. Alternative explanations include chronic symptoms increasing personality scores or psychological distress because of pain, or a selection bias whereby those

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doi:10.1016/S0022-3999(02)00599-8

with particular types of personality are more likely to seek health care. Furthermore, it is conceivable that IBS may in part have a hereditable component, as do certain personality traits (e.g., extroversion and neuroticism), and that any link between the two may be entirely coincidental. There remain a lack of population-based studies that have evaluated the relationship between personality and chronic GI symptoms.

Other studies have suggested that between 20% and 61% of patients with functional bowel disorders who attend gastroenterology outpatient clinics have a current psychiatric diagnosis if formally assessed [3,13,14]. However, the literature remains controversial as to whether psychiatric diagnoses are truly increased in subjects with IBS from the community. A study conducted as part of the NIMA epidemiological catchment area study found that psychiatric diagnosis was higher in subjects who had symptoms suggestive of a functional GI disorder [15]. However, a population birth cohort study failed to detect an association with psychiatric diagnosis and IBS as defined by standard symptom based diagnostic criteria [16].

We therefore aimed to evaluate the relationship between GI symptoms in the community characterised by abdominal pain with personality traits and psychiatric co-morbidity. We postulated that if personality traits and psychiatric history were causally linked to functional GI syndromes, then associations would be detected within a population comprising those with abdominal pain related symptoms. Rather than defining symptoms groupings *a priori*, we elected to undertake an empiric based approach to symptom grouping applying factor and cluster analysis, in order to reduce any bias from preconceived classification of symptomatic subjects.

Subjects and methods

Subjects

Participants were members of the Dunedin Multidisciplinary Health and Development Study (DMHDS). This was a longitudinal investigation of the health, development and behaviour of a complete cohort born between April 1, 1972 and March 31, 1973, in Dunedin, a city of approximately 120,00 on New Zealand's South Island [17].

Perinatal data were obtained at delivery. The children were traced for follow-up at the age of 3 years and 91% of the eligible births participated in the assessment. This provided a base sample of 1037 (52% male) for the longitudinal study. The cohort has been assessed with a diverse array of psychological, medical and sociological measures at ages 3 ($n=1037$), 5 ($n=991$), 7 ($n=954$), 9 ($n=955$), 11 ($n=925$), 13 ($n=850$), 15 ($n=976$), 18 ($n=993$), 21 ($n=992$) and most recently at age 26 ($n=980$).

The children's fathers were representative of the social class distribution in the general population of similar age in New Zealand. The study members were predominantly of European ancestry. Fewer than 7% of the sample identified themselves at age 18 as Maori or Polynesian, which matches the ethnic distribution of the South Island of New Zealand.

Measurement of GI symptoms

An abbreviated version of the Bowel Symptom Questionnaire (BSQ) was included in the assessment at age 26 years. The BSQ is a reliable and valid instrument that has been used extensively in epidemiological studies of disorders of the GI tract [18]. The abbreviated version was self-administered and took approximately 10 min to complete.

The abbreviated BSQ contained 18 items, which described the features of abdominal pain or discomfort. These included 10 items assessing symptoms associated with pain (change in stool frequency ($\times 2$), change in stool form ($\times 2$), nocturnal pain, fullness and early satiety, retching, nausea and vomiting), 5 items assessing features associated with the relief of pain (bowel movement, medication ($\times 2$), food and belching) and 3 items assessing the relationship of food and meals to the onset of pain. Items were scored on a five-point Likert scale with the following response options: *not at all*, *sometimes*, *often*, *very often*, *almost always*. All symptoms were evaluated over the preceding 12 months, and this approach provides data comparable to a physician interview [18].

Natural symptom groupings were identified using factor analysis and *k*-means clustering; this approach identifies groups or "clusters" of individuals who share common symptom profiles. The analysis was applied to the full cohort using all 18 items (including those used to evaluate IBS). The methods have been described in the analysis section.

Psychiatric diagnoses

Mental health diagnoses were obtained at ages 18 and 21 years using a modified version of the Diagnostic Interview Schedule [19]. The modifications consisted of: (i) including only those questions pertaining to the assessment of DSM-III-R criteria; (2) assessing only the symptoms that occurred within the past 12 months; (3) assessing only the more commonly occurring diagnoses for this age group; and (4) limiting options to "0 = no," "1 = yes, sometimes" and "2 = yes, definitely." Only those responses receiving a "2" were considered severe enough to be entered into the diagnostic algorithms. Diagnoses were determined using computer-run algorithms that followed explicit criteria specified by the *DSM-III-R*.

Diagnoses were derived for the following 15 disorders at age 21: (a) 6 anxiety disorders: generalised anxiety disorder ($n=18$, 1.9%), obsessive-compulsive disorder ($n=67$, 7.1%), panic disorder ($n=6$, 0.6%), agoraphobia ($n=36$, 3.8%), social phobia ($n=92$, 9.7%) and simple phobia ($n=80$, 8.4%); (b) 3 mood disorders: major depressive episode ($n=161$, 16.8%), manic episode ($n=19$, 2.0%) and dysthymia ($n=28$, 3.0%); (c) 2 eating disorders: anorexia

nervosa ($n=4$, 0.4%) and bulimia nervosa ($n=9$, 1.0%); (d) 2 substance disorders: alcohol dependence ($n=94$, 9.8%) and cannabis dependence ($n=91$, 9.6%); (e) 1 Axis-II *DSM-III-R* disorder: antisocial personality disorder ($n=31$, 3.2%); and (f) 1 category of nonaffective psychosis ($n=39$, 4.1%), which consisted of the positive psychotic symptoms (Criterion A of the *DSM-III-R*, pp. 194–195) for the diagnosis of schizophrenia and schizophreniform disorders, with the exclusion of such symptoms occurring solely under the influence of alcohol or drugs, or during a major depressive episode.

The same set of diagnoses were derived at the age 18 assessment, with the exception that information concerning mania and nonaffective psychosis was not sought, and conduct disorder ($n=51$, 5.5%) was diagnosed instead of the Axis II, *DSM-III-R* antisocial personality disorder category used at age 21.

The mental health interviewers were tertiary qualified and were trained in the administration of the DIS. Reliability of the ages 18 and 21 mental health assessments was very good; at age 18, the average κ coefficient across three families of disorders (anxiety, depressive and substance dependence disorder) was .70 and was $>.85$ across the same three families at age 21 [20].

Three categories of psychiatric diagnosis were evaluated:

(a) Any psychiatric disorder — this included all subjects who had received a diagnosis of psychiatric disorder (any DSMIII-R Axis I or Axis II diagnosis) at age 18 and/or 21 years;
(b) Any anxiety disorder — all subjects who had received a diagnosis of anxiety disorder (i.e., any one or more of generalised anxiety disorder, obsessive-compulsive disorder, panic disorder, agoraphobia, social phobia or simple phobia) at age 18 and/or 21 years;
(c) Any depression — all subjects who had received a diagnosis of major depressive episode or dysthymia at age 18 and/or 21 years.

Subjects with psychiatric comorbidities were included in all analyses and were coded to each diagnostic category as appropriate. For example, subjects with a diagnosis of both anxiety and depression were included in three categories — any psychiatric diagnosis, any anxiety disorder and any depression. We recognise that subjects with comorbid conditions may represent a more severe psychiatric group. However, sample numbers were too small to evaluate these subjects separately.

Personality

As part of the age 18 assessment, study members completed a modified version (Form NZ) of the Multidimensional Personality Questionnaire (MPQ) [21]. Rationale and description of the modifications of the MPQ for use in NZ were approved by Tellegen and have been described elsewhere [22]. The MPQ is a self-report personality instrument designed to assess a broad range of individual differences in affective and behavioural style. The 177-item version of the MPQ (Form NZ) yielded 10 different scales [23]. These scales have been shown to possess good psychometric properties in this population [22]. The internal consistency coefficients (α's) ranged from .63 to .80, with an average value of .73. The scale intercorrelations for male study members ranged from $-.30$ to .50, with a mean absolute value of .16. The scale intercorrelations for female study members ranged from $-.38$ to .41, with a mean absolute value of .17. The low magnitudes of these intercorrelations are similar to those obtained with the original instrument and illustrate the relative independence of the 10 MPQ scales [22].

For each scale, subjects were grouped according to a quartile split of scale scores. In all analysis, subjects appearing in the fourth quartile were compared to remaining sample. Preliminary analyses showed that this approach had little effect on the outcome when compared to an approach based on treating each scale as a continuous variable. It had the advantage of providing more meaningful odds ratios. A scale description appears in Table 1.

Statistical analysis

Development of cluster solution

Latent symptom factors were extracted using principal components analysis, with the criterion of an eigenvalue

Table 1
MPQ personality scales

	Description of the fourth quartile
Positive Emotionality scales	
Well being	Has a happy, cheerful disposition; feels good about self and sees a bright future.
Social closeness	Is sociable; likes people and turns to others for comfort.
Social potency	Is forceful and decisive; fond of influencing others; fond of leadership roles.
Achievement	Works hard; enjoys demanding projects and working long hours.
Negative Emotionality scales	
Alienation	Feels mistreated, victimised, betrayed and the target of false rumours.
Stress reaction	Is nervous, vulnerable, sensitive, prone to worry.
Aggression	Hurts others for own advantage; will frighten and cause discomfort for others.
Constraint scales	
Traditionalism	Desires a conservative social environment; endorses high moral standards.
Harm avoidance	Avoids excitement and danger; prefers safe tasks even if tedious.
Control	Is reflective, cautious, careful, rational, planful.

greater than or equal to one. Varimax rotation was used to obtain a solution containing orthogonal (or statistically independent) factors. A *k*-means cluster analysis was then applied to the latent factors. The *k*-means analysis began with a three-cluster solution and proceeded by generating increasingly complex cluster solutions (i.e., four, then five clusters). The choice of an appropriate starting point (i.e., three clusters) was guided by knowing that the items assessed features associated with both upper and lower GI syndromes; a health group was also expected.

Three criteria were used to select the appropriate cluster solution. First, comparisons were made of cluster membership across increasingly complex cluster solutions: if the more complex solution seemed to systematically break a large cluster into substantive subclusters, the complex solution was adopted; however, if the more complex solution seemed to randomly allocate members of several clusters to a new cluster or clusters, the simpler solution was adopted. Second, the distance metric (Euclidean distance) method was used to judge whether the within-cluster homogeneity was enhanced by moving to a more complex cluster solution. If the average distance metric was substantively reduced with a more complex solution, the more complex solution was favoured. Third, to preserve the reliability of within-cluster estimates, no cluster could be made up of less than 5% of the entire sample.

The interpretation of each cluster was aided by describing a cluster profile that comprised the mean score per factor per cluster. For each cluster, there is a series of mean scores centred about zero. A mean of zero indicates that the cluster is average (i.e., undistinguished) on that particular factor. The unit of measurement is the SD, because of the unit normal distribution of factor scores: a score of ± 2.0 indicates that the cluster is within the top or bottom 5% in terms of that factor. Scores of less than -1.0 or greater than $+1.0$ were interpreted as indicating clear differentiation; scores between 0.5 and 1.0 (positive or negative) were interpreted as indicating possible differentiation.

The relationship of cluster membership to psychiatric disorder and personality

The relationship of psychiatric disorder and personality measures to cluster group membership was described using gender-adjusted odds ratios. Logistic regression was used to estimate the odds ratios of being in a diseased cluster (vs. being in the health group), given differences in psychiatric history and personality characteristics. In the latter case, each diseased cluster was compared separately to the health group.

Results

The factor and cluster analysis was performed on the 971 subjects (499 males and 472 females), who provided GI symptom data. Psychiatric history was available for 888 of these subjects (452 males and 436 females) and personality data was available for 915 subjects (466 males and 449 females). For the psychiatric and personality measures, nonresponse was statistically independent of subject gender ($P = .32$ and $P = .25$, respectively) and GI cluster group membership ($P = .31$ and $P = .52$, respectively).

Cluster analysis

Factor structure

Details of the factor structure are shown Table 2. Five latent factors were identified from the principal components

Table 2
Latent GI symptom factors identified by principal components analysis

	Latent symptom factors				
Abdominal pain or discomfort associated feature	Pain associated with dysmotility	Pain associated with diarrhea	Pain associated with vomiting or nausea	Pain associated with constipation	Pain associated with ulcer-like symptoms
Made worse by food	0.69	0.33	0.15	0.05	0.13
Fullness	0.69	0.07	0.14	0.40	0.10
Early satiety	0.67	0.06	0.20	0.26	0.05
Occurring after meals	0.66	0.36	0.03	0.17	0.19
Nocturnal pain	0.49	0.28	0.27	−0.08	−0.01
Relieved by prescribed medicines	0.40	−0.04	0.11	−0.15	0.30
Increased bowel movements	0.16	0.90	0.14	0.03	0.09
Looser bowel movements	0.27	0.85	0.16	0.01	0.08
Relieved by bowel movement	0.12	0.71	0.01	0.46	0.18
Retching	0.12	0.06	0.83	0.06	0.11
Vomiting	0.14	0.11	0.82	0.03	0.03
Nausea	0.43	0.15	0.66	0.17	0.12
Harder bowel movements	0.03	0.16	0.04	0.86	0.12
Fewer bowel movements	0.24	0.05	0.08	0.82	0.06
Relieved by food or milk	−0.10	0.10	0.08	0.07	0.81
Occurring before meals	0.14	0.17	0.08	0.11	0.66
Relieved by indigestion medication	0.32	−0.10	−0.02	−0.04	0.65
Relieved by belching	0.12	0.26	0.09	0.27	0.48

analysis; these were labelled abdominal pain with (i) dysmotility-like symptoms such as early satiety and fullness; (ii) diarrhea; (iii) vomiting or nausea; (iv) constipation; (v) ulcer-like symptoms such as pain related to meals or belching. The minimum eigenvalue was 1.12 and the five factors accounted for 63.1% of the total variance (30.7%, 9.6%, 8.5%, 8.0% and 6.2%, respectively).

Cluster structure

Table 3 shows the results of the k-means cluster analysis. The cluster parameters represent mean factor scores for each latent symptom variable within each cluster of individuals.

The *k*-means analysis produced a six-cluster solution, which was made up of a health group, and five disease clusters. The disease clusters were defined by higher than average scores on a single symptom, and were labelled according to that symptom — i.e., pain associated with dysmotility-like symptoms, pain associated with diarrhea, pain associated with vomiting and nausea, pain associated with constipation and pain associated with ulcer-like symptoms.

Cluster membership was significantly related to subject gender (Table 4: $\chi^2 = 45.02$, $P < .0001$). The differences across gender were most evident in four groups: prevalence estimates were higher for males compared to females for the health group (71.7% vs. 57.8%), but were higher for females compared to males for pain associated with dysmotility symptoms (8.9% vs. 3.4%), pain associated with constipation (10.2% vs. 4.2%) and pain associated with nausea or vomiting (8.7% vs. 3.4%).

Psychiatric disorder and symptom cluster groups

Table 5 shows the associations between cluster membership and psychiatric history. In these analyses, subjects who are allocated to any disease cluster are compared to those who were allocated to the health group. Each disease cluster is then separately compared to the health group.

A diagnosis of *any* psychiatric illness was not associated with GI symptoms overall (i.e., of being a member of any cluster) and was not consistently linked to the membership of individual cluster groups. Of all effects considered, only two emerged as significant: a diagnosis of any psychiatric disorder at both ages 18 *and* 21 years increased the odds of membership of the dysmotility group (OR = 2.49, 95% CI: 1.29–4.77), while a diagnosis at age 18 *or* 21 years increased the odds of membership of the constipation group (OR = 2.00, 95% CI: 1.08–3.70).

Table 4
Characteristics of cluster groups according to subject gender

Cluster group pain associated with	Gender		
	Total, % (*n*)	Female, % (*n*)	Male, % (*n*)
Dysmotility	6.1 (59)	8.9 (42)	3.4 (17)
Diarrhea	8.3 (81)	8.3 (39)	8.4 (42)
Vomiting or nausea	6.0 (58)	8.7 (41)	3.4 (17)
Constipation	7.1 (69)	10.2 (48)	4.2 (21)
Ulcer-like	7.5 (73)	6.1 (29)	8.8 (44)
Health group	65.0 (631)	57.8 (273)	71.7 (358)
Total	100 (971)	100.0 (472)	100.0 (499)

Similar results were observed with respect to anxiety disorders. When diagnosed at either (but not both) 18 *or* 21 years, anxiety disorder significantly increased the odds of GI illness overall (OR = 1.42, 95% CI: 1.01–1.99), and significantly increased the odds of membership of the constipation group (OR = 2.11, 95% CI: 1.17–3.79). No further effects were observed.

Depressive disorder was clearly linked to GI illness overall, and was also linked to membership of the dysmotility and constipation clusters. A diagnosis of depression and age 18 *or* 21 years was associated with increases in the odds of 1.69 (95% CI: 1.27–2.25) for all GI illnesses, of 2.16 (95% CI: 1.12–4.16) for dysmotility and of 2.07 (95% CI: 1.13–3.80) for constipation. The corresponding odds ratios for a diagnosis of depression at both 18 *and* 21 years were 2.75 (95% CI: 1.97–3.84), 7.55 (95% CI: 3.47–16.42) and 4.18 (95% CI: 1.82–9.61). A diagnosis of depression at 18 *or* 21 years was also associated with an increase in the odds of belonging to the ulcer-like group (OR = 1.82, 95% CI: 1.00–3.29).

MPQ personality scales and cluster group membership

Table 6 shows the associations between cluster membership and fourth quartile scores on the subscales of the Multiphasic Personality Questionnaire. In these analyses,

Table 3
Cluster analysis of factors identified by principal components analysis

Cluster group pain associated with	Latent symptom factors				
	Pain associated with dysmotility	Pain associated with diarrhea	Pain associated with vomiting or nausea	Pain associated with constipation	Pain associated with ulcer-like symptoms
Dysmotility-like symptoms	2.54	0.53	0.003	− 0.17	− 0.37
Diarrhea	− 0.25	2.34	− 0.24	− 0.03	0.23
Vomiting or nausea	− 0.02	0.20	2.99	0.17	0.13
Constipation	0.31	0.04	− 0.28	2.70	− 0.05
Ulcer-like symptoms	0.49	− 0.42	− 0.27	− 0.36	2.67
Health group	− 0.30	− 0.32	− 0.18	− 0.25	− 0.31

Table 5
The association of psychiatric disorder to symptom cluster group

Psychiatric disorder	Cluster group: All cluster groups combined[a] OR (95% CI)	Dysmotility-like symptoms[b] OR (95% CI)	Diarrhea[b] OR (95% CI)	Nausea or vomiting[b] OR (95% CI)	Constipation[b] OR (95% CI)	Ulcer-like symptoms[b] OR (95% CI)
Any disorder						
No diagnosis	1.00	1.00	1.00	1.00	1.00	1.00
Diagnosis at 18 *or* 21	1.21 (0.88–1.68)	1.20 (0.60–2.43)	0.92 (0.54–1.59)	1.33 (0.68–2.60)	2.00 (1.08–3.70)*	1.05 (0.58–1.90)
Diagnosis at 18 *and* 21	1.22 (0.86–1.74)	2.49 (1.29–4.77)**	0.57 (0.28–1.15)	1.55 (0.76–3.14)	1.54 (0.76–3.13)	0.90 (0.46–1.78)
Anxiety disorder						
No diagnosis	1.00	1.00	1.00	1.00	1.00	1.00
Diagnosis at 18 *or* 21	1.42 (1.01–1.99)*	0.96 (0.46–1.96)	1.04 (0.57–1.90)	1.82 (0.94–3.55)	2.11 (1.17–3.79)*	1.42 (0.76–2.65)
Diagnosis at 18 *and* 21	1.47 (0.94–2.30)	1.88 (0.90–3.93)	0.92 (0.39–2.16)	2.16 (0.97–4.81)	1.21 (0.50–2.91)	1.31 (0.55–3.09)
Depressive disorder						
No diagnosis	1.00	1.00	1.00	1.00	1.00	1.00
Diagnosis at 18 *or* 21	1.69 (1.27–2.25)**	2.16 (1.12–4.16)*	1.10 (0.61–1.99)	1.63 (0.85–3.15)	2.07 (1.13–3.80)*	1.82 (1.00–3.29)*
Diagnosis at 18 *and* 21	2.75 (1.97–3.84)***	7.55 (3.47–16.42)***	0.54 (0.12–2.37)	2.58 (0.97–6.86)	4.18 (1.82–9.61)***	1.26 (0.36–4.38)

[a] Odds ratio represents the odds of being in any "diseased" group relative to the "health" group.
[b] Odds ratio represents the odds of being in the specified "diseased" group relative to the "health" group.
* $P < .05$.
** $P < .01$.
*** $P < .001$.

subjects who are allocated to "any" disease cluster are compared to those who were allocated to the health group. Each individual disease cluster is then separately compared to the health group.

The personality subscales were not strongly linked GI illness overall, or to membership of individual GI disease clusters. Membership of "any" diseased cluster was associated with a reduced odds of being in the fourth quartile for the well-being scale (OR = 0.64, 95% CI: 0.46–0.88), as was membership of the dysmotility cluster (OR = 0.43, 95% CI: 0.20–0.92); in contrast, membership of "any" diseased cluster was associated with an increased odds of being in the fourth quartile for the social potency scale (OR = 1.64, 95% CI: 1.18–2.28), as was membership of the dysmotility cluster (OR = 2.58, 95% CI: 1.42–4.70) and the diarrhea cluster (OR = 2.05, 95% CI: 1.21–3.45). Membership of the constipation cluster was associated with an increased odds of being in the 4th quartile for the Stress Reaction scale

Table 6
The association of personality measures to symptom cluster group

MPQ	Cluster group: All cluster groups combined[a] OR (95% CI)	Dysmotility-like symptoms[b] OR (95% CI)	Diarrhea[b] OR (95% CI)	Nausea or vomiting[b] OR (95% CI)	Constipation[b] OR (95% CI)	Ulcer-like symptoms[b] OR (95% CI)
Subscale						
Well being	0.64 (0.46–0.88)**	0.43 (0.20–0.92)*	0.62 (0.34–1.11)	0.58 (0.29–1.19)	0.78 (0.43–1.43)	0.76 (0.43–1.36)
Social potency	1.64 (1.18–2.28)**	2.58 (1.42–4.70)**	2.05 (1.21–3.45)**	1.27 (0.63–2.56)	1.70 (0.94–3.09)	0.92 (0.48–1.77)
Achievement	1.20 (0.86–1.68)	0.84 (0.38–1.86)	1.55 (0.91–2.67)	1.68 (0.87–3.22)	0.74 (0.35–1.55)	1.28 (0.72–2.30)
Social closeness	1.02 (0.76–1.38)	1.30 (0.74–2.31)	1.27 (0.76–2.10)	0.91 (0.49–1.67)	0.83 (0.46–1.47)	0.99 (0.57–1.74)
Stress reaction	1.32 (0.96–1.82)	1.53 (0.84–2.80)	1.10 (0.62–1.96)	1.44 (0.78–2.66)	1.76 (1.01–3.05)*	0.87 (0.45–1.69)
Alienation	1.00 (0.74–1.35)	1.16 (0.64–2.12)	0.66 (0.38–1.16)	1.46 (0.82–2.60)	1.01 (0.57–1.80)	0.96 (0.56–1.65)
Aggression	1.17 (0.84–1.65)	1.08 (0.50–2.32)	1.14 (0.65–2.03)	1.51 (0.75–3.03)	1.40 (0.73–2.68)	0.90 (0.49–1.65)
Control	1.32 (0.99–1.76)	1.11 (0.62–1.99)	1.33 (0.81–2.20)	1.42 (0.80–2.51)	1.07 (0.62–1.85)	1.66 (0.99–2.77)
Harm avoidance	0.80 (0.58–1.10)	1.48 (0.84–2.62)	0.82 (0.47–1.43)	0.79 (0.42–1.47)	0.63 (0.34–1.15)	0.57 (0.29–1.10)
Traditionalism	1.02 (0.72–1.45)	0.63 (0.27–1.43)	1.23 (0.68–2.21)	1.52 (0.80–2.90)	0.88 (0.44–1.74)	1.01 (0.52–1.94)

[a] Odds ratio represents the odds of being in any "diseased" group relative to the "health" group.
[b] Odds ratio represents the odds of being in the specified "diseased" group relative to the "health" group.
* $P < .05$.
** $P < .01$.

(OR = 1.76, 95% CI: 1.01–3.05). None of the remaining comparisons achieved statistical significance.

Discussion

This is the first population-based study to investigate the association of psychiatric disorders and personality traits with empirically derived GI "disease" clusters. We have found that chronic GI symptoms are common in this young population and that subjects fall into distinct and mutually exclusive symptom groups. Similar findings have been reported elsewhere [24]; we have now observed common clusters of upper and lower GI illnesses in four nations (Australia, the USA, Germany and Sweden), applying separate cross-sectional population-based surveys [24]. Thus, the data support our contention that the groupings observed in the current birth cohort are distinct and probably represent different pathophysiological abnormalities. The observation that specific psychiatric disorders were linked to some but not other clusters further supports this hypothesis.

We have observed that specific GI syndromes, as defined by cluster analysis, were strongly linked to depression (constipation and dysmotility in particular) and moderately linked to anxiety (constipation). These findings are consistent with data reported from the Epidemiologic Catchment Area (ECA) project in the US [15,25,26]; in the latter study, lifetime (and probably functional) GI symptoms were linked to lifetime psychiatric diagnoses, including a diagnosis of depression and specific anxiety disorders. However, data was not reported for individual symptoms in ECA and the use of lifetime prevalence data remains problematic: in particular, it remains uncertain as to whether subjects are reporting recurrent or even current symptoms, and the temporal proximity of GI symptoms and psychiatric history remains unknown. Thus, there remains a general lack of population-based data on the association between chronic GI symptoms or symptom complexes and psychiatric disorder as determined by DSM-III or -IV criteria.

The notion that functional GI syndromes have a psychiatric basis rests largely on studies of IBS in patient samples. These have frequently reported high rates of psychiatric illnesses (including anxiety and depression) in patients with IBS, with estimates ranging from 10% to 100%. The lowest prevalence of depression in IBS reported is 10%, which was found in a study by Dinan et al. [3] who evaluated consecutive IBS patients recruited from outpatient clinics. Anxiety disorders, in particular panic disorder, have also been linked to IBS in outpatient studies; it has been reported that approximately one-third of IBS patients have experienced an anxiety disorder sometime in their life as measured by standardised diagnostic criteria [11,13]. However, this is controversial. For example, Heitkemper et al. [10] evaluated female volunteers and did not find any significant differences between those persons with IBS and healthy controls on any of the DSM-III anxiety disorders including generalised anxiety disorder, agoraphobia, panic disorder or phobic disorder. The broad range of estimates for psychiatric disorder suggests that selection bias may be influencing some of the findings [3,13,14]. Moreover, many of the early studies also relied on clinical judgement to assess psychiatric illness in IBS patients, which may be heavily subject to interviewer bias [27,28].

The associations observed in the present study are very unlikely to be explained by selection bias, since the subjects were not selected based on current or prior health care seeking. An unresolved issue remains the causal chain in the relationship between psychiatric illness and GI symptoms. It is conceivable that chronic GI pain actually causes psychiatric illness, however, we do not have detailed GI symptom data on the cohort prior to age 26; thus, we cannot assess whether psychiatric diagnoses preceded symptoms from this study. An alternative explanation is that there is a common underlying predisposition to both psychiatric disorder (particularly depression) and GI illness (e.g., genetic or common environment), rather than one directly causing the other. Thus, while antidepressants appear to be useful therapeutic agents in patients with chronic unexplained GI symptom complexes such as the IBS, nonulcer dyspepsia and noncardiac chest pain [29], they also modulate GI function at the peripheral level and appear to be efficacious in sub-antidepressant doses. This implies they may work for reasons other than lifting depression.

The relationship between IBS and psychiatric illness in this cohort has previously been reported [16]. In our earlier study, we observed that there was no association between psychiatric diagnosis at ages 18 and 21 years (including separate diagnoses of anxiety or depression) and IBS at age 26 years, as assessed according to Manning and Rome II criteria. The concordance between our empirically derived clusters and IBS was generally quite good; roughly 55% of subjects, who met the Rome II criteria for IBS, were allocated to the dysmotility or constipation group; a further 28% were allocated to the diarrhea group (data not shown). We conclude that these clusters represent the underlying functional symptoms that form functional GI syndromes such as the IBS. We acknowledge that organic disease has not been excluded in this cohort; however, it is recognised that organic disease is rare in younger individuals [30]. An important implication of our finding is that psychiatric disorders may be more closely related to the symptoms that underlie the functional syndromes than specifically to the syndromes themselves.

The current study failed to detect clear-cut major personality differences amongst subjects with GI syndromes. These findings are consistent with data on IBS in patient samples. In studies that have used the Minnesota Multiphasic Personality Inventory (MMPI), IBS patients have been found to score higher than healthy controls on the following subscales:

hypochondriasis, depression, hysteria, schizophrenia, social introversion, ego strength and conversion [7–9]. Patients with IBS, however, have been found to have similar scores on the MMPI, except for psychoathenia, compared with inflammatory bowel disease, an organic disorder [7]. In studies using the EPI, IBS patients report higher mean scores on the neuroticism subscale vs. controls [2], although this difference just failed to reach significance in another population-based study of people with IBS, people with some bowel symptoms and controls [1]. Dinan et al., using the Eysenck Personality Questionnaire, found IBS patients to be more neurotic and introverted than patients with peptic ulcer disease [3]. On the other hand, no difference in personality characteristics were observed between IBS and peptic ulcer disease patients using the Edwards Personal Preference Schedule (EPPS), which measures the relative strength of 14 psychological needs [12]. Overall, the present study supports the literature, and suggests whether patients or community subjects are evaluated, people with functional GI symptoms do not have a unique personality profile.

The present study was community based and applied valid, standardised measures of GI symptoms, personality and psychiatric diagnoses. The results are likely to be generalizable to other Western nations, as the cohort is sociodemographically comparable with for example US whites. The cohort, however, was young (aged 26), and hence the results may not apply to older age groups. Another limitation was an inability to estimate the exact causal chain, although with further follow-up of this cohort such information should become available.

In conclusion, this community study adds support for the contention that psychiatric disease plays a pivotal role in the pathogenesis of chronic GI symptoms. However, no unique personality profile appears to characterise those with GI symptoms.

Acknowledgments

The Dunedin Multidisciplinary Health and Development Research Unit is supported by the Health Research Council of New Zealand. Data collection was partially supported by a NIMH grant no. MH-45070. The authors are indebted to the Study members for their participation and continued support.

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