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Words Fail Me: The Verbal IQ Deficit in Inflammatory Bowel Disease and Irritable Bowel Syndrome

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Background: Many chronic illnesses are accompanied by impaired cognitive functioning. In people with Inflammatory Bowel Disease (IBD), there is some research to suggest a decrement in verbal IQ (VIQ), when compared to people with Irritable Bowel Syndrome (IBS) and healthy controls. Although this is an important finding, it is necessary to ensure that such deficits are not due to methodological problems such as the failure to take into account pre-morbid functioning.

Methods: A total of 88 people (IBD, N = 29; IBS, N = 29; Controls, N = 30) completed the Wechsler Abbreviated Scale of Intelligence (WASI), the Wechsler Test of Adult Reading (WATR), the Trait Rumination Questionnaire (TRQ), the Center for Epidemiologic Studies Depression Scale (CES-D), and the General Health Questionnaire (GHQ-12).

Results: We found evidence of a VIQ decrement in both IBD and IBS groups when measured against both healthy controls and against their own pre-morbid IQ scores (WTAR-Predicted WAIS-III IQ measures). However, the decrement was larger (and of clinical significance) in the IBD group but not in the IBS group.

Conclusion: Some tentative evidence is presented which suggests that poor VIQ performance may be due in part to interference from excessive rumination.

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Key Words: IBD, IBS, neuropsychological function, IQ, anxiety, rumination, illness

Many chronic illnesses are accompanied by impaired cognitive functioning, eg, multiple sclerosis,[1,2] sickle cell disease,[3] chronic kidney disease,[4] type 2 diabetes mellitus,[5] and inflammatory bowel disease (IBD).[6] However, cognitive impairment is also evident in chronic illnesses in which no clear disease process has been identified, for example chronic fatigue syndrome[7,8] and irritable bowel syndrome (IBS).[6] The nature, quality, and quantity of deficits found depend on the type of illness studied and the instruments used to investigate cognitive function. For example, whereas multiple sclerosis is sometimes accompanied by a working memory deficit[9,10] and accuracy and speed in everyday activities,[11] it is IQ and attentional memory that are more often impaired in sickle cell disease.[12,13] Information processing speed may be reduced in people with diabetes,[5] people with chronic fatigue syndrome,[8] and in those with systemic lupus erythematosus,[14] while hepatitis C sufferers may experience problems with sustained attention and psychomotor speed.[15] Finally, verbal IQ deficits have been found in people with IBD and IBS,[6] while mild verbal memory deficits have been reported in patients with Lyme disease.[16] While depression and medication are potential protagonists in influencing neuropsychological performance,[7,17] cognitive deficits have nevertheless been found in studies that have controlled for these factors, eg, in studies investigating IBD.[6,18] Clearly, there is the need for a cognitive-based account of such dysfunction. Among populations with multiple sclerosis and chronic fatigue syndrome, such problems have been attributed to impaired cognitive processing[19] or limited cognitive resources.[20,21] The latter argument finds support in the work of Blomhoff et al,[22] who showed that people with IBS make increased use of attentional resources. Similarly, Matotek et al[9] argued that verbal fluency problems in people with multiple sclerosis may also be due to capacity limitations in working memory.

The findings reported by Attree et al[6] are an important contribution to our understanding of the cognitive effects of chronic illness. However, there are a number of methodological issues that need to be addressed in order to make firmer foundations on which to build our understanding. Specifically, Attree et al did not report premorbid measures of IQ that might otherwise be used to strengthen the case for cognitive deficits in these groups. Furthermore, they did not report the extent to which the deficit in verbal IQ was clinically significant. In the study that follows, we address both these questions. However, we also take the opportunity to speculate on a possible cause of the reduced verbal fluency, namely rumination. Scott and McIntosh[23] suggested that rumination uses cognitive resources in a way that necessarily

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TABLE 1. Means, Standard Errors, F, and *P*-values for ANOVA on Demographic and Psychosocial Variables

	IBS (n=29)		IBD (n=29)		Healthy (n=30)		Overall Comparison	
Variable	M	SE	M	SE	M	SE	F	*P*
Age	45	2.80	45	3.33	39	3.31	1.43	0.25
Years of education	12.2	.39	11.7	.36	12.3	.38	.87	0.42
Anxiety	15.10	.79	14.7	.71	12.5	.66	3.76	0.03
Depression	38.69	2.44	40.00	2.39	34.37	1.87	1.75	0.18
TRQ	33.31	1.30	31.03	1.38	32.01	1.47	.70	0.50
Motivation	10.03	.83	7.55	.91	7.83	.77	2.62	0.08
Distraction	10.41	.64	11.65	.83	11.33	.78	7.17	0.49
Emotion	12.86	.91	11.82	.77	12.33	.75	4.20	0.66
Years of illness	10.05	1.81	13.52	2.19	—	—	1.49	0.23

results in a decrement in cognitive performance and it is on this basis that we hypothesize that many of the cognitive impairments reported above are a direct consequence of rumination. In particular, we investigate the possibility that the impairment in verbal fluency in IBS reported by Attree et al[6] is due to excessive rumination, and so in the remainder of this introduction we examine the available evidence in support of this hypothesis.

The finding that people with chronic illness ruminate is well documented, as are its psychophysical effects. For example, Sullivan et al[24] showed that pain-rumination was the strongest predictor of perceived pain and disability among patients with soft-tissue injuries. Devoulyte and Sullivan[25] showed that people with upper respiratory tract illness also ruminate about their pain, and that this variable shows the strongest relationship with illness severity, even when controlling for initial symptom severity, duration of prior symptoms, and depression. In more direct support of our hypothesis, rumination has also been shown to affect cognitive processing: for example, there is a wealth of evidence showing that pain, or even the threat of pain, disrupts attentional processes.[26,27]

Whereas IBD is a rare organic disease with a clear medical pathology, IBS is a more common functional disorder with no clear cause and no lasting effective treatment. Although they have different etiologies, the conditions nevertheless have some symptom overlap and so IBS and IBD are often used as matched controls for one another. Rumination is very often related to a generalized anxiety associated with worries and concerns about an illness, particularly in people with chronic functional illness. Moreover, according to Brosschot and Thayer,[28] most patients suffering from medically unexplained somatic complaints spend a great deal of time worrying about their condition and therefore we expect to find higher levels of rumination in sufferers of IBS than IBD. In the following experiment we further investigated the possibility that the deficit found in verbal IQ[6] may be attributed, at least in part, to mental distraction caused by rumination.

MATERIALS AND METHODS

Participants

A total of 88 volunteers participated in the study (see Table 1 for full participant details). All participants met the inclusion criteria (people with IBD or IBS with an illness duration of at least 1 year, diagnosed by a qualified medical practitioner). None of the volunteers (illness or control groups) were suffering from a comorbid illness nor were they taking any psychoactive medication. Institutional ethical guidelines were followed and informed consent was obtained from all participants. People with IBD and IBS were recruited from a database that consisted of research-volunteers and also from local community-media notices. As an additional control, members of the healthy group were recruited from friends and relatives of the IBD and IBS participants. There were no statistical differences in mean age or years of education between the 3 groups.

Measures

Each participant completed 2 types of cognitive-function assessment (described below) as well as completing a series of depression, rumination, and anxiety scales (details also given below). All testing took place in the participants' homes and assessments were administered in a random order and executed according to the published instructions.

Neuropsychological Tests

The Wechsler Abbreviated Scale of Intelligence (WASI) was used to assess the participants' verbal, nonverbal and general cognitive functioning. The WASI was developed to ensure that the 4 subtest items in this test battery have

different but parallel forms to the Wechsler full-scale counterparts, thus the Verbal IQ, Performance IQ, and Full Scale IQ scores obtained from the WASI are linked to the Wechsler Adult Intelligence Scale (WAIS), Third Edition.[29]

Premorbid intellectual functioning was assessed by the Wechsler Test of Adult Reading (WTAR), UK Adaptation.[29] The WTAR is an assessment tool for estimating premorbid intellectual functioning of adults ages 16–89. This reading test comprises 50 words that have atypical grapheme to phoneme translations, which means that the participant is unlikely to be able to pronounce the words using previous learning. The rationale for using this test is:

"Unlike many intellectual and memory abilities, reading recognition is relatively stable in the presence of cognitive declines associated with normal aging or brain injury. The purpose of the $WTAR^{UK}$ is not for the assessment and diagnosis of developmental reading disorders, but rather for an initial estimation of pre-morbid intellectual and memory abilities (assuming a normal development of reading skills prior to injury or cognitive decline)" (WTAR manual, p 2).[29]

In order to summarize performance in terms of actual and WTAR-predicted WAIS-III scores, the participants' ages were needed in order to use the correct norms table given in the WTAR manual. The WTAR raw scores were converted to standard scores by means of the norms table. For instance, if the WTAR raw score was 30, for 16–24-year-olds, the standard score equivalent would be 94, whereas for 80–89-year-olds, the standard score would be 90.

Predicted VIQ and Observed VIQ Difference

This involved simply subtracting the observed VIQ scores from the predicted VIQ scores. The procedure involves comparison of actual difference scores with the expected difference score given in the WTAR manual. For each participant the actual difference score is compared with a minimum critical difference score necessary for statistical significance at a criterion level of $P < 0.15$ or $P < 0.05$ (see WTAR manual, p 272).[29] We used the more stringent $P < 0.05$ criterion level. Further details of the WTAR are given in the WTAR Manual.[29]

WTAR-Predicted WAIS-III Full IQ (FIQ), Performance IQ (PIQ), and VIQ

These scores were calculated by using tables given in the WTAR manual, using demographic and the WTAR scores to give the correct WTAR-predicted WAIS-III IQ values.

Scores in the text are reported as either observed-VIQ, as measured by the WASI or predicted-VIQ scores, as measured by the WTAR (WTAR-predicted WAIS-III VIQ). In the analyses of covariance carried out the data were analyzed with illness group (IBD versus IBS versus healthy) and type of VIQ score (observed VIQ versus predicted VIQ) as the between-participants and repeated measures variables, respectively. The covariates were age, sex, years of education, and anxiety.

Assessment of Rumination

The Trait Rumination Questionnaire (TRQ)[23] was used to measure participants' tendency to ruminate across subscales of emotionality, motivation, and distraction. Responses are on a 7-point Likert scale, which ranges from "does not describe me well" to "does describe me well."

Assessment of Depression

The Center for Epidemiologic Studies Depression Scale[30] is a 20-item self-report scale designed to measure depressive symptomatology in the general population. Responses are on a 4-point Likert scale. This scale was designed to measure current level of depressive symptomatology, with emphasis on the affective component, depressed mood.

Assessment of Anxiety

A shortened version of the General Health Questionnaire (GHQ-12[31] was used as a measure of anxiety. The questionnaire comprises 12 questions, and asked participants about their general level of happiness, experience of depressive and anxiety symptoms, and sleep disturbance over the last 4 weeks. Interpretation of the answers was based on a 4-point Likert response scale (symptom present: "not at all" = 0, "same as usual" = 1, "more than usual" = 2 and "much more than usual" = 3).

RESULTS

A 1-way ANOVA was carried out on the demographic and psychosocial variables. Since there was a significant difference in anxiety level between the groups, a post-hoc test using Tukey's HSD was carried out. It emerged that people with IBS were significantly more anxious than healthy controls ($P = 0.034$). Comparisons between the illness groups (IBS versus IBD) and between healthy controls and the IBD group were nonsignificant ($P = 0.900$ and $P = 0.092$, respectively). None of the other demographic or psychosocial variables were significantly different between the groups (see Table 1).

Although neither of the main effects was significant, there was a strong interaction between illness group and type of VIQ (observed VIQ versus predicted VIQ), $F_{(2,81)} = 13.14$, $P < 0.001$, $Eta^2 = 0.245$ (see Fig. 1). Although the groups differed very little in their predicted VIQ scores, the observed VIQ scores showed a large difference between groups: While healthy controls scored higher than predicted, the illness groups showed a strong decrement in observed VIQ, both relative to the healthy control group (thus replicating the pattern reported by Attree et al[6]) and relative to their own predicted VIQ scores.

To gain further insight into this finding, we used the

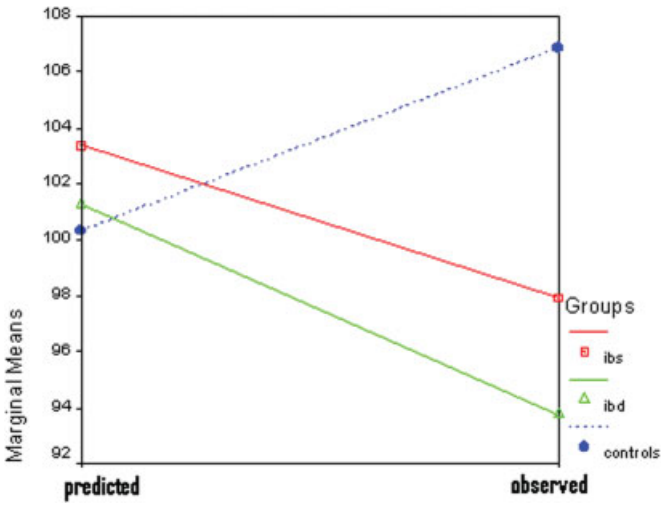

FIGURE 1. Predicted Verbal IQ and observed Verbal IQ for all groups.[Color figure can be viewed in the online issue, which is available at www.interscience.wiley.com.]

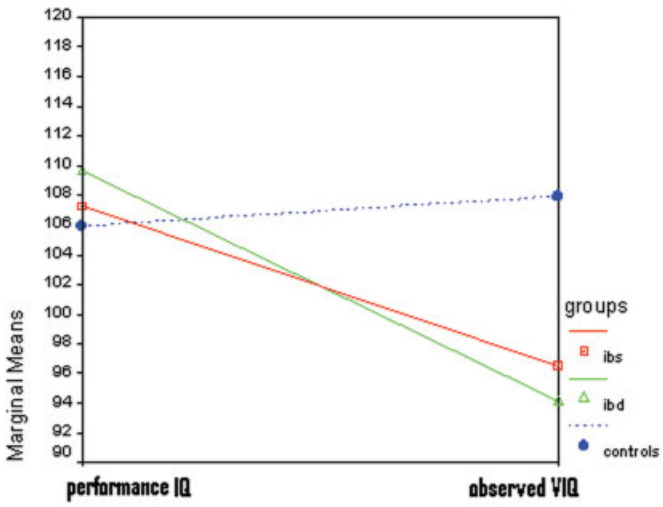

FIGURE 2. Performance IQ and observed Verbal IQ for all groups. [Color figure can be viewed in the online issue, which is available at www.interscience.wiley.com.]

difference between the predicted VIQ and the observed VIQ scores for each participant. The number of participants in each group who showed a statistically significant decrement was counted. This led to a finding of 27 (93.1%), 22 (77.3%), and 8 (27.3%) for IBD, IBS, and healthy controls, respectively. However, it is debatable whether this equates to true clinical significance, since many participants who showed a statistically significant difference were in the normal range of distribution for both PIQ and observed VIQ, and some were quite high. We therefore took a more cautious approach in deciding whether these statistically significant decrements were clinically significant by setting a criterion level for clinical significance: individuals were considered to have an unambiguous clinically significant observed VIQ deficit if their PIQ scores were over 89 and their observed VIQ scores were lower than 90; all of these individuals had at least a 10-point observed VIQ decrement. There were 15 participants who met this criterion. Of these, the majority (13; more than 80%) were in the IBD group. In fact, only 1 healthy comparison participant (3%) and 1 person with IBS (3%) showed a clinically significant deficit.

The difference scores were then entered as the repeated measure in an ANCOVA in which illness group was once again the independent factor and the covariates were age, sex, years of education, and anxiety. We once again found a strong interaction effect, $F_{(2,81)} = 11.517$, $P < 0.001$, $Eta^2 = 0.217$. Although all 3 groups were similar in terms of PIQ, the 2 illness groups (IBD and IBS) showed a marked deficit in observed VIQ relative to their own PIQ. In the healthy comparison group, the PIQ and observed VIQ were similar. These results, displayed in Figure 2, are very similar to those reported by Attree et al[6] even when the premorbid IQ (WTAR-predicted WAIS-III FIQ) was taken into account.

Given that the observed VIQ score is composed of 2 component tests, vocabulary and similarities, it was of interest to determine if just 1 or both of these tests added significantly to the overall observed VIQ decrement. To this end, multiple regression was carried out using scores on the vocabulary and similarities subtests as predictors. Only the vocabulary subtest was found to be a significant predictor of the PIQ-observed VIQ difference score (b = −0.40, beta = −0.36, t = −2.81, $P = 0.007$). The similarities subtest showed no relationship with PIQ-observed VIQ (b = 0.08, beta = 0.07, t = 0.54, $P = 0.595$).

Finally, we tested our hypothesis with regard to rumination. The rumination data revealed an interesting, although statistically nonsignificant pattern. In terms of the effort that participants used to try to reduce rumination (motivation subscale of the TRQ), IBS participants scored more highly (10.0) than either the IBD (7.6) or control (7.8) groups. This was supported by a strong, although nevertheless nonsignificant main effect, $F_{(2,85)} = 2.62$, $P = 0.08$. For the other 2 measures of rumination, distraction and emotion, there were no effects approaching significance, $F_{(2,85)} = 0.717$, $P = 0.49$ and $F_{(2,85)} = 0.420$, $P = 0.66$, respectively. A direct correlation between measures of rumination and observed VIQ for the groups separately showed that for the IBD group the distraction component of rumination negatively correlated with observed VIQ ($r = -0.44$; $P = 0.02$) and for the IBS group only the total of the rumination components correlated with observed VIQ ($r = +0.37$; $P = 0.05$). There was no significant correlation between any of the rumination measures and observed VIQ for the control group.

DISCUSSION

The results of the present study confirm Attree et al's[6] finding of a decrement in VIQ compared with PIQ in both IBD and IBS participants. Moreover, the observed VIQ decrement in these groups was found relative to both their own PIQ and to the observed VIQ/PIQ scores of the control group. It should be noted that in spite of the observed VIQ decrement, the mean observed VIQ scores for both illness groups were still within the normal range and therefore the decrement cannot be seen as indicative of more general impairment. However, this does not detract from the finding that both illness groups showed a relative observed VIQ deficit that could not be predicted on the basis of predicted VIQ. Whereas almost half of the IBD participants were shown to have a significant clinical decrement, only 1 participant in the IBS group showed a clinical decrement.

It is interesting to note that of the 2 components of observed VIQ, only the vocabulary subscale was able to predict the PIQ-observed VIQ difference score. The observed VIQ deficit in the illness groups therefore seems to reflect performance on the individual vocabulary subtest in particular. Whereas the similarities subtest assesses abstract verbal reasoning, the vocabulary subtest determines the degree to which a person has acquired, understood, and is able to express vocabulary. Given that the predicted VIQ was normal in both illness groups, we can assume that the problem lay either in comprehension or expression, the most likely of which would be verbal expression given the absence of any observable neurological damage. So we must ask why IBD and IBS patients might have a decrement in their verbal expression abilities? Our own tentative hypothesis was that illness groups, particularly the IBS group, would show a higher propensity to ruminate about their illness. However, we found that only motivation to reduce rumination (and not distraction) was marginally different between the groups. Given that the other measures of rumination were similar, there is therefore circumstantial evidence for a higher level of rumination among the IBS group, for which these participants expend more cognitive effort in trying to reduce. However, given the nature of the questionnaire the rumination in the present context may have been directed toward performance on the cognitive tasks, rather than illness-related.

The lack of significant differences in rumination may in part be attributed to the choice of instrument. We had predicted that IBS patients would suffer greater distraction from ruminative thought; however, our selection of the TRQ was useful in allowing us to measure motivation to overcome rumination (something that would be expected among illness groups who had not previously been predisposed to rumination) and this cognitive effort in itself may draw on limited resources. On the other hand, the TRQ may not have been sensitive enough to distinguish between the groups and so a health-based rumination questionnaire may be useful in future investigations of this kind. The finding that our rumination measure appeared to differentiate the 2 illness groups from one another, rather than from the control group, adds weight to the argument that different mechanisms underlie the observed VIQ deficit in the 2 types of illness, particularly in the light of our finding that the IBD group was far more likely to display clinical levels of observed VIQ deficit, whereas the IBS group remained within the normal range. Moreover, the finding that rumination was correlated with observed VIQ only in the illness groups, albeit in a complex pattern, warrants further research. For now, we tentatively suggest that, whereas observed VIQ deficits in IBS patients may have an underlying cognitive cause, those found in IBD patients may have a more organic basis, as suggested by Hollerbach et al.[18] They found that 75% of the IBD group in their study ($n = 24$) showed short-term memory dysfunction and focal white matter lesions. They concluded that the disease process itself was responsible for these effects.

In summary, our main findings are of a strong and clear observed VIQ decrement in people with IBD and IBS compared to healthy controls that is unlikely to be due to premorbid levels of intellectual functioning. Moreover, the decrement is particularly strong for people with IBD. Although it is clear that further assessments of neuropsychological functioning, addressing the underlying causes of observed VIQ decrement in IBD patients, are necessary, the present findings have revealed further insights regarding the pattern of intellectual functioning in people with IBD and people with IBS.

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