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Cognitive Function in People With Chronic Illness: Inflammatory Bowel Disease and Irritable Bowel Syndrome

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Recent research has shown that people with chronic illnesses often experience cognitive deficits, such deficits may be specific to a particular type of illness, reflecting the disease process itself, or they may be deficits that are common across a number of chronic illnesses. Our study investigated whether people with an organic disease (Inflammatory Bowel Disease) show cognitive dysfunction relative to the control group and people with a functional illness (Irritable Bowel Syndrome), and if so, to elucidate the mechanisms by which such dysfunction occurs. A quasi-experimental design using three groups of participants provided scores on IQ, memory, and cognitive flexibility. Differences in absolute scores were slight. However, a noticeable interaction effect was found between group and IQ: The illness groups showed a deficit in verbal IQ relative to both their own performance IQ and to that of the control group's verbal IQ. This verbal deficit cannot be explained by depression, cognitive load, or medication.

Key words: neuropsychological function, IBS, IBD, cognitive deficit, VIQ, PIQ

Chronic illnesses conditions that are generally characterized by symptoms that vary both in frequency and intensity over a long time span. Many chronic illnesses are invisible to others—that is, others are unaware that the individual has an illness unless they have been specifically told. Such illnesses are termed Invisible Chronic Illnesses and include Multiple Sclerosis (MS), Chronic Fatigue Syndrome (CFS), Systemic Lupus Erythematosus (SLE), Eosinophilia Myalgia Syndrome, Chronic Lyme Disease, Inflammatory Bowel Disease (IBD), and Irritable Bowel Syndrome (IBS). Chronic illnesses are often classified as organic or functional depending on whether a clear disease process has been identified (Thompson, 2001; Wessely, Nimnuan, & Sharpe, 1999). Although the usefulness of this distinction has not gone unchallenged (Lechin, van der Dijs, & Lechin, 1996), researchers have found the distinction beneficial—when looking for differences between a specific illness group and others, it is useful to include a matched illness group—people who suffer from similar symptoms, but who differ on the organic/functional domain.

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Although the psychosocial concomitants and consequences of chronic illness are increasingly being studied and taken seriously, much of the research into the cognitive aspects of chronic illness is limited both in quantity and to a small number of conditions. This body of research has been consistent in finding that cognitive deficits accompany such illnesses, although the underlying mechanisms are not fully understood and may be different according to the disease studied. Such effects may be subtle, and therefore difficult to find.

Research on cognitive deficits has focused mainly on the area of memory (Fischer, 1988; Gomborone, Dewsnap, Libby, & Farthing, 1993; Hanly et al., 1992; Joyce, Blumenthal, & Wessely, 1996; Marshall, Forstot, Callies, Peterson, & Scheck, 1997; O'Brien, Sahakian, & Checkley, 1993; Rissenberg & Chambers, 1998). However, studies have also been carried out on other cognitive domains such as attentional processes (Daly, Komaroff, Bloomingdale, Wilson, & Albert, 2001; Ginsburg et al., 1992), spatial ability (Daly et al., 2001; O'Brien et al., 1993), cognitive flexibility (Daly et al., 2001; Ginsburg et al., 1992), cognitive speed (Marshall et al., 1997), and concentration and reading ability (Daly et al., 2001; Ray, Phillips & Weir, 1993; Wearden & Appleby, 1997). A wide range of measures

have been used to investigate the extent to which cognitive deficits exist in chronic illness, including verbal tests, such as letter and category fluency and word association learning (Joyce et al., 1996); verbal memory and intelligence tests (DeLuca, Barbieri-Berger & Johnson, 1994); and language ability (Daly et al., 2001).

Most research in this area has been conducted on CFS (Daly et al., 2001; Joyce et al., 1996; Wearden & Appleby, 1997), MS (Bagert, Camplair, & Bourdette, 2002; DeLuca et al., 1994; Fischer, 1988; Foong & Ron, 1998), and SLE (Ginsburg et al., 1992; Hanly et al., 1992; Leritz, Brandt, Minor, Reis-Jensen, & Petri, 2002). Although some researchers report negative findings, the general pattern of research shows that cognitive deficits are clearly associated with chronic illnesses. What is unknown is the extent of, and the causal explanation for, such deficits.

Early reports of cognitive deficits in CFS had originally been interpreted as exaggerated complaints by people with CFS or personality difficulties (Buckley et al., 1999), but Lawrie, MacHale, Cavanagh, O'Carroll, and Goodwin (2000) suggested such cognitive deficits reflect impaired neuropsychological function. People with MS show deficits in learning and long-term memory, complex concentration (DeLuca, Johnson, & Natelson, 1993; Fischer, 1987), and verbal intelligence (DeLuca et al., 1994). More recently, the meta-analysis carried out by Zakzanis (2000) revealed that patients with MS have impairment across a range of neurocognitive domains. The pattern of these impairments differs between the different subtypes of MS: impairments relating to frontal–executive tasks are more commonly found in patients with chronic–progressive MS, whereas memory impairments tend to have a greater frequency in patients with relapsing–remitting MS. Zakzanis concluded that his meta-analysis illustrated the need for neuropsychological assessments which are tailored for the different MS sub-types.

Persons with SLE have been found to have poor performance on attention and visuospatial ability (Carbotte, Denburg, & Denburg, 1995; Ginsburg et al., 1992; Hanly et al., 1992); Carbotte et al. (1995) found that the impairment reflects an immune-mediated compromise of an underlying neural substrate, rather than the nonspecific effects of chronic illness or its treatment.

Attentional deficits have also been found in patients with Lyme disease (Rissenberg & Chambers, 1998), whereas patients with Seasonal Affective Disorder exhibited slowed response on spatial memory tests, which were unrelated to level of depression (O'Brien et al., 1993).

Although there is a growing body of evidence that cognitive impairments are found in chronic illnesses that are not thought to directly affect the central nervous system, there is a paucity of research concerning IBD and IBS.

IBD is composed ulcerative colitis (UC) and Crohn's disease (CD), both of which show a clear disease process (ulcers and inflammation of the colon, small bowel, or both in UC and CD, respectively, although CD can also affect the initial sections of colon in the region just beyond the ileum). The prevalence rate is low—approximately 1 in 600 suffering from UC and 1 in 1600 suffering from CD. Men and women are affected equally (National Association of Crohn's Disease & Colitis, 2001).

However, psychosocial factors may influence the course of the disease and the ways in which the patient copes. As the intestines become swollen, inflamed, and ulcerated, those affected may suffer from pain, weight loss, tiredness, and swollen joints, and symptoms can vary unpredictably both in frequency and intensity over a long period of time.

IBS is a chronic disorder affecting an estimated 15% to 22% of Western populations with a male to female sex ratio of 2:1 (Talley, Boyce, & Jones, 1997). Symptoms include abdominal pain (varies in intensity from mild to extremely severe), altered bowel habits (diarrhea, constipation, or an alternation of both), a feeling of urgency when needing a bowel movement, a feeling of incomplete evacuation, and flatulence. There is no consensus of opinion as to the cause or causes of IBS, and no treatment that is lastingly effective (Phillips, 1996). IBS is therefore classified as a functional disorder. IBD and IBS are often used as controls for each other, as there is symptom overlap between them. However, IBD is a progressive disease, and is therefore far more serious than IBS.

There are very few published studies investigating cognitive function in people with IBD or IBS. Hollerbach, Kullman, Geissler, Schoelmerich, and Andus (2000) investigated the extraintestinal involvement of the central nervous system (CNS) and its contribution to the pathophysiology of IBD, comparing 26 IBD patients with 24 controls. They found that the IBD group showed short-term memory dysfunction and morphologic CNS abnormalities, which were assessed as follows. Cognitive evoked cortical event potentials (p300-EP) were studied by using standard auditory evoked event-related cerebral potentials. Mean peak latency of the p300 wave was

significantly increased in people with IBD. Morphologic brain abnormalities were assessed by magnetic resonance imaging (MRI), which revealed focal white matter lesions in 75% of the patients studied, and these were associated with increased p300 latencies. Because they excluded patients with preexisting neurological disorder, who smoked, or who were taking CNS active medication, they concluded that the disease process itself was responsible for such effects. The differences were independent of disease type (UC or CD).

Gomborone et al. (1993) investigated IBS patients' ability to recall emotionally varying words and used IBD patients as an illness-control group. They also included healthy volunteers and a depressed group. The healthy volunteers showed a pattern of recognizing more neutral than positive and more positive than negative words. Both the depressed and the IBS group recognized more emotionally negative words than the healthy or IBD group. They concluded that there was, therefore, a confirmatory bias for negative words in people with IBS. However, their focus of study was not cognitive function, and their results were interpreted as being a psychosocial characteristic of people with IBS.

More recently, Fent et al. (1999) investigated the hemispheric preference and cognitive style of IBS patients by the way of the stimulation of afferent visceral signals. They tested the preferred hemisphere of the participants using two different methods. First, participants (IBS and control) were categorized as to whether their performance on a cognitive test was better for spatial or verbal tasks. The dependent variable was colonic distension sensitivity. Results were analyzed by an independent two-way analysis of variance. The only significant effect was the interaction: In the control group, the spatial participants had higher colonic distension thresholds than the verbal ones; and in IBS patients, the opposite was the case. The researchers also measured hemispheric preference by conjugate lateral eye movement: IBS and control participants were grouped as to whether their eye movements were to the right or the left. An identical analysis was performed using eye movements as the independent variable instead of the verbal–spatial factor. This effect was significant: right movers had higher thresholds than left movers. There was also a significant interaction effect. Fent et al. therefore concluded that their findings indicated that IBS patients exhibited a different interrelationship among hemispheric preference, colon sensitivity, and spatial–verbal information processing when compared with a small group of healthy control participants. They concluded that sensory changes in the gut are related to subtle changes in cognition, although the mechanisms are unclear. None of these studies, however, used both a matched illness group and a control group within the same study. This is necessary to clarify whether the deficits are specific or general, and to begin to elucidate the possible mechanisms for such deficits.

Several hypotheses have been proposed to account for cognitive dysfunction in persons with chronic illness. First, consider the impact of depression and medication uses: the assessment of cognitive functioning in chronic illness is complicated by the fact that measurements may be affected by depression (Martin, 1997) and also medication use (Lichter, Richardson, & Wyke, 1986) because both may affect neuropsychological performance. Cognitive deficits may not be entirely the result of these factors, however, because studies designed to tease out such factors show that persons with chronic illness continue to show such deficits (Carbotte et al., 1995; Gaudino, Masur, Kaufman, Sliwinski, & Krupp, 1995). Therefore, although depression is not the primary etiology for cognitive dysfunction, it does play a role in cognitive functioning (Gaudino et al., 1995; Lawrie et al., 2000)

Second, the disease process may itself be responsible for cognitive deficits. This would certainly apply to diseases such as primary MS, in which spinal cord damage and axonal loss within plaques in the brain are the hallmarks of this disease. Both Staples and Lincoln (1979) and Trimble and Grant (1982) claimed that MS patients with more advanced illness had more pronounced cognitive deficits. However, cognitive dysfunction has also been found in illnesses in which no CNS damage is believed to occur (Carbotte et al., 1995). If people with IBD show cognitive deficits relative to people with IBS, this suggests that the cognitive dysfunction may be specific rather than general, and it would be important to discover whether this was the result of the disease process itself.

Third, there is cognitive load. A further explanation for the cognitive deficits experienced by persons with chronic illness may be provided by cognitive load theory (e.g., Sweller, 1988). *Cognitive load* refers to the total amount of mental activity imposed on working memory at any one time. Baddeley (1992) suggested that "worry and self-concern" primarily used the resources of the central executive and the articulatory loop. Persons with chronic illness have an additional cognitive load of monitoring varying and unpredictable levels of their symptoms, which may account for the "mild deficit in effortful cognition" noted by DeLuca

et al. (1995). Because working memory is extremely limited in both capacity and duration, this additional cognitive load will, under some conditions, have an impact on memory, thought, learning, and problem solving. Blomhoff, Spetelan, Jacobsen, and Malt (2001) found that people with IBS have an "increased use of brain-attentional resources," and both Litvan et al. (1988) and Marshall et al. (1997) proposed that the most reasonable explanation for MS and CFS patients' poor cognitive performance was a working memory deficit as a consequence of cognitive resource limitations.

Although a body of research describes the type of cognitive dysfunction associated with chronic illness in several illness groups, the mechanisms by which such deficits arise are still unclear. To begin to clarify these, studies that include matched illness groups and a control group are needed. In this study, neuropsychological tests on people with IBD enable the nature and extent of any cognitive difficulties to be quantified objectively by comparing cognitive function on a sample of people with an organic disease (IBD) against not only a sample of healthy persons, but a control sample of persons with a functional illness as well (IBS). IBD and IBS illness groups are ideal groups on which to test such hypotheses because they suffer from similar symptoms, but differ in the organic/functional aspect. In a small pilot study conducted by the authors, specific deficits were found in verbal IQ only. This study was carried out with 14 participants (10 women and 4 men, with a *M* age of 60.2), who had all been diagnosed with IBS. Test scores of the sample were analyzed with reference to normative data for the Wechsler Abbreviated Scale of Intelligence (WASI), and a highly significant effect regarding Verbal IQ (VIQ) in relation to Performance IQ (PIQ) was found (VIQ < PIQ). This finding was unexpected, because it is not clear why VIQ alone would be affected by illness. If further studies confirmed this effect, doubt would be cast on the cognitive load theory, because this would predict a relative deficit in PIQ rather than VIQ. To extend this investigation, tests of attention and incidental memory have been included to provide a broader profile of neuropsychological assessment.

Based on the literature relating to other chronic illnesses, we hypothesize that cognitive deficits will occur in both IBS and IBD groups, and that if the disease process is involved, the IBD group will show greater deficits. Based on findings from a previous pilot study, we hypothesize that both the IBD and IBS groups will score significantly lower on VIQ than the control group.

Method

Participants

A total of 92 volunteers participated in the study. Persons with IBS or IBD fulfilled the following inclusion criteria: diagnosis by a qualified medical practitioner and illness duration of at least 1 year. Participants (with IBS or IBD or control group) were excluded if they were taking psychoactive medications or suffering from a comorbid illness. All participants gave informed consent, which followed institutional ethics guidelines. Following the application of the inclusion and exclusion criteria, 70 participants remained.

A total of 27 people with IBS took part in the study (24 women, 3 men). The mean age of the IBS group was 45.3 years, and they had an average of 12.3 years of education. Their average duration of illness was 9.6 years. The participants with IBS were recruited from a database consisting of people with IBS who had previously expressed an interest in taking part in research.

The IBD group consisted of 16 people (12 women, 4 men). UC and CD were considered together, as they share many symptoms, have a similar clinical course, and are considered to be one group by researchers in the area (Hollerbach et al., 2000; Searle & Bennett, 2001). The mean age of the IBD group was 40.4 years, and they had an average of 11.8 years of education. Their average duration of illness was 15.2 years. Participants in the IBD group were recruited from media advertisements in the local community.

The 27 control individuals (24 women, 3 men) had a mean age of 42.3 years and an average of 12.41 years of education. They were recruited from friends and relatives of the participants in the IBS and IBD groups. The participants in the three groups did not differ significantly in terms of age or years of education.

Measures

Each participant completed three types of cognitive function assessment and a depression scale. To control for possible order effects, such as fatigue, the assessments were administered in a Latin square order; for example, Participant 1 received the WASI, which has four subtests, followed by the incidental memory assessment, the Stroop Color Word Test, and the Center for Epidemiologic Studies Depression Scale (CES–D); Participant 2 received the incidental memory assessment, followed by the Stroop Color Word Test, the CES–D, and the WAIS, and so on. All testing took place in the participants'

homes, and took between 1.5 and 2 hr to complete. All tests were administered according to the published instructions. Brief details of the measures follow.

Neuropsychological tests. The WASI was used to assess the participants' verbal, nonverbal, and general cognitive functioning. The WASI was developed to ensure that the four subtest items in this test battery have different but parallel forms to the Wechsler full-scale counterparts, thus the VIQ, PIQ, and Full Scale IQ scores obtained from the WASI are linked to the Wechsler Adult Intelligence Scale–Third Edition (Weschler, 1999).

Assessment of incidental memory, that is, spatial and object recognition memory, was carried out using a computer-generated virtual environment (VE). Pugnetti et al. (1998) found that this form of assessment could identify "aspects of behaviour that are normally inaccessible during traditional formal psychometric testing." The VE used in this research has identified cognitive impairments in patients with vascular brain injury (Rose et al., 1999) and traumatic brain injury (Rose, Attree, Brooks, & Andrews, 2001) compared to healthy control participants. The VE was constructed using Superscape VRT software and run on a desktop computer. Exploration of the VE was controlled by an analog joystick. The environment depicted four interconnected rooms in a bungalow—a bedroom, a music room, a lounge, and a kitchen. In the rooms were 20 objects (e.g., a camera, an abstract picture, a bottle of wine). Performance on the spatial recognition test was scored on a predetermined criterion that allocated marks according to number and shapes of rooms, entry doorway positions, exit walls, and exit passageway positions, that participants correctly identified; this gave a total maximum score of 20. Object recognition memory was also scored out of a possible 20 points. To correct for guessing, incorrectly recognized lure objects were subtracted from correctly recognized target objects (Baddeley, 1997).

The color word test was used as a general test of selective attention and cognitive flexibility (Graf & Utl, 1997; Stroop, 1935); that is, it assessed performance on a task in the presence of conflicting or ambiguous stimuli. This test consisted of stimuli with two dimensions, color and verbal meaning, and involved the use of two cards. Each card measured 11 in. × 8.5 in. and contained 28 words (four rows of seven words). The Word Card showed a series of color words (red, green, yellow, blue) printed in black ink; The Incongruent Word Card displayed the color words printed in mismatched ink (e.g., the word red printed in green letters). Participants were asked to focus on the color of the words and name these colors while ignoring the conflicting word meaning. Scores on the Stroop Color Word Test were obtained by calculating the difference between the time taken to complete the Word Card and the Incongruent Word Card.

Assessment of depression. The CES–D (Radloff, 1977) 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. The CES–D has been shown to be both reliable and valid (Beekman et al., 1997).

Results

Preliminary analysis showed that groups differed significantly on depression, $F = 9.56$, $p < .001$, and length of illness, $F = 21.77$, $p < .001$. To reduce the variance attributable to these variables and other social demographic measures (age, sex, and years of education) these were entered as covariates in further analyses. Summary statistics for the cognitive function measures and depression scale are shown in Table 1.

First, the IQ measures were considered. Data were entered into a split plot two-way analysis of covariance (ANCOVA) with IQ (Performance and Verbal) as the repeated measures factor and groups as the independent factor. There was a significant interaction between the groups and type of IQ, $F2, 62 = 12.858$, $p < .001$, $\eta^2 = .286$ (Figure 1).

Whereas the control participants scored identically on PIQ and VIQ, the illness groups scored considerably lower on the VIQ measure, both in relation to their own PIQ scores and in relation to the control group. It can be seen that there was little difference between groups on performance IQ. Because verbal deficits had been hypothesized to occur in the illness groups, a planned comparison was made between IBD and IBS groups combined and the control group on VIQ. Results showed there was a significant difference, $F1, 62 = 11.295$, $p = .001$.

The object and spatial recognition measures were considered next. An identical analysis was performed on these measures. A significant interaction was found between the group and the recognition measures, $F2, 62 = 5.503$, $p = .006$, $\eta^2 = .151$. Compared to the other groups, the IBD group scored significantly lower on object recognition but significantly higher on spatial recognition (Figure 2). A series of simple effects post-hoc tests were carried out to

Table 1. *Test Score Means and Standard Errors for Persons With IBS, IBD, and Control Participants*

	IBS[a]		IBD[b]		Control[c]	
Test Name	**M**	***SE***	**M**	***SE***	**M**	***SE***
CES–D	18.63	2.33	20.81	2.57	8.78	1.41
WASI						
Full IQ	102.79	2.26	101.79	3.44	107.71	2.67
Performance IQ	109.62	2.29	109.65	3.48	107.07	2.71
Verbal IQ	95.79	2.08	94.24	3.16	107.04	2.46
Recognition Memory						
Object	11.12	.61	9.03	.93	10.97	.73
Spatial	12.79	.63	14.73	.95	11.97	.74
Stroop Test	12.68	1.07	10.60	1.62	11.92	1.26

Note: IBS = irritable bowel syndrome; IBD = inflammatory bowel disease; CES–D = Center for Epidemiologic Studies Depression Scale; WASI = Wechsler Abbreviated Scale of Intelligence. Neuropsychological test scores were adjusted for depression, length of illness, and demographic variables.
[a]$n = 27$. [b]$n = 16$. [c]$n = 27$.

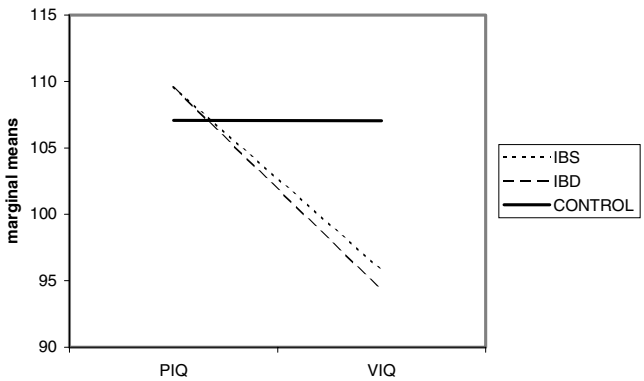

Figure 1. This figure demonstrates the significant interaction between the groups (IBS = Irritable Bowel Syndrome; IBD = Inflammatory Bowel Disease) and IQ score (VIQ = mean verbal IQ; PIQ = mean performance IQ). The data were covaried for depression, length of illness, and demographic variables.

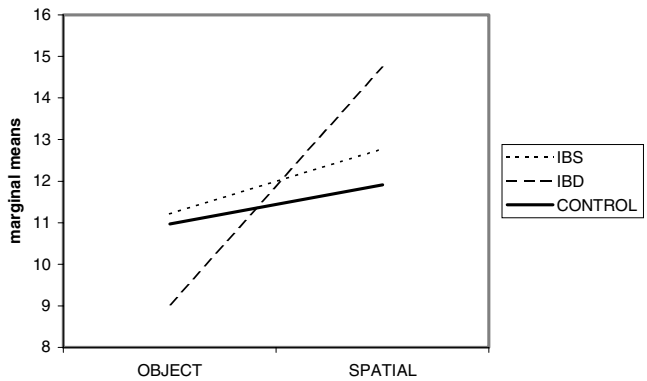

Figure 2. This figure demonstrates the significant interaction between the groups (IBS = Irritable Bowel Syndrome; IBD = Inflammatory Bowel Disease) and recognition score (object = mean object recognition score; spatial = mean spatial recognition score). The data were covaried for depression, length of illness, and demographic variables.

further examine the differences. A Bonferroni correction was made using $p = .10$ for an exploratory study with small sample size (Stevens, 2002). This gave a criterion probability value of .033.

For all groups and both measures, scores were in the normal range. Post-hoc tests gave F values with associated probability values all more than .033. Finally, a one-way ANCOVA was then performed on the Stroop measure. This showed that groups were not significantly different, $F2, 62 = .657$, $p = .522$.

Discussion

Results showed that there were only slight differences between the groups in terms of absolute IQ and recognition scores. Looking at the adjusted means in isolation, it might be thought that there are no differences in cognitive function between the illness and control groups. For instance, it is clear that there are no important differences between groups on the Stroop Color Word Test, or on absolute scores of recognition memory. In relation to IQ, the interesting finding is in the interaction: Whereas the control group score almost identically on Full Scale IQ, PIQ, and VIQ (as would be expected), the illness groups show a deficit in VIQ relative to both their own PIQ and to the IQ scores attained by the control group. This finding confirmed results of a pilot study carried out previously by the authors.

This verbal deficit showing up in both illness groups relative to the control group and confirming previous

results is not only interesting, but also potentially may indicate some clinical significance. In a normal population, the expected mean VIQ–PIQ discrepancy is zero (Weschler, 1992). For example, Bornstein (1983) argued that the Verbal and Performance scales are the most valid Weschler index of dysfunction, and consequently have an important clinical role; other authors have found that a low VIQ relative to PIQ may be an accurate indicator of left hemisphere dysfunction, and a low PIQ relative to VIQ is a more nonspecific indicator of brain damage or dysfunction (Warrington, James, & Maciejeweski, 1986; Whelan, 1998).

However, in relation to this study, the reasons for the discrepancy between VIQ and PIQ are unknown. Indeed, this unusual pattern of PIQ being higher than VIQ for both of the illness groups is found in only a few other populations. For example, Hill (1995) found that people with early-onset diabetes mellitus exhibited a VIQ decrement, relative to PIQ, although this pattern was not found in adults with later-onset diabetes. Siegal, Minshew, and Goldstein (1996) pointed out that a VIQ < PIQ profile has traditionally been thought to be a typical pattern associated with autism, although this profile has not been universally demonstrated.

However, the studies by Hill (1995) and Siegal et al. (1996) do not offer any obvious explanation for the relative PIQ < VIQ deficit found in the IBS and IBD groups. The relative deficit is unlikely to be the result of cognitive load, because PIQ was not affected, and indeed, was high. It is also unlikely to be the result of attentional or motivational factors, because here again, PIQ would have been expected to show a deficit. Medications cannot have affected participants, because persons on CNS active medications were not included in the study. Depression and other relevant psychosocial variables were covaried out, and in any case, depression would have affected other measures as well as VIQ.

In respect of recognition memory, results from this study show that the IBD group was different from the other two groups, scoring low on object recognition, but unexpectedly high on spatial recognition—again, rather than absolute differences, the interesting finding was in the interaction. This finding, if replicated, may indicate that the disease process is involved in this type of cognitive function. Belger et al.'s (1998) study using fMRI indicated that spatial and object working memory tasks use different hemispheric networks; is it, then, the case that persons with IBD have a different pattern of hemispheric preference? Fent et al.'s (1999) study found that people with IBS exhibited a different interrelationship among hemispheric preference, colon sensitivity, and spatial–verbal information processing, and their finding warrants further investigation with other patient groups. Nevertheless, although the interaction between group and type of recognition memory was statistically significant, further work clearly must be carried out to determine whether this finding is robust.

Scores on the Stroop Color Word Test were not different between the groups, confirming that persons with IBS or IBD do not have apparent deficits in focused attention or mental speed. However, this does not mean the cognitive load theory is entirely discounted. As mentioned previously, persons with chronic illness must constantly monitor the varying and unpredictable levels of their symptoms, which is assumed to have an impact on the limited capacity and duration of working memory. Any impact of cognitive load on cognitive function may be apparent only in tasks that clearly tap the ability to divide attention—which reduces processing capacity. Further research is indicated using a test of divided attention, such as the Paced Auditory Serial Addition Task (Gronwall & Sampson, 1974).

In summary, this preliminary study suggests that there is a subtle effect of brain–gut interactions on neurological function: there appear to be commonalities in cognitive impairments within the IBS and IBD groups that are unexplained by disease activity, and the IBD group's scores on the object and spatial recognition tests need further investigation. There is no published work investigating cognitive deficits in IBS, and our finding that people with IBS show a pattern of results similar to that of IBD but different from healthy controls is highly significant, for it suggests that IBS might be similar to IBD in respect of biophysical markers. These speculations must be tested experimentally.

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