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Bulimia nervosa with co-morbid avoidant personality disorder: behavioural characteristics and serotonergic function

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ABSTRACT

Background. Separate lines of research link lowered serotonin tone to interpersonal submissiveness and bulimia nervosa (BN). We explored the impact of co-morbid avoidant personality disorder (APD), as a proxy for submissiveness, on behavioural inhibition and serotonin function in women with BN.

Method. Participants included women with BN with co-morbid APD (BNA+, $N=13$); women with BN but without APD (BNA−, $N=23$), and control women with neither BN nor APD ($N=23$). The women were assessed for psychopathological tendencies and eating disorder symptoms, and participated in a computerized laboratory task that measured behavioural inhibition and disinhibition. Participants also provided blood samples for measurement of serial prolactin responses following oral administration of the partial 5-HT agonist meta-chlorophenylpiperazine (m-CPP).

Results. The BNA+ group had higher scores than the other groups on self-report measures of submissiveness, social avoidance, restricted emotional expression, affective instability and self-harming behaviours. Compared with the other groups, the BNA+ group tended to be more inhibited under cues for punishment on the computerized task and to have blunted prolactin response following m-CPP. The bulimic groups did not differ from each other on current eating symptoms or on frequencies of other mental disorders.

Conclusions. Findings indicate that women with BN and co-morbid APD may be characterized by interpersonal submissiveness and avoidance, affective instability, self-harm, behavioural inhibition in response to threat and lower sensitivity to serotonergic activation. These findings may indicate common, serotonergic factors, associated with social submissiveness, behavioural inhibition to threat and BN.

INTRODUCTION

Alterations in the functioning of the central serotonin (5-hydroxytryptamine, 5-HT) system have been widely implicated in bulimia nervosa (BN). For example, women with BN have reduced binding of 5-HT uptake inhibitors in platelets (Marazziti *et al.* 1988; Steiger *et al.* 2000), decreased levels of 5-HT metabolite 5-HIAA in cerebrospinal fluid (CSF) (Jimerson *et al.* 1992) and blunted neuroendocrine responses to 5-HT agonists like meta-chlorophenylpiperazine (m-CPP) (Brewerton *et al.* 1992; Steiger *et al.* 2001*a*, *b*) and fenfluramine (Jimerson *et al.* 1997). Furthermore, acute depletion of the 5-HT precursor tryptophan results in a temporary exacerbation of symptoms

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in women with active BN (Weltzin *et al.* 1995) and in women with BN in remission (Smith *et al.* 1999).

Serotonergic function in BN appears to be associated with several factors. First, 5-HT function may correlate with severity of BN symptoms. For example, blunting of neuroendocrine response to fenfluramine is correlated with binge-eating and purging symptoms (Monteleone *et al.* 2000; but compare Steiger *et al.* 2001*a*, *b*). Secondly, 5-HT function in BN may be influenced by the presence of co-morbid symptoms such as anxiety or depression (Brewerton, 1995) as per neuroendocrine response to tryptophan but not m-CPP (Brewerton *et al.* 1992). Personality traits may also influence 5-HT function in BN. For example, Waller *et al.* (1996) found that self-reported hostility in women with BN was related to smaller neuroendocrine responses following administration of the 5-HT_{1a} receptor agonist, buspirone. Furthermore, our group has shown that greater self-reported impulsivity predicts reductions in platelet paroxetine binding in BN (Steiger *et al.* 2001*a*), and that a history of self-mutilation or suicidal behaviour coincides with further blunting of m-CPP stimulated neuroendocrine responses (Steiger *et al.* 2001*b*). Such findings demonstrate that, while serotonergic function is lowered in BN, further reductions of 5-HT status in BN are associated with hostile and impulsive personality traits.

It is important to note that some indices of 5-HT function 'normalize' along with symptom remission. For example, neuroendocrine responses to fenfluramine (Wolfe *et al.* 2000) and m-CPP (Kaye *et al.* 1998) in women recovered from BN are comparable with those of healthy women with no history of BN. Such findings suggest that some indices of 5-HT functioning in active BN may reflect nutritional or state-related factors. Other indices of 5-HT function remain altered even after symptomatic remission. For example, compared with control women, women remitted from BN have increased levels of CSF 5-HIAA (Kaye *et al.* 1998) and reductions of medial orbital frontal cortex 5-HT_{2A} binding (Kaye *et al.* 2001). In summary, some indices of 5-HT status in BN may reflect state factors (such as nutritional factors or severity of active BN symptoms) while others may be markers of trait vulnerability in BN. It may be that 5-HT function in BN reflects the combination of dieting, binge-eating, purging, as well as psychosocial–interpersonal stressors, perturbing and interacting with an already vulnerable 5-HT system (Brewerton, 1995).

The current report was designed to further explore the implications of personality pathology for 5-HT status in BN. In this light, we list some important observations: (1) a recent meta-analysis of 28 studies on personality disorders (PDs) in BN (Rosenvinge *et al.* 2000) found that DSM-IV (American Psychiatric Association, 1994) Cluster B (i.e. antisocial, borderline, histrionic and narcissistic) and Cluster C (i.e. avoidant, dependent, obsessive–compulsive) PDs were both – and equally – common (at 44% respectively) in BN; (2) compared with research on Cluster B PDs, there is very little research on the role of PDs from Cluster C in BN; (3) of the Cluster C PDs, Avoidant PD (APD) appears to be the most common in both BN (e.g. Gartner *et al.* 1989; Yates *et al.* 1989) and in the general population (Torgersen *et al.* 2001); and (4) there is indirect evidence on clinical and preclinical fronts that APD may be related to reduced serotonergic tone. The clinical evidence is that serotonin re-uptake inhibitors have been shown to reduce frequency of APD symptoms. For example, APD symptoms are shown to reslove after citalopram and sertraline in patients with depression (Ekselius & von Knorring, 1998), after venlafaxine in patients with social phobia (Altamura *et al.* 1999) and after fluvoxamine in patients with body dysmorphic disorder (Phillips & McElroy, 2000). Preclinical evidence suggests that behavioural traits significant for APD may involve reduced 5-HT activity. For example, increased 5-HT levels in brain, particularly in forebrain, have been implicated in dominant behaviours in rodents (File *et al.* 1981), small reptiles (Baxter *et al.* 2001) and monkeys (Raleigh & McGuire, 1991). Evidence suggests that serotonin plays a similar role in the regulation of dominant and submissive behaviour in humans as well. For example, in studies of healthy participants, Moskowitz *et al.* (2001) found increases in self-reported dominant behaviours following a 2-week period of tryptophan loading while Tse & Bond (2002) found decreases in other-rated submissiveness with citalopram. An increase in 5-HT thus increases dominance and decreases

submissiveness in humans; such findings are interesting given that APD is characterized by patterns of behavioural, interpersonal and emotional restraint or avoidance (American Psychiatric Association, 1994).

The current study was designed to examine the implications for co-morbid APD for behaviour and serotonergic function in BN. Three groups of women participated: those with BN and co-morbid APD, those with BN but without co-morbid APD, and normal-eater control women with no PD. Serotonergic function was measured by serial prolactin responses to the partial 5-HT agonist, meta-chlorophenylpiperazine (m-CPP). The hypotheses were that bulimic patients would show decreased 5-HT function and that bulimic women with APD would show further reductions. A related but secondary goal was to compare the groups on behavioural measures of inhibition and disinhibition, as well as and self-report measures of eating symptoms and personality pathology. Here the expectations were that the group with APD would be more behaviourally inhibited and would report more trait submissiveness and social avoidance.

METHOD

Participants

Women with BN

Women ($N=36$) aged 18–45, and meeting DSM-IV (American Psychiatric Association, 1994) criteria for a bulimic-spectrum eating disorder, were recruited through out-patient consultations at a specialized hospital-based eating disorders programme. DSM-IV criteria were established using the Eating Disorders Examination (EDE) (Fairburn & Cooper, 1993). Twenty-eight of the women met criteria for BN, purging subtype; four of the women met criteria for BN, non-purging subtype; and four of the women met criteria for 'subclinical' BN, purging subtype (where participants reported bingeing and purging within the past 3 months, but less than twice a week on average). Given characteristic weight fluctuations in BN, modest deviations from normal body mass index (BMI) (Beaumont *et al.* 1988) were accepted, but cases with extreme BMIs (under 17 or over 28), and individuals meeting DSM-IV criteria for anorexia nervosa, were excluded. To test our primary and secondary hypotheses, bulimic participants were subdivided into two groups, based on the presence (BNA+, $N=13$) or absence (BNA−, $N=23$) of APD (with SCID-II criteria) (First *et al.* 1996). There were no differences in terms of BN subtype (described above) across the two groups, with chi-square testing.

Controls

Normal-eating women (control, $N=23$) were recruited who had no identifiable eating or personality (according to responses on the ED and SCID-II) disorder. Control women also denied a history of psychological/psychiatric treatment.

Participants were recruited from an ongoing project examining the implications of personality and other variables for response to m-CPP in BN. The first report from this dataset examined impulsivity and affective instability (BN, $N=26$; control, $N=22$) (Steiger *et al.* 2001*a*) and data are shared with the current report for 23 BN and 21 control participants. The second report examined self-destructiveness (BN, $N=40$; control, $N=21$) (Steiger *et al.* 2001*b*) and data are shared with the current report for 36 BN and 21 control participants. The third report examined childhood abuse (BN, $N=35$; control, $N=25$) (Steiger *et al.* 2001*c*) and data are shared with the current report for 31 BN and 23 control participants. Participant inclusion for each project depended on availability and on the specific measures needed (e.g. controlling for different psychopathology and developmental experiences).

Mean ages of the three groups were compared using univariate ANOVA, and there was a non-significant trend towards a group difference ($F(2,56=2{\cdot}77$, $P=0{\cdot}07$), however pairwise testing with least significant difference (LSD) tests revealed no significant age differences among the groups. Mean ($\pm$S.D.) for the control, BNA− and BNA+ groups were 24·9 ($\pm$7·5), 22·3 ($\pm$3·4) and 26·5 ($\pm$4·2) years respectively. Further, the three groups did not differ significantly on mean BMI using univariate ANOVA ($F(2,56)=0{\cdot}04$, $P=0{\cdot}96$). Mean ($\pm$S.D.) for the control, BNA− and BNA+ groups were respectively 22·0 ($\pm$2·0), 21·8 ($\pm$3·1) and 21·9 ($\pm$3·0) kg/m^2.

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Measures

Eating disorder diagnoses and symptoms

Eating Attitudes Test-26 (*EAT*-26) (Garner *et al*. 1982)

This is a 26-item measure of eating disorder symptoms and has established psychometric properties (e.g. Garner *et al*. 1982; Banasiak *et al*. 2001).

Eating Disorder Examination (*EDE*) (Fairburn & Cooper, 1993)

The EDE establishes presence and severity of all criterion BN symptoms (e.g. bingeing, purging, body-image disturbances, menstrual function and dietary restraint). The interview yields valid estimates of average weekly frequency of bingeing and purging (episodes of vomiting, laxative abuse, compulsive exercise, diuretic or 'diet pill' abuse). Reliability exceeds 0·90 on all but three of 62 EDE items, and internal consistency and discriminant validity figures are excellent (Fairburn & Cooper, 1993). We have developed a French EDE translation, and equivalence in English and French versions is indicated according to conventional indices (*t* tests, coefficient alphas).

Psychopathology

Diagnostic Interview Schedule, version 4 (*DIS*-4) (Bucholz *et al*. 1991)

The DIS-4 is a DSM-IV version of the NIMH Diagnostic Interview Schedule (Robins *et al*. 1981) and is a self-administered interview used to assess DSM-IV (APA, 1994) Axis I disorders. Given a bilingual population, we also used a validated French-language version of the DIS-4 (Lepage *et al*. 1996). Modules relating to major depression and anxiety disorders were used.

Structured Clinical Interview for DSM-IV Axis II (*SCID-II*) (First *et al*. 1994)

The SCID-II is a face-to-face interview that yields good inter-rater reliabilities and differentiation of main PD classifications. The SCID-II for DSM-III-R has test–retest reliabilities from 0·74 to 0·87 and kappas from 0·43 to 1·00 (Segal *et al*. 1994), while for DSM-IV, inter-rater kappas range from 0·48 to 0·98 for categorical diagnosis, and intraclass correlation coefficients range from 0·90 to 0·98 for dimensional judgements; internal consistency coefficients range from 0·71 to 0·94, overall, SCID-II has adequate inter-rater reliability and internal consistency (Maffei *et al*. 1997). The interview guides decision-tree branching for the interviewer and scores automatically. For consistency with our prior studies in this population (e.g. Steiger *et al*. 1992), we disregarded the borderline PD criterion referring to 'overeating'. Given a bilingual population, we also used a validated French-language version of the SCID-II (see Steiger *et al*. 2001*c*). In the current study, interviews were conducted by a primary interviewer and a random sampling of 50% of the APD sections of interviews were scored by a second interviewer (kappa 0·84).

Personality pathology

Dimensional Assessment for Personality Pathology-Basic Questionnaire (*DAPP-BQ*) (Livesley *et al*. 1992)

The DAPP-BQ is an extremely well constructed questionnaire providing a comprehensive dimensional assessment of personality pathology. Content is derived from expert-based lists describing DSM-III and DSM-III-R personality-disorder features, and content analysis of interviews with patients. The current 'Basic Questionnaire' comprises 18 subscales (12–16 items each, with alphas ranging from 0·87 to 0·94) (Livesley *et al*. 1992), representing factor-based dimensions obtained in large, independent population samples. With permission of the author, we have developed a French-language translation of the DAPP, performance of which (via *t* tests, coefficient alphas) resembles that of the English version.

Behavioural inhibition and disinhibition

Go/No-Go discrimination task (Newman *et al*. 1985)

The Go/No-Go is a well-established laboratory measure of behavioural response inhibition and disinhibition. We used a version of the task modified to so as to permit comparative examination of the differential effects of four combinations of monetary reward and punishment (Iaboni *et al*. 1995; LeMarquand *et al*. 1999). The task as employed herein has four conditions. Each condition contains a different set of eight stimuli (two-digit numbers). Within each condition, four of the stimuli are designated as

positive (S+), and four of the stimuli are negative (S−). Individuals must learn (through a process of trial and error) whether to respond (by pressing a button) to stimuli presented one at a time on a computer screen. Correct responses are signalled with a high pitched tone and the word 'correct' on the computer screen. Incorrect responses are signalled by a low-pitched tone and the word 'wrong' on the screen. Errors of commission (i.e. failure to inhibit responses to S−) and errors of omission (failure to respond to S+) are tracked, under each of four randomized conditions: (1) reward–punishment, in which subjects start with 50 cents, and are rewarded (win 5 cents) for responses to S+ and punished (lose 5 cents) for responses to S−; (2) punishment–punishment, in which subjects begin with $2.00, have no opportunity to win more money, but lose money (5 cents) of they respond to S− or fail to respond to S+; (3) reward–reward, in which subjects begin with no money, but earn rewards (5 cents) by responding appropriately to S+ and S−; (4) punishment–reward, in which subjects start with 50 cents, and a non-response to S+ is punished (loss of 5 cents) and a non-response to S− is rewarded (winning of 5 cents). The overall task can be learned and completed in about 30 min.

Validity of the task has been established in laboratory studies implicating several and diverse clinical and non-clinical populations. On the Go/No-Go task, impulsive or aggressive (e.g. psychopaths, aggressive mothers, impulsive normals) subjects show consistently greater response disinhibition (as measured by errors of commission on the task (e.g. Newman *et al.* 1985, 1990; Newman & Kosson, 1986; Patterson *et al.* 1987; Iaboni *et al.* 1995; LeMarquand *et al.* 1999)). As concerns response inhibition, introverted individuals make fewer commission errors than extroverts while neurotic introverts make more omission errors than other subjects (Helmers *et al.* 1995).

Serotonergic functioning

m-CPP challenge (Mueller *et al.* 1985)

Since 5-HT promotes prolactin secretion from the pituitary, it is conventional to draw inferences about central 5-HT functioning from 5-HT-induced alterations in plasma prolactin levels (Yatham & Steiner, 1993). We measured prolactin levels before and after oral administration of the partial 5-HT agonist m-CPP, which (since it binds with highest affinity to 5-HT_{2c} receptors (see Mueller *et al.* 1985) is thought to be a fairly specific 5-HT probe (Yatham & Steiner, 1993). Participants, tested as out-patients, were required to have been free of psychoactive medications for at least 6 weeks and were tested in follicular phase of menses (i.e. 5 to 14 days following start of last menses). Before testing, participants were asked to refrain from alcohol, exercise, or street drugs for 48 h and from binge-eating for 24 h. On the test morning, participants underwent a urine screen for drug (cocaine, amphetamine, opiates, cannabinoids (marijuana), and benzodiazepines) use with an enzyme-multiplied immunoassay technique kit. Samples were drawn after an overnight fast. Detailed procedures for biochemical assays are described elsewhere (Steiger *et al.* 2001*a*). Baseline measures on hormones constitute the mean of two values obtained from samples drawn at 8.30 and 9.00 a.m. before m-CPP administration. Prolactin levels were determined by radioimmunoassay, using the corresponding, validated Amersham radioimmunoassay kits from Johnson and Johnson, Markham, Ontario.

Procedure

The protocol was approved by hospital Research Ethics Board. Bulimic participants were contacted and recruited for participation at entry to treatment at a specialized Eating Disorders Program. Normal eater controls were recruited through community newspapers. All participants gave informed consent prior to participating, and completed testing in three or four sessions that took place at the outpatient clinic of the Eating Disorders Program. Procedures for the Go/No-Go and m-CPP challenges were as described in the respective sections above. Participants were reimbursed modestly ($5/h) for their time and expenses.

Statistical analyses

Participants were organized into three subgroups: BN with avoidant PD (BNA+; $N=13$), BN without avoidant PD (BNA−; $N=23$), controls (normal eaters) ($N=23$). Groups were compared on eating and psychopathological

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symptoms using ANOVAs and (except as indicated) with Newman–Keuls tests (to control family-wise error). Where EDE measures (e.g. binge frequencies) yielded zero values in normal eaters, we applied *t* tests to examine pairwise differences across avoidant and non-avoidant bulimic women only. We used pairwise chi-square tests to explore group effects on categorical measures of pathology (Axis I and II). We used one-way ANOVA to explore group effects on dimensional indices of personality pathology and repeated-measures ANOVAs to test for group effects on serial prolactin. Repeated measures ANOVAs treated group (BNA+, BNA−, controls) and time (baseline and at 30, 60, 90, 120, 150, 180, 210 and 240 min) factors. Mauchly's test of sphericity was used to test for homogeneity of covariance. We tested the groups for differences on variables (season of testing, contraceptive use) known or thought to influence prolactin responses after m-CPP administration. Because our sample size was not sufficiently large to allow for stable estimation of seasonal effects, we turned to published findings: prolactin response after m-CPP administration is reportedly larger in winter than in summer and at intermediate levels in spring and fall (Brewerton, 1989) and our own data (e.g. Steiger *et al.* 2001*c*) has shown a similar trend. To control for the effect, we entered two dummy variables as covariates, which compared samplings obtained in summer (and then fall and spring) to those obtained in winter. Statistical tests were two-tailed and conducted at the 0·05 level of significance. To balance type I and type II errors, we report pairwise group comparisons with and without Bonferroni corrections.

RESULTS

Eating symptoms

To compare the three groups on overall eating symptomatology, they were compared on mean EAT-26 final scores. As expected, we found a significant group effect using univariate ANOVA ($F(2,56) = 145{\cdot}1$, $P < 0{\cdot}0001$). *Post hoc* tests using Newman–Keuls test indicated that both bulimic groups differed from controls ($P < 0{\cdot}05$), but did not differ from each other. Mean ± S.D. EAT-26 final score for the control, BNA− and BNA+ groups were respectively: 2·8 ± 2·9, 40·3 ± 9·8 and 40·6 ± 11·9. Thus, the two bulimic groups had higher EAT-26 final scores than controls.

On EDE measures, the two BN subgroups were contrasted using *t* tests and did not differ on any of the EDE items examined. Included here were the average number of episodes per month of binge eating 33·6 *v.* 30·4 ± S.D. 28·0 *v.* 32·0 ($t = 0{\cdot}31$, $P = 0{\cdot}94$) or self-induced vomiting, 38·6 *v.* 31·4 ± S.D. 33·0 *v.* 38·7, ($t = 0{\cdot}11$, $P = 0{\cdot}56$), over the preceding 3 months.

Table 1. *Number of cases and percentage of groups meeting current* (*preceding* 12 *months*) *criteria for select Axis I psychopathology according to the DIS*-4

	Control ($N = 23$)		BNA− ($N = 23$)		BNA+ ($N = 13$)	
	Cases *N*	(%)	Cases *N*	(%)	Cases *N*	(%)
Simple phobia	1	(4·3)	2	(8·7)	2	(15·4)
Social phobia	1	(4·3)	5	(21·7)	2	(15·4)
Agoraphobia	0	(0)	0	(0)	0	(0)
Panic disorder	0	(0)	0	(0)	2	(15·4)
OCD	0	(0)	2	(8·7)	0	(0)
GAD	0	(0)	1	(4·3)	0	(0)
PTSD	1	(4·3)	1	(4·3)	3	(23·1)
MDD	2	(8·7)	7	(30·4)	2	(15·4)

OCD, obsessive–compulsive disorder; GAD, generalized anxiety disorder; PTSD, post-traumatic stress disorder; MDD, major depressive disorder.

Psychopathology

Axis I

The groups were compared for presence of major depression and for anxiety disorders over the preceding 12 months. Results are shown in Table 1. There were no significant ($P < 0{\cdot}05$) differences detected among the groups on any of the disorders using pairwise chi-square tests, indicating that the groups did not differ in terms of current frequency these Axis I disorders.

Axis II

The BNA+ and BNA− groups had originally been formed based on presence or absence of co-morbid avoidant PD, while control group participants were excluded if they met criteria for a PD. Numbers of participants meeting criteria for a PD for the BNA− and BNA+ groups were: paranoid (0,0), schizoid (0,0),

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Table 2. *Group means and statistics for DAPP scales*

DAPP scale	Control	BNA −	BNA +	*F*(2,56)
Affective instability	2·48a	3·48b	3·95c	21·5***
Antisocial aggression	2·06a	2·82b	3·06b	12·1***
Anxiousness	2·33a	3·70b	4·09b	26·6***
Callousness	1·77	1·93	1·93	NS
Cognitive distortion	1·64a	2·36b	2·73b	10·6***
Compulsivity	3·15	3·17	3·86	NS
Conduct problems	1·42	1·78	1·72	NS
Identity problems	1·82a	3·30b	3·83b	30·0***
Insecure attachment	1·95a	2·79b	3·14b	9·71***
Intimacy problems	1·80	2·32	2·79	NS
Narcissism	2·69a	3·63b	3·54b	12·4***
Rejection sensitivity	2·39	2·94	2·35	NS
Restricted expression	2·21a	2·84b	3·62c	19·2***
Self-harming behaviours	1·09a	2·2a	2·84b	20·1***
Social avoidance	2·20a	3·02b	3·95c	23·4***
Stimulus seeking	2·55	2·87	2·88	NS
Submissiveness	2·25a	2·87b	3·67c	17·0***
Suspiciousness	1·59a	2·56b	2·54b	10·8***

Postscripts (a, b, c) identify significantly different ($P<0·05$) group means (Newman–Keuls *post hoc* test).

*** $P<0·001$.

Table 3. *Group means and statistics for the Go/No-Go Discrimination Task*

	Control	BNA −	BNA +	*F*(2,56)
Commission errors				
RR	11·3	9·13	14·7	NS
RP	7·21	6·3	8·69	NS
PR	12·9	14·7	9·84	NS
PP	7·60	8·17	12·2	NS
Omission errors				
RR	3·22	1·57	2·23	NS
RP	3·52	2·78	4·31	NS
PR	8·17	6·39	8·23	NS
PP	3·65a	3·43a	8·85b	3·86*

Postscripts (a, b) identify significantly different group means ($P<0·05$, Newman–Keuls).

RR, reward/reward condition; RP, reward/punishment condition; PR, punishment/reward condition; PP, punishment/punishment condition.

* $P=0·037$.

schizotypal (0,0), antisocial (2,0), borderline (5,3), histrionic (0,1), narcissistic (0,0), dependent (1,2) and obsessive–compulsive (5,3). Using pairwise chi-square tests, both bulimic groups were found to have higher proportions ($P<0·05$) than controls meeting criteria for borderline PD and for obsessive–compulsive PD. The bulimic groups did not differ from each other on these PDs, however, and there were no significant group differences found for the remaining PDs. In summary, the bulimic groups were more likely than controls to meet criteria for borderline and obsessive–compulsive PDs, and the bulimic groups did not differ from each other on PDs beyond avoidant PD used for grouping.

Personality pathology

The groups were compared on each of the 18 DAPP subscales using separate univariate ANOVAs with an Bonferroni adjusted *P* value of 0·0027 (0·05/18) for significance on the ANOVAs for multiple comparisons. The results (group means and statistics) are shown in Table 2. Consistent with our hypotheses, the BNA + group reported significantly (Newman–Keuls *post hoc*, $P<0·05$) more restrictive (emotional) expression, social avoidance and submissiveness than the other groups. Inconsistent with our expectations, the BNA + group also reported significantly (Newman–Keuls *post hoc*, $P<0·05$) more affective instability and self-harming behaviours than the other groups. Furthermore, both bulimic groups reported significantly (Newman–Keuls *post hoc*, $P<0·05$) more antisocial aggression, anxiousness, cognitive distortion, identity problems, narcissism and suspiciousness than did the controls but did not differ from each other on these scales.

Behavioural inhibition and disinhibition

The three groups were contrasted on their performance on the Go/No-Go task. Eight separate ANOVAs were run: for commission errors (one for each of the four conditions) and for omission errors (also one for each of the four conditions). The results (group means and statistics) are shown in Table 3. The BNA + group made significantly more omission errors in the punishment–punishment condition than the other groups (Newman–Keuls *post hoc*, $P<0·05$), and that the latter two groups did not differ from each other. Thus, the BNA + group was more inhibited than the other groups specifically when faced only with cues for punishment (here, the prospect of losing money or not winning money). The significance level of this effect did not remain after Bonferroni correction (adjusted alpha, $0·05/8=0·00625$) and no other differences were significant with or without correction.

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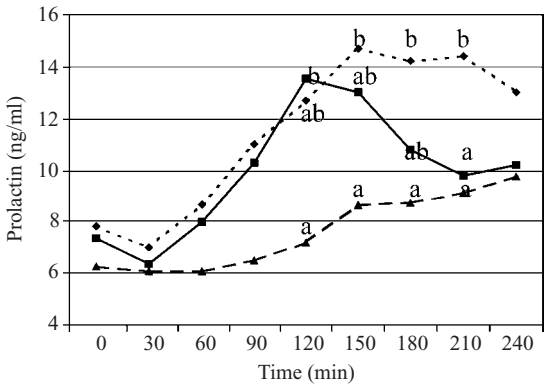

FIG. 1. Mean group serial prolactin levels following m-CPP challenge. ANOVA results indicate a significant group × time interaction ($F(16, 98df) = 1{\cdot}79$, $P = 0{\cdot}042$). *Post hoc* testing results are depicted with a and b letters representing significantly different means using Fisher's least significant difference. (◆---◆, healthy controls (HC); ■—■, bulimia nervosa without avoidant personality (BNA−); ▲---▲, bulimia nervosa with avoidant personality (BNA+).)

Serotonergic function

Groups were compared on prolactin response to m-CPP challenge using ANOVA (group × time with time as a repeated-measures variable). The results are shown in Fig. 1. We found a significant group by time interaction ($F(16,98) = 1{\cdot}79$, $P = 0{\cdot}042$), a main effect of time ($F(8,49) = 7{\cdot}57$, $P < 0{\cdot}001$) and a non-significant trend for a main effect of group ($F(2,56) = 2{\cdot}63$, $P = 0{\cdot}081$). Pairwise *post hoc* analyses were performed using Newman–Keuls but no group differences could be detected. Using Fisher's LSD tests, the BNA+ group was found to have a significantly ($P < 0{\cdot}05$) lower mean prolactin level than the BNA− group at 120 min. The BNA+ group also significantly ($P < 0{\cdot}05$) lower mean prolactin levels than the control group at 150 and 180 min. Both bulimic groups had lower mean prolactin levels than the control group at 210 min. No other differences were significant. In summary, the prolactin data indicate that: (*a*) the BNA+ group was blunted relative to the BNA− group 120 min after m-CPP ingestion and relative to the control group 150 and 180 min after m-CPP ingestion; and, (*b*) both bulimic groups were blunted relative to controls 210 min after m-CPP ingestion. We also verified responses to m-CPP by examining the simple effects of time for each of the three groups; effects were significant for control and BNA−, but not for BNA+. *Post hoc* testing using Newman–Keuls tests indicated that the control and BNA− groups experienced an increase in prolactin levels following m-CPP relative to their respective baselines while the values for the BNA+ group did not increase relative to its baseline.

Covariates

We compared the groups in terms of season of testing. Numbers according to seasons (winter, spring, summer, fall) of testing were: for HC (3,4,9,7); BNA− (4,7,4,8); and BNA+ (8,4,1,0). Chi-square analysis was significant ($P < 0{\cdot}006$), indicating differences in group sizes across season. However, when we re-ran the prolactin results covarying season, the covariate effect was not significant, indicating season did not affect group differences. We were also interested in controlling for possible effects due to contraceptive medication and numbers of participants taking contraceptives were: control, BNA−, BNA+ (11, 8, and 2). Chi-square analysis was not significant ($P = 0{\cdot}148$), indicating the groups did not differ significantly in terms of contraceptive use. Also, given that the groups differed on affective instability and self-harming scores (not traditionally associated with APD), we tested for these as well in covariance analyses. Neither covariate effect was significant, implying that differences on prolactin response in the BNA+ group were not due to variance from affective instability and self-harming behaviours.

DISCUSSION

We compared three groups of women (women with BN and co-morbid APD, women with BN but without co-morbid APD and normal eater control women) on self-report measures of psychopathology and personality pathology, on eating symptomatology, on behavioural measures of inhibition and disinhibition, and on serial prolactin responses to m-CPP. Compared with the other groups, women in the BNA+ group differed on several measures that appear to be consistent with our hypotheses. First, the BNA+ group reported relatively more emotional restriction, social avoidance and interpersonal submissiveness as assessed with the DAPP (Table 2). Such dimensional findings appear generally consistent with categorical

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DSM-IV criteria for APD. Secondly, the BNA+ group also made more omission errors on the Go/No-Go task under cues for punishment (Table 3), indicating that the BNA+ group was characterized by behavioural inhibition specifically in the face of monetary 'threat.' We note that the significance level of the result is modest ($P=0{\cdot}037$) and the group difference would no longer be significant with Bonferroni corrections for the multiple tests. That the BNA+ group should make more omission errors (but not more commission errors) appears to be consistent with the result of Helmers *et al.* (1995), who found that 'neurotic introverts' – which seems like a fitting description of those with APD – made more total omission errors than other groups and did not differ from other groups on commission errors. Furthermore, that the group differences were selective to the punishment–punishment condition appears to complement a recent finding from our group (Bruce *et al.* 2003), wherein laxative abusing women with BN made more commission errors than non-abusers, but only in the punishment–punishment condition. Such findings could indicate that some women with BN have altered sensitivity to punishment with no change in sensitivity to reward. Thirdly, the BNA+ group evinced a somewhat blunted (prolactin) response to 5-HT activation compared with both of the other groups (Fig. 1). This result was statistically significant between BNA+ *v.* controls 150, 180 and 210 min after ingestion of m-CPP. We also found differences between BNA+ and BNA− at 120 min after ingestion of m-CPP. We must highlight that these effects were also modest (blunting found at 3/8 time points after baseline *v.* controls and 1/8 time points after baseline *v.* BNA−, and using LSD tests without correction for multiple tests). We interpret the m-CPP results as potentially consistent with our primary hypothesis that APD may be related to reductions in serotonergic activation in BN. We also found that, compared with controls, the BNA− group had blunted prolactin response at 210 min following m-CPP. Although arguably a marginal effect (at 1/8 time points after baseline), such a finding is consistent with the previous literature showing that active BN (regardless of psychiatric co-morbidity) is associated with at least modestly blunted prolactin response following m-CPP (e.g. Brewerton *et al.* 1992; Levitan *et al.* 1997; Steiger *et al.* 2001*a*, *b*, *c*).

The bulimic groups did not differ significantly from each other (or from controls) on frequency of (current) major depression or anxiety disorders so it appears that differences between the bulimic groups were not due to confounding effects of depression or anxiety symptoms. Our result seems consistent with those of Brewerton *et al.* (1992) who found that co-morbid major depression did not influence neuroendocrine response to m-CPP in BN. Also, the bulimic groups did not differ from each other in terms of the frequency of other PDs, indicating that the results for the BNA+ group were not likely to be due to confounds in additional DSM-IV Axis II co-morbidity. The BNA+ group (as well as the BNA− group) was more likely than controls to meet criteria for BPD and obsessive–compulsive PD. Such findings seem consistent with the Rosenvinge *et al.* (2000) meta-analysis that showed Cluster B and Cluster C PDs are more common in BN than in controls. The BNA+ group did differ from the BNA− group on two measures that do not fit neatly with the APD diagnosis, namely affective instability and self-destructiveness (Fig. 1). Such results could mean that affective instability and/or self-harming behaviours (elevated in the BNA+ group) might be associated with the Go/No-Go and m-CPP challenge results. Indeed, previous results from a partly overlapping dataset (Steiger *et al.* 2001*a*, *b*) demonstrated the importance of these personality traits, particularly impulsivity and self-destructiveness, for (blunted) 5-HT status in BN. We note, however, that the BNA+ group was not more likely to meet criteria for BPD, and that the m-CPP results were unaffected by covarying out affective instability and self-harming behaviour, suggesting that the current m-CPP results were not due to increases in these traits in the BNA+ group. Further, previous research has found that self-harming behaviours are quite common in BN, even among individuals not meeting criteria for BPD (Paul *et al.* 2002).

We note the various limitations to the findings. First, as we have alluded to, the significance levels of two of our key findings (Go/No-Go and m-CPP) were modest. We interpret this result as primarily due to our small sample size, but it is of course possible that we have

capitalized on chance error. The overall pattern of results seems consistent internally (i.e. APD co-aggregating with behavioural, interpersonal and serotonergic inhibition) as well as consistent with other clinical and pre-clinical APD literature outlined previously suggesting implication of the 5-HT system. Secondly, there is considerable sample overlap with some of our prior publications. Findings from those reports suggested that self-destructiveness (Steiger *et al.* 2001*b*) and other tendencies (Steiger *et al.* 2001*a*, *c*) were associated with blunted prolactin function. While we controlled for these effects statistically, the current results may be limited in their generalizability by overlap with these prior reports. Thirdly, we were unable to control fully for nutritionally-related and 'illness state' effects in our bulimic participants. For example, we did not measure serial m-CPP concentrations along with prolactin, nor were we able to control for the specific time of testing within the menstrual cycle. Nutritional status has been shown to be important in neuroendocrine response to 5-HT agonists in BN (e.g. Kaye *et al.* 1998; Wolfe *et al.* 2000), and we are unable to deterimine whether the effects of avoidant PD on 5-HT function would remain significant when symptoms of BN remit following treatment. Furthermore, moderate dieting can alter prolactin response to m-CPP in otherwise healthy women (Cowen *et al.* 1996), and there is a tendency for prolactin response to m-CPP to normalize with short-term weight gain in anorexia nervosa (Brewerton & Jimerson, 1996). It is therefore possible that nutritional effects influenced our bulimic *v.* control and BNA+ *v.* BNA− differences. We found that the bulimic and control groups did not differ on age or BMI, and that the two bulimic groups had comparable current BN symptomatology. Finally, while our groups did not differ on their use of contraceptives, we were unable to determine whether the groups may have differed on a more detailed hormonal comparison.

In conclusion, women with BN and avoidant PD may be characterized by interpersonal submissiveness and avoidance, affective instability, self-harm, cautiousness when faced with perceived threats and lower sensitivity to serotonergic activation. We hypothesize that – among other factors including altered nutritional status, severity of bulimic symptoms, Axis I and II pathology, contraceptive use and season of the year – presence of concurrent avoidant PD may contribute to behavioural and post-synaptic serotonergic dysregulation in BN. Implications of the results are that co-morbid avoidant PD has important biological and behavioural effects in individuals with BN and such individuals might benefit from assessments of whether interventions that increase serotonin activity offer additional therapeutic benefit.

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REFERENCES

Altamura, A. C., Pioli, R., Vitto, M. & Mannu, P. (1999). Venlafaxine in social phobia: a study in selective serotonin reuptake inhibitor non-responders. *International Clinical Psychopharmacology* **14**, 239–245.

American Psychiatric Association (1994). *Diagnostic and Statistical Manual of Mental Disorders, 4th edn.* APA: Washington, DC.

Banasiak, S. J., Wertheim, E. H., Koerner, J. & Voudouris, N. J. (2001). Test–retest reliability and internal consistency of a variety of measures of dietary restraint and body concerns in a sample of adolescent girls. *International Journal of Eating Disorders* **29**, 85–89.

Baxter, L. R. Jr., Clark, E. C., Ackermann, R. F., Lacan, G. & Melega, W. P. (2001). Brain mediation of Anolis social dominance displays. II. Differential forebrain serotonin turnover, and effects of specific 5-HT receptor agonists. *Brain, Behavior and Evolution* **57**, 184–201.

Beaumont, P., Al-Alami, M. & Touyz, S. (1988). Relevance of a standard measurement of undernutrition to the diagnosis of anorexia: use of Quetelet's body mass index. *International Journal of Eating Disorders* **7**, 399–405.

Brewerton, T. D. (1989). Seasonal variation of serotonin function in humans: research and clinical implications. *Annals of Clinical Psychiatry* **1**, 153–164.

Brewerton, T. D. (1995). Toward a unified theory of serotonin dysregulation in eating and related disorders. *Psychoneuroendocrinology* **20**, 561–590.

Brewerton, T. D. & Jimerson, D. C. (1996). Studies of serotonin function in anorexia nervosa. *Psychiatry Research* **62**, 31–42.

Brewerton, T. D., Mueller, E. A., Lesem, M. D., Brandt, H. A., Quearry, B., George, D. T., Murphy, D. L. & Jimerson, D. C. (1992). Neuroendocrine responses to m-chlorophenylpiperazine and L-tryptophan in bulimia. *Archives of General Psychiatry* **49**, 852–861.

Bruce, K. R., Koerner, N. M., Steiger, H. & Young, S. N. (2003). Laxative misuse and behavioral disinhibition in bulimia nervosa. *International Journal of Eating Disorders* **33**, 92–97.

Bucholz, K. K., Robins, L. N., Shayka, J. J., Przybeck, T. R., Heltzer, J. E., Goldring, E., Klein, M. H., Greist, J. H., Erdman, H. P. & Skare, S. S. (1991). Performance of two forms of a computer psychiatric screening interview; Version 1 of the DISSI. *Journal of Psychiatric Research* **25**, 117–129.

Cowen, P. J., Clifford, E. M., Walsh, A. E., Williams, C. & Fairburn, C. G. (1996). Moderate dieting causes 5-HT_{2C} receptor supersensitivity. *Psychological Medicine* **26**, 1155–1159.

Ekselius, L. & von Knorring, L. (1998). Personality disorder comorbidity with major depression and response to treatment with sertraline or citalopram. *International Clinical Psychopharmacology* **13**, 205–211.

Fairburn, C. G. & Cooper, P. (1993). The eating disorders examination. In *Binge Eating: Nature, Assessment and Treatment, 12th edn.* (ed. C. Fairburn and G. Wilson), pp. 317–360. Guilford Press: New York.

File, S. E., James, T. A. & MacLeod, N. K. (1981). Depletion in amygdaloid 5-hydroxytryptamine concentration and changes in social and aggressive behaviour. *Journal of Neural Transmission* **50**, 1–12.

First, M. B., Spitzer, R. L., Gibbon, M., Williams, M., Janet, B. W. & Benjamin, L. (1996). Structured clinical interview for DSM-IV Axis II Personality Disorders (SCID-II) version 2.0. Biometrics Research Department, New York State Psychiatric Institute: New York.

Garner, D. M., Olmsted, M. P., Bohr, Y. & Garfinkel, P. E. (1982). The eating attitudes test: psychometric features and clinical correlates. *Psychological Medicine* **12**, 871–878.

Gartner, A. F., Marcus, R. N., Halmi, K. & Loranger, A. W. (1989). DSM-III-R personality disorders in patients with eating disorders. *American Journal of Psychiatry* **146**, 1585–1591.

Helmers, K. F., Young, S. N. & Pihl, R. O. (1995). Assessment of measures of impulsivity in healthy male volunteers. *Personality and Individual Differences* **19**, 927–935.

Iaboni, F., Douglas, V. I. & Baker, A. G. (1995). Effects of reward and response costs on inhibition in ADHD children. *Journal of Abnormal Psychology* **104**, 232–240.

Jimerson, D. C., Lesem, M. D., Kaye, W. H. & Brewerton, T. D. (1992). Low serotonin and dopamine metabolite concentrations in cerebrospinal fluid from bulimic patients with frequent binge episodes. *Archives of General Psychiatry* **49**, 132–138.

Jimerson, D. C., Wolfe, B. E., Metzger, E. D., Finkelstein, D. M., Cooper, T. B. & Levine, J. M. (1997). Decreased serotonin function in bulimia nervosa. *Archives of General Psychiatry* **54**, 529–534.

Kaye, W. H., Greeno, C. G., Moss, H., Fernstrom, J., Fernstrom, M., Lilenfeld, L. R., Weltzin, T. E. & Mann, J. J. (1998). Alterations in serotonin activity and psychiatric symptoms after recovery from bulimia nervosa. *Archives of General Psychiatry* **55**, 927–935.

Kaye, W. H., Frank, G. K., Meltzer, C. C., Price, J. C., McConaha, C. W., Crossan, P. J., Klump, K. L. & Rhodes, L. (2001). Altered serotonin 2A receptor activity in women who have recovered from bulimia nervosa. *American Journal of Psychiatry* **158**, 1152–1155.

LeMarquand, D. G., Benkelfat, C., Pihl, R. O., Palmour, R. M. & Young, S. N. (1999). Behavioral disinhibition induced by tryptophan depletion in nonalcoholic young men with multigenerational family histories of paternal alcoholism. *American Journal of Psychiatry* **156**, 1771–1779.

Lepage, D., Jolicoeur, F. B., Ggheysen, F., Caillard, V., Diener, J. M., Castagnet, F., Lomazzi, H., Raffrey, G. & Zarafian, E. (1996). Diagnostic interview schedule: validation d'une version française informatisée. (Diagnostic interview schedule: validation of a computerized French version.) *Annales de Psychiatrie* **11**, 5–13.

Levitan, R. D., Kaplan, A. S., Joffe, R. T., Levitt, A. J. & Brown, G. M. (1997). Hormonal and subjective responses to intravenous meta-chlorophenylpiperazine in bulimia nervosa. *Archives of General Psychiatry* **54**, 521–527.

Livesley, W. J., Jackson, D. N. & Schroeder, M. L. (1992). Factorial structure of traits delineating personality disorders in clinical and general population samples. *Journal of Abnormal Psychology* **101**, 432–440.

Maffei, C., Fossati, A., Agostoni, I., Barraco, A., Bagnato, M., Deborah, D., Namia, C., Novella, L. & Petrachi, M. (1997). Interrater reliability and internal consistency of the structured clinical interview for DSM-IV axis II personality disorders (SCID-II), version 2.0. *Journal of Personality Disorders* **11**, 279–284.

Marazziti, D., Macchi, E., Rotondo, A., Placidi, G. F. & Cassano, G. B. (1988). Involvement of serotonin system in bulimia. *Life Sciences* **43**, 2123–2126.

Monteleone, P., Brambilla, F., Bortolotti, F. & Maj, M. (2000). Serotonergic dysfunction across the eating disorders: relationship to eating behaviour, purging behaviour, nutritional status and general psychopathology. *Psychological Medicine* **30**, 1099–1110.

Moskowitz, D. S., Pinard, G., Zuroff, D. C., Annable, L. & Young, S. N. (2001). The effect of tryptophan on social interaction in every day life: a placebo-controlled study. *Neuropsychopharmacology* **25**, 277–289.

Mueller, E. A., Murphy, D. L. & Sunderland, T. (1985). Neuroendocrine effects of m-chlorophenylpiperazine, a serotonin agonist, in humans. *Journal of Clinical Endocrinology and Metabolism* **61**, 1179–1184.

Newman, J. P. & Kosson, D. S. (1986). Passive avoidance learning in psychopathic and nonpsychopathic offenders. *Journal of Abnormal Psychology* **95**, 252–256.

Newman, J. P., Widom, C. S. & Nathan, S. (1985). Passive avoidance in syndromes of disinhibition: psychopathy and extraversion. *Journal of Personality and Social Psychology* **48**, 1316–1327.

Newman, J. P., Patterson, C. M., Howland, E. W. & Nichols, S. L. (1990). Passive avoidance in psychopaths: the effects of reward. *Personality and Individual Differences* **11**, 1101–1114.

Patterson, C. M., Kosson, D. S. & Newman, J. P. (1987). Reaction to punishment, reflectivity, and passive avoidance learning in extraverts. *Journal of Personality and Social Psychology* **52**, 565–575.

Paul, T., Schroeter, K., Dahme, B. & Nutzinger, D. O. (2002). Self-injurious behavior in women with eating disorders. *American Journal of Psychiatry* **159**, 408–411.

Phillips, K. A. & McElroy, S. L. (2000). Personality disorders and traits in patients with body dysmorphic disorder. *Comprensive Psychiatry* **41**, 229–236.

Raleigh, M. J. & McGuire, M. T. (1991). Bidirectional relationships between tryptophan and social behavior in vervet monkeys. In *Kynurenine and Serotonin Pathways: Progress in Tryptophan Research, Advances in Experimental Medicine and Biology*, **294** (ed. R. Schwarcz, S. N. Young and R. R. Brown), pp. 289–298. Plenum Press: New York.

Robins, L. N., Heltzer, J. E., Croughan, J. & Ratcliff, K. S. (1981). National Institute of Mental Health Diagnostic Interview Schedule. *Archives of General Psychiatry* **38**, 381–389.

Rosenvinge, J. H., Martinussen, M. & Ostensen, E. (2000). The comorbidity of eating disorders and personality disorders: a meta-analytic review of studies published between 1983 and 1998. *Eating and Weight Disorders* **5**, 52–61.

Segal, D. L., Hersen, M. & van Hasselt, V. B. (1994). Reliability of the Structured Clinical Interview for DSM-III-R: an evaluative review. *Comprehensive Psychiatry* **35**, 316–327.

Smith, K. A., Fairburn, C. G. & Cowen, P. J. (1999). Symptomatic relapse in bulimia nervosa following acute tryptophan depletion. *Archives of General Psychiatry* **56**, 171–176.

Steiger, H., Leung, F. Y. & Houle, L. (1992). Relationships among borderline features, body dissatisfaction and bulimic symptoms in nonclinical females. *Addictive Behaviors* **17**, 397–406.

Steiger, H., Young, S. N., Kin, N. M., Koerner, N., Israel, M., Lageix, P. & Paris, J. (2001*a*). Implications of impulsive and affective symptoms for serotonin function in bulimia nervosa. *Psychological Medicine* **31**, 85–95.

Steiger, H., Koerner, N., Engelberg, M. J., Israel, M., Ng Ying Kin, N. M. & Young, S. N. (2001*b*). Self-destructiveness and serotonin function in bulimia nervosa. *Psychiatry Research* **103**, 15–26.

Steiger, H., Gauvin, L., Israel, M., Koerner, N., Ng Ying Kin, N. M., Paris, J. & Young, S. N. (2001*c*). Association of serotonin and cortisol indices with childhood abuse in bulimia nervosa. *Archives of General Psychiatry* **58**, 837–843.

Steiger, H., Leonard, S., Kin, N. Y., Ladouceur, C., Ramdoyal, D. & Young, S. N. (2000). Childhood abuse and platelet tritiated-paroxetine binding in bulimia nervosa: implications of borderline personality disorder. *Journal of Clinical Psychiatry* **61**, 428–435.

Torgersen, S., Kringlen, E. & Cramer, V. (2001). The prevalence of personality disorders in a community sample. *Archives of General Psychiatry* **58**, 590–596.

Tse, W. S. & Bond, A. J. (2002). Serotonergic intervention affects both social dominance and affiliative behaviour. *Psychopharmacology* **161**, 324–330.

Waller, D. A., Steinberg, A., Gullion, C., Moeller, F. G., Cannon, D. S., Petty, F., Hardy, B. W., Orsulak, P. & Rush, A. J. (1996). Impulsivity and neuroendocrine response to buspirone in bulimia nervosa. *Biological Psychiatry* **39**, 371–374.

Weltzin, T. E., Fernstrom, M. H., Fernstrom, J. D., Neuberger, S. K. & Kaye, W. H. (1995). Acute tryptophan depletion and increased food intake and irritability in bulimia nervosa. *American Journal of Psychiatry* **152**, 1668–1671.

Wolfe, B. E., Metzger, E. D., Levine, J. M., Finkelstein, D. M., Cooper, T. B. & Jimerson, D. C. (2000). Serotonin function following remission from bulimia nervosa. *Neuropsychopharmacology* **22**, 257–263.

Yates, W. R., Sieleni, B., Reich, J. & Brass, C. (1989). Comorbidity of bulimia nervosa and personality disorder. *Journal of Clinical Psychiatry* **50**, 57–59.

Yatham, L. N. & Steiner, M. (1993). Neuroendocrine probes of serotonergic functions: a critical review. *Life Sciences* **53**, 447–463.