Implications of compulsive and impulsive traits for serotonin status in women with bulimia nervosa

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Abstract

Studies of bulimia nervosa (BN) often report decreased brain serotonin (5-hydroxytryptamine: 5-HT) activity. Across populations, impulsivity has been linked to reduced 5-HT activity, but compulsivity has been associated (at least inconsistently) with an increase. We therefore became interested in the association between behavioral-trait variations and 5-HT status in BN. In 56 bulimic and 29 non-bulimic women, we measured eating symptoms, personality traits, platelet paroxetine binding, and neuroendocrine responses following oral meta-chlorophenylpiperazine (m-CPP). Relative to normal eaters, bulimic women showed reduced density (Bmax) of platelet paroxetine-binding sites, blunted prolactin (PRL) responses following m-CPP, and (as a trend) lower basal PRL levels. However, after effects of binge–purge frequencies, body mass, and other extraneous factors were controlled, PRL levels at baseline and other moments in the serial sampling varied systematically with presence of impulsive and compulsive traits. PRL was generally low in BN, but ‘high-compulsive’/‘low-impulsive’ traits were associated with higher (normal-range) PRL values. Comparable trait-related variations were not observed on paroxetine-binding indices. Our findings suggest that 5-HT status in BN may correspond to impulsive or compulsive traits, and they encourage multidimensional modeling of the pathophysiological role of 5-HT in BN.

Keywords: Bulimia nervosa; Serotonin; Impulsivity; Compulsivity; Neurobiology; Personality traits

1. Introduction

Manipulations that reduce central serotonin (5-hydroxytryptamine: 5-HT) activity often precipitate compulsive or ‘binge’ eating (Brewerton, 1995). Such effects have led to the hypothesis, corroborated by various forms of clinical evidence, that bulimia nervosa (BN) involves reduced 5-HT neurotransmission. For example, as a group, BN patients display reduced platelet binding of 5-HT uptake inhibitors (Marazziti et al., 1988; Steiger et al., 2000, 2001a) and blunted neuroendocrine responses to 5-HT precursors and agonists (Brew-
inferences about central 5-HT functioning based on 5-
HPRL.

Intriguing as they are, such findings are ame-
nable to various causal interpretations: Dieting,
bingeing and purging have all been thought to
promote secondary reductions in brain 5-HT activ-
ity, and hence to underlie reduced 5-HT tone in
BN (Anderson et al., 1990; Brewerton, 1995).
However, in non-eating-disordered populations,
low brain 5-HT activity has been associated with
psychopathological-trait variations on dimensions
like impulsivity and aggression (Asberg et al.,
1987; Coccaro et al., 1989). Prominence of impuls-
vity and aggression in bulimic patients (Grilo,
2002; Steiger et al., 2003) raises the possibility
that decreased 5-HT tone in the population could
be (in part) a trait correlate. In keeping with this
idea, studies in BN have indicated 5-HT activity
to be inversely linked to levels of impulsivity or
hostility (Carrasco et al., 2000; Steiger et al.,
2001a,b; Verkes et al., 1996; Waller et al., 1996).

If the preceding points to a link between impul-
sivity and reduced 5-HT function, a question arises
concerning a substantial subgroup of BN sufferers
who display compulsive (rather than impulsive)
traits (Grilo, 2002; Steiger et al., 2003). Findings
in non-eating-disordered samples have sometimes
(Swedo et al., 1992; Fineberg et al., 1998)—but
not always (Baumgarten and Grosdanovic, 1998;
Stein et al., 1996)—associated compulsivity with
increased 5-HT tone, supporting the proposal that
‘impulsive’ and ‘compulsive’ traits may occupy
opposite poles of a continuum of 5-HT under-
to overactivity (Cloninger et al., 1993). The present
study applied this notion to 5-HT findings in BN.
If 5-HT status in BN corresponds to impulsive or
compulsive traits, then impulsivity should coincide
with decreased 5-HT activity, and compulsivity,
the converse. In contrast, if 5-HT status in BN is
largely a consequence of bulimic behavior, then 5-
HT indices in active bulimics should covary more
strongly with ‘eating-symptom’ rather than ‘trait’
variations.

We assessed 5-HT function in two ways: (1)
Given relatively direct connection between sero-
tonergic stimulation and release of prolactin
(PRL), it has become conventional to draw infer-
ences about central 5-HT functioning based on 5-
HT agonist-stimulated changes in peripheral PRL
(Yatham and Steiner, 1993). Meta-chlorophenyl-
piperazine (m-CPP) binds with highest affinity to
5-HT₂ receptors, and lesser affinity to 5-HT₁ and
alpha₂-noradrenergic receptors—and is therefore
thought to constitute a fairly specific probe of postsynaptic 5-HT function. PRL responses fol-
lowing administration of m-CPP have been used
to study 5-HT neurotransmission in several studies
on BN (Brewerton et al., 1992; Steiger et al.,
2001a,b,c). (2) Binding in platelet membranes of
5-HT reuptake inhibitors is believed to model
aspects of central (presynaptic) 5-HT reuptake
(Coccaro et al., 1996). Studies using platelet imip-
ramine binding (Marazziti et al., 1988) or parox-
etine binding (Steiger et al., 2000, 2001a) have
shown transporter density (Bmax), but not affinity
(Kd), to be reduced in bulimic vs. non-bulimic
women. A similar association has been document-
ed between aggressiveness, in personality-disor-
dered patients, and reduced density (Bmax) of
platelet paroxetine uptake sites (Coccaro et al.,
1996).

2. Methods

2.1. Participants

This study received institutional ethics-board
approval, and involved participation by informed
consent. An initial group of 67 bulimics was
recruited through a specialized Eating Disorders
(ED) program on the basis of the following crite-
ia: Female, aged 18–40, actively bingeing,
and not pregnant, anorexic, obese (body mass index
of 28 or less), or on psychoactive medications
(in the last 6 weeks). ED symptoms were confirmed
using the Eating Disorders Examination (Fairburn
and Cooper, 1993). We excluded six individuals
due to EKG abnormalities or other medical con-
ditions that would contraindicate endocrine chal-
lenge, and one more case with an EMIT urine
screen revealing amphetamine abuse. In four more
cases, a vein could not be obtained for blood
draws. We thus completed full assays in 56 wom-
en, 45 (80.4%) of whom met DSM-IV (American
Psychiatric Association, 1994) criteria for BN-
purging subtype, 6 (10.7%) for BN-nonpurging
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