Olfactory Identification Ability in Anorexia Nervosa

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Objective: **The hypothesis tested was that patients with severe eating disorders would demonstrate olfactory identification deficits as a result of zinc deficiency or malnutrition.** *Method*: **The University of Pennsylvania Smell Identification Test (UPSIT) was administered to 27 hospitalized female patients with anorexia nervosa and 50 normal control female subjects. For a subgroup of patients, serum zinc levels and body mass indices were obtained at pre- and post-nutritional repletion phases.** *Results*: **UPSIT scores for patients with eating disorders were equivalent to normal control subjects in spite of the fact that the patients were nutritionally compromised as determined by body mass index. Serum zinc levels were not significantly different at pre- and post-nutritional repletion.** *Conclusions*: **In contrast to patients with schizophrenia, patients with severe eating disorders have intact olfactory function. This finding suggests that transient metabolic or nutritional disturbances alone cannot account for previously reported olfactory deficits.**

Key Words: olfaction, anorexia nervosa, schizophrenia

INTRODUCTION

Olfactory identification has been examined in patients with a number of psychiatric conditions including schizophrenia (Hurwitz et al 1988; Kopala et al 1989; 1990; 1992; in press; Seidman et al 1992; Wu et al 1993), depression (Amsterdam et al 1987), and bipolar affective disorder (Hurwitz et al 1988). The deficits originally reported in male patients with schizophrenia and subsequently in older female patients with this diagnosis may reflect abnormalities of the orbital frontal cortex. In this regard, assessment of olfactory identification ability could serve as a behavioral measure of the functional integrity of this brain region. Previous investigators have not examined olfactory identification ability in patients with anorexia nervosa.

Malnutrition or zinc deficiency are factors that could potentially contribute to olfactory dysfunction in patients with eating disorders. Although zinc deficiency has been shown to diminish gustatory sensitivity, the evidence for a relationship between zinc status and olfactory performance is conflicting (Russell et al 1993; Krueger and Krueger 1980; Vreman et al 1980; Deems et al 1991; Doty et al 1991; Prasad 1992). Anorexia nervosa is thought to be associated with compromised zinc status in some individuals (Humphries et al 1989) while purging behavior has been reported to damage taste receptors on the palate (Rodin et al 1990).

The purpose of this study was to examine olfactory identification ability and zinc status in patients with anorexia nervosa by employing the University of Pennsylvania Smell

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Table 1

UPSIT scores and mean ages for the two groups

Diagnostic Category	*N*	Mean UPSIT	Age
Anorexia Nervosa	27	38.5 (sd = 1.4 range 36-40)	20.1 (sd = 3.6 range 14-29)
Normal Control Females	50	38.2 (sd = 1.3 range 35-40)	27.4 (sd = 11.1 range 10-56)
t		0.93	-4.20*

*$p < 0.001$

Identification Test (UPSIT) (Doty et al 1984). Olfactory function was assessed in 27 hospitalized female patients with severe anorexia nervosa. For a subgroup of patients, serum zinc levels were measured in the pre- and post-treatment phases of hospitalization. The hypothesis tested was that patients with severe eating disorders would demonstrate olfactory identification deficits as a result of zinc deficiency or malnutrition as measured by abnormal body mass index (BMI).

METHODS

Subjects

All patients were consecutively admitted to an Eating Disorders Unit at the University of British Columbia Hospital, UBC Site, or St. Paul's Hospital in Vancouver. No males were excluded but only females were referred. This study was approved by the University of British Columbia ethics committee, and all patients and subjects signed informed consent. None of the patients received financial remuneration.

Initially, 30 hospitalized female patients with anorexia nervosa and 50 control women were recruited for the study. All patients met DSM-III-R criteria (American Psychiatric Association 1987) for a diagnosis of anorexia nervosa. Of the 27 patients who met inclusion criteria, 19 were classified as nonbulimic (DSM-IV) while eight were of the bulimic subtype. None received any other Axis I or II diagnosis. Demographic data such as age and smoking habit were recorded. The mean age for the patients with anorexia nervosa was 20.1 years (sd 3.6) while that of the normal control group was 27.4 years (sd 11.1).

Exclusion criteria included feeding by nasogastric tube (n = 3), upper respiratory tract infection, recent nasal cocaine use or other substance abuse, persistent rhinitis, head injury, facial trauma or other medical conditions which could interfere with normal olfactory ability (for example, hypothyroidism). All patients were examined within the first three days of hospitalization.

Normal weight female control subjects were recruited from the hospital and university staff through notices placed on bulletin boards. All control subjects met the same exclusion criteria as did patients, and none had a past history of or was currently suffering from an eating disorder or other psychiatric disorder. The former was verified by a semi-structured psychiatric interview for eating disorders (Goldner et al 1992) while the latter was determined by subjective account.

Measures

Olfactory identification ability was assessed by administering the UPSIT (Doty et al 1984) after a method described in detail elsewhere (Kopala et al 1989; 1992). Patients were physically examined on admission and smell-tested within the first three days of hospitalization prior to the initiation of treatment. General laboratory investigations included complete blood count with differential, urinalysis, electrocardiogram and a determination of thyroid functioning (T_4 or TSH). Body mass indices were calculated by dividing weight in kilograms by height in meters squared (kg/m^2). For 15 of the patients, pre-treatment serum zinc levels were obtained at the same time that the UPSIT was completed. Subsequently, ten of these 15 patients had serum zinc levels repeated along with the UPSIT in the post-nutritional repletion phase.

RESULTS

The mean UPSIT scores and ages for the two groups are presented in Table 1. The UPSIT scores of the patients with anorexia nervosa and the normal control subjects did not differ significantly. The mean score of both groups, and the individual scores of all patients and controls would be classified as normosmic (scoring 35 or more out of a possible 40) (Doty et al 1984). 38.5% of patients and 16.7% of normal control subjects were smokers.

The normal control females were significantly older than the patients ($t_{65} = -4.20, p < 0.001$). However, standardization data (Doty et al 1984) indicates that mean UPSIT scores for these two age groups are not different. The mean BMI for patients was 16.0 (range 13.5 kg/m^2 to 20.5 kg/m^2) which is much lower than published normative values (18 kg/m^2 to 24 kg/m^2) (Treasure et al 1985). Additionally, BMI for a subgroup of 11 young control females was calculated (mean age 15.5 years) at 18.9 (range 15.7 to 23.0). This was significantly higher than that of the patients ($t_{24.0} = -3.65, p < 0.001$).

Table 2

Mean serum zinc levels and UPSIT scores at pre - and post-treatment phases

	Pre-Treatment	Post-Treatment	t
Zinc (pmol/L)	13.4 (2.8)	12.5 (2.1)	0.87
UPSIT	38.3 (1.2)	38.2 (1.2)	0.24

For the 15 patients who had pre-treatment zinc levels analyzed in addition to completing the UPSIT, the mean pre-treatment serum zinc level was 13.4 mol/L (sd = 2.8). This value was considered to be within the laboratory's normal range. There were no significant differences between mean serum zinc levels at pre- and post-treatment ($t_{(9)} = 0.87$, $p > 0.05$). Finally, mean UPSIT scores did not differ at pre- versus post-treatment (38.3 versus 38.2) ($t_{(8)} = 0.24, p > 0.05$) (see Table 2).

DISCUSSION

The cardinal finding of this study is that hospitalized female patients with severe anorexia nervosa have intact olfactory identification ability despite abnormal body mass indices. In addition, only one of the patients had abnormal zinc status as determined by laboratory standards at both pre- and post-treatment. One possible caveat to the interpretation of these data is that serum zinc levels may not adequately reflect total body zinc. In this regard, zinc is believed to be predominantly an intracellular trace element. Other measures of zinc status such as hair analysis may be equally unreliable (Meftah et al 1991; Sandstead 1991).

Although olfactory deficits have been reported in patients with schizophrenia, patients with eating disorders have not been studied. Moreover, the relationship between olfactory status and serum zinc levels has not been clearly established. A case report (Krueger and Krueger 1980) described hyposmia as being associated with low serum zinc levels. Vreman et al (1980) reported no correlation between zinc concentrations in taste or smell mean detection levels in patients with chronic renal failure. Subsequently, Russell et al (1983) concluded that there is limited evidence to support the notion that zinc deficiency could result in impairment of olfactory performance in humans. Furthermore, Deems et al (1991) reported that zinc therapy may provide no benefit to patients with olfactory and gustatory dysfunction.

More patients than normal control subjects were smokers. If smoking did interfere substantially with olfactory identification ability then it would be expected that patients with eating disorders would have lower UPSIT scores. However, the patients had higher UPSIT scores as compared to normal controls. Therefore, smoking did not appear to influence olfactory performance negatively in this group of patients. This finding supports previous results for patients with schizophrenia (Kopala et al 1992; Wu et al 1993).

The uniformly excellent performance on the UPSIT by these women with eating disorders was unexpected. It had been hypothesized that abnormalities would exist in this group because most patients were nutritionally compromised, as indicated by their low BMI. It is possible, however, that the UPSIT did not allow for discrimination between subjects and patients who score in the normal range. In this regard, the UPSIT was designed to identify individuals with olfactory deficits (Doty et al 1984). The finding of intact olfactory identification ability in women with anorexia nervosa and severe nutritional depletion suggests that olfactory ability is a stable phenomenon, unlikely related to state. This finding could further our understanding of the potential significance of olfactory identification deficits in patients with schizophrenia. It has been previously suggested that the olfactory identification deficits in patients with schizophrenia are more likely a reflection of trait abnormalities than a consequence of psychotic symptoms (Kopala et al 1989; 1992). In this regard, Seidman et al (1992) have speculated that the UPSIT may serve as a behavioral probe for orbital frontal cortex functioning in patients with schizophrenia. If such is the case, patients with eating disorders and nutritional depletion do not demonstrate altered function in orbital frontal regions or in other brain regions subserving olfactory pathways. Furthermore, the observed olfactory function in anorexia lends credence to the notion that the deficits in schizophrenia are not likely to be related to transient metabolic or nutritional imbalance which could accompany psychotic illness. Rather, for some patients with schizophrenia, a more enduring, stable brain abnormality may be present.

Finally, the results of this study add to a steadily growing body of literature concerning the involvement of the olfactory system in patients with psychiatric disorders. Specifically, this group of psychiatrically and nutritionally compromised women showed no evidence for deterioration in central nervous system processing of olfactory stimuli.

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