Auditory P300 In College-Aged Females At-Risk For Eating Disorders

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Abstract

Neurological impairment in eating disorders has been widely documented. The P300 component of event-related potentials has demonstrated efficacy in identifying individuals who suffer from mental disorders and cognitive deficits. More recently, a number of researchers have used P300 event-related potentials (ERPs) to elucidate the effects of food intake and glucose metabolism on brain function and performance on cognitive tasks. Minor food deprivation decreases P300 amplitude and increases P300 latency, indicators of impaired cognitive performance. Short-term memory disruptions correlate with such ERP decrements. The present study examined the auditory P300 in females who are at-risk for developing an eating disorder as defined by a score of 14 or more on the Drive for Thinness subscale of the Eating Disorder Inventory-2 (EDI-2). Memory performance was also examined. The results failed to support the hypothesis that at-risk females, compared to normal controls would restrict their food intake and would therefore exhibit smaller P300 amplitudes, longer P300 latencies, and impaired short-term memory. There was also no statistically significant difference between the at-risk and normal controls group on a self-report measure of amount of and time since last food consumption. Interestingly, the at-risk and normal controls groups also differed significantly on EDI-2 scores for the bulimia, interoceptive awareness, and asceticism subscales.

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At-Risk For Eating Disorders

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The Auditory P300 In Females At-Risk For Eating Disorders

The precise etiology of the eating disorders has remained elusive. Both major variants, anorexia nervosa and bulimia nervosa, are characterized by an extreme drive for thinness that ultimately leads to dangerously abnormal eating behaviors (Garner & Olmstead, 1984; Raciti & Norcross, 1987; Garner, 1991). Eating disordered individuals effectively starve themselves, whether it be through the extreme dietary restraint exhibited by anorexics, or the incessant bingeing and purging of bulimics.

Food deprivation in the eating-disordered individual leads to impaired cognitive performance on memory, focusing/execution, verbal, and visuospacial tasks (Pendleton Jones et al., 1991). Such deficits have lead researchers to investigate neurological correlates of eating-disordered thought and behavior for additional tests to screen for eating disorders. Because the P300 component of the event-related potential (ERP) may be sensitive to food intake, and has proved useful in diagnosing disorders such as schizophrenia, depression, and Alzheimer's dementia in individuals who exhibit memory and attention deficits, the P300 may also be an effective screening device for eating disorders. Accordingly, a review of the eating disorders, the P300, and their link to cognitive impairment and food intake follows.

EATING DISORDERS

It is estimated that up to 20% of school-aged (10 to 18 years) children have an eating disorder (Kinoy, 1984; Phelps & Wilczenski, 1993). Estimates of incidence range up to 14% for college-aged (18-22 years) females (Szmukler, 1983; Herzog,

Norman, Rigotti, & Pepose, 1986). These disorders often result in serious health concerns. Abnormal glucose tolerance, cardiovascular problems, anemia, liver damage, epileptic seizures and renal failure are some of the complications (Hsu, 1990). Additionally, the eating disorders are marked by considerable rates of morbidity and are difficult to treat.

Physiological, psychological, biological and sociocultural factors all contribute to the development of eating disorders. It is widely accepted that the incessant bombardment of children from an early age with media images of idealized beauty has led to a greater occurrence of both anorexia nervosa and bulimia nervosa in western cultures. Indeed, rates of incidence are highest in those populations that place the greatest emphasis on dieting or thinness (Garner, Garfinkel, Schwartz, & Thompson, 1980; Hsu, 1990). There is a clear gender bias; the female to male ratio of eating disorders is 10:1 (Garner, 1991). Additionally, higher prevalence among professional dancers (Brooks-Gunn, Burrow & Warren, 1988), figure skaters (Brooks-Gunn et al., 1988), gymnasts (Rosen & Hough, 1988) and other athletes has been widely documented. The number of cases is growing, particularly among adolescents and those in early adulthood (Leichner & Gertler, 1988).

Eating disorders are typically split into two subtypes: anorexia nervosa, which is characterized by self-starvation to the point of malnutrition, and bulimia nervosa, which involves constant bingeing and purging. At the heart of these disorders is the possession of distorted perceptions of weight, body shape, and eating as well as extreme asceticism and impulsiveness (Killen et al., 1994; Striegel-Moore, Schrieber, Pike, Wilfley, & Rodin, 1995). Thus, the two variants of eating disorders share these

key aberrant thoughts and perceptions. It is the behavioral manifestations that differentiate anorexia from bulimia.

There has been recent discussion and research regarding subclinical variants of the eating disorders or "at-risk" individuals (Bunnell, Shenker, Nussbaum, Jacobson, & Cooper, 1990; Herzog, Hopkins, & Burns, 1993; Williams, Schaefer, Shisslak, Gronwaldt, & Comerci, 1986). Individuals who are classified as at-risk typically score high on measures of eating disorder symptoms but fail to meet the strict diagnostic criteria of the DSM-IV. It has been suggested that the eating disorders occur on a continuum, with normal dieters at one end of the scale and eating disordered patients at the other (Garner & Olmstead, 1984; Raciti & Norcross, 1987). Herzog, Hopkins & Burns (1993) investigated the presenting symptoms of 33 eating disordered women who failed to meet the DSM-III-R criteria, and were therefore classified "subdiagnostic". The researchers reported that the most commonly missed DSM-III-R criterion for their anorexic sample was amenorrhea. The most commonly missed criterion for their bulimic sample was bingeing and purging frequency, even though the subclinical individuals were bingeing and purging one or two times a week. At follow-up (mean 41 months) 79% of the subjects still exhibited significantly disordered eating behaviors and 46% had progressed to meet the DSM-III-R criteria for anorexia nervosa or bulimia nervosa.

Yager, Landsverk, and Edelstein (1987) obtained similar results. At a 20-month follow-up, 33% of their at-risk subjects had progressed to develop a full eating disorder. Clearly, at-risk behaviors such as "subclinical" bingeing and purging or dietary restraint are stressful and dangerous. Numerous outcome studies have reported that such at-

risk behaviors do persist (Hsu & Sobkiewicz, 1989; Herzog, Keller & Lavori, 1988), and in up to 40% of these individuals, progress to a clinical eating disorder (Garner, Olmstead, Garfinkel, 1983, Garner, 1991) pointing to the need for early identification and intervention.

The border between eating disorders and other forms of psychopathology is not always clear. The literature describes eating disordered individuals as possessing irrational beliefs regarding eating and body issues as well as abnormal thoughts in general (Hsu, 1990; Crisp, 1980). Both bulimics and anorexics tend to see inner hunger and eating sensations in a "black and white" manner. Anorexics, however, tend to extend this absolutist thinking to all facets of life (Butow, Beumont, & Touyz, 1993). Considerable rates of depression (Cooper & Fairburn, 1986; Laessle, Kittl, Ficter, Wittchen & Pirke, 1987), obsessionality (Beumont, George, & Smart, 1976), anxiety (Mitchell, Davis & Goff, 1985), and borderline personality disorder (Piran, Lerner, Garfinkel, Kennedy, & Brouilette, 1988) have been observed in anorexia nervosa and bulimia nervosa. Hsu, Kaye, and Weltzin (1993) recently presented the case for a possible link between the eating disorders and obsessive-compulsive disorders (OCD). Individuals with these disorders share many personality traits and there does appear to be a higher incidence of OCD in those who suffer from eating disorders. OCD and the eating disorders may also share an underlying neurobiological disturbance; serotonin reuptake inhibitors have proven effective in treating both OCD and eating disordered patients.

Classic descriptions of eating disorder patients reported the same stereotypic symptoms and beliefs clinicians and researchers report today (Pope and Hudson,

1988), including the 10 to 1 ratio of female to male victims. Vitousek and Hollon (1990) examined cognitive content and processing in individuals with eating disorders. They argue that anorexia is represented in cognitive structures that combine extremely perfectionistic views of the self with abnormal beliefs about weight. These strict, weight-related self-schemata, in turn, influence cognitive processing. In other words, the anorectic's strong preference for extreme thinness pervades her thinking, influencing her perceptions, attributions, expectations and memories. Strangely enough, the anorectic patient will often acknowledge that eating disordered behavior in other persons is abnormal, but will not apply the same assessment to herself. There is also a constant drive toward concealment of perceived imperfections, which pushes the anorectic to excel at everything they do, especially dieting. Bulimics, like anorectics, are in a struggle to maintain control over their weight and body shape, and have decided that bingeing and purging is the best way to maintain their body ideal. They persist in these beliefs despite the fact that they eventually show outward signs of damage. Factors such as these undoubtedly contribute to the refractory nature of the eating disorders.

Screening And Diagnosis

Another characteristic of eating disorders is heterogeneity of the causes and presenting symptoms among patients. Because there is no universal protocol for diagnosing the eating disorders, assessment becomes a complicated exercise. Most diagnostic methods rely upon self-report measures and clinical interviews. Given the heterogeneous presentation and etiology within the eating disordered population, the most appropriate route to diagnosis is the structured clinical interview, which allows the

clinician to ascertain which DSM-IV diagnostic criteria are met. However, a complete medical work-up would be quite lengthy and not practical for screening purposes. There are a number of instruments available for the purpose of measuring the existence and severity of symptoms commonly encountered in anorexia nervosa and bulimia nervosa. Such instruments are useful in screening populations and obtaining a preliminary picture of the unique constellation of symptoms in a particular individual(s). One commonly used instrument of this type is the Eating Disorder Inventory-2 (EDI-2)(Garner, 1991).

The EDI-2 is a 91 item, self-report instrument which measures the symptoms most frequently observed in eating disordered individuals. Responses to individual items are obtained through a six-point scale ranging from "always" to "sometimes" to "never". The EDI-2 consists of the original 64 items of the EDI (Garner & Olmstead, 1984, 1986) which are three subscales measuring attitudes and behaviors revolving around eating, body shape, and body weight (Body Dissatisfaction, Bulimia, Drive for Thinness) and five which assess clinically significant traits or constructs (Ineffectiveness, Interoceptive Awareness, Interpersonal Distrust, Maturity Fears, and Perfectionism). Twenty-seven new items are found in the EDI-2, containing three provisional subscales (Asceticism, Impulse Regulation, and Social Insecurity) (Garner, 1991). The instrument's 11 subscales and the constructs they measure are: Drive for Thinness - measuring the degree to which an individual is excessively focused on the pursuit of a thin body; Bulimia – assessing the tendency to binge or eat excessive amounts of food; Body Dissatisfaction – measuring dissatisfaction with the size and shape of the stomach, hips, thighs and buttocks; Ineffectiveness – measuring the

degree to which an individual feels a lack of control over their life and attendant feelings of inadequacy and worthlessness; Perfectionism – measuring the tendency for eating disordered individuals to believe that all of their actions should be perfect; Interpersonal Distrust – measuring an individual's need to remain at an emotional distance from others; Interoceptive Awareness – assessing uncertainty and confusion in identifying and appropriately responding to emotional and visceral states such as hunger cues; Maturity Fears – measuring the need to escape to the safety of childhood; Asceticism – assessing the pursuit of virtue through self-denial and control of urges and drives; Impulse Regulation – measuring the existence of poor impulse regulation as demonstrated by recklessness and self-destructive behavior; and Social Insecurity – assessing distrust of social relationships.

The EDI-2 is useful for obtaining a picture of the psychological and behavioral factors operating in the patient, as well as setting up a treatment plan and determining its effectiveness and patient progress. The descriptive information provided by the inventory is organized into a patient profile that can be compared to a set of norms from both normal and eating disordered populations. The EDI-2 is effective at screening nonpatient samples and may be used to study factors related to the constructs measured by the instrument (Garner, 1991).

There have been a number of studies, in addition to the work on the original EDI (Garner & Olmstead, 1984), looking at the internal consistency of the EDI and the EDI-2 provisional subscales. In the original eating disordered sample, Garner and Olmstead obtained reliability coefficients (Cronbach's alpha) for the EDI subscales ranging from 0.83 to 0.93. Internal consistency was also found to be respectable in a

large Swedish sample (exact range of alphas reported as "similar to the original study"; see Norring, 1990; Norring & Sohlberg, 1988) and patients from New Zealand (Welch, Hall, & Norring, 1990), with alphas in the range of 0.70 to 0.81.

Samples of nonpatient college women were used to provide reliability estimates; coefficients ranged from 0.72 to 0.92 (Garner & Olmstead, 1984), 0.79 to 0.92 (Raciti & Norcross, 1987), and 0.69 to 0.93 (Vanderheyden & Boland, 1987). Reliability estimates for the provisional subscales of the EDI-2 were 0.70, 0.77, and 0.80 for the eating disordered sample, and 0.44, 0.79, and 0.80 for the nonpatient college female sample for Asceticism, Impulse Regulation and Social Insecurity respectively (Garner, 1991).

Test-retest reliabilities were obtained through several studies. Welch, Hall, & Walkey, (1988) examined two administrations of the EDI a week apart and reported coefficients ranging from 0.79 to 0.95 for all subscales with the exception of the Interoceptive Awareness subscale that had a reliability coefficient of 0.67. Wear and Pratz (1987) reported even higher coefficients, all (with the exception of Maturity fears) greater than 0.80, at retest three weeks later. The EDI-2, then, appears to be a reliable instrument that measures a collection of stable traits exhibited by eating disordered individuals.

The EDI-2 demonstrates appropriate levels of validity. During development of the instrument, only items that discriminated between eating disordered and healthy subjects were retained, providing evidence of criterion-related validity (Garner, 1991). Concurrent validity was demonstrated by way of correlations between anorexia nervosa patients' subscale scores and clinicians' ratings. Statistically significant correlations

($p>0.001$) for the Drive for Thinness and the Body Dissatisfaction subscales were 0.53 and 0.44 (Garner, 1991).

Finally, construct validity has been reported in the form of convergent and discriminant validity, subscale intercorrelations, and factor analysis. A factor analysis performed by Welch, Hall and Walkey (1988) using three nonpatient samples resulted in a three-factor solution. Factor loadings resulted in the identification of the Perfectionism subscale, a factor composed of the Drive for Thinness, Bulimia, and Body Dissatisfaction subscales, and a factor formed by the collapse of the Ineffectiveness and Interoceptive Awareness subscales. Several other studies resulted in the identification of the original eight factors (Welch, Hall, & Norring, 1990; Norring, 1990; Williams, Schaefer, Shisslak, Gronwaldt, & Comerci, 1986).

The instrument has been successfully used to identify individuals who are at-risk for developing an eating disorder. The construct of drive for thinness has been described as one of the key motivational variables in the development of eating disorders (Polivy & Herman, 1993) and is related to dietary restraint (Striegel-Moore, Schreiber, Pike, Wilfley, & Rodin, 1995). Cooper, Cooper & Fairburn (1985) provided evidence that the Drive for Thinness, Body Dissatisfaction, and Bulimia subscales actually do measure core psychological and behavioral traits of individuals who are suffering from eating disorders. Vanderheyden & Boland (1987) used a discriminant function analysis to show that a drive for thinness, along with dietary restraint and negative self-image, were the strongest predictors of which individuals were eating disordered. Garner, Olmstead, Polivy, & Garfinkel (1984) used the Drive for Thinness subscale to identify female college and ballet students who were "weight preoccupied".

Clinical interviews were then performed to assess the subjects' diagnostic status. 75% of those who had elevated scores on the Drive for Thinness subscale either had an eating disorder in the past or at the time of testing. In a group of female ballet students, Garner, Garfinkel, Rockert, & Olmstead (1987) found that Drive for Thinness scores predicted the development of eating disorders. In these studies the anorexia nervosa comparison groups had mean scores of 15 on the Drive for Thinness subscale. Norring and Sohlberg (1988) reported that by using an even more stringent cutoff of 17 (97th percentile) they were able to identify a group of women all of whom had an eating disorder.

Adult females with high Drive for Thinness scores consume fewer calories than those with normal scores (Laessle, Tuschl, Kotthaus, & Pirke, 1989a, 1989b) and it is believed that such successful past dieting is crucial for the development of eating disorders (Garner et al., 1984). Rezek & Leary (1991), using a liberal cutoff score of 8, found that women who had elevated DT scores (mean DT score: 12.25) and were placed in a situation where they experienced low levels of control over the outcome of an interpersonal interaction, ate less sweetened cereal and planned to eat less dinner than controls. Finally, Williamson et al. (1995) found that high DT scores showed a statistically significant correlation with a measure of dietary restraint (the Dietary Restraint scale of the TFEQ). Thus, the Drive for Thinness subscale is a reliable tool for identifying females with a preoccupation with weight and body size and who are restricting their food consumption - two factors that have been shown to predict the presence or propensity towards an eating disorder.

Garner (1991) recommends a cutoff score of 14 (which corresponds to the 90th percentile in college-aged female samples) on the Drive for Thinness subscale when screening for eating disorders. Of those identified by such a score, up to 40 percent can be expected to have an eating disorder of clinical severity. Garner, Olmstead, and Garfinkel (1983) reported that such a sample of weight-preoccupied females includes subgroups of "normal dieters" as well as those who exhibit psychopathology similar to anorexia nervosa.

Brain Function And Eating Disorders

The eating disorders have been linked to brain dysfunction. The neurological complications are numerous and diverse. Hsu (1990) reviewed brain metabolic disturbances associated with anorexia nervosa and bulimia nervosa. Chief among these are abnormal glucose tolerance, faulty temperature regulation, and electrolyte imbalances, all of which may produce EEG abnormalities. Indeed, there are electroencephalographic disturbances in 40% to 50% of patients, most believed due to electrolyte imbalances (Crisp, 1980;Crisp, Fenton, & Scotton, 1968). These abnormalities in EEG background rhythms appear to be associated with low serum potassium and sodium values. This may be due to reduced intake of electrolytes or increases in cerebral ventricular volume. Numerous investigators have observed the concomitant cerebral atrophy on computerized tomography scans (Datlof, Coleman, Forbes, & Kreipe, 1986; Krieg, Pirke, Laver & Backmund, 1988). While these abnormalities were observed in clinical settings at advanced stages where the patients were severely emaciated, it is possible that some of these disturbances may appear at earlier stages to a lesser degree.

It is well documented that there are marked central nervous system abnormalities in individuals with eating disorders. The demonstrated link between affective disorders and anorexia nervosa (Biederman et al., 1985; Hurt, Brun-Eberentz, Commerford, Samuel-Lajeunesse, & Halmi, 1997) has led some to investigate right parietal lobe abnormalities (Kinsbourne and Bemporad, 1984; Bradley et al., 1996). Delvenne et al. (1997a), in a recently published a positron emission tomography (PET) study, found parietal lobe hypometabolism of glucose in normal weight bulimics compared to healthy subjects. In addition, these glucose effects did not correlate with Body Mass Index (BMI) and all subjects were in a "stable metabolic state" indicating that the effects were independent of the consequences of starvation. In a previous study Delvenne et al. (1995) uncovered the same decrease in parietal glucose metabolism in anorexic patients. These parietal lobe deficits are particularly important since it is believed that this region is involved in body size and appearance perception. A distorted body image is a key feature of the eating disorders (Horne, Van Vactor, & Emerson, 1991).

Abnormalities in neurotransmitters (Halmi, Dekirmenjian, Davis, Casper, & Goldberg, 1978), neuropeptides (Gold, Kaye, Robertson, & Ebert, 1983), and visual evoked responses (Aono & Kumashiro, 1983) have also been reported. Serotonin (5-HT) plays a key role in eating and postprandial satiety; increasing intrasynaptic serotonin decreases food consumption (Berk, Kessa, Szabo, & Butkow, 1997). Serotonin synthesis and release into the synaptic cleft are driven by the diet-provided amino acid tryptophan, which is a 5-HT precursor. Thus, there has been much attention paid to this neurotransmitter as it relates to eating disorders. Disturbances in

serotonin regulation have been widely documented for both bulimia nervosa and anorexia nervosa patients (Goldbloom, Hicks & Garfinkel, 1990; Brewerton et al., 1992; Kaye et al., 1988; & Weizman et al., 1986), and the efficacy of 5-HT reuptake blockers in treating eating disorders is well known (Leibowitz, 1990).

Pendleton-Jones et al. (1991) reported impaired performance by underweight anorectics on a comprehensive battery of neuropsychological tests. Anorectics performed more poorly in memory, verbal, focusing/execution, and visuospacial domains. Fox (1981) found decreased arithmetic performance and Brouwers et al. (1986) uncovered performance problems on both verbal and non-verbal tasks in anorexia nervosa subjects. Recent research has focused on identifying which of these performance deficits are actually features of the eating disorders and which are a result of eating disorder-related problems such as hormonal imbalances (Hsu, 1990) and other psychological pathologies such as depression and anxiety (Hsu, Kaye, & Weltzin, 1993).

It is also difficult, in many cases, to determine whether the deficits reported above are a result of malnutrition and starvation or if they existed prior to onset. There is at least some evidence that starvation can lead to eating disorder-like symptoms. The classic example of this is the landmark study of World War II conscientious objectors by Keys, Brozek, Henschel, Mickelsen, and Taylor (1950). These starved individuals exhibited depression, preoccupation with food, apathy, isolation, and abnormal eating behaviors – classic eating disorder symptoms. In addition, Maloney, Brunner, Winget, and Farrell (1983) found that adolescent anorexics who gained at least 3.4 kg showed an improvement in 6 of the 7 symptoms reported by Keys et al.

P300

The P300 component of the event-related potential, the primary dependent variable in this study, has been shown to correlate with memory and attentional deficits similar to those observed in eating disordered individuals. It is sensitive to relatively minor variations in daily food intake and brain glucose metabolism (Geisler & Polich, 1992). The abnormal eating patterns of both at-risk and clinically diagnosed eating disordered individuals point to the P300 as a potential screening instrument.

Event-related potentials (ERP) are manifestations of underlying brain activity recorded at the scalp via an electrode montage. More specifically, these potentials represent a summation of synchronous electrical activity of open fields of neurons, most likely graded post-synaptic potentials, because of their longer duration in contrast to the high frequency, shorter duration presynaptic spikes. The investigational importance of the ERP lies in the fact that the eliciting stimuli are external and are thus amenable to scientific manipulation. The P300 component of the ERP is a positive peak with a latency of 300 msec. The functional value of the P300 comes from the conceptualization of the component as an electrophysiological measure of cognitive function. More specifically, variations in P300 have been characterized as representing attentiveness, uncertainty reduction, processing demand, task demand, value, context-updating, and task difficulty (Johnson, 1989; Donchin, 1981; Goodin, 1990). The P300 is produced when an infrequent, task-relevant stimulus is encountered. Additionally, the P300 appears to be endogenous, produced primarily in response to the nature of information processing required of the subject (Johnson, 1986), and it is largely independent of the physical stimulus.

The P300 is believed to emanate from sources in the temporal lobe, in particular the hippocampus (McCarthy et al., 1989) and superior temporal gyrus (Knight et al., 1987; McCarley et al., 1993). Other brain structures, such as parietal lobe, are also likely to contribute. A large body of research posits that the putative brain generators involved in the auditory P300 production are hippocampus and structures of the temporal cortex (Geisler & Polich, 1992; McCarley, Faux, Shenton, Nestor, & Adams, 1991; McCarley, Shenton, O'Donnell, Faux, Kikinis, Nestor, & Jolesz, 1993). Temporal lobe has also been implicated in the abnormal unilateral P300 findings in schizophrenics with suspected temporal lobe damage (McCarley et al., 1993).

Knight, Scabini, Woods, and Clayworth (1989) studied the auditory P300 in subjects who had lesions of either: 1) temporal-parietal junction, or 2) lateral parietal lobe. P3a waves were recorded to target stimuli while subjects performed an oddball detection task, and P3b waves were recorded to novel stimuli as subjects performed a dichotic listening task. The subjects with lateral parietal lobe lesions produced normal P300s compared to controls. Subjects with unilateral lesions at the temporal-parietal junction, more specifically, in the posterior superior temporal plane, did not produce P3a nor P3b waves over the Pz and Cz electrode sites. There was, however, some P300 activity at Fz, pointing to the possibility of multiple P300 generators. These data correlate with a reduced ability to orient to distracting stimuli in the same subjects suggesting that the system that produces the auditory P300 is responsible for behaviors that involve orientation to environmental stimuli.

Differences in P300 wave amplitude and latency have been found in disorders associated with abnormal cognitive functioning, such as schizophrenia (McCarley,

Faux, Shenton, Nestor, & Adams, 1991), depression (Swanwick, Rowan, Coen, & O'Mahoney, 1995), obsessive compulsive disorder (Towey, Tenke, Bruder, Leite, Friedman, Liebowitz, & Hollander, 1994), attention deficit hyperactivity disorder (Kuperman, Johnson, Arndt, & Lindgreen, 1996), bipolar disorder (Blackwood, Sharp, Walker, & Goody, 1996), and Alzheimer's type dementia (McCarley, Shenton, O'Donnell, Faux, Kikinis, Nestor, & Jolesz, 1993). Therefore, the P300 component of the event related potential is believed to emanate from structures in the temporal-parietal area and is affected when these areas are in some way compromised. The P300, then, may prove to be a useful indicator of underlying neural functioning in eating disordered individuals who exhibit central nervous system dysfunction such as sulcal widening (Krieg, Lauer, & Pirke, 1989) and regional hypometabolism of glucose in parietal lobe (Delvenne et al., 1995, 1997a, b), which may play a role in the distorted body images eating-disordered individuals possess.

P300, Attention, And Memory

Memory and attentional deficits are clinically important signposts to neuropathology. The widely documented role of the P300 as a measure of attention and memory processes in healthy individuals (Gordon et al., 1986; Donchin & Coles, 1988; Goodin, 1990; McPherson, 1995) as well as those with significant neuropathology (Baribeau-Braun et al., 1983; Pfefferbaum et al., 1984; Polich, Ladish, & Bloom, 1990) underscores its clinical relevance as a measure of cognitive impairment. A review of these studies follows.

P300 latency has been described as being reflective of speed of information processing (Polich et al., 1983). More specifically, Magliero et al. (1984) found P300

peak latency reflects stimulus evaluation time; longer reaction times correlate with longer stimulus evaluation times. McCarthy and Donchin (1981) altered stimulus discriminability by manipulating extraneous noise levels in target stimuli. P300 latency increased with the inclusion of noise. Numerous researchers have reported evidence of P300 latency increases in conjunction with increases in memory load (Brookhuis, Mulder, Mulder, & Gloerich, 1983; Ford, Pfefferbaum, Tinklenberg, & Kopell, 1982). Finally, Kramer, Schneider, Fisk, & Donchin (1986), in an investigation into the habituation effect observed over repeated trials, found that P300 latency decreased as reaction times became quicker, indicating that the "development of automatic processing substantially reduced stimulus evaluation time".

Polich, Howard and Starr (1983) suggest that P300 latency is sensitive to individual differences in memory. Subjects were administered the Digit Span subtest of the Weschler Adult Intelligence Scale and P300s were recorded to a simple auditory counting task. P300 latency was negatively correlated with memory score, indicating that latencies were smaller for individuals who showed better memory performance. These findings suggest that P300 latency indexes memory capacity in healthy subjects and is in agreement with studies that have found longer latencies in individuals with cognitive impairment caused by various types of brain damage. A review of these findings will be presented later in this paper.

P300 amplitude has been described as being sensitive to the cognitive demands of the task (Kramer, Wickens, & Donchin, 1983; Johnston, Miller, & Burleson, 1986); the more difficult the task, the larger the amplitude. In an influential paper, Ray Johnson (1986) proposed a triarchic model of P300 amplitude whereby all experimental

influences on P300 amplitude could be described by three components: subjective probability, stimulus meaning, and information transmission. Subjective probability is the unexpectedness of the stimulus; the less expected the stimulus, the larger the P300 amplitude. Amplitude increases for stimulus meaning are due to stimulus complexity, task complexity and valuation of the stimulus. Finally, the greater the amount of stimulus information transmitted to the subject the greater the P300 amplitude. Stimulus probability and stimulus meaning are believed to be independent influences on P300 amplitude.

Donchin (1981) conceptualized the functional significance of the P300 as being indicative of memory context-updating processes. This hypothesis predicts that unanticipated or rare events will result in the updating of memory with the goal of maintaining a current and up-to-date representation of the environment. P300 amplitude also appears to be sensitive to the perceptual/cognitive resource demands of a task (Kramer et al., 1986); increases in amplitude occur with increases in the difficulty of the primary task. Karis, Fabiani, & Donchin (1984) tested the hypothesis that P300 is a measure of the context updating of working memory. Such short-term information storage is a basic feature of many cognitive actions. The rare event or stimulus presentation allows an updating of the event in working memory. Such updating theoretically provides cues or details for later retrieval. If P300 is a measure of the degree of updating, then it should predict recall. Subjects were presented with a list of words to be recalled later and ERPs were recorded both during list presentation and during word recall. When subjects used simple rote memorization strategies, P300 amplitude was larger during both presentation and word recall. For subjects who used

a more elaborate memorization strategy, P300 amplitudes were smaller at recall and there was no relationship between the amplitude at recall and the amplitude at presentation. For both groups, recalled words generated larger P300 amplitudes than words not recalled.

Fabiani, Karis and Donchin (1986) replicated the above findings while using an incidental recall task to ensure that subjects did not use elaborate memorization strategies. They again found that recalled words produced larger P300s both during recall and at presentation. Rote memorizers are constantly updating their working memory for subsequent recall. Those who use elaborate memorization schemes are tapping into processes and features unrelated to such stimulus context updating. That is, they are not so much updating their representation of the word in memory as they are fitting it into an elaborate story or relating it to other themes. These elaborate memorization activities would occur well outside of the 250 to 600 msec P300 window. Such findings point strongly toward the P300 as a measure of more basic memory processes.

As mentioned earlier, the P300 has been used to index perturbations of memory and attentional processes observed in healthy volunteers. It has been widely documented that schizophrenic patients have smaller P300 amplitudes than healthy controls subjects (Baribeau-Braun et al., 1983; Brecher & Begleiter, 1983; Pfefferbaum et al., 1984) as well as left < right topographic asymmetry (McCarley et al., 1993). Schizophrenics are also reported to have prolonged P300 latencies (Pfefferbaum et al., 1984). Depressed patients have demonstrated reduced P300 amplitude as well (Patterson, Michalewski, & Starr, 1988; Swanwick, Rowan, Coen, & O'Mahoney, 1995;

Pfefferbaum et al. 1984). Finally, Towey et al. (1990) presented evidence of P300 latency indexing the cortical hyperarousal typical of OCD patients. Using an auditory oddball task they gradually increased task difficulty, which would theoretically lengthen P300 latencies. OCD patients displayed shorter latencies than controls as the task became more difficult.

It is well known that patients with Alzheimer's disease present with severe short-term memory loss. Polich, Ladish, & Bloom (1990) investigated P300 assessment in the early stages of Alzheimer's disease in 16 patients. P300 amplitudes were significantly smaller and latencies were significantly larger for these patients. The latency findings are especially interesting given the fact that Alzheimer's patients have difficulty learning new information and tasks; problems with the ability to update working memory, which the P300 indexes, would clearly interfere with the transfer from short-term to long-term memory needed for learning and memory formation. Thus, the P300 is a useful, on-line measure of a number of attentional and cognitive processes which has demonstrated utility in differentiating between normal subjects and those who are experiencing disruptions in memory and/or attention.

P300, Eating, And Metabolism

Glucose administration improves performance on a number of memory-related neuropsychological tests in groups that traditionally show memory impairment, such as the elderly (Hall et al., 1989; Manning, Hall, & Gold, 1990). There is also evidence that low glucose levels are associated with minor impaired memory performance in normal subjects. A number of studies have demonstrated that the P300 is sensitive to food intake and brain glucose levels. In the short term, P300 amplitude is larger in

subjects who have recently eaten compared to those who have not eaten (Geisler & Polich, 1992). Additionally, P300 latency is delayed in subjects experiencing insulin induced hypoglycemia (Blackman, Towle, Lewis, Spire, & Polonsky, 1990; Jones et al., 1990). However, when fasting induces hypoglycemia, the latency effects are described as small (Geisler & Polich, 1990). Gallai, Mazzotta, Firenze and Del Gatto (1988) offered further support to the above latency findings, in addition to reporting a decrease in P300 amplitude with increased levels of hypoglycemia. Such variations were recorded at hypoglycemic levels where substantial cognitive disturbance was not yet observed (subjects were lucid), pointing to the utility of the P300 as a highly sensitive measure of metabolic processes. Finally, Dustman et al. (1990) reported that P300 amplitude increases were associated with exercise-related increases in metabolic activity.

Geisler and Polich (1992) investigated the link between food deprivation, P300, and memory performance across two experiments employing an auditory oddball paradigm. In one experiment, subjects were tested either within 3 hours of food consumption or after 6 hours of food deprivation. P300 amplitudes to target stimuli were smaller for those who had not recently eaten in comparison to those who had. The second experiment measured ERPs and memory performance in subjects before, immediately after (food-1), and 30 minutes following (food-2) food consumption to determine whether the previously observed changes in P300 were associated with variations in memory performance. The relationship of these variables to more general physiological responses to food consumption, namely blood glucose level, heart rate and body temperature, was also examined to determine if the previously observed

P300 variations might be due to a food-caused, overall general physiological arousal. P300 amplitude for the target stimuli initially increased, and then leveled off, following food consumption while latency was marginally decreased.

Memory performance improved after food consumption for the last five words in a list of 20, which is consistent with impaired short-term memory during food deprivation. Blood glucose, heart rate, and body temperature were not correlated with P300 amplitude or with memory performance. Glucose levels did, however, significantly increase across the three measurement times, as did heart rate.

It appears that P300 amplitude may be a sensitive index of the metabolic processes underlying eating behavior. The case for memory performance is not as strong, although it does appear that, at least for recently presented words, food consumption does exert a statistically significant influence. It should be noted that Gallai, Mazotta, Firenze, & Del Gatto (1988) and Blackman et al. (1990) did not replicate Polich's findings using a visual task. The reasons are unclear, but they may be unrelated to the visual modality.

Baldeweg, Ullsperger, Pietrowsky, Fehm and Born (1993) did find visual P300 correlates of hunger and satiety. Subjects were tested while satiated and after a 16 hour fasting period. Area of the visual P300 waveform was smallest when subject's answers were in agreement with their actual state of hunger. The results further underscore the importance of the P300 as a sensitive measure of internal state, specifically in this case, perception of the state of hunger.

While the effects of extreme food deprivation are undoubtedly important, it is also important to look at the starvation-independent cognitive impairment exhibited by

eating-disordered individuals, as well as, perturbations to cognitive functioning caused by mild hypoglycemia in healthy individuals. As mentioned earlier, Geisler & Polich (1992) uncovered variations in P300 amplitude and concomitant memory performance in normal subjects based upon elapsed time since food consumption. Using a similarly basic oddball paradigm, Jones et al. (1990) found significant effects in mildly hypoglycemic subjects. P300 amplitudes were decreased compared to controls, and P300 latencies were increased. These studies demonstrate that, even at very low levels of food deprivation and with very simple cognitive tasks, there are observable nutritional state effects on brain function.

Bradley et al. (1996) presented evidence of more complex cognitive abnormalities and their relationship to P300 in a recent study of adolescent anorexics. The research group recorded ERPs for both a verbal and a non-verbal memory task in anorexic patients both during their illness and when nutritionally recovered and in healthy control subjects. P300 latencies were longer for anorexics on the verbal task and on the non-verbal task the anorexic group exhibited a right > left hemispheric P300 symmetry where normals showed an asymmetry. More specifically, the anorexic subjects demonstrated decreased right hemisphere response to the nonverbal task. These differences were maximal over the central/parietal region. At nutritional recovery follow-up, the anorexics showed continued non-verbal processing problems. Such evidence is in line with previous findings of left hemisphere, high verbal ability in anorexic patients (Maxwell, Tucker & Townes, 1984; Hsu, 1990). In addition, P300 amplitude was significantly correlated with Body Mass Index (BMI) in anorexics on the non-verbal task. Lower BMI was significantly related to decreased processing of

nonverbal information in the right hemisphere, as indicated by a decrease in P300 amplitude. These data lend additional support to the notion that nutritional state does affect the production of P300.

Since P300 amplitude and latency are sensitive to typical variations in feeding behavior, memory performance, glucose level, and hypoglycemia in normal subjects (Geisler & Polich, 1992), as well as non-verbal performance decrements in anorexics, it may prove to be useful in detecting at-risk individuals who frequently restrict dietary intake (Laessle, Tuschl, Kotthaus, & Pirke, 1989a, 1989b; Rezek & Leary, 1991; Striegel-Moore, Schreiber, Pike, Wilfley, & Rodin, 1995; Williamson, Lawson, Brooks, Wozniak, Ryan, Bray, & Duchmann, 1995) and who may demonstrate perturbations to normal memory function (Pendleton Jones et al., 1991). Indeed, one of the most salient behavioral manifestations of the eating disorders is self-starvation, in both bulimic and restrictive anorectic individuals.

Rationale And Hypothesis Of Study

As reviewed above, existing ERP studies with eating disordered individuals reflect extreme levels of starvation and the attendant neurological disruptions. Such studies raise the possibility that the P300 ERP may be useful as a clinical screening method in individuals at-risk for developing an eating disorder. If so, then the P300 findings should be correlated with paper and pencil measures of at-risk behavior currently in use. This study will examine the P300 in subjects who are at-risk and not at-risk for developing an eating disorder as measured by the EDI-2 Drive for Thinness scale.

It has been demonstrated that the P300 is sensitive to food deprivation and that it correlates with the associated short-term memory impairment. Eating disordered individuals also exhibit decreased metabolism primarily in parietal regions. Because such food deprivation leads to smaller P300 amplitudes compared to those in subjects who have recently eaten, and since individuals who are at-risk for eating disorders generally restrict food intake, and may exhibit parietal region impairment, we would expect there to be a significant difference between the at-risk individuals and normal controls for P300 amplitude, latency and short term verbal memory performance. It was therefore predicted that, compared to the normal controls group, the at-risk group would exhibit a statistically significant decrease in P300 amplitude and increase in P300 latency that would be greatest over the parietal (Pz) electrode site. It was also predicted that the at-risk group would show a decrease in the number of words recalled from the last half of the presented list due to generally lower blood-glucose levels resulting from food deprivation and/or a parietal lobe hypometabolic condition compared to controls.

It is clear that many of the disruptions to normal cognitive functioning found in eating disorders are amenable to ERP studies. Missing from the literature is an investigation into the pathogenesis of the eating disorders. By examining the P300 in individuals who exhibit a propensity towards an eating disorder, we hope to elucidate the cognitive deficits that may precede the starved state and associated pathologies that arise from a full-blown case of eating disorders.

Method

Thirty-one female college students, drawn from a pool of 227 female college students enrolled in Introductory Psychology labs at Drake University volunteered for the study. The students were screened for participation using the Eating Disorder Inventory-2 (EDI-2) and, based upon their score on the Drive for Thinness scale, were placed in one of two groups: control and at-risk. Fifteen subjects from the control group and sixteen from the at-risk group were randomly chosen to participate in the EEG recording portion of the experiment. The average age for the at-risk subjects was 18.94 years of age and for controls, 18.67 years of age. The two groups did not differ statistically by age (t [29]=0.87, p<0.30). All subjects were between the ages of 18 and 21 years of age and right-handed as determined through self-report. Each of the 227 students who were screened gave informed consent. To ensure confidentiality, a unique code number on all questionnaires and forms identified subjects. The informed consent forms (bearing the subjects' signatures) were kept separate from the questionnaires to further protect subject's privacy

Screening for Eating Disorders.

For the present study the EDI-2 was administered at the end of a weekly Introductory Psychology lab section and all volunteers were given extra credits points to be counted toward their final lab grade. At-risk subjects were defined as those scoring at the 90th percentile on the Drive for Thinness (DT; raw score cutoff: 14) scale of the Eating Disorder Inventory-2. Garner (1991) identified this cutoff score as appropriate for identifying individuals who are "at-risk". Using this cutoff, 10% of college females are identified, and up to 40% of these may have an eating disorder of

clinical severity. As mentioned earlier, the utility of the DT scale for identifying at-risk individuals who are restricting their food intake has been demonstrated in a number of studies. The EDI-2 Drive for Thinness scale identified 26 (11.45 %) of the 227 females as at-risk for an eating disorder. This percentage is in line with the 10% figure reported by Garner (1991) when using the Drive for Thinness scale for screening for at-risk individuals.

As expected, placing subjects into the two groups based upon their scores on the Drive for Thinness scale resulted in a main effect of "group" for scores on the scales of the Eating Disorder Inventory-2 instrument using an ANOVA analysis with scores as the within-subjects factor (F [1,29]=11.22, p<0.001). Follow-up, one-way ANOVA analyses revealed a statistically significant difference between the two groups on the Drive for Thinness scale (F [1,29]=109.26, p <0.001), Bulimia scale (F [1,29]=5.31, p<0.03), Interoceptive Awareness scale (F [1,29]=5.23, p<0.03), and Asceticism scale (F [1,29]=5.90, p<0.02). On all four of these scales, the at-risk group had significantly higher scores than the control group. Means and standard deviations for the individual scales of the EDI-2 are listed in Table 1.

Subjects who met the selection criteria were randomly selected, telephoned, and invited to participate in the EEG recording session for extra-credit points. Of the 26 subjects identified as at-risk, eighteen agreed to participate and eight declined. Of the 201 control subjects, twenty were randomly chosen and called; fifteen agreed to participate and five declined. Subjects were also asked to wear glasses instead of contact lenses when necessary in order to avoid excessive eyeblinking, and that the experiment would last approximately two hours.

Since meals consumed at lunchtime are usually smaller than the meals consumed at dinnertime, an attempt was made to counterbalance time of testing. Subjects were assigned to either a 3 p.m. session (after lunch), or a 7 p.m. (after dinner) session to control for time-of-day effects related to these meals. Due to the small number of available subjects, it was necessary to allow four subjects from each group to pick a time slot other than the one that was offered. This resulted in nine at-risk subjects being tested at 7pm and seven at 3pm. Nine normal controls subjects were tested at 7pm and six were tested at 3pm. Subjects were not given specific instructions on what to eat or not eat prior to the testing session; they were tested while maintaining their daily eating routines.

The two groups did not differ statistically on the measured food consumption variables: "FOOD"-the amount of food consumed at the last meal, and "LASTFOOD"-time since last food intake. One-way ANOVAs revealed no main "group" effect for the FOOD variable ($F[1,29]=.59$, $p<0.45$) nor the LASTFOOD variable ($F[1,29]=.45$, $p<0.51$). Means and standard deviations for the food consumption variables are listed in Table 3.

Medical History.

All but five of the subjects reported having regular menstrual periods. Of these, four were in the at-risk group and one was in the normal-control group. This last individual, while not possessing a score of 14 on the DT subscale which would warrant inclusion in the at-risk group, did have scores on all of the subscales which were higher than other control subjects. Eight subjects reported that they were smokers.

Physical Factors.

The two groups did not differ on the two key physical measurements performed - height and weight. One-way ANOVAs revealed no significant difference in height ($F[1,29]=2.31$, $p<0.14$) nor weight ($F[1,29]=.31$, $p<0.58$) between the two groups. Means for the height and weight factors are listed in Table 2.

Procedure

Questionnaires

Upon arrival at the lab, subjects were given an informed consent form and a food consumption questionnaire. Following the design of Geisler and Polich (1990), subjects' food intake in the 6 hours preceding testing was scored as follows: a zero was given for no food consumed, a one for juice, milk, or other beverage, a two for a small snack (toast, a piece of fruit, etc.), a three for a standard meal, a four for a complete meal and a snack, and a five for a complete meal and numerous snacks (a snack and a soda for example).

A questionnaire gathered the following information: 1) whether contact lenses were being worn, 2) age, 3) whether subjects played collegiate sports, 4) whether they had regular menstrual periods, 5) date of last menstrual period, 6) whether they were smokers, and if yes 7) how many cigarettes they smoke per day, 8) which prescription medications they were currently taking (including oral contraceptives), 9) whether they usually eat breakfast, 10) whether they were on the university meal plan, 11) whether they were vegetarian, and the food rating scale, with time since eating anything. All subjects received extra-credit points regardless of level of participation.

EEG Recording.

Subjects were comfortably seated during recordings and testing. ERPs were elicited by presenting a series of target and standard tones binaurally through a pair of earphones inserted into the ear canal. Target tones were presented at a frequency of 1000-Hz and standard tones at 2000-Hz at 60dB (9.9 msec r/f, 50 msec plateau) once every 2 seconds. Target tones occurred randomly 15% of the time. There were a total of 600 stimulus presentations.

Subjects were instructed to press a mouse button with the index finger of their right hand as quickly as possible when the target tone was detected. Subjects practiced the task over the course of 30 stimulus presentations; the experimenter watched the practice session to ensure that the subject was indeed performing the task. During the recording session, subjects were asked to focus on a mark placed on a computer screen to control vertical and horizontal eye movement. To control the amount of noise recorded, subjects were asked to minimize the number of times they blinked their eyes.

ERPs were recorded during presentation of the auditory stimuli from 3 scalp electrodes that are a subset of the International 10-20 system using linked ears. Recordings were made at midline locations over parietal (Pz), central (Cz), and frontal (Fz) scalp sites. Vertical EOG (eye movement) was recorded from right eye supra-orbital and infra-orbital electrodes. Horizontal EOG was recorded through electrodes placed at the right and left external canthi. Electrode impedances were less than 6 Kohms. The EEG was filtered using a bandpass of 0.03 to 35 Hz (12dB/octave rolloff). ERP averages were constructed for each channel from 256 msec. samples over a

1,000 msec. time interval. Sampling commenced 100 msec. prior to stimulus presentation; this established the baseline. Single trial epochs from any channel that had voltages in excess of +-60 microvolts were removed. Single trial epochs with vertical EOG contamination were corrected using regression-based and subject-unique weighting coefficients. P300 component peak amplitude was defined as the data point at each site with the largest positive voltage between 250 and 600 msec. P300 component peak latency was measured in milliseconds as the largest positive voltage between 250 and 600 msec.

Button presses were recorded and accuracy rates calculated for each subject. Accuracy averaged 98.60% across both groups; the at-risk group had an average accuracy of 97.92% and the normal controls group had an average accuracy of 99.32%.

The preceding subject selection and experimental design resulted in a two-factor (group x electrode) with repeated measures design with two groups, three electrode sites, N=15 in the normal controls group and N=16 in the at-risk group.

Memory Testing and Additional measurement.

Following the recording of the ERPs, memory performance was tested. Each subject was presented binaurally by way of a standard, single speaker tape recorder with the same list of twenty, two syllable words, randomly selected from the Toronto word pool (Murdock, 1976). Words were presented at a rate of one per second. The subject was immediately given an answer sheet and asked to write down as many words as she could recall within five minutes.

Subjects were then weighed and their height recorded. All subjects were finally debriefed, and in cases where the EDI-2 profile warranted, they were told that their score profile was similar to individuals who score in the at-risk range for developing an eating disorder and they were instructed that if they had any concerns along these lines they should call an on-campus counselor, trained in working with eating disordered individuals. The counselor's phone number was provided.

Results

I. Behavioral Measures

Memory Testing. The means and standard deviations for the memory data are listed in Table 4. One-way ANOVAS revealed no significant difference in the total number of words recalled for each group ($F[1,29]=1.17$, $p<.29$). A two-factor ANOVA with "wordblock" as the within subjects variable revealed no main effect for the "group" variable among the four five-word blocks ($F[1,29]=1.46$, $p<.24$). In other words, the two groups did not differ in the number of words they were able to recall from any of the four word blocks.

In line with Geisler and Polich's (1990) findings, primacy and recency effects were observed for both groups. Subjects remembered more words from blocks 1 (primacy) and 4 (recency) indicating the presence of normal memory function. Memory performance data are presented in graphical form in Figure 2.

II. Event-Related Potential Data

Grand averaged waveforms to target tones for the at-risk and the normal controls groups for FP1, Fz, Cz, and Pz electrode sites are shown in Figure 1.

P300 Amplitude Analyses. P300 amplitude did not differentiate the two groups. The planned two-factor ANOVA using "electrode site" as the within-subjects variable, revealed no statistically significant difference in mean P300 amplitude values between the two groups. There was no main effect of "group" (F [1,29]=1.03, p<.32) nor was there a significant "group x electrode site" interaction (F[1,29]=0.17, p<.85). The mean P300 amplitudes among the 3 mid-sagittal (Fz, Cz, and Pz) electrode sites as well as the standard deviations are presented numerically in Table 5 and graphically in Figure 3.

P300 Latency Analyses. The at-risk group did not differ from the control group on P300 latency. The planned two-factor ANOVA, with "electrode site" as the within-subjects variable revealed no statistically significant effect for "group" (F[1,29]=0.33, p<.57) nor for the "group x electrode site" interaction (F[1,29]=2.23, p<.12). Mean P300 latencies and standard deviations among the 3 mid-sagittal (Fz, Cz, and Pz) electrode sites are presented numerically in Table 6 and graphically in Figure 4.

Correlations. Pearson product-moment correlation coefficients were calculated between the "lastfood" variable and P300 latency and amplitude. There was no significant correlation between P300 amplitude and the amount of time since last food intake. There was no significant correlation between P300 latency and the lastfood variable.

Pearson product-moment correlation coefficients were also calculated between Drive for Thinness scores and P300 amplitude and latency. There were no significant correlations between Drive for Thinness scores and ERP data.

Discussion

P300 amplitudes and latencies did not differ significantly for the at-risk individuals compared to controls. There was no significant main effect for the group variable, or the electrode site variable nor was there any significant interaction (refer to Figures 3 and 4). These data suggest that P300 data are not likely to be useful as a routine screening device for college females who are at-risk for developing an eating disorder. The problem with clinical usage probably lies in: 1) the difficulty in assessing actual food intake because at-risk populations are more likely to be dishonest about their eating history, 2) a lack of understanding of food intake patterns in at-risk populations due to the paucity of research, and 3) the difficulty in identifying possible bulimic subjects. Previous reports of cognitive deficits in eating disordered individuals may be the result of starvation and not a part of the eating disorder itself. Therefore, it is possible that at-risk subjects are not in a sufficient state of starvation to affect memory and P300 production. This study possibly could have benefited from blood glucose screenings as confirmation of food intake; unfortunately, such screenings were not feasible in the current study. The self-reported food intake analyses revealed no significant difference between the two groups. Given the fact that weight-preoccupied individuals frequently engage in dietary restriction, the food consumption data would seem to be of considerable importance. Caution is required in the interpretation of self-

reported food intake since it is notoriously unreliable in eating disordered subjects. Williamson et al. (1995), in a study of body mass and dietary restraint, found that the DT scale of the EDI-2 correlated strongly with the Dietary Restraint scale of the Three Factor Eating Questionnaire (TFEQ)(Stunkard & Messick, 1985) and that the Bulimia subscale was highly correlated with the Overeating scale of the TFEQ. Even though bulimics do engage in dietary restraint, the at-risk sample may have contained both dietary restrainers and overeaters. The statistically significant difference between the at-risk and normal controls groups on the Bulimia subscale would seem to point to this possibility. Additionally, there was no significant difference in memory performance between the two groups.

Another possible explanation for the current results is that the P300 may not be sensitive to the type and pattern of dietary restraint exhibited by at-risk populations. The work of Geisler & Polich (1990, 1992,1994) reported that P300 amplitudes are reduced in subjects who had not eaten within the last six hours, compared to those who had. Gallai, Mazzotta, Firenze, & Del Gatto (1988) reported smaller P300 amplitudes and longer latencies in subjects experiencing insulin-induced minor hypoglycemia over the relatively short period of 135 minutes. If we take the self-reported food intake of the present study to be true, then the absence of a significant difference in P300 amplitude between the two groups is not surprising; the two groups did not differ significantly on self-reported food intake on testing day, so there should not be an amplitude difference, nor should there be a difference in memory performance. It is possible, however, that P300 is sensitive to the dietary restraint that takes place over longer time periods of months or even years. This must be addressed by future research.

The brain glucose hypometabolism reported for eating disorders occurs at near-starvation levels. The subjects in the current study were clearly not starved in the extreme sense of the word, but there may have been within-group differences in how they were restricting food intake. Delvenne et al. (1997a, 1997b), in two separate studies, reported global and regional hypometabolism of glucose in both bulimic and anorexic patients compared to healthy controls. Both groups showed lower relative glucose metabolism in the parietal cortex region of interest (ROI). However, the absolute glucose levels reported for the anorexic subjects were significantly correlated with body mass index (BMI), a ratio of weight to squared height. No such relationship between hypometabolism and BMI existed for bulimics.

The relationship of brain glucose hypometabolism to BMI in anorexics but not in bulimics raises the question of what particular metabolic perturbations, if any, might be operating in a weight-preoccupied sample such as the one examined in the present study. The anorexic eating pattern results in extreme starvation; bulimics, until very late stages of their illness, tend to be of normal weight. Is there a meaningful difference in the pattern of dietary restraint between at-risk subjects with anorectic tendencies and at-risk subjects with a propensity towards bulimia that may have masked an effect had the two subvariants been examined separately? Anorexia and bulimia patients both exhibit an overconcern with body weight and shape (Hsu, 1990; Palmer, 1993). Both groups exhibit dietary restraint; but they vary in degree and timing. The degree of restraint in anorexics is extreme, and is a defining variable for this subtype. Bulimic patients, on the other hand, eventually give in to their desire to restrain and end up bingeing and purging (Palmer, 1993). It is clear, then, that at any

given time, anorexics are in a state of chronic food deprivation, whereas bulimics tend to be less extreme in the amount of restraint they show. In the present study, the high number of at-risk subjects with bulimia issues might have appeared closer to normal control subjects in food intake, thus masking an effect that might have shown up had the study been restricted to at-risk for anorexia subjects. Subjects with high scores on the Bulimia subscale could have been omitted. There is also evidence that underweight anorexics demonstrate greater cognitive impairment than normal-weight bulimics (Fox, 1981). More specifically, anorexics showed greater verbal ability impairment compared to bulimics and controls, findings later replicated by Bradley et al. (1996) and Pendleton Jones et al. (1991). It therefore makes sense to control for or separate out subjects who display bulimic tendencies. Unfortunately, there are no studies investigating the differences in eating patterns between these two types of at-risk populations. Additionally, there are no PET studies examining brain glucose metabolism in at-risk populations, making it unclear whether there are any significant metabolic effects of less extreme levels of dietary restraint.

It is possible that the focus of the present study on food intake is not the ideal way to examine the problem. At-risk subjects also demonstrated significantly elevated scores on the interoceptive awareness subscale, an indication that they were focused on and keenly aware of their inner sensations of hunger and satiety. Heilbrun & Worobow (1990) have proposed that this extreme focus on turbulent inner sensations and the stress it generates in the at-risk person leads to the opposite reaction in the eating disordered person, or complete disattention to inner sensations, which allows them to more easily restrain eating. The stress of such inner focus and the fear of

giving into the associated urges in the at-risk individual is therefore given as an explanation for the reduced or dysfunctional attention reported in eating disordered individuals (Hamsher, Halmi, & Benton, 1981; Pendleton Jones et al., 1991; Small, Madero, Teagno, & Ebert, 1983). Other methods of generating P300 may be more sensitive to an at-risk state. The only published P300 study of eating disorders uncovered perturbations to the normal left/right hemispheric function using more challenging verbal and non-verbal tasks but they also used a more severely disturbed patient population, suffering from anorexia nervosa (Bradley et al., 1996).

Another clinical feature of eating disordered individuals is chronic vigilance regarding eating and body-related issues (Hsu, Kaye, & Weltzin, 1993). Eating disordered individuals' heightened levels of vigilance have been reported to extend beyond food and body issues into all other facets of life (Hsu, 1990), at least in early stages of the disease. Indeed, this feature has lead many researchers to propose a link between the eating disorders and OCD (Hsu, Crisp, Harding, & Hartshorn, 1980; Hsu, Kaye, & Weltzin, 1993). Such OCD-related hyper-vigilance has been shown to relate to P300 amplitudes (Towey et al., 1994). P300 amplitudes are larger in OCD patients compared to normal controls, indicating a cognitive overarousal. Eating disordered individuals' obsession with food and body shape and bulimics' compulsive overeating and vomiting would seem to point to the feasibility of a P300 investigation with a focus on this psychological variable.

As mentioned earlier, the Bradley et al. (1996) study uncovered longer P300 latencies for anorexics on a verbal task and hemispheric differences between anorexics

and controls on a non-verbal task. Numerous researchers have proposed a right posterior hemispheric dysfunction in anorexics due mainly to demonstrated impaired non-verbal performance on tasks such as an abstract figure recognition test (Kinsbourne & Bemporad, 1986; Lezak, 1983). Future P300 studies of at-risk individuals might also benefit from a larger electrode montage that would allow comparisons of hemispheric performance.

The issue of whether or not the neuropsychological impairment observed in eating disordered patients might be due to the effects of starvation was raised earlier in the paper. Obviously, ethical constraints have resulted in a paucity of studies on the topic. The Keys et al. (1950) study, while providing much valuable information, presents some problems when it comes to generalizing to eating disordered populations. First, the subjects in the Keys study were all male. Secondly, the researchers excluded subjects who exhibited any psychological illness, as many eating disordered individuals do. In the Pendleton Jones et al. (1991) study, the impaired attention reported in the two groups of anorexic patients was not observed in the weight restored anorexics, suggesting that attentional deficits arise from the dietary/metabolic effects of extreme starvation. In addition, Bradley et al. (1996) reported improvement on a verbal task when anorexic subjects recovered weight. It is possible, then, that females who are at-risk for developing eating disorders do not display the same cognitive deficits as eating disordered individuals simply because they are not severely restricting their food intake. The lack of evidence of significant memory impairment in the present study supports this notion.

In sum, the present study was intended to be a preliminary investigation into the auditory P300 in females who are at-risk for eating disorders. Given the fact that a fairly stringent selection criterion was used and the fact that the at-risk group also had significantly elevated scores on three additional subscales it is highly likely that we examined two different and distinct subgroups of the population. The likely reason for the non-significant findings is the apparent insensitivity of the P300 to the type of dietary restraint engaged in by the at-risk group. Food consumption measures that capture long-term dietary restraint, such as food diaries and blood glucose screenings are necessary to document actual food intake. Future P300 studies of eating disorders should make every effort to control for nutritional state and inclusion of bulimics. Such steps would have added considerable clarity to the results of the current study in looking at at-risk individuals. It is important for future studies to focus on the cognitive deficits shared by at-risk and eating disordered individuals and their ERP correlates. Food intake, while important and certainly a salient feature, is difficult to isolate, especially in a population that is expert at concealing such behavior. The task for future researchers should be to identify the precursors to abnormal cognitive structures and styles, such as hyper-vigilance, in eating disordered individuals. The P300 brain potential may be a sensitive measure of cognitive performance, but future research will be required to elucidate any sensitivity it may have to eating disorder etiology.

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Table 1

EDI-2 Subscale Scores for Control (C) and At-Risk (ED) Subjects

Subscale	Control (n=15) M	Control (n=15) SD	At-Risk (n=16) M	At-Risk (n=16) SD	
DT	3.6	4.3	16.4	2.2	*p<0.001
B	0.5	1.3	3.6	4.9	*p<0.03
BD	10.8	8.9	15.8	7.6	
I	1.5	2.8	3.3	3.9	
P	8.1	4.0	7.9	4.2	
ID	3.4	4.1	2.2	2.2	
IA	1.8	3.7	5.8	5.8	*p<0.03
MF	2.1	2.3	3.1	2.7	
A	3.9	1.5	6.6	3.9	*p<0.02
IR	1.2	1.5	2.6	4.2	
SI	3.2	3.3	2.7	2.8	

Table 2

Height and Weight for Control and At-Risk Subjects

	Control (n=15)		At-Risk (n=16)	
	M	SD	M	SD
Height (in inches)	66.6	2.8	65.2	2.2
Weight (in Lbs.)	148.5	26.0	144.1	16.5

Table 3

Amount (Food) and Time (Lastfood) of Last Food Consumption for Control and At-Risk Subjects

	Control (n=15)		At-Risk (n=16)	
	M	SD	M	SD
Food	3.3	0.8	3.0	1.1
Lastfood (in hours)	1.7	1.2	2.4	4.2

able 4

Vord Recall by 5-Word Block for Control and At-Risk Subjects

	Control (n=15)		At-Risk (n=16)	
	M	SD	M	SD
Block 1	2.9	0.8	2.4	0.8
Block 2	1.8	1.4	1.8	1.1
Block 3	2.0	0.9	1.6	1.0
Block 4	2.2	1.1	2.1	1.1
Total	8.7	1.7	8.0	2.0

Table 5

P300 Amplitude (in microvolts) at Midsagital Electrode Sites for Control and At-Risk Subjects

	Control (n=15)		At-Risk (n=16)	
	M	SD	M	SD
Electrode Site				
FZ	7.4	5.0	9.2	3.4
CZ	12.6	7.0	15.2	6.8
PZ	15.5	5.8	17.4	8.2

Table 6

P300 Latency (in milliseconds) at Midsagital Electrode Sites for Control and At-Risk Subjects

	Control (n=15)		At-Risk (n=16)	
Electrode Site	M	SD	M	SD
FZ	320.1	31.2	342.1	18.4
CZ	335.1	68.4	337.5	21.6
PZ	352.7	54.3	350.3	20.5

P300 Grand Average Waveforms for Control (n=15) and and At-Risk (n=16) Subjects, nes.

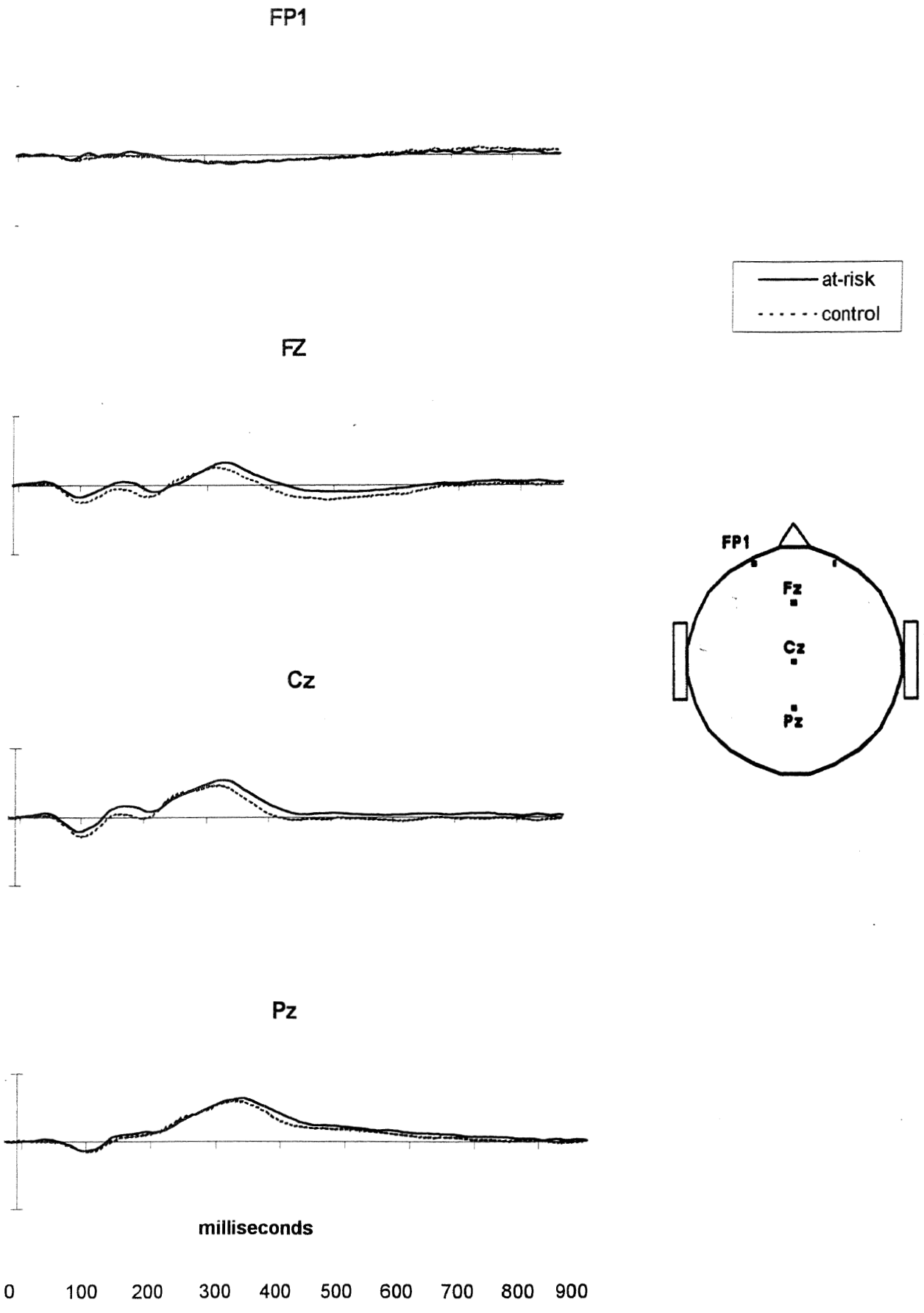

Figure 2: Memory - Words Recalled For Each Word Block

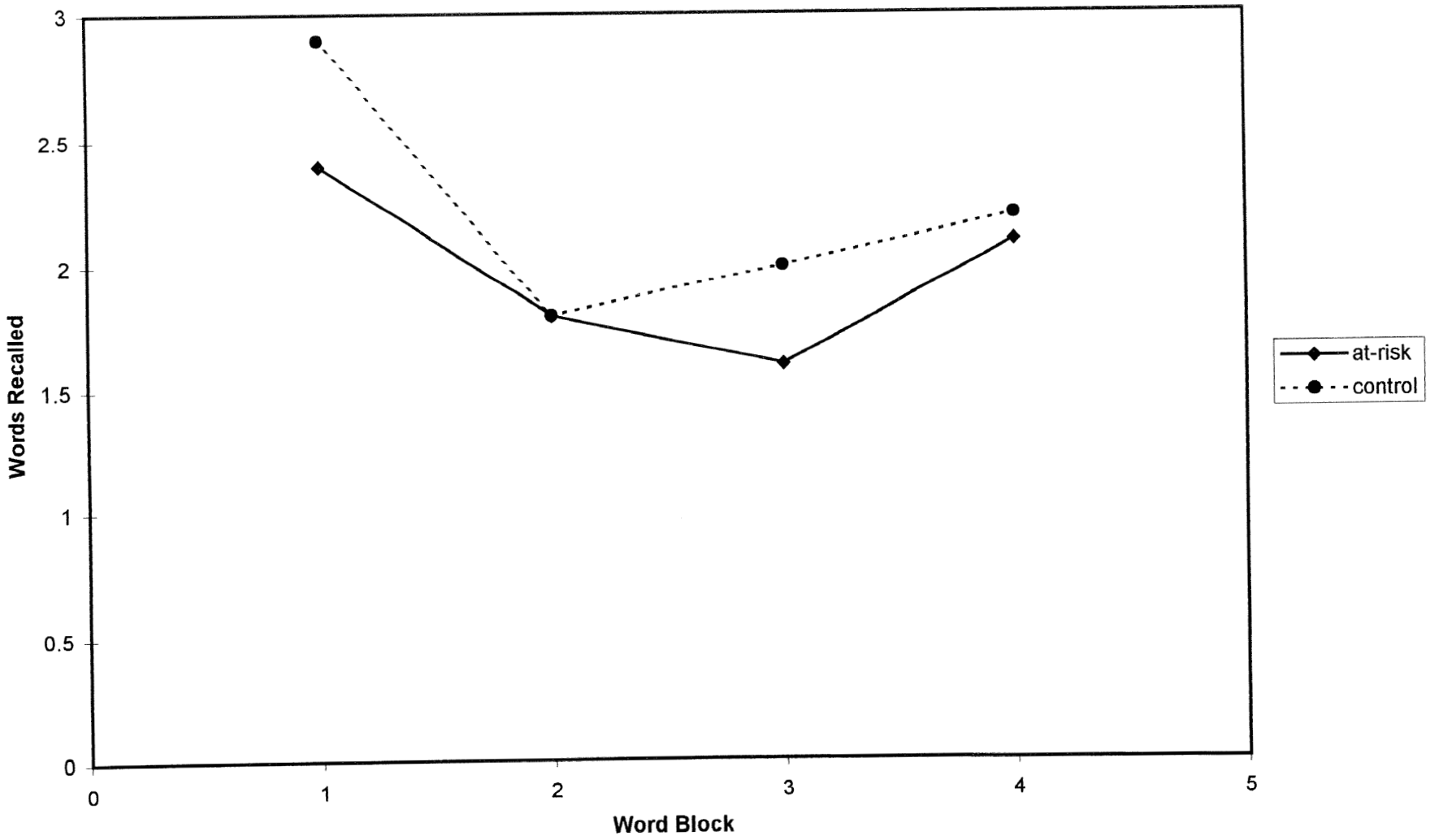

Figure 3: P300 amplitude

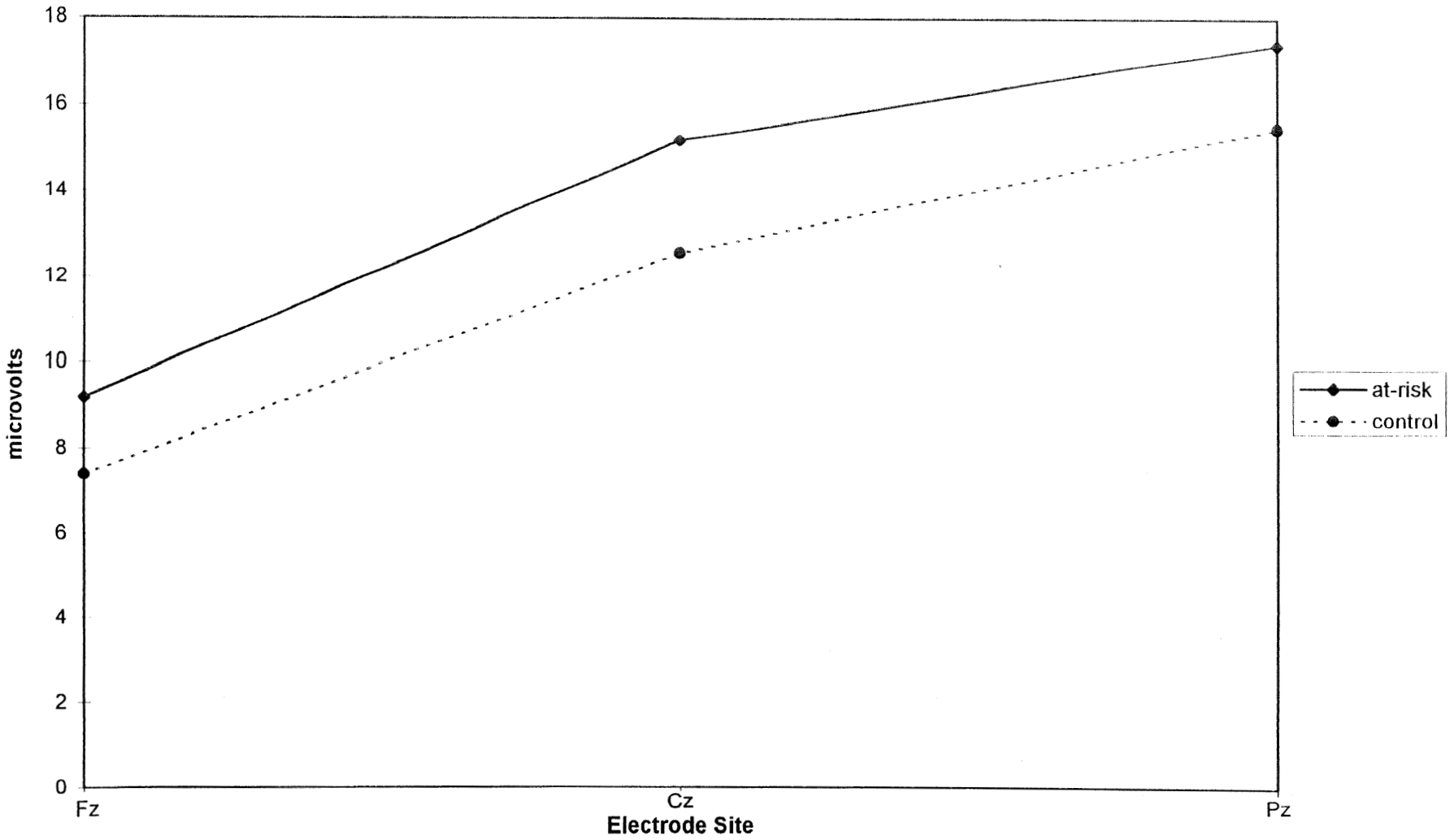

Figure 4: P300 Latency