THE ROLE OF BODY MASS INDEX
AND ITS COVARIATES IN EMOTION RECOGNITION

A dissertation submitted
to Kent State University in partial
fulfillment of the requirements for the
degree of Doctor of Philosophy

by

Angela Nicole Roberts Miller

August, 2013
Dissertation written by
Angela Nicole Roberts Miller
A.A.S., Metropolitan Community Colleges, 2001
B.S., Wichita State University, 2003
B.A., University of Missouri- Kansas City, 2007
M.P.H., Wichita State University, 2005
M.A., Kent State University, 2010
Ph.D., Kent State University, 2013

Approved by

___________________________________, Co-Chair, Doctoral Dissertation Committee
Joel Hughes
___________________________________, Co-Chair, Doctoral Dissertation Committee
John Gunstad
___________________________________, Members, Doctoral Dissertation Committee
Katherine Rawson
___________________________________,
Scott Olds
___________________________________,
Colleen Novak

Accepted by

___________________________________, Chair, Department of Psychology
Maria Zaragoza
___________________________________, Associate Dean, College of Arts and Sciences
Raymond A. Craig
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DEDICATION

To my grandfather, Jere L. Roberts who believed long before this dream became a reality.

I wish you were here.

“For I know the plans I have for you,” declares the Lord

(Jeremiah 29:11)
ACKNOWLEDGEMENTS

It would have been impossible for me to have completed this work without the continual support and encouragement I received. I would like to thank my advisor, Dr. Joel Hughes, and my dissertation committee co-chair, Dr. John Gunstad. From you, I was inspired to pursue the integration of health psychology and neuropsychology with my background in public health. You believed in my abilities and my vision. The mentorship and professional freedom you provided allowed me to become the psychologist I am today.

Thank you to the members of my dissertation committee: Dr. Katherine Rawson, Dr. R. Scott Olds, and Dr. Colleen Novak. Your time and thoughtful feedback challenged me and made this work better. Thank you to Dr. Mary Beth Spitznagel and Dr. John Linton. Words cannot express how much I appreciate your willingness to share your expertise. Thank you to Dr. Misty A.W. Hawkins, a fellow scholar on the journey and a dear friend. You gave me the momentum and perspective I needed to actually cross the finish line.

Thank you to my parents, Larry and Debbie Roberts, my grandparents, and all my extended family. Without you, none of this would have been possible. You believed when I doubted, hoped through all trials, and loved unceasingly. I am eternally grateful.

In a special way, thank you to my husband, Jason Miller, and my children. You willingly sacrificed time with me, home cooked meals, a cleaner house, and most of your discretional income to allow me to pursue this dream. There were no limits to your love and support. This achievement is as much yours as it is mine. You made this worth doing.

Lastly, I am lost without the love of my Creator. All that I have and all that I am are a gift from Him and without Him every success I have is empty.  Ad maiorem Dei gloriam!
INTRODUCTION

Obesity is a chronic and debilitating medical condition that results from a complex mix of genetic, physiological, psychological, and social factors. It is a condition that is both costly and difficult to manage and it has been associated with an increased risk for numerous other chronic health problems including heart disease, Type 2 diabetes, cancer, and musculoskeletal degeneration (World Health Organization, 2000). With 68% of men and 51% of women in the United States overweight or obese, it has become increasingly important to understand the factors that underlie an individual’s development of obesity as well as those that contribute to its maintenance (Kaiser Foundation, 2007). Many studies have been conducted to (a) better understand obesity’s link to psychopathology (b) propose various pathways explaining the likelihood to develop obesity, (c) examine the association between obesity and psychosocial factors, and, more recently, (c) observe obesity’s impact on the brain. Despite a recognized consensus regarding the complexity of obesity, little is known about how various demographic, medical, and cognitive performance variables interact in this population, especially in relation to factors which may contribute to the maintenance of obesity over time. Research has supported that one key aspect of this process is eating in response to psychological rather than physiological cues. Given the increased prevalence of psychopathology, particularly mood disorders, in obese individuals, the question arises as
to whether there exists an underlying impairment in emotion recognition. Such impairment could not only potentially reflect morphological changes (e.g., cerebral atrophy) in the brain, but may also have significant implications for the individual’s ability to interact socially and the degree of distress they experience in that context. Unmitigated, such distress may have important implications for understanding emotional eating and subsequently obesity, itself.

**Obesity Prevalence and Comorbidities**

In adults, obesity is defined as having a body mass index (BMI) of 30.0 kg/height in m² or greater (National Institutes of Health [NIH], 2000). Among children, a BMI percentile equal to or greater than the 95th percentile is defined as overweight (Centers for Disease Control [CDC], 2007). Since 1980, the rate of obesity has doubled with one in three adults in the United States being obese (Hedley et al., 2004). An even more rapid pattern of increase has been observed among the young. In children and teens, body mass index is plotted on the Centers for Disease Control’s BMI-for-age growth charts to obtain a percentile ranking. These percentiles indicate the relative position of the child's BMI number among children of the same sex and age. Currently, 34% of children are considered overweight or at-risk for becoming overweight (≥85th percentile) and 17% are considered obese (>95th percentile) (Ogden et al., 2006). This represents a tripling of childhood weight issues over the past thirty years (Hedley et al. 2004). In the United States, as with other developed nations, environmental (e.g., readily available, convenient, high calorie foods), behavioral (e.g., sedentary lifestyle), and emotional
factors (e.g., emotional eating as a coping mechanism) have contributed greatly to the increasing prevalence of obesity.

Some ethnic groups are disproportionately affected. Among children ages 2–19, Mexican-Americans and Non-Hispanic blacks are more likely than Caucasian youth to be overweight (Ogden et al., 2006). In addition, Freedman and colleagues (2005) found that overweight African American youth are more likely than their overweight Caucasian peers to become obese adults. Currently, 76% of African American adults are overweight and almost half (45%) are obese (Ogden et al., 2006). African American adult women (14.7%) are more likely than women of other ethnic background to be morbidly obese (BMI ≥ 40; Ogden et al., 2006).

Obesity among adults has been associated with a variety of significant health conditions including cardiovascular disease, sleep apnea, hypertension, type 2 diabetes, stroke, chronic pain, impaired fertility, and some cancers (Li, Bowerman, & Heber, 2005; NIH, 2000). Similarly, these diseases, including type 2 diabetes, arthritis, and gallstones, once linked only to advancing age, are found even among the youngest of overweight children (Gennuso, Epstein, Paluch, & Cerny, 1998). Sadly, compared to their non-overweight peers, these children are over 20 times more likely to be obese as adults, increasing their risk of disease and early death (Whitaker, Wright, Pepe, Seidel, & Dietz, 1997). Researchers predict that given obesity’s growing prevalence and extensive comorbidities, obesity and overweight may lead to the first decline in life expectancy in more than two centuries (Olshansky et al., 2005).
Obesity and the Brain

Whereas research has long supported the deleterious impact of obesity on physical health, recent literature has also shown that obesity has a negative impact on the brain. Multiple longitudinal studies have demonstrated that overweight middle-aged individuals are more likely than their normal weight counterparts to experience a significant decline in brain function in later life (Dahl, et al., 2009; Whitmer, et al., 2008). Obesity has been shown to be an independent risk factor for Alzheimer’s disease and has been linked to white matter disease and increased atrophy in the temporal lobes of older adults (Gustafson, Lissner, Bengtsson, Bjorkelund, & Skoog, 2004; Gustafson, Rothenberg, Blennow, Steen, & Skoog, 2003; Gustafson, Steen, & Skoog, 2004).

Although atrophy of brain tissue is considered part of normal aging it appears that obesity accelerates this process.

Deficits have been demonstrated across a variety of cognitive domains, including attention, processing speed, memory, and executive functioning among patients with diabetes, hypertension, and obstructive sleep apnea (Battersby et al., 1993; Manschot et al., 2006; Ostrosky-Solis, Mendoza, & Ardila, 2001; Salorio, White, Piccirillo, Duntley, & Uhles, 2002). Whereas these conditions are often comorbid with obesity, more recent research indicates that cognitive deficits may exist in obese individuals independent of such medical conditions (Gunstad et al., 2007; Whitmer, Gunderson, & Barrett-Connor, 2005).

An increasing number of studies suggest a link between obesity and deficits in executive function. Increases in body mass index (BMI) have been associated with
cognitive decline across the adult life-span specifically in the areas of executive function and memory (Elias, Elias, Sullivan, Wolf, & D’Agostino, 2003; Gunstad et al., 2007). Half of bariatric surgery candidates demonstrate impaired executive functioning (Chelune, Ortega, Linton, & Boustany, 1986). Executive functions are comprised of the skills necessary for planning, organization, cognitive flexibility, problem solving, and behavioral monitoring. Further, studies have linked these deficits with difficulties regulating food consumption and increases in food cravings (Spinella & Lyke, 2004). Additionally, Davis and colleagues (2004) found a positive association between BMI and impaired decision making. This may contribute to the difficulties obese individuals report in controlling their eating, despite a desire to lose weight and maintain long-term lifestyle change.

Many researchers have speculated that obesity may magnify dementia risk by worsening cerebral atrophy even in cognitively intact individuals. The most commonly proposed mediators within these pathways include impaired respiratory function (Guo et al., 2006), impaired oxygenation secondary to sleep apnea (Lim & Veasey, 2010), hypercortisolemia (Lupien et al., 1998), inflammation (Benson, Janssen, Hahn, Tan, Dietz, Mann, et al., 2008; Convit, 2005; van Dijk et al., 2005), lack of exercise (Colcombe et al., 2003), cardiovascular disease (Breteler et al., 1994; Swan et al., 1998), insulin resistance (Convit, 2005; Elias, Elias, Sullivan, Wolf, & D’Agostino, 2005; Greenwood & Winocur, 2005), and Type 2 diabetes (den Heijer et al., 2003; Ferguson et al., 2003). Additionally, adipocytes are known to secrete proteins (e.g., cytokines, leptin)
that may alter cognitive functioning when present in abnormal levels (Harvey, 2007; Wilson, Finch, & Harvey, 2002).

Raji et al. (2010) found that higher body fat was strongly associated with brain volume deficits in a sample of cognitively normal elderly, even after the effects of age, sex, and race were removed. Interestingly, whereas the negative correlations were most prominent in the obese subjects, they were also observed in those that were overweight. If brain changes occur with low to moderate amounts of weight gain and cumulatively worsen, it may imply that significant changes exist long before cognitive deficits are observed and age may not be a predictive factor. Studies among younger obese subjects support this contention, suggesting that increases in adiposity are linked with decreases in whole brain volume and specifically white matter changes and atrophy in the frontal lobes, caudate, temporal lobes, precuneus, hippocampus, and thalamus (Gazdzinski et al., 2008; Gustafson et al., 2004; Gunstad, Paul, Cohen, Tate, Spitznagel, & Grieve, 2008; Pannacciulli et al., 2006; Taki et al., 2008; Walther, Birdsell, Glisky, & Ryan, 2010). These brain regions are responsible for a wide variety of critical functions including attention, emotion regulation, emotion recognition, memory, executive processes, taste perception, and the regulation of feeding behaviors.

**Emotion Recognition**

**An Introduction to Emotion**

Most researchers define emotion as consisting of a physiological, evaluative, and/or expressive reaction to a personally significant event (Frijda, 1986). The
implication is that emotional experience is multi-faceted. Physiological changes in the autonomic nervous system (e.g., fluctuations in heart rate, blood pressure, respiration, gastrointestinal and urinary activity) interplay with the actions of the endocrine system (e.g., hormonal release) and the brain. The associated somatic perceptions associated with these fluctuations may subsequently influence, either independently or in combination with external cues, a subjective emotional experience. Labels of these experiences result from a cognitive interpretation of the physiological and/or environmental information (Schachter & Singer, 1962). These labels often engage the familiar emotional terminology (e.g., sadness or happiness) used to verbally convey emotional states. Finally, the emotional experience is also characterized by observable changes in posture, eye contact, vocalizations (e.g., sighs, groans), and/or facial expression. These outward manifestations function as a form of critical currency in social interaction, facilitating human relationships (Keltner et al., 2003; Keltner & Kring, 1998).

The Face of Emotion

The outward expression of emotion facilitates social interaction by serving informative, evocative, and incentive functions (Keltner et al., 2003; Keltner & Kring, 1998). For example, an individual’s facial expression has the ability to communicate contextual information about emotional state and intention that words alone may not convey. In addition, it can provide information which defines the relationship between the parties involved. Much more than just informational exchange, facial expressions have the potential to also evoke emotions in others (Batson & Shaw, 1991; Dimberg & Öhman, 1996; Keltner & Kring, 1998). By expressing or withholding emotion, humans
reinforce or inhibit social behavior. However, for this to be successful, the production and interpretation of emotion must be accurate and unambiguous. Disturbances in either production or interpretation of facial expression can alter social relationships by eliminating valuable contextual information or by leading to miscommunication.

The Brain and Emotion Recognition

Evidence from lesion studies has shown that damage to the occipital and temporal lobes, including areas of the limbic system, can lead to impairments in affect recognition by disrupting the individual’s ability to selectively attend to faces (Adolphs, 2002; Phillips, 2003; Posamentier & Abdi, 2003). If an individual is not attending, or paying attention to the face, they will not process more subtle aspects of its expression. This has the potential to hinder the higher order processing necessary for the identification of emotion and the further attribution of intent. In a social context, such deficits in emotion recognition could result in the interpersonal difficulties associated with multiple forms of psychopathology. Research has shown that individuals with depression, autism, bipolar disorder, and schizophrenia have difficulty accurately recognizing emotions in the faces of others (Archer, Hay, & Young, 1992; Bolte & Poustka, 2003; Edwards et al., 2002; Leppänen, Milders, Bell, Terriere, & Hietanen, 2004; Mandal et al., 1998; Morrison et al., 1988; Phillips, Drevets, Rauch, & Lane, 2003). In addition, these disorders have been linked to abnormalities in the brain structures responsible for the processing of emotion. Given the prevalence of psychological co-morbidity, especially depression (Benson et al., 2008), in obese populations, these findings have particularly important implications as the presence of psychopathology may reflect underlying changes in brain morphology.
**Eating Pathology and Emotion Recognition**

Emotional difficulties are clinically understood as a significant component of eating pathology. Patients with anorexia and bulimia have an impaired ability to differentiate their emotional stimuli from their physical and physiological states and subsequently convey their emotions to others (Bruch, 1985; Davis & Marsh, 1986). Often, this misperception of emotions and bodily sensations is accompanied by confusion of hunger and satiety cues and frequently it occurs in the context of a crisis involving their pathology (Bruch, 1985; Davis & Marsh, 1986). For example, an individual with bulimia may misinterpret negative affect and its associated physiological arousal with intense hunger resulting in binging behavior.

Research has shown that recognizing facial expressions of emotion is difficult for individuals with an eating disorder. Zonnevijlle-Bender, van Goozen, Cohen-Kettenis, van Elburg, and van Engeland (2002) found that individuals with anorexia demonstrated difficulty labeling emotions when verbal cues were lacking. This was subsequently supported by studies utilizing the Reading the Mind in the Eyes task (Baron-Cohen et al. 2001), which demands that emotions are labeled based on viewing only a person’s eyes (Harrison et al. 2009; Oldershaw, 2009; Russell et al. 2009). Women with eating pathology, regardless of diagnosis (anorexia, bulimia, eating disorder not otherwise specified), perform significantly worse than controls on tasks of emotion recognition. In addition, Kucharska-Pietura, Nikolaou, Masiak, and Treasure (2004) found those with anorexia have a decreased ability to recognize facially expressions that are negative or involve complex emotions, including ‘shame’ and ‘contempt.’ It is unknown whether
these difficulties can be attributed to deficits in perception or cognition. The answer to this question is of significant interest because research has linked emotion recognition to lack of social competence (Addington & Addington, 1998; Bryson, Bell, & Lysaker, 1997; Knappmeyer, Thornton, & Bulthoff, 2003) and factors which predict and perpetuate eating pathology (Bohle, von Wietersheim, Wilke, & Feiereis, 1991; Hilbert & Tuschen-Caffier, 2004; Leon, Lucas, Colligan, Ferdinande, & Kamp, 1985).

It has been suggested that the cognitive and perceptual components of emotion recognition reflect different skill sets which uniquely impact social ability (Kessler et al., 2006). However, research testing this hypothesis is limited, inconsistent in findings, and almost exclusively focused on individuals with anorexia (Bydlowski et al., 2005; Kucharska-Pietura et al., 2004; Zonnevijlle-Bender et al., 2002). If, as Kessler and colleagues (2006) claim, abilities in perceptual and cognitive emotion recognition are different and the perception of emotion in faces is not impaired in individuals with an eating pathology, then the link between social competence and emotion recognition (Kucharska-Pietura et al., 2004), could be explained by cognitive bias. Legenbauer, Vocks, and Ruddel (2008) spoke to the plausibility of this by citing research showing that individuals with eating disorders demonstrate an overall negative cognitive bias (Dritschel, Williams, & Cooper, 1991), particularly in the context of situations where cues are ambiguous and refer to weight/shape constructs (Cooper, 1997).

In their study comparing components of emotion recognition in individuals with bulimia and healthy controls, Legenbauer and colleagues (2008) found that the cognitive-affective aspect of emotion recognition was significantly impaired in
individuals with bulimia compared to controls. In contrast, perception of emotion was similar for all participants with the exception of the recognition of ‘surprise.’ Furthermore, no difference in emotion recognition was found between individuals with and without co-morbid depression, supporting the belief that perception of emotion recognition is not solely linked to depressive symptoms. Interestingly, they also found that contradictory to the nonsignificant findings regarding clarity and attention for the feelings of others, participants with an eating disorder reported, with a large effect size, less ability to distinguish their own feelings. This inability to distinguish personal emotions was inversely associated with negative body image for both groups. Legenbauer and colleagues (2008) speculated that if participants are unsure as to their emotions, they may also harbor insecurities which may be reflected in a higher degree of body dissatisfaction. This dissatisfaction could lead to attempts to reduce distress through maladaptive coping, including emotional eating or manipulation of weight.

**Obesity, Emotion, and Eating**

Studies have shown that obese individuals often have difficulty managing negative emotions and tend to direct feelings of anger and discomfort against themselves and subsequently rely on food for comfort (Castelnuovo-Tedesco & Schiebel, 1975). Contrary to the typical physiological response to emotional arousal, which is loss of appetite, emotional eating behaviors represent a pattern of enlarging food consumption as a means of reducing negative affect (van Vreckem & Vandereycken, 1995). According to psychosomatic theory, emotional eating results from learned behaviors in early life in which food becomes a primary coping mechanism is the result of learning experiences.
early in life in which food was used as a way of coping with psychological problems (Kaplan & Kaplan, 1957). As a direct result, some individuals develop impairments in their ability to recognize and accurately identifying emotions as well as sensations of hunger and satiety (i.e. introceptive awareness; Bruch, 1973).

Sim and Zeman (2005) reported that such dysregulated eating behaviors and body dissatisfaction was mediated, in part, by impaired emotional awareness, frequent negative emotional experience, and maladaptive coping in response to negative affect. The precise mechanisms underlying this relationship are unknown, but it has been suggested that individuals with poor emotional recognition and coping skills attribute negative emotions to their body and use eating to reduce their discomfort. Another possibility is that body dissatisfaction compromises a person’s ability to identify and cope with negative emotions, and therefore, the individuals use eating to manage the ambiguity of their emotional state (Hayaki, Friedman, & Brownell, 2002; Morosin & Riva, 1997).

**Obesity, the Brain, and Emotion Recognition: A Summary**

As mentioned earlier, recent developments in the literature have shown that obesity has a negative impact on the brain. Although it is known that the effects of the normal aging process as well as comorbid conditions such as hypertension and diabetes may contribute to the structural brain changes found in obese individuals (Gold et al., 2007; Kramer et al., 2007; Nash & Fillit, 2006; Raz. Rodgrigue, & Acker, 2003), studies controlling for these potential confounds suggest that mechanisms more directly related to adiposity and increased body mass account for more variance in these structural brain changes (Elias et al., 2003; Elias et al., 2005; Gunstad, Paul et al., 2007; Gunstad et al.,
2006; Wolf et al., 2007). These relationships are foundational to future mechanistic research in obesity.

The structural brain changes observed in obese populations have been linked not only with generalized cognitive and functional decline, but also with significant, specific neurocognitive deficits. The most frequently cited neuropsychological finding in obesity research suggests an association between increases in body mass and executive dysfunction, including reduced cognitive inhibition, organization, and flexibility (Boeka & Lokken, 2008, Cserjési et al., 2009; Cournot et al., 2006; Fergenbaum et al., 2009; Gunstad, Paul et al., 2007; Sabia et al., 2009; Wolf et al., 2007). Taken together with deficits in attention, psychomotor and information processing speed, and working memory, these impairments may have serious implications for an obese individual’s ability to recognize emotions in all forms—whether in the faces and behaviors of others, in images, in themselves—by impacting their ability to attend to and decipher facial expressions, make appropriate interpretations and attributions based on the information gained, attach appropriate cognitive labels, and effectively express emotion in social context. Thus, difficulties with emotion recognition may not only serve a role in the etiology of obesity, but may also serve to perpetuate it over the course of the lifespan.

The Current Study

No study to date has examined performance on tests of emotion recognition in an obese population while concurrently examining variables which may mediate emotion recognition in this population. The purposed study seeks to examine the associations among demographic and medical variables (i.e. age, BMI, gender, estimated premorbid
IQ, and diagnosis of diabetes, hypertension, or sleep apnea) as well as performance on cognitive tests of memory, attention, executive function, sensory-motor, and verbal skills.

**Hypotheses**

*Aim 1:* The first aim of the present study is to examine the relationship between performance on tasks of emotion recognition and body mass index. Consistent with previous research, it is hypothesized that:

*Hypothesis 1:* Body mass index will be significantly related to performance on tasks of emotion recognition. More specifically, body mass index will be inversely related to accuracy on tests of emotion recognition. It is also anticipated that as BMI increases, reaction time will increase.

*Aim 2:* The second and central aim of the proposed study is to examine demographic and medical variables as well as performance on neuropsychological tests as mediators of emotion recognition in this population.

*Hypothesis 2:* Elevated body mass index will be associated with reduced neurocognitive performance on tests of attention, fine motor and processing speed, executive function, and memory.

*Hypothesis 3:* Emotion recognition performance will be significantly related to performance on neurocognitive tasks as well as medical and demographic variables.

1. Performance on tasks of emotion recognition will be inversely correlated with age, estimated premorbid IQ, education, and presence of medical conditions.
2. Deficits in emotion recognition will be mediated by performance on tests of executive function, attention, processing speed, but not with tasks of motor, memory or language abilities after controlling for demographic variables and medical conditions.
METHOD

Overview

Data for this study were extracted from The Brain Resource International Database (BRID) [Gordon, 2003a; 2003b] and the Longitudinal Assessment of Bariatric Surgery (LABS) dataset [Gunstad et al., 2011]. The BRID is a quality-controlled database that contains demographic, medical, cognitive, and psychiatric data for healthy individuals across the lifespan. The LABS database contains demographic, medical, cognitive, and psychiatric data for morbidly obese candidates for bariatric surgery as well as demographically and medically similar obese individuals who are not seeking surgery. The same standardized and quality-controlled computerized neuropsychological test battery was used to obtain the neurocognitive test data for both datasets. These data were combined to form the current study sample.

Participants

Data from the BRID include healthy adults recruited from the community surrounding six sites (New York, Rhode Island, Holland, London, Adelaide, and Sydney). Original BRID exclusion criteria included history of psychiatric [e.g., schizophrenia, mood disorder, ADHD, substance use disorder; assessed with the Somatic and Psychological Health Report (Hickie, Davenport, Naismith, Hadzi-Pavlovic &
Koschera, 2001), medical (e.g., hypertension, diabetes, cardiac disease, sleep apnea, thyroid disorder), and neurologic (e.g., traumatic brain injury, dementia) conditions that are known to have an impact on brain structure and/or cognitive performance.

Data from the LABS database include morbidly obese adult participants who were recruited from LABS sites, including medical centers in New York and Fargo, North Dakota. Adults who were English-speaking and medically qualified for bariatric surgery were eligible to participate. Original LABS exclusion criteria included current psychiatric/neurological conditions and sensory deficits that are known to result in cognitive deficits independent of obesity or its related medical conditions (e.g., schizophrenia, stroke, head injury, dementia, mood disorder, alcohol/drug abuse). The Structured Clinical Interview for DSM-IV-TR Disorders (SCID; First, Spitzer, Gibbon, & Williams, 2002) was used to screen for potential psychiatric disorders.

The combined study sample consisted of 393 participants (70% women; age range 18-88; BMI range 18-75) who underwent standardized assessment of cognitive functioning and relevant medical/demographic covariates. See Table 1 for demographic and medical characteristics of study participants separated by data source.

**Procedures**

Informed consent was obtained from all participants prior to participation. At each site, participants were asked to undergo neuropsychological assessment and provide information regarding medical and psychiatric history. Participants’ body weight and height were measured using standardized protocols. Body mass index (BMI) was calculated using the formula: \([\text{weight in lbs} / (\text{height in inches})^2]\) x 703. At all sites, the
### Table 1

**Descriptive Statistics (M, SD), for Demographic and Medical Characteristics and Neurocognitive Composite Variables**

<table>
<thead>
<tr>
<th></th>
<th>BRID (n = 202)</th>
<th>LABS (n = 191)</th>
<th>Combined (n = 393)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender (% female)*</td>
<td>59%</td>
<td>81%</td>
<td>70%</td>
</tr>
<tr>
<td>Age (years)*</td>
<td>52.81 (19.94)</td>
<td>42.53 (11.42)</td>
<td>47.81 (17.13)</td>
</tr>
<tr>
<td>Spot the Word (IQ)*</td>
<td>50.85 (5.86)</td>
<td>46.39 (6.18)</td>
<td>48.67 (6.41)</td>
</tr>
<tr>
<td>BMI (kg/m$^2$)*</td>
<td>25.76 (4.21)</td>
<td>44.59 (6.84)</td>
<td>34.91 (10.98)</td>
</tr>
<tr>
<td>Hypertension*</td>
<td>0%</td>
<td>46.6%</td>
<td>22.6%</td>
</tr>
<tr>
<td>Type 2 Diabetes*</td>
<td>0%</td>
<td>23.6%</td>
<td>11.5%</td>
</tr>
<tr>
<td>Sleep Apnea*</td>
<td>0%</td>
<td>27.7%</td>
<td>13.5%</td>
</tr>
<tr>
<td>Attention/Processing</td>
<td>0.04 (0.41)</td>
<td>-0.07 (0.37)</td>
<td></td>
</tr>
<tr>
<td>Speed*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Executive Function*</td>
<td>-0.05 (0.92)</td>
<td>0.04 (1.04)</td>
<td></td>
</tr>
<tr>
<td>Motor Function*</td>
<td>0.04 (0.99)</td>
<td>-0.03 (1.01)</td>
<td></td>
</tr>
<tr>
<td>Language*</td>
<td>-0.03 (1.06)</td>
<td>0.04 (0.93)</td>
<td></td>
</tr>
<tr>
<td>Memory*</td>
<td>-0.01 (0.76)</td>
<td>0.02 (0.73)</td>
<td></td>
</tr>
<tr>
<td>Emotion Recognition</td>
<td>31.93 (6.52)</td>
<td>33.21 (3.93)</td>
<td></td>
</tr>
<tr>
<td>Accuracy (total correct)*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Emotion Recognition</td>
<td>2.27 (0.54)</td>
<td>2.05 (0.43)</td>
<td></td>
</tr>
<tr>
<td>Reaction Time (seconds)*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Emotion Recognition</td>
<td>14.79 (4.45)</td>
<td>16.82 (3.73)</td>
<td></td>
</tr>
<tr>
<td>Ratio (Accuracy/Reaction Time)*</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*denotes a significant (p < .05) difference between BRID and LABS groups. *Neurocognitive composite variables have been standardized within the current sample (i.e, z-scores: M = 0, SD = 1).
standardized test battery was administered in a fixed order using pre-recorded instructions and a touch-screen computer with automated verbal responses. Testing was monitored by trained research staff and took approximately 45-60 minutes to complete. Scoring was conducted using automated software for most tests and hand scoring by trained researchers for recorded verbal responses.

**Measures**

The computerized test battery *IntegNeuro* (Gordon 2003a; 2003b) was used to assess functioning in multiple cognitive domains. This battery is highly standardized, allowing for administration across sites, and has been shown to possess strong reliability and validity (Paul et al, 2005; Williams et al., 2005) with similar age-related patterns of performance. It has also differentiated lean and otherwise healthy obese persons in similar studies (Gunstad et al., 2006; Gunstad et al., 2007). Specific tests within the computerized battery are listed below by cognitive domain:

**Estimated IQ**

**Spot-the-Word.** This test is a computerized adaptation of the Spot the Real Word test which asks individuals to distinguish a valid word in the English language (“true” target word) from a non-word foil to render an estimated premorbid intelligence quotient which served as a dependent variable.
Attention

**Digit Span.** This test assesses basic auditory attention and working memory. Participants are presented with a series of digits on the touch-screen, separated by a one-second interval, and then asked to enter the digits on a numeric keypad. Forward and backward trials are presented, gradually increasing from 3 to 9 items, with two sequences at each level. The total number of correct forward and backward trials served as dependent variables.

**Span of Visual Memory.** This is a test of reaction time and sustained performance similar to the Wechsler Memory Scales-III Spatial Span. It requires forward and backward re-creation of illuminated patterns of circles on the screen by touching the screen in the same order. Twenty trials were administered with a random delay of 2-4 seconds between trials. Mean reaction time served as a dependent variable.

Executive Function

**Switching of Attention.** This test is a computerized adaptation of the Trail Making Test (Reitan, 1958) and consists of two parts. In the first part, participants are presented with a pattern of 25 numbers in circles and asked to touch them in ascending order assessing psychomotor speed and visual scanning. In the second part, an array of 13 numbers (1-13) and 12 letters (A-L) is presented. Participants are asked to touch numbers and letters alternately in ascending order assessing set-shifting demands. Time to completion (in seconds) of the two tests served as dependent variables.
**Verbal Interference.** This test assesses ability to inhibit automatic and irrelevant responses, similar to a Stroop task (Golden, 1978). The test is comprised of two parts. In the first part, the subject is asked to identify the name of each word as quickly as possible after it is presented. In the second part, they are asked to quickly name the color of each word. Each segment lasts for one minute. The dependent variable was a ratio of correctly identified words (Trial 2 divided by Trial 1).

**Maze Task.** This test is a computerized adaptation of the Austin Maze (Walsh, 1991). Participants are asked to identify a hidden path through an 8X8 matrix. The trial ends upon completion of the maze twice without error or after 10 minutes. The number of maze errors and the number of maze overruns (i.e. perseverative errors) were included as dependent variables.

**Memory**

**Verbal List-learning.** Participants are read a list of 12 words a total of 4 times, and asked to recall as many words as possible after each trial. Following a distraction list, participants are asked to recall words from the original list. After a 20-minute filled delay, participants are again asked to recall target words. Finally, a recognition trial comprised of target words and foils is completed. The following dependent variables were generated from this task: Learning (Trial 4 minus Trial 1 Recall), Delayed Recall, and Discrimination (true positives minus false positives on the recognition trial).
Language

Category Fluency. The test asks participants to generate as many animal names in a 60 second time period. Total number of correct words served as the dependent variable.

Motor

Motor Tapping. This test asks participants to tap a circle on the touch-screen as quickly as possible for 60 seconds. The dependent variable was the total number of taps with the dominant hand.

Explicit Emotion Recognition

This test employs 72 facial expression stimuli comprised of 12 different individuals (6 females, 6 males), each depicting neutral and evoked happiness, fear, sadness, anger, and disgust, from the Gur et al. (2002) standardized and normed set. Stimuli are equated for size, luminance, and central alignment of the face within the image (with eyes as midpoint reference). In the administration of this task, 48 of the face stimuli (8 different individuals, depicting the 6 expressions) were presented in a pseudorandom sequence, for 2 seconds each, and participants identified (via computer mouse click) the verbal label for each emotional expression from among the six expression options. Number of correct response, average reaction time for correct responses, and a ratio of correct responses to reaction time were used as dependent variables.
Data Analyses

Preliminary Analyses

Descriptive data regarding participant demographic characteristics, clinical conditions, and neuropsychological test performance were generated and the distribution of these variables were examined for potential violations of normality. Extreme values were truncated to 3 SD from the mean [Choice Reaction Time and Verbal Interference] and square root transformations were employed [Maze Errors and Maze Overruns] to address violations found to the assumptions of normality. Raw scores were employed for each neurocognitive task. Scores were then standardized (z-scores) and averaged into composite variables according to the factor model of neurocognitive domains which had previously been confirmed (Stanek, 2011).

Primary Analyses

Bivariate associations between BMI, demographic variables, emotion recognition, and neurocognitive test performance were performed. Correlation analyses were followed by a series of hierarchic linear regressions to examine whether cognitive performance in the above identified domains (e.g., executive function, attention, etc.) mediates the relationship between BMI and emotion recognition.

The following factors with known importance for cognitive function independent of obesity were employed as covariates in the primary analyses in order to examine the independent effects of BMI on emotion recognition tasks: sex, age, premorbid I.Q. estimate, and diagnosis of diabetes, hypertension, and/or sleep apnea.
RESULTS

Preliminary Correlation Analyses

Examination of zero-order correlations revealed that increased age was associated with poorer performance across all neurocognitive domains and emotion recognition tasks. Higher pre-morbid IQ estimates were found to be associated with better Language (r = .13, p < .01), Attention/Processing Speed (r = .11, p < .05), Emotion Recognition Accuracy (r = .14; p < .01), Emotion Recognition Reaction Time (r = .10; p < .05) and Executive Function (r = -.17, p < .01). Female gender was associated with better performance on tasks of Emotion Recognition (Accuracy r = .13; p < .01; Reaction Time r = -.18; p < .01). See Table 2 for full correlations among the control variables, neurocognitive factors, and Emotion Recognition Tasks.

Zero-order correlation analyses demonstrated that slower Emotion Recognition Reaction Times were associated with poorer Attention/Processing Speed (r = .20, p < .01), Executive Function (r = .15, p < .01), Language (r = -.29, p < .01) and Memory (r = -.23, p < .01) performance. Improvements in Emotion Reaction Accuracy were associated with better performance across all neurocognitive domains. See Table 3 for full correlations among emotion recognition tasks and neurocognitive factors.

Zero-order correlation analyses revealed significant associations between BMI and Emotion Recognition Reaction Time (r = -.17, p < .01), but no other cognitive factors. These effects were similar after controlling for age, gender, and pre-morbid IQ
Table 2

*Correlations among Age, Gender, Premorbid IQ Estimates, Emotion Recognition (ER) Tasks and Neurocognitive Domain Factors*

<table>
<thead>
<tr>
<th></th>
<th>Attention/Processing Speed</th>
<th>Executive Function</th>
<th>Motor Function</th>
<th>Language</th>
<th>Memory</th>
<th>ER Accuracy</th>
<th>ER Reaction Time</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>.316**</td>
<td>.352**</td>
<td>-.122*</td>
<td>-.353**</td>
<td>-.401**</td>
<td>-.538**</td>
<td>.279**</td>
</tr>
<tr>
<td>Gender</td>
<td>- .075</td>
<td>.036</td>
<td>-.046</td>
<td>.057</td>
<td>.159**</td>
<td>.130**</td>
<td>-.178**</td>
</tr>
<tr>
<td>Premorbid IQ Estimates</td>
<td>.114*</td>
<td>-.165**</td>
<td>.050</td>
<td>.035</td>
<td>.100*</td>
<td>.143**</td>
<td></td>
</tr>
</tbody>
</table>

*denotes significance at $p < .05$, **denotes significance at $p < .01$

Table 3

*Correlations among Emotion Recognition (ER) Tasks and Neurocognitive Domain Factors*

<table>
<thead>
<tr>
<th></th>
<th>Attention/Processing Speed</th>
<th>Executive Function</th>
<th>Motor Function</th>
<th>Language</th>
<th>Memory</th>
<th>ER Accuracy</th>
<th>ER Reaction Time</th>
</tr>
</thead>
<tbody>
<tr>
<td>Attention/Processing Speed</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Executive Function</td>
<td>__</td>
<td>__</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Motor Function</td>
<td>-0.01</td>
<td>-0.16</td>
<td>__</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Language</td>
<td>-0.16</td>
<td>-0.31</td>
<td>0.19</td>
<td>__</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Memory</td>
<td>-0.17</td>
<td>-0.35</td>
<td>0.05</td>
<td>0.31</td>
<td>__</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ER Accuracy</td>
<td>-0.22</td>
<td>-0.43</td>
<td>0.18</td>
<td>0.30</td>
<td>0.38</td>
<td>__</td>
<td></td>
</tr>
<tr>
<td>ER Reaction Time</td>
<td>0.20</td>
<td>0.15</td>
<td>-0.10</td>
<td>-0.29</td>
<td>-0.23</td>
<td>0.00</td>
<td>__</td>
</tr>
</tbody>
</table>

Bolded, shaded coefficients are significant at $p < .01$ level.
estimates \((r = -0.14, p < 0.05)\). See Table 4 for full results of partial correlation analyses of BMI, medical variables, and neurocognitive factors. See footnote for additional analyses.¹

**Main Effects of BMI on Emotion Recognition Accuracy**

Results of a hierarchal multiple regression analysis predicting Accuracy of Emotion Recognition from BMI while controlling for gender, age, pre-morbid IQ estimates, hypertension, sleep apnea, diabetes, and performance in all neurocognitive domains demonstrated significant overall model fit \([R^2 = 0.37, F(4, 384) = 56.81, p < 0.001]\). Increases in BMI did not significantly predict accuracy of Emotion Recognition \((\beta = 0.02)\) and the addition of BMI to the model did not improve overall model fit \((\Delta R^2 = 0.00, \Delta F = 1.37, p = 0.71)\), indicating that BMI was not an independent predictor of Emotion Recognition Accuracy. See Table 5 for a depiction of main effects for accuracy and speed of Emotion Recognition.

**Main Effects of BMI on Emotion Recognition Accuracy Accounting for Speed**

A ratio of Emotion Recognition Accuracy to Emotion Recognition Reaction Time was generated to represent a possible speed/accuracy trade-off. A hierarchal multiple

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¹ Results of the main regression analyses include control of relevant medical conditions within the LABS dataset, which according the literature may influence the impact the interpretation of the effects of BMI on emotion recognition given the established independent effects of these distinct, yet comorbid conditions on neurocognition in general (see Table 2 for correlations in the present sample). Although the aim of the present study was to examine the independent effects of BMI on performance on tasks of emotion recognition, analyses were also conducted to examine the overall cumulative impact of obesity without parsing out the impact of these medical conditions. Results indicated that the effects of BMI on Emotion Recognition Accuracy and Emotion Recognition Reaction Time in the absence of medical covariates were similar to the results of the primary analyses, which included these variables.
Table 4

*Partial Correlations among BMI, Medical Conditions, and Neurocognitive Test Performance Controlling for Age, Gender, and Pre-Morbid IQ Estimates*

<table>
<thead>
<tr>
<th></th>
<th>BMI</th>
<th>Hypertension</th>
<th>Type 2 Diabetes</th>
<th>Sleep Apnea</th>
</tr>
</thead>
<tbody>
<tr>
<td>ER Accuracy</td>
<td>0.00</td>
<td>0.13</td>
<td>0.06</td>
<td>0.01</td>
</tr>
<tr>
<td>ER Reaction Time</td>
<td>-0.14</td>
<td>-0.06</td>
<td>-0.06</td>
<td>0.05</td>
</tr>
<tr>
<td>Attention/Processing</td>
<td>-0.07</td>
<td>-0.03</td>
<td>-0.07</td>
<td>-0.07</td>
</tr>
<tr>
<td>Speed</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Executive Function</td>
<td>0.00</td>
<td>0.03</td>
<td>0.07</td>
<td>-0.06</td>
</tr>
<tr>
<td>Motor Function</td>
<td>0.10</td>
<td>0.07</td>
<td>-0.04</td>
<td>0.01</td>
</tr>
<tr>
<td>Language</td>
<td>-0.00</td>
<td>0.09</td>
<td>-0.06</td>
<td>-0.09</td>
</tr>
<tr>
<td>Memory</td>
<td>-0.04</td>
<td>-0.15</td>
<td>-0.06</td>
<td>-0.12</td>
</tr>
</tbody>
</table>

Bolded, shaded coefficients are significant at $p < .05$ level.

Table 5

*Main Effects of BMI on Emotion Recognition (ER) Variables Controlling for Age, Gender, Pre-Morbid IQ Estimates, Medical Conditions, and Performance on Neurocognitive Domains (n = 393)*

<table>
<thead>
<tr>
<th></th>
<th>BMI</th>
<th>SE</th>
<th>$\beta$</th>
</tr>
</thead>
<tbody>
<tr>
<td>ER Accuracy</td>
<td>-0.03</td>
<td>0.03</td>
<td>-0.05</td>
</tr>
<tr>
<td>ER Reaction Time</td>
<td>-0.01*</td>
<td>0.00</td>
<td>-0.14</td>
</tr>
<tr>
<td>ER Ratio (Accuracy/Time)</td>
<td>0.03</td>
<td>0.02</td>
<td>0.09</td>
</tr>
</tbody>
</table>

*$p < 0.05$*
regression analysis predicting this ratio from BMI while controlling for gender, age, pre-morbid IQ estimates, hypertension, sleep apnea, diabetes, and performance in all neurocognitive domains demonstrated overall significant model fit \( R^2 = .49, F(12, 243) = 19.44, p < .001 \). Increases in BMI did not significantly predicted this ratio (\( \beta = .09 \)) and adding BMI to the model did not improve overall model fit (\( \Delta R^2 = .01, \Delta F = 2.56, p = .11 \)), indicating that BMI was not an independent predictor of Emotion Recognition Accuracy when speed was taken into account.

**Main Effects of BMI on Emotion Recognition Reaction Time**

Results of a hierarchal multiple regression analysis predicting Emotion Recognition Reaction Time from BMI while controlling for gender, age, pre-morbid IQ estimates, hypertension, sleep apnea, diabetes, and performance in all neurocognitive domains demonstrated significant overall model fit \( R^2 = .23, F(9, 246) = 8.15, p < .001 \). Increases in BMI significantly predicted faster Emotion Recognition Reaction Times (\( \beta = -.13 \)). In addition, adding BMI to the model improved overall model fit (\( \Delta R^2 = .02, \Delta F = 4.84, p < .05 \)), indicating that BMI was an independent predictor of speed of Emotion Recognition.

**Mediation Models**

It had originally been postulated that deficits on tasks of emotion recognition would be mediated by performance on tests of executive function, attention, processing speed, but not by performance on tasks of motor, memory or language abilities after controlling for demographic variables and medical conditions. However, the earlier
correlations demonstrated that BMI was not significantly associated with performance in any neurocognitive domain. As such, the basic criteria for mediation had not been met (Baron and Kenny, 1986; Judd and Kenny, 1981). Therefore, although BMI was found to be a significant predictor of speed of Emotion Recognition, this relationship was not mediated by performance in other cognitive domains.

**Post Hoc Analyses**

Given that BMI independently predicted speed of Emotion Recognition, post hoc analyses were conducted to explore possible moderators of this association. Cross-product terms were created between BMI and each of the following variables: Age, Gender, Premorbid IQ Estimate, Attention/Processing Speed, Executive Function, Memory, Motor, and Language performance. In this series of regression analyses, none of the interactions significantly improved model fit. These findings indicate that the relationship between BMI and speed of Emotion Recognition is robust. Regardless of the individual’s gender, age, estimated premorbid IQ or assessed neurocognitive performance, increases in BMI are associated with faster speed of Emotion Recognition tasks. See Table 6 for complete depiction of the interaction effects.

For illustrative purposes, BMI was categorized into five groups based on established BMI classifications: Normal Weight (BMI=18.0 - 24.9), Overweight (BMI=25.0 - 29.9), Class I Obesity (BMI= 30.0 - 34.9), Class II Obesity (BMI= 35.0 - 39.9), and Class III Obesity (BMI= 40.0 or higher). A one-way ANOVA was run to examine between group differences in Emotion Recognition Speed. Individuals with BMI of 40 or higher were significantly faster at recognizing emotions than individuals whose BMIs fell
Table 6

*Results of Adjusted Regression Analyses Examining Interaction Effects of BMI with each of the Demographic and Neurocognitive Variables on Emotion Reaction Speed (n = 393)*

<table>
<thead>
<tr>
<th>Interaction</th>
<th>B</th>
<th>SE</th>
<th>β</th>
<th>R²</th>
<th>ΔR²</th>
<th>ΔF</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI x Executive Function*</td>
<td>.001</td>
<td>.003</td>
<td>.024</td>
<td>.126</td>
<td>.001</td>
<td>.221</td>
</tr>
<tr>
<td>BMI x Attn/Processing Speed*</td>
<td>.009</td>
<td>.007</td>
<td>.063</td>
<td>.132</td>
<td>.004</td>
<td>1.631</td>
</tr>
<tr>
<td>BMI x Memory*</td>
<td>-.006</td>
<td>.003</td>
<td>-.090</td>
<td>.138</td>
<td>.007</td>
<td>3.152</td>
</tr>
<tr>
<td>BMI x Language*</td>
<td>.001</td>
<td>.002</td>
<td>.013</td>
<td>.169</td>
<td>.000</td>
<td>.064</td>
</tr>
<tr>
<td>BMI x Motor*</td>
<td>-.001</td>
<td>.002</td>
<td>-.020</td>
<td>.125</td>
<td>.000</td>
<td>.152</td>
</tr>
<tr>
<td>BMI x Age**</td>
<td>.000</td>
<td>.000</td>
<td>.010</td>
<td>.118</td>
<td>.000</td>
<td>.029</td>
</tr>
<tr>
<td>BMI x Gender**</td>
<td>.000</td>
<td>.000</td>
<td>.058</td>
<td>.189</td>
<td>.002</td>
<td>.941</td>
</tr>
<tr>
<td>BMI x IQ**</td>
<td>-.001</td>
<td>.000</td>
<td>-.068</td>
<td>.189</td>
<td>.004</td>
<td>2.099</td>
</tr>
</tbody>
</table>

*effects of age, IQ, gender, medical variables, and performance in all other neurocognitive domains removed

**effects of medical variables, performances in all neurocognitive domains, and all other demographic variables removed
in the normal, overweight, or class I obesity categories. There was no statistically
significant difference between any other group pairings, indicating that the significant
main effects observed between BMI and Emotion Recognition Speed were driven by the
performance of the sample. Refer to Table 7 and Figure 1 for the full depiction of the
between group differences.

Table 7
One-way ANOVA Examining Between Groups Differences among BMI Categories

<table>
<thead>
<tr>
<th></th>
<th>Sum of Squares</th>
<th>df</th>
<th>Mean Square</th>
<th>F</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between Groups</td>
<td>3.843</td>
<td>4</td>
<td>.961</td>
<td>3.858</td>
<td>.004</td>
</tr>
<tr>
<td>Within Groups</td>
<td>84.935</td>
<td>341</td>
<td>.249</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>88.778</td>
<td>345</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Figure 1
Mean Emotion Recognition across BMI Categories
DISCUSSION

The current study sought to examine the associations among demographic and medical variables (i.e. age, BMI, gender, estimated premorbid IQ, and diagnosis of diabetes, hypertension, or sleep apnea) as well as performance on cognitive tests of memory, attention, executive function, sensory-motor, and verbal skills. The first aim was to explore the relationship between performance on tasks of emotion recognition and body mass index. Contrary to the hypothesis that BMI would be inversely related to performance on tests of emotion recognition, results indicated that as BMI increases, reaction time to complete these tasks decreases. This finding was noted even after the effects of age, gender, estimated pre-morbid IQ, pre-existing medical conditions, and performance in all neurocognitive domains was removed. With each standard deviation increase in BMI, emotion recognition speed was 0.14 standard deviations faster. In addition, when examined across BMI categories, it was observed that participants with BMIs greater than 40 kg/m² showed the fastest reaction times.

Further, the primary aim of the proposed study was to study demographic, medical and neurocognitive performance variables as mediators of emotion recognition in this population. Examination of zero-order correlations revealed that decreased age, higher pre-morbid IQ, female gender, and better performance in the domains of Attention/ Processing Speed, Executive Function, Language and Memory were
associated with better performance across all emotion recognition tasks. However, contrary to the hypotheses that elevated BMI would be associated with reduced neurocognitive performance on tests of attention, fine motor and processing speed, executive function, and memory, it was found that in this population BMI was not significantly associated with performance in any neurocognitive domain. As such, emotion recognition speed was not mediated any of these variables. Post-hoc moderation analyses demonstrated that regardless of the individual’s gender, age, estimated premorbid IQ or assessed neurocognitive performance, increases in BMI are associated with faster speed of Emotion Recognition tasks. Overall, these findings extend current understanding of emotion-processing as it relates to BMI and also raise questions regarding the mechanisms underlying the evolution of improved emotion recognition speed in obese individuals.

**Consistency with Current Research**

To date, no studies have examined performance on tasks of emotion recognition based on BMI. Therefore, the study’s findings regarding emotion recognition are novel and represent an extension of current knowledge. However, it is important to note that in this study correlational analyses revealed no significant associations between BMI and neurocognitive performance. These effects are contrary to the findings of other studies which have identified associations between increases in BMI and neurocognitive deficits (Boeka & Lokken, 2008, Cserjési et al., 2009; Cournot et al., 2006; Fergenbaum et al., 2009; Gunstad, Paul et al., 2007; Sabia et al., 2009; Stanek, Strain, et al., 2013; Wolf et al., 2007). One possible explanation for this disparity is lack of sufficient power due to
sample size. In this study, the sample consisted of 393 individuals. Of these participants, 204 met criteria for obesity (BMI ≥ 30). Given the small magnitude of main effects noted in the existing literature when associations between BMI and neurocognitive performance are found, it is anticipated that a much larger sample would be required to observe similar results.

**Emotion Theory and Heightened Emotional Sensitivity as an Explanation of Study Findings**

The current findings provide support for contemporary theories of emotion which generally agree that emotions evolved to facilitate adaptation to environmental threats (Plutchik, 2003). Main effects for BMI emerged in the examination of emotion recognition speed while accuracy remained unaffected. Initially, it may appear surprising that the time taken to recognize emotions decreases as BMI increases given the literature which has identified specific neurocognitive deficits in executive function, attention, working memory, and psychomotor and information processing speed in obese individuals (Boeka & Lokken, 2008, Cserjési et al., 2009; Cournot et al., 2006; Fergenbaum et al., 2009; Gunstad, Paul et al., 2007; Sabia et al., 2009; Wolf et al., 2007). However, if the functional capacity of rapid emotion recognition is considered in the context of the psychological experience of obesity, this finding can be more readily explained. Whether through psychopathology, the insidious effects of stigma and discrimination, or a combination of the two, obese individuals may have developed heightened sensitivity to facial expressions of emotion as a means of self-protection.
Faster emotion recognition would likely have been initially adaptive, aiding the individual in avoiding perceived threat.

**Psychological Implications of Obesity**

Obesity and overweight have been associated with many psychological and psychosocial consequences and are associated with significant emotional distress. These consequences include greater report of psychological difficulties such as depression, pathological eating behaviors, societal stigma, and reduced quality of life (Bean, Stewart, & Olbrisch, 2008; Fabricatore & Wadden, 2006). Women, patients pursuing bariatric surgery, and the morbidly obese are at greater risk for experiencing these negative outcomes (Bean, Stewart, & Olbrisch, 2008). An understanding of these relationships can inform a comprehensive conceptualization of how faster emotion recognition evolve as BMI increases.

**Psychopathology and Obesity**

The association between obesity and psychiatric disorders in the general population is an ongoing debate (Muhlhaus, Horbach, & de Zwaan, 2009). The literature is mixed, with some studies indicating that obesity is linked to an increased risk of psychopathology while others find no relationship or a negative relationship (Muhlhaus & de Zwaan, 2008). However, lack of homogeneity exists in the methodology used and this is combined with multiple potential moderators such as gender, severity of obesity, or socioeconomic status and differing populations which may provide a possible explanation for the disparity in findings and confound a comparison of associations.
between obesity and different psychiatric states (Faith, Matz, & Jorge, 2002; Friedman & Brownell, 1995; Muhlhans, Horbach, de Zwaan, 2009).

Recent research had found positive associations between obesity and depression (Benson et al. 2008), bipolar disorder (Alciati et al., 2007), anxiety disorders (Kalarchian, Marcus, Levine, Courcoulas, et al., 2007), eating pathology (Ramacciotti, Coli, Bondi, Burgalassi, Massimetti, & Dell’Osso, 2008), attention-deficit/hyperactivity disorder (Kalarchian, Marcus, Levine, Courcoulas, et al., 2007), personality disorders (Amann et al., 2009; Jonge, Van Furth, Lacey, & Waller, 2003; Mobbs, Crepin, Thiery, Golay, & Van der Linden, 2010; Sansone, Wiederman, & Sansone, 2000), increased substance use (Kalarchian, Marcus, Levine, Courcoulas, et al., 2007), and post-traumatic stress disorder and trauma history (Gunstad, Paul, Spitznagel, Cohen, Williams, Kohn, et al., 2006; Perkonigg, Owashi, Stein, Kirschbaum, & Wittchen, 2009). These clinical presentations have the potential to not only influence emotional experience, but further compound the stigma and discrimination experienced due to weight.

**Psychosocial Difficulties of Obesity**

Stigma and discrimination against overweight and obese individuals are common despite the increasing prevalence of these conditions. These negative attitudes and behaviors extend across the population and encompass even health professionals. Research has associated overweight and obesity with specific disadvantages in the areas of getting into college, renting a home, and getting married (Pi-Sunyer, 1998). In addition, these individuals are often believed to be “mean, stupid, ugly, unhappy, less competent, socially isolated, and lacking in self-discipline, motivation and personal
control” (Puhl & Brownell, 2006). These beliefs are learned early and children as young as age five rate overweight and obese children as less likeable, particularly obese females (Penny & Haddock, 2007). These biases persist into adulthood and have a potentially significant impact on the individual’s economic, professional, and personal opportunities.

**Heightened Sensitivity to Emotional Stimuli**

Taken together, obesity’s psychological and psychosocial sequelae foster an environment which poses the potential for an almost continuous emotional threat. This becomes increasingly important as the process of acquiring emotion recognition is considered. At an early age, humans begin developing the ability to detect, discriminate and recognize emotions (Nelson, 1987; Walker-Andrews, 1997). Despite an incomplete visual system at birth, newborns instinctively orient towards face-like stimuli as well as possess the ability to mimic simple facial movements such as opening of the mouth (Meltzoff & Moore, 1983; Valenza, Simion, Macchi-Cassia, & Umilta, 1996). After a few months, as their visual system matures, infants begin to discriminate between basic facial expressions. Research has found that at three months of age infants can discriminate between smiling and neutral faces (Kuchuk, Vibbert, & Bornstein, 1986) and that by four and five months they can discriminate between facial expressions of joy and anger (LaBarbera, Izard, Vietze, & Parisi, 1976). The ability to recognize other basic expressions of emotion appears to develop during the second half of the first year (Walker-Andrews, 1997). However, full maturation of these skills occurs gradually across the lifespan and is not isolated to one developmental stage. More subtle expression of emotion may, in fact, continue to develop well into early adulthood (Herba & Phillips,
2004). The implication of this trajectory is that as obese individuals undergo repeated exposure to negative emotional stimuli, they have the potential to develop a learned, heightened sensitivity to emotional expression. The more an individual is exposed to face of disgust, for example, the more readily he or she can recognize it as it develops.

Similar findings have been found in individuals suffering with Borderline Personality Disorder (BPD). Lynch, Rosenthal, and colleagues (2006) examined emotional sensitivity in BPD by comparing 20 individuals with BPD and 20 normal controls on their accuracy in identifying facial expression of emotion. Their results demonstrated that, as facial expressions morphed from neutral to maximum intensity, participants with BPD correctly identified facial affect at an earlier stage than did healthy controls. In addition, participants with BPD were more sensitive than healthy controls in identifying emotional expressions in general, regardless of valence. As in the current study, no corresponding sacrifice in accuracy was observed to account for the increase in speed. Apart from studies of BPD, faster speed of facial affect recognition has been rarely reported for clinical groups, the exception being paranoid schizophrenia (Davis & Gibson, 2000).

While these disorders and obesity may appear vastly disparate, there are commonalities. Both BPD and obesity have been associated with a higher prevalence of trauma history and/or the presence of a chronically invalidating environment (Gunstad, et al., 2006; Perkonigg et al., 2009; Zanarini, Dubo, Lewis, & Williams, 1997). Over time as these stimuli are repeatedly presented, the potential for learning increases. Theoretically, the more familiar the individual is with the stimulus the quicker the
recognition of that stimulus becomes. This could reasonably explain observed increases in speed of negative emotion recognition. However, it is unclear what mechanisms would lead to the generalization of this ability to the recognition of positive emotions in the same individual assuming they were experienced with less frequency. As this study did not parse out differences in emotion recognition speed or accuracy based on emotion type, future research will be needed to further explore these differences.

**Limitations**

The results of the current studying and the corresponding conclusions must be interpreted in the context of several limitations. Importantly, the current study used a task of explicit emotion recognition, which presumably serves a proxy for an individual’s ability to recognize emotion in real world settings. However, problems have arisen in the study of emotion recognition and facial expressions due to a lack of a widely accepted, standardized measure of emotion recognition. While the majority of studies in this area rely on slight variations of the basic experimental protocol used here, dissimilar stimuli make it difficult to compare and contrast the results of these studies and limit generalizability (Edwards et al., 2002; Elfenbein & Ambady, 2002; Morrison et al., 1988; Russell, 1994; Russell, Bachorowski, & Fernandez-Dols, 2003). Further, additional problems related to the stimuli themselves have been identified.

Although emotions are expressed in a variety of ways, studies of facial affect recognition typically utilize prototypes of facial emotions as stimuli (Russell, 1994, 2003). It is argued that the use of such prototypes compromises the studies’ validity. In fact, research comparing spontaneous expressions of emotion with posed expressions
supports the claim that posed facial expressions lack ecological validity (Ekman, 1993; Moscovitch & Olds, 1982; Motley & Camden, 1988). Researchers cite that posed facial expressions are found to be more symmetrical than spontaneous expressions and are characterized by the absence of action by certain facial muscles as an explanation for these claims (Ekman, 1993; Moscovitch & Olds, 1982). Spontaneous facial expressions are, in contrast, more ambiguous than staged displays of basic emotion as individuals seldom experience emotions in isolation (Ekman et al., 1987; Halberstadt, 2003). This ambiguity could account for findings demonstrating a greater accuracy of emotion recognition among individuals judging posed rather than spontaneous expressions (Gosselin, Kirouac, & Dore, 1995; Motley & Camden, 1988; Russell et al., 2003).

As mentioned earlier, there are also differences in the response format used in these studies. In this study, a forced choice option is used where participants select their responses from a list of emotions. Some researchers argue that a forced-choice methodology produces artifacts by inflating agreement (Russell et al., 2003). However, even with all of these potential variants, the majority of the literature finds that under these conditions demographically diverse individuals reach above chance levels of agreement in identifying displayed emotions (Elfenbein & Ambady, 2002; Russell, 1994).

Furthermore, the cross-sectional design of the present research limits the interpretation of findings, especially in regards to mechanisms and potential causal links. Additional longitudinal studies are needed to observe emotional/psychological, cognitive and structural brain changes in obesity as well as quasi-experimental studies to explore
changes in emotion recognition corresponding to changes in weight. Additionally, longitudinal research is needed to more fully explore the life factors which may influence the relationship between elevated BMI and faster speeds of emotion recognition.

The sampling methodology in the current study allowed for a wide range of BMIs among study participants to address study aims and increase generalizability of findings; however, the exclusion of common medical and psychological conditions of the BRID may have created a sample that was qualitatively different from the LABS sample. For example, the BRID sample may have been more physically fit, less prone to physical and/or mental fatigue, or less likely to have experienced a history of trauma or discrimination. These possible differences may not be fully accounted for through statistical controls and they thus may complicate results through statistical artifacts.

The current study also only utilized one index of obesity creating another potential area of weakness in the methodology. Despite BMI’s function as the gold standard in the measurement of obesity for clinical and research purposes, this index does not distinguish lean muscle mass from fat composition, and it may not be a particularly optimal measure of adiposity. Future studies should incorporate multiple indices of obesity to more fully explore relationships between adiposity and emotion recognition.

Lastly, although the demonstrated effects on speed of emotion recognition were statistically significant and correspond to theoretically supported speculation regarding potential mechanisms, the magnitude of the independent BMI effects are small ($\beta = -.14$). However, given the large number of covariates in the analyses this test is conservative,
thus the results are quite robust. Additionally, given the nature of the results significance can be inferred regarding important clinical implications.

**Conclusions**

Results of the present study suggest important clinical implications for the assessment, treatment, and management of obesity. In summary, findings indicated associations between elevated BMI and faster emotion recognition reaction times. The improvements in speed of emotion recognition may be mild, as indicated by the relatively small effect sizes. However, even minor increases in speed of emotion recognition may correspond to more significant difficulties in interpersonal functioning. For example, while being able to more rapidly identify emotions may have been initially adaptive, it may, over time, result in a hypervigilant state in which the individual experiences persistent physiological arousal secondary to constant scanning of the environment for emotional cues. Physiological arousal alone is sufficient to trigger negative emotional appraisal and a subjectively distressful experience. In addition, high sensitivity to emotional cues together with a potential for biased perception towards social rejection or threat may also lead to situational avoidance.

While these difficulties may be relatively mild and may not be universally apparent in obese individuals, health care providers should be aware of the potential impact of these findings and tailor treatments and clinical strategies appropriately. Given the potential for a negative pattern of interpersonal perception, clinicians need to be aware of their nonverbal signals and their patients’ possible misinterpretation thereof. If a patient is primed to detect and avoid the negative appraisals of others, they will be
much less likely to fully engage in treatment and may require more support in generating successful behavioral changes.

Additional research is needed regarding the functional implications of increased emotion recognition speeds in obesity as current conclusions are speculative. Studies such as these could afford clinicians and researchers the opportunity to fully appreciate the psychosocial impact of obesity related changes in emotion perception. Similarly, increased understanding of the specific mechanisms underlying these observations may eventually improve the effectiveness of the treatment and management of obesity and obesity-related psychological distress.
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