Does a sensitive palate beget sensitive mood?
The relation between supertasting and disordered mood

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ABSTRACT

ANNA VAN METER: Does a sensitive palate beget sensitive mood?
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Objective

Prevalence rates of bipolar disorder may be as high as 11% (Angst et al., 2003); currently, research is being conducted on biologically-based traits, with the goal to find ways to ascertain a person's risk for bipolar disorder, or to lend greater certainty to a diagnosis. One trait of interest is an individual's ability to taste phenothioureas, a family of bitter-tasting compounds (Wooding, 2006). The aim of the present study is to determine whether this taste sensitivity has utility as a biomarker for mood disorder risk and, if so, whether emotional reactivity and regulation moderate this relation.

Method

Participants (*N*=499) were undergraduates at the University of North Carolina at Chapel Hill. Participants completed a series of questionnaires related to their mood, emotion regulation, and family history of psychiatric disorder. Next, participants completed a mood induction paradigm. Finally, participants' taste sensitivity was measured.

Results

Three groups, based on taste sensitivity, were identified. Ratings of hypomania, family history of psychiatric disorder, psychological treatment seeking, and emotion

regulation did not differ across groups. Scores on the BDI were related to taste sensitivity ($p<.05$), but this relation was driven primarily by outliers. Using regression, tasting predicted stronger responses to both positive and negative mood inductions ($p<.05$). Additionally, the interaction of negative emotion regulation and tasting predicted weaker responses to the mood inductions. Finally, emotion regulation strategies were predictive of both depression and hypomania scores ($p<.05$). Testing the effect sizes against the zone of indifference ($r= \pm 0.2$), only the emotion regulation strategies showed promise as predictors of mood disorder.

Discussion

The present study represents the largest sample investigating mood and supertasting. Therefore, the low – or absent – effect size of taste sensitivity in the present analyses sheds doubt on the utility of taste sensitivity as a biomarker for mood disorder risk. However, there were trends to suggest that supertasters are more sensitive to their environment than nontasters and that they may have increased risk for depression. Additionally, taste – or threat – sensitivity may interact with negative emotion regulation strategies in intriguing ways. Future studies, using a clinical sample, may help to better elaborate the trends found in this study.

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Mood disorders affect a large proportion of the population; lifetime prevalence rates for depression are estimated at >19% (Kessler et al., 2010) and rates for bipolar spectrum disorders may be as high as 11% (Angst, Gamma, Benazzi, et al., 2003). These disorders are associated with substantial human suffering, as well as cost to society, in the form of lost productivity and healthcare costs (WHO, 2008). Perhaps most concerning is the high rate of suicidal behavior among people with mood disorders; among those with bipolar disorder, up to 50% may attempt suicide and the completion rate is 20 times higher than that of other attempters (Baldessarini & Tondo, 2003; Bostwick & Pankratz, 2000; Goldstein et al., 2005).

Unfortunately, bipolar disorder can be difficult to diagnose, and – on average – people go ten years between their introduction to mental health services and an accurate diagnosis (Hirschfeld et al., 2003; Lish, Dime-Meenan, Whybrow, Price, & Hirschfeld, 1994). During this time, individuals are likely receiving insufficient and/or inappropriate treatment, which may worsen their prognosis (McElroy, Strakowski, West, Keck, & McConville, 1997; Schraufnagel, Brumback, Harper, & Weinberg, 2001). Diagnosis is further complicated by the fact that the vast majority, as many as 90%, of people with bipolar disorder also suffer from another Axis I or II disorder (Faedda, Baldessarini, Glovinsky, & Austin, 2004; Kowatch, Youngstrom, Danielyan, & Findling, 2005; Wozniak et al., 1995; Youngstrom, 2009).

Currently, diagnosis relies on clinical interview. Unique to bipolar disorder is the fact that retrospective reporting of symptoms is equally important to the diagnostic formulation as current symptomatology: the clinician must ascertain whether or not both manic and depressive symptoms have *ever* been present. Unfortunately, many clinicians fail to inquire about previous episodes of mania or depression, or the patient may not accurately remember (Maughan & Rutter, 1997; Youngstrom, 2010; Youngstrom, Birmaher, & Findling, 2008). Though other information – such as family history, prevalence rates, and reports from other informants – may be helpful in improving diagnostic accuracy (Youngstrom & Duax, 2005; Youngstrom & Youngstrom, 2005), these are not often included in an assessment.

Objective Measure of Risk

There is a serious need for objective measures of mood disorder risk. Currently, research is being conducted on a number of biologically-based traits, with the goal to find ways in which to ascertain a person's risk for bipolar disorder, or to lend greater certainty to a diagnosis. Among these constructs are temperament and the behavioral activation system (BAS), both of which have been linked to increased risk for mood disorder and may offer improved diagnostic discrimination (Akiskal & Akiskal, 1992; Akiskal et al., 1995; Alloy et al., 2008; Goto, Terao, Hoaki, & Wang, 2010; Urosevic, Abramson, Harmon-Jones, & Alloy, 2008). However, using current methods, both BAS and temperament are measured using self-reports. Though valuable information can be gleaned from self-reports, mood symptoms are also measured through self-report (interview); and the addition of more self-report information does not add to interview data as much as a distinct method can (Campbell & Fiske, 1959). In addition, data collected from both self report and interview are subject to

bias of interpretation. An objective way by which to assess risk for mood disorders would be a valuable addition to the field.

Genes. There is little doubt that genes play an important role in the development of mood disorders, but to date, the identification of genes specific to mood disorders has proved elusive (Hasler, Drevets, Gould, Gottesman, & Manji, 2006). The search for gene candidates for mood disorder is active, but there are hundreds of candidates (Johansson et al., 2001; Smoller & Finn, 2003), and studies have failed to replicate most findings; therefore, there is no consistent support for any one gene or family of genes (Anguelova, Benkelfat, & Turecki, 2003; Cho et al., 2005; Schulze & McMahon, 2009). Someday, a genetic test may be available for bipolar and other mood disorders, but as yet, that possibility remains elusive. Most agree that bipolar disorder does not result from a homogenous etiology (Hasler, et al., 2006); rather it is the confluence of genetic and environmental risk that results in bipolar disorder (Faraone, Glatt, & Tsuang, 2003; Mick & Faraone, 2009; Serretti & Mandelli, 2008; Smoller & Finn, 2003). The identification of a component implicated in the gene*environment interplay that can be easily and inexpensively tested would be an important intermediate step in the development of an objective, reliable method of diagnosis.

Brain structure and function. In addition to genes, studies have investigated brain structure and function as possible biomarkers of bipolar disorder. Though some promising results have been found (Chang et al., 2005; Chang, Wagner, Garrett, Howe, & Reiss, 2008; DelBello, Zimmerman, Mills, Getz, & Strakowski, 2004; Pavuluri, O'Connor, Harral, & Sweeney, 2008), most are not consistent across studies, nor are they specific to bipolar disorder (Terry, Lopez-Larson, & Frazier, 2009). Additionally, there is evidence to suggest that the use of mood stabilizers in people with bipolar disorder may affect brain composition

and function, making it difficult to determine whether brain abnormalities are the cause or consequence of bipolar disorder (Savitz & Drevets, 2009). Finally, the brain abnormalities found in people with bipolar disorder can be different in adults and children, which confuses the implications of these findings from a developmental perspective (Pavuluri, O'Connor, Harral, & Sweeney, 2007). At this point, the limited utility of brain structure and function information cannot justify the great expense associated with the fMRI tests or other brain scans necessary to find abnormalities. A biomarker that can be identified quickly, and with limited time and monetary resources is needed.

Supertasting

One biomarker that can be easily and inexpensively measured, and that has been investigated in relation to alcoholism, and to a lesser extent, depression, is taste sensitivity. One's sensitivity to specific bitter compounds can be easily and inexpensively tested and is linked to a single gene, making it a very promising genetic marker (Wooding, 2006). The evidence of a relation between taste and depression, though intriguing, has not been widely investigated, nor has research expanded to determine whether or not supertasting may also be associated with other mood disorders, such as bipolar disorder. This branch of research has not been developed, but the theory and results to date lend support to the additional investigation of the relation between one's taste sensitivity and mood.

Though it may seem like a stretch to propose taste sensitivity as a vulnerability for disordered mood, links between taste and affect are, in fact, well-established (Dess & Edelheit, 1998). Our taste experiences are sent to regions of the brain responsible for motivation, reinforcement, memory, and emotion (Dess & Edelheit, 1998; Scott, 1987). Our mood and emotions can also affect our taste; many people crave sweet or fatty foods when

feeling stressed or low, and find that eating these "comfort foods" improves their mood. Food consumption for the purpose of regulating emotion, known as instrumental eating, is found predominantly during negative mood states (Greimel, Macht, Krumhuberc, & Ellgring, 2006; Macht & Simons, 2000). Related, overweight is associated with depression (Petry, Barry, Pietrzak, & Wagner, 2008). Our taste preferences, in turn, are related to our affect and behavior. For example, people who are at high levels of stress or who are depressed, are more sensitive to sweet tastes, are more likely to report unpleasant tastes, and are more likely to be picky eaters (Dess, 1991; Dess & Edelheit, 1998).

TAS2R38. Preference or distaste for some flavors is genetically determined; knowledge about the genes responsible for specific sensitivities can tell us about a person's genetic profile without having to subject them to expensive genetic testing. Research has shown that people who are particularly sensitive to the bitter taste associated with phenothioureas, a group of about 40 compounds, share a genetic profile; one's ability to taste these compounds is due to a single gene (Fox, 1932). The association between this gene and tasting phenothioureas was first made by Fox (1932) due to a mistake in his lab; he was synthesizing phenylthiocarbamide and some of it flew into the air, Fox's colleague complained of how bitter it tasted, but Fox didn't taste anything. Others in the scientific community quickly latched on to the idea, as there were (are) few Mendelian markers, and finding one offered great utility as an organizing mechanism for the study of the genome (Bartoshuk, Duffy, & Miller, 1994; Wooding, 2006). It took 70 years for the specific gene to be identified; in 2003, Kim et al. determined that the *TAS2R38* gene is responsible for one's ability to taste phenothioureas. The finding has since been replicated multiple times and has led to the discovery of other genetic modifiers (Duffy et al., 2004; Hayes et al.; Mennella,

Pepino, Duke, & Reed; Reed et al., 2010). The identification of *TAS2R38* has resulted in the initiation of new studies focused on phenotypes related to *TAS2R38* and surrounding genes, rather than to taste sensitivity alone (Duffy, Davidson, et al., 2004; Duffy, Peterson, & Bartoshuk, 2004; Wooding, 2006).

Those with a dominant allele *T* on *TAS2R38* will be able to taste the phenothiourea compounds; two dominant *T*s will result in a person being a *supertaster*, someone who is extremely sensitive to phenothioureas. A single *T* will be a *medium taster*, someone who can taste phenothioureas, but for whom it is not extremely unpleasant. People, who have two recessive *t*s are known as *nontasters*; they cannot taste phenothioureas (Bartoshuk, et al., 1994; Joiner & Perez, 2004). The compounds used most frequently to test for tasting are phenylthiocarbamide (PTC) and 6-n-propylthiouracil (PROP). Recently, PROP has gained favor over PTC because it has no aroma, and it is used as a treatment for hyperthyroidism, so it has been tested for safety by the FDA (DiCarlo & Powers, 1998; Duffy et al., 2010). Studies using the method of supertaster identification described in the present study have consistently shown that about 25% of the population are supertasters, 25% are nontasters, and the remaining 50% are medium tasters (Bartoshuk, et al., 1994; DiCarlo & Powers, 1998; Wooding, 2006). However, it is acknowledged that these groupings may be somewhat arbitrary (Bartoshuk, et al., 1994; B. J. Tepper, 2008).

Food preference. Supertasters tend to be characterized as picky eaters; contrary to early beliefs, their taste sensitivity extends beyond bitter flavors (Bartoshuk, et al., 1994). They tend to prefer fatty foods and bland flavors. Previous research has linked supertasting to low consumption of vegetables, due to the often bitter flavors associated with greens, and to dislike of alcohol (DiCarlo & Powers, 1998; Duffy, Davidson, et al., 2004; Duffy, et al.,

2010; Duffy, Peterson, et al., 2004). Interestingly, some prime sources of omega-3 fatty acids are green, leafy vegetables and fish – flavors that supertasters find particularly off-putting; omega-3 deficits have been hypothesized as a risk factor for mood disorders (Hakkarainen et al., 2004; Logan, 2004; Montgomery & Richardson, 2008; Parker et al., 2006). Supertasting has also been linked to a number of physical health issues, including cancer and cardiovascular disease (Duffy, 2007). Finally, since the 1960s, there has been research linking one's taste sensitivity to a range of other traits, including personality, psychopharmacologic reactions, and smoking behaviors (Bartoshuk, et al., 1994; Mascie-Taylor, McManus, MacLarnon, & Lanigan, 1983). Each of these correlates have also been studied in relation to bipolar disorder (Bagby et al., 1997; Hasler, et al., 2006; Kandel et al., 1997).

Evolutionary role. The reason for the relation between taste sensitivity and other traits is not known, but one theory, rooted in evolution, has gained support. People sensitive to bitter tastes tend to exhibit an automatic, physical reaction to bitter substances. This is true from infancy through adulthood, and cannot be masked initially, though a more socially-acceptable response usually follows (Greimel, et al., 2006). It is thought that the reason for this physical manifestation of "yuck" is to provide a way by which approach and avoidance messages, regarding food, can be communicated. Before foods were processed, when one might encounter an unknown green in the wild, it would have been adaptive to be very sensitive to bitter tastes – poisons often share a flavor profile with the phenothiourea compounds (Boyd, 1950; Wooding, 2006). This theory also explains the generally picky eating found among supertasters – "when in doubt, spit it out" (Dess, 1991). Finally, it may also offer some insight into the preference for fatty foods (Tepper & Nurse, 1998; Yackinous

& Guinard, 2002), since fats are a dense source of easily-stored calories, and would have been beneficial to consume in times when food was not always easily accessible. Though this evolutionary adaption now results in a dislike of many vegetables and a preference for fatty foods, the extreme response to potentially-threatening foods could have been protective in another era.

Mood

A hypothesis, to be explored in the present study, is that supertasting represents a heightened sensitivity, not only to foods, but also to other environmental stimuli. People who are especially sensitive are more likely to react strongly to emotional stimuli, and these reactions may then influence subsequent interactions and reactions, creating something like a domino effect, whereby one's mood and emotional state are highly affected (out of proportion to any one stimulus). This may come across as moodiness and irritability, both characteristics of supertasters (Joiner & Perez, 2004), and eventually, act as a trigger to an episode of disordered mood. Dess (1991) also makes an argument for the relation between mood and eating more generally, saying that food is inextricably linked to our social and emotional development, and that our preferences and relationship to food is the product of a complicated interplay between genes and experience – not unlike the development of disordered mood. In order to begin fleshing out the link between supertasting and disordered mood, the question of *whether or not taste sensitivity is related to emotional sensitivity* must be answered first.

Studies have shown that depressive symptoms are more common among people who are highly sensitive to bitter tastes, specifically those associated with compounds such as phenylthiocarbamide (PTC) and 6-n-propylthiouracil (PROP) (Dess & Chapman, 1990; Dess

& Edelheit, 1998; Whittemore, 1986) (cf. Jones, 2009). The ability to taste these compounds is genetic; people with the dominant *T* allele of the *TAS2R38* gene can taste the flavor, whereas those without this allele cannot. Mood disorders are also highly heritable. Among family members of people affected, the rate of bipolar disorder is five times higher than the general population (Hodgins, Faucher, Zarac, & Ellenbogen, 2002; Tsuchiya, Byrne, & Mortensen, 2003). Similarly, the rate of depression among family members of people with major depression is three times higher than otherwise expected (Sullivan, Neale, & Kendler, 2000). The heritability of mood disorder is nonspecific, meaning that family members of people with bipolar disorder are more likely to have not only bipolar disorder, but are also at much higher risk for depression and other psychiatric disorders (DelBello & Geller, 2001). The genetics of mood disorder are more complicated than that of supertasting, which is due to a single gene, but the transmittal of the supertasting allele may be a way to measure a portion of genetic risk, in lieu of a more specific genetic test for mood disorders.

Taste sensitivity has also been explored in relation to anxiety disorders, but a relation was not found, suggesting that PROP tasting may be a marker specific to mood disorders (Heath, Melichar, Nutt, & Donaldson, 2006). Still, not all supertasters suffer from mood disorders, and not all people with mood disorders are supertasters; the presence of the supertasting gene may indicate a specific subtype of disordered mood, which could have important implications for treatment. Specifically, some research has indicated that the presence of the supertaster gene, in people suffering from depression, is associated with more familial depression, perhaps indicating a more heritable form of the disorder (Whittemore, 1990) (cf. Joiner & Perez, 2004). In a study of supertasting and depression, an effect of tasting was found, such that depressed tasters reported an earlier age of onset than depressed

nontasters (Whittemore, 1990). This result has not been further explored, but if replicated, could offer a valuable way of aiding diagnosis in people with early onset mood disorder. The symptoms of mood disorder are often difficult to diagnosis in young people, and there has been a consistent call for objective measures of disordered mood in young people (Mascie-Taylor, et al., 1983). In other studies, familial mood disorder has been associated with an earlier age of onset (Faraone, et al., 2003); therefore, in order to investigate *whether or not supertasting might be associated with a more heritable subtype of mood disorder*, age of symptom onset and family history of psychiatric illness will be assessed in the present study.

Alcoholism

Comorbid alcoholism and depression is pervasive in the community (Grant & Harford, 1995), which seems to contradict the fact that supertasters are more likely to be depressed than nontasters, but less likely to be alcoholics (Whittemore, 1986). However, if supertasting is associated with a specific subtype of mood disorder, it may help to explain individual differences in comorbidity. DiCarlo and Powers (1998) conducted a study on alcoholism, depression, and supertasting; they found that among people who were alcoholics, or had a family history of alcoholism, nontasters were most common. Among people who were depressed, or had depression in their families, tasters were more common, but the group with the highest prevalence of supertasters was the group who had symptoms and/or a family history of *both* alcoholism and depression. The authors conclude that there may be two genetically-distinct types of alcoholism. The same argument may be made for depression; based on these results, it may be that that there are genetically distinct types of depression that can be differentiated by their tasting profile. Specifically, among people who are depressed, those who are supertasters may be more likely to use alcohol (and perhaps food /

other substances) to alleviate their mood symptoms, resulting in comorbid alcoholism and depression. Though this hypothesis has not been directly investigated, there are other data that offer reason to explore this: women are more sensitive to tastes than men (Bartoshuk, et al., 1994), are more likely to have comorbid depression and disordered eating, and often have comorbid depression and alcoholism (Bartoshuk, et al., 1994; Dawson & Grant, 1998; DiCarlo & Powers, 1998). If this hypothesis were supported, it could have important implications for diagnosis and treatment.

Mania

Research on eating behaviors and mood has consistently found a relation between negative mood and cravings and eating behaviors, but positive mood does not seem to affect appetite in the same way (Macht & Simons, 2000). However, research on bipolar disorder (or mania) and its relation to supertasting, or eating / appetite more generally, has not been done; therefore, a relation between supertasting and symptoms of mania cannot be assumed. However, there are some data that suggest this is a relation worth investigating. Studies have found an impact of dominance, an emotion associated with mania, on taste and eating (Dess, 1991; Dess & Edelheit, 1998); specifically, people who feel dominant are more sensitive to bitter tastes during stressful situations. Impulsivity, defined broadly as rash action based on reward-seeking without consideration of consequences, is also a key symptom of mania (Carlson & Kashani, 1988; Holmes et al., 2009; Peluso et al., 2007; Swann, Pazzaglia, Nicholls, Dougherty, & Moeller, 2003), and is often implicated in alcohol use and binge eating disorders (Dawe & Loxton, 2004; Rosval et al., 2006); people who have trouble with alcohol or drugs often exhibit poor insight for consequences, and a preference for smaller, immediate rewards than larger, longer term rewards. Similarly, those with bulimia or binge

eating disorder, tend to experience an inability to resist food cravings, a failure to appropriately weigh consequences, and a loss of control when binging. This type of impulsivity, with its focus on immediate rewards, may also be related to previous research suggesting that supertasters are more pleasure-seeking than non-tasters (Joiner & Perez, 2004). Again, there may be a subgroup of people for whom there is a problematic relation between mood and taste that can play out in multiple ways.

Perhaps a more compelling argument for the exploration of the relation between taste and mania is that, when specific depressive symptoms have been explored across tasters and non-tasters, differences that may be indicative of bipolar disorder emerge. Specifically, symptoms associated with atypical depression, often thought to be a prodrome to bipolar disorder (Perugi, Toni, Travierso, & Akiskal, 2003; Stewart et al., 2006), are more common and more severe among tasters than non-tasters (Whittemore, 1990). One of the primary symptoms of atypical depression is craving for dessert foods. Research has shown, across both animal and human models, that some individuals experiencing depressive symptoms will crave sweet and/or fatty foods and often "self-medicate" by indulging in desserts and other sweets (Willner et al., 1998). Interestingly, though the craving for sweets increases, the subjective pleasure of eating the sweet does not, which can lead to over-eating, as the person tries, unsuccessfully, to satiate their craving. The relation between negative emotion and using food as an emotion regulation technique is strong; in previous studies, it has held regardless of individuals' gender, body weight, or dietary restraint (Macht & Simons, 2000). Furthermore, this association is found in both clinical and community samples; and, though it has not been linked to supertasting, it seems possible that an association exists, given supertasters' preference for foods high in fat and sugar (Bajec & Pickering, 2010). If this

preference translates to craving, it may be that supertasters are more likely to experience atypical depression and, therefore, are at greater risk for bipolar disorder.

In addition to the symptoms of atypical depression, people who are supertasters and suffer from depression are more likely to have a prolonged course of illness (greater than two years) and to suffer from more severe symptoms (Whittemore, 1990). Early onset depression and recurrent episodes are both signs that a depression may later convert into a bipolar spectrum disorder (Strober et al., 1995).

Neurotransmitters

Other studies have looked at the mood disorder-taste relation a different way, linking neurotransmitters known to be implicated in mood and anxiety disorders; serotonin (5-HT) and noradrenaline (NA) specifically. The rationale for this line of investigation comes from the fact that human taste cells both release and absorb 5-HT in response to different tastes, and both 5-HT and NA influence taste cell excitability (Heath, et al., 2006). In an innovative study, Heath et al (2006) investigated the effect of two antidepressants (a serotonin reuptake inhibitor and a noradrenaline reuptake inhibitor) on taste sensitivity. The results showed that both agents increased taste sensitivity in participants. This is important because it provides foundation for other theories linking taste and mood. Interestingly, the authors found evidence that tasting of very bitter tastes (like PROP) may occur through an additional pathway as well as through the one that sweet, umami, and average-range bitter tastes are transmitted; previous research has suggested this as well (Dotson, Roper, & Spector, 2005), indicating that PROP tasting may be a viable marker *whether or not* an individual is taking psychopharmaceuticals, and that the ability to taste bitter compounds is evolutionarily important enough to be supported through multiple taste systems. This could be valuable to

diagnosis, as people may be medicated with psychotropic drugs before they are assessed for bipolar, which can affect other characteristics associated with the disorder including mood, sleep, and even brain anatomy (Chang, et al., 2005; Pfeifer, Welge, Strakowski, Adler, & Delbello, 2008).

Bipolar Spectrum and Supertasting

Though bipolar disorder and depression are diagnostically distinct, there is evidence to support the idea of a spectrum of mood disorders, without clear boundaries (Akiskal et al., 2000; Angst et al., 2010; Angst, Gamma, Benazzi, et al., 2003; Goto, et al., 2010; Perugi, et al., 2003; Youngstrom, Van Meter, & Perez Algorta, 2010). In fact, many people with bipolar disorder will experience significant episodes of depression, and a commonly-held belief in the field is that bipolar disorder is often misdiagnosed as depression, due to poor assessment of hypomanic symptoms (Akiskal, et al., 2000; Angst, et al., 2010; Angst, Gamma, Benazzi, et al., 2003; American Psychiatric Association, 2002). Though the research on supertasting and mood is limited to depression, there is good reason to hypothesize that the relation between supertasting and bipolar might, in fact, be stronger. The two most significant reasons are the relation between supertasting and emotional response, which is exaggerated in bipolar disorder, and the relation between supertasting and symptoms of atypical depression that are seen more often in people who also experience periods of hypomania or mania. The exploration of supertasting, in relation to the spectrum of mood disorders, including hypomania and bipolar disorder, is a novel addition to the field.

Emotion Reaction

In animal studies, sensitivity to bitter tastes is related to increased emotional reactions to (non-taste) threatening stimuli (Dess & Minor, 1996; Macht & Mueller, 2007). Similarly,

studies of humans have shown a relation between supertasting and tension, stress, and depressive symptoms (Dess & Chapman, 1990; Macht & Mueller, 2007; Mascie-Taylor, et al., 1983; Whittemore, 1986). A growing body of evidence suggests that supertasting is related to a greater readiness to respond to the environment (Macht & Mueller, 2007), an emotional "hair trigger."

In an important study on the relation between tasting status and emotional reactivity, Macht and Mueller (2007) found that supertasters showed the strongest emotional response to an anger-inducing film clip, when compared to medium tasters and non-tasters. Smaller effects were found for other negative emotions, including fear, tension, and sadness. The findings were consistent for both genders. This result is particularly interesting because it lends credibility to the theory that there is an evolutionary reason for supertasters' reaction: these individuals are more reactive to emotional stimuli, and may react particularly strongly to negative stimuli across senses. If the same pattern were found among people with mood disorders, it would support the hypothesis that sensitivity to emotional stimuli encountered in every day life may have a greater impact on those at risk for, or diagnosed with, mood disorders. There is, in fact, a body of research that suggests that stress is a significant risk factor for the onset and maintenance of mood episodes (Cohen, Hammen, Henry, & Daley, 2004; Dienes, Hammen, Henry, Cohen, & Daley, 2006; Garnefski, Kraaij, & Spinhoven, 2001; Johnson & Miller, 1997; Tsuchiya, Byrne, & Mortensen, 2003).

Previous research has found that mood induction tends to be more effective in people with bipolar disorder than in controls; the mood induced is rated more strongly, and lasts longer (Roiser et al., 2009). Additionally, mood induction, in euthymic people with bipolar disorder, tends to result in increased impulsivity and poorer judgment (Roiser, et al., 2009).

This suggests that whether or not a person is currently in a mood episode, those with bipolar tend to have stronger reaction to emotional stimuli, which may result in negative consequences. The hypothesis is that *stronger emotional reactions seen in people with disordered mood may be attributed, in part, to an overall sensitivity, including taste*.

Brain structure and function

Amygdala. Though reactions to all emotional stimuli may be related to one's taste sensitivity, most compelling is the likely relation to negative, threatening stimuli. Data from multiple fields of research, including mood disorder, schizophrenia, emotion regulation, and aggression, implicate the amygdala as the brain's threat response center, responsible for mood regulation, emotional memory, and rage reactions (Altshuler, Bartzokis, Grieder, Curran, & Mintz, 1998). Across the lifespan, the amygdala shows both structural and functional abnormalities in people with mood disorders (Blumberg et al., 2005; Chang, et al., 2005; Chang, et al., 2008; DelBello, et al., 2004; Dickstein et al., 2010; Kalmar et al., 2009; Pfeifer, et al., 2008). Interestingly, a recent study showed a strong association between the 5HTTLPR allele (serotonin, like that related above to taste sensitivity) and extreme amygdala response to perceived threat (Hariri, Tessitore, Mattay, Fera, & Weinberger, 2002). The authors describe a theory in which the heightened threat response, coupled with environmental stress, leads to overstimulation of the amygdala, resulting in additional abnormal function. This process maps nicely onto the phenomenon of exaggerated reactivity to stress and consequential mood dysregulation described above. Interestingly, in rat studies, manipulation of the amygdala, (by altering levels of noradrenaline and serotonin) resulted in altered food preferences among rats, and in impairment of a previously learned taste aversion. These results indicate that the amygdala plays an important role in the preference

for novel vs. familiar foods, and in one's ability to learn that ingesting certain foods is associated with sickness, providing further evidence that similar systems may be involved in taste and mood (Borsini & Rolls, 1984).

Amygdalar function can be measured using fMRI and could offer clues about an individual's risk for mood disorder (Chang, et al., 2008; DelBello, et al., 2004; Kalmar, et al., 2009), but it is expensive and impractical as a diagnostic aid currently. However, if an association can be made between amygdala dysfunction and taste sensitivity, which is easy and inexpensive to measure, it could have significant utility.

Limbic system. The amygdala is not the only region of the brain that is related to both taste and mood; relative to the other four senses, the gustatory system is reflected in parts of the brain typically responsible for motivation, emotion, and autonomic processes (Dess & Edelheit, 1998; Norgren, 1985). The primary area of reception for the taste system is the limbic system; the termination points for nerves of the gustatory system are the amygdala, thalamus, and hypothalamus (Norgren, 1985). The hypothalamus controls both hunger and thirst, it affects how much we eat and when we eat. Lesions of the hypothalamus may result in uncontrolled hunger, or cessation of eating. The hypothalamus is also the center of other body systems implicated in mood disorders. Of these, the hypothalamic control of our circadian rhythms is most important. People with bipolar disorder often experience sleep disturbance coinciding with mood episodes (Geller et al., 2002; Harvey, Mullin, & Hinshaw, 2006), or even chronic circadian rhythm instability, and are sensitive to environmental and other influences on their sleep patterns (Harvey, et al., 2006; Hasler, et al., 2006). This disruption may be caused by a mutation of the CLOCK gene, which studies have shown to be

related to both circadian rhythm instability and bipolar mood recurrence (Benedetti et al., 2003).

The thalamus is responsible for taste detection and recognition (Reilly, 1998), it is implicated in learned taste aversion, and food-related behaviors (e.g., seeking or saving food in the face of an anticipated shortage). Based on the theory, described above, that supertasting may be an evolutionary artifact related to toxin avoidance, the importance of the thalamus' roles of learning preferred or aversive foods, along with planning for necessary sustenance are evident. In addition to its role in the several sensory systems, the thalamus is thought to play a crucial role in the communication between the midbrain and the cerebral cortex; in bipolar disorder, the theory is that there is overactivation of subcortical regions, including the thalamus, and insufficient control by the cortex, which leads to dysregulation of mood and emotion (Blumberg et al., 2003; Chang et al., 2004; Chang, et al., 2008; Hariri, et al., 2002). Laboratory studies of the thalamus and its role in taste typically destroy the thalamus' function through lesions, whereas in people with bipolar disorder, *increased activation* of the thalamus is often found during mood (both depressed and manic) episodes (Strakowski, DelBello, & Adler, 2005). So, though the thalamus is implicated in both the mood and gustatory systems, current research methods do not accommodate direct testing of the relation.

If there is an abnormality in any of these brain regions, the amygdala, hypothalamus, or thalamus, it is unlikely that it would affect only one system. The fact that taste is governed by some of the same mechanisms that are thought to be responsible for the more "typical" characteristics of bipolar disorder supports the theory that the gustatory system may provide

a way by which to assess and better understand brain abnormalities implicated in bipolar disorder.

Behavioral inhibition and behavioral activation systems

Previous research has shown that tasting sensitivity may interact with other traits, such as personality and temperament (Dess & Edelheit, 1998; Mascie-Taylor, et al., 1983), to make individuals more reactive to emotional stimuli and stress. Perhaps related is evidence suggesting that for people with a temperament characterized by arousability and dominance, stress increases sensitivity to bitter tastes (Dess & Edelheit, 1998). This gives some credence to the theory that trait sensitivity, which may also manifest in arousability, can lead to heightened reactivity in the face of stress (as one would expect from a threatening stimuli). The behavioral activation (BAS) and behavioral inhibition (BIS) systems are also implicated in excessive reactivity; both are often found to be dysregulated in people with mood disorders. Specifically, dysregulated BAS may lead to impulsivity and approach behaviors, whereas dysregulated BIS may lead to inappropriate reactions in the face of threat or stress (Alloy, et al., 2008; Johnson, Turner, & Iwata, 2003; Meyer, Johnson, & Winters, 2001). Though BIS and BAS have not been investigated in relation to supertasting, they are well-established relative to bipolar disorder and likely play a role in the overall relation between supertasting, emotional reactivity, and mood disorder.

The relation between supertasting and mood disorder likely lies in the "hair trigger" response described above and the subsequent inflated emotion. Imagine an individual who is naturally more arousable, and s/he is a supertaster, with the associated heightened reactivity. If s/he encounters an emotional stimulus, it will produce a reaction and increase his/her arousal level, thereby increasing the likelihood that, in the face of subsequent challenges, the

individual's reaction may grow out-of-proportion. Rather than an additive effect of multiple stressors, people who tend toward strong reactions may experience a more extreme non-linear increase in their emotion (Macht & Mueller, 2007). This potentially-rapid ramping up of emotion could have implications for the development of disordered mood. Another important factor is the fact that people with mood disorders, in addition to being highly emotional and reactive, often lack the ability to appropriately temper their reactions, leading to the prolonged, extreme moods that characterize the disorder (Henry, 2010; Hlastala et al., 2000; Meyer, et al., 2001).

Emotion regulation

Emotion regulation (ER) plays a key role in the maintenance of euthymic mood; people with mood disorders (depression and bipolar disorder) consistently show mastery of fewer emotion regulation strategies, and a tendency to have poorly regulated emotions (Angst, Gamma, & Endrass, 2003; Dickstein, Brazel, Goldberg, & Hunt, 2009; Green, Cahill, & Malhi, 2007), regardless of mood state (Gross, 1998). Dysfunctional emotion regulation is thought to develop as a result of many of the same factors that contribute to mood disorder, including both heritable, internal traits and external risk factors (Angst, Gamma, & Endrass, 2003; Calkins, 1994; Silk, 2006). One reason people with mood disorders tend to have poor emotion regulation may be that they were not taught good emotion regulation strategies (perhaps due to parental mood disorder)(Calkins, 1994). Another contributor to emotion regulation is the amygdala, in conjunction with the pre-frontal cortex. As described above, people with mood disorders tend to have abnormal amygdalar size and function (Blumberg, et al., 2005; Chang, et al., 2005; Eippert et al., 2007; Hariri, et al., 2002; Leppanen, 2006). So, in addition to experiencing more extreme emotions,

people with mood disorders are less able to effectively moderate those responses, which may lead to other problems, including interpersonal challenges and mood episodes (Yap, Allen, & Sheeber, 2007).

Emotion regulation occurs at multiple levels, within a number of different systems. As a result, there are many ways to measure one's ER. For the present study, in which participants are presented with an anticipated stimulus, against which they might be expected to use cognitive methods of emotion regulation, a self-report measure of emotion regulation strategies, the Cognitive Emotion Regulation Questionnaire (Garnefski, Kraaij, & van Etten, 2005; Jermann, Van der Linden, d'Acremont, & Zermatten, 2006), will be used to assess participants' repertoire and mastery of a set of common emotion regulation strategies.

Emotion regulation should moderate an instinctual overreaction to an emotional stimulus, but if one had poor emotion regulation, the reaction could be explosive. It is not hard to see how the unique characteristics described above – arousability, tendency toward extreme reactions to threat, and an inability to temper emotion – could create a kind of "perfect storm" to initiate disordered mood.

Objective

The broad aim of the present study is to determine whether or not taste sensitivity has utility as a biomarker for propensity towards disordered mood and, if so, whether emotional reactivity to threat and the regulation of that reaction show promise as the mechanism by which disordered mood develops.

The exploration of the relation between supertasting and mood disorder is not going to reveal a direct causal relationship; not everyone who is a supertaster will have disordered mood, and not everyone with disordered mood will be a supertaster, but the relation may

provide important information about a specific vulnerability that could inform future prevention and intervention work. In addition, if people who are both supertasters and suffer from disordered mood are found to be different from others on measures of age of onset and / or family history of psychiatric disorder, this could be an important step toward identifying a subgroup of people for whom sensitivity to the environmental and genetic risk translates more often into psychopathology.

Hypotheses.

- Supertasters will have an earlier age of onset of emotional or behavioral problems than medium and non-tasters.
- Supertasters will have a higher rate of familial psychiatric disorder than medium and non-tasters.
- Supertasters will have higher BDI scores than medium and non-tasters.
- Supertasting will predict heightened response to the threat mood induction.
- Heightened response to the mood induction will predict increased mood symptomatology.
- People who are supertasters will be less adept at regulating their emotions.
- The relation between supertasting and mood induction response will be moderated by emotion regulation, such that those who are proficient emotion regulators will react less than predicted by their tasting status.
- The relation between threat reactivity and mood symptomatology will be moderated by emotion regulation, such that high scores on the measure of emotion regulation will be associated with less mood symptomatology than predicted by threat response.

Method

Participants

Participants (*N*=499) were undergraduates at the University of North Carolina at Chapel Hill. The study was advertised on the UNC Human Participation in Research website as an opportunity to participate in a study examining individual differences in taste and mood. Participants received course credit in their psychology class in exchange for their participation. Participants' average age was 19 years, 63% were female. Seventy-one percent of participants identified as Caucasian, 13% as African or African American, 11% as Asian, 1% as Native American, and 4% identified as "Other."

Studies often come under criticism for the use of college students in investigations of psychopathology (Coyne, 1994; Gotlib, 1984; Vredenburg, Flett, & Krames, 1993). The present study is not about mood disorders per se; it is about the relation between disordered mood and supertasting. There is no reason to think that students are less likely to be supertasters, or are at lower risk for mood disorder than the population at large (Blanco et al., 2008; Buckle, 1972) and, in other undergraduate studies, participants meeting research criteria for bipolar disorder have been reliably recruited through two-stage subject pool studies (Alloy, et al., 2008; Shen, Alloy, Abramson, & Sylvia, 2008) or through university mental health services (Stangler & Printz, 1980). Additionally, in community studies, bipolar disorder and subthreshold bipolar disorder is prevalent among young adults (Lewinsohn, Seeley, Buckley, & Klein, 2002; Merikangas et al., 2007).

Measures

Study questionnaires were administered online using Qualtrics (Qualtrics Labs Inc., 2009). The order of presentation for the questionnaires was randomized to reduce the potential for confounding effects created by priming or other ordering effects. Computer administration facilitated data management and decreased opportunities for entry mistakes and misplaced data. In addition, given the sensitive nature of some of the questions, computer administration has shown to elicit more honest, less distorted responses from participants than traditional interview formats (Evan & Miller, 1969; Richman, Kiesler, Weisband, & Drasgow, 1999).

Beck Depression Inventory (BDI). The BDI (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) has been used for almost 50 years as a reliable method for both detecting and measuring the intensity of depression (Beck, Steer, & Carbin, 1988). The BDI was developed based on clinical observations. It consists of 21 items, each indicating a specific symptom or attitude. The items are rated on a 0-3 point scale to provide information regarding the intensity of the symptom or attitude. Scores are produced by summing the 21-items (for the present study, the item assessing for suicidal thoughts was omitted). Scores less than 10 indicate no or minimal depression, mild to moderate depression is 10-18; moderate to severe depression is 19-29; and severe depression is 30-63. The BDI has shown good internal consistency, in a meta-analysis of 25 studies, the mean coefficient alpha for nonpsychiatric populations was .81, for psychiatric populations .86 (Beck, et al., 1988). In addition, a meta-analysis of 35 studies looking at validity found a mean correlation between clinical ratings and the BDI for psychiatric patients of .72, for nonpsychiatric patients .60. Similarly, in our sample, the reliability was good, Cronbach's alpha .88.

Hypomanic Checklist (HCL-32). The HCL-32 was developed primarily as a self-report to help clinicians identify hypomanic symptoms in patients presenting with depression. It consists of 32 questions regarding symptoms of hypomania, to which individuals respond "yes" or "no." In an international validation study of three diverse populations, the HCL-32 had a reliability score of .82 (Cronbach's alpha). In our sample, the reliability was comparable, Cronbach's alpha .80. The HCL performed moderately well at discriminating between participants with MDD and those with bipolar disorder with an area under the curve of .74. Factor analysis revealed two classes of hypomanic symptoms, an active/elated factor and a risk-taking/irritable factor. This is comparable with the structure found in a study of an earlier 20-item version of the HCL (Hantouche, Angst, & Akiskal, 2003) and that associated with the hypomanic symptoms assessed by the Mood Disorder Questionnaire (Benazzi & Akiskal, 2003).

Age of onset. In order to investigate whether or not tasters, who self-identify as having struggled with mood or emotional problems, experienced those difficulties earlier than non-tasters (perhaps indicating a subtype of the disorder) the following questions were posed, following the administration of the BDI and HCL:

- Have you ever seen a mental health professional (psychologist, psychiatrist, counselor) for concerns about your emotions or mood?
- If so, how old were you when you first saw a mental health professional?

Additionally, if the person answered 'yes,' 'sometimes,' 'frequently,' or 'all the time,' to any question on the BDI or HCL, they were routed by Qualtrics to answer these additional questions:

- How old were you when you first felt that your mood or emotions became difficult for you?
- How old were you when someone else first noticed or commented that your mood or emotions might be difficult for you (e.g., how old were you when someone asked or suggested that you might be depressed, or suggested that your mood might be unusually high)?

Cognitive Emotion Regulation Questionnaire (CERQ). The CERQ is a reliable and valid method by which to measure individual differences in emotion regulation strategies (Jermann, et al., 2006). The CERQ consists of nine distinct scales – self-blame, other-blame, acceptance, refocus on planning, positive refocusing, rumination, positive reappraisal, putting into perspective, and catastrophizing. The scales with higher scores indicate which cognitive strategies the participant uses most; the scales represent trait emotion regulation strategies, not the specific strategies used during the research session or in some other specific situation. High scores on self-blame, rumination, and catastrophizing are associated with increased risk of psychopathology (e.g., I feel that I am the one to blame for it), high scores on positive refocusing and positive appraisal are thought to be protective (e.g., I think that other people go through much worse experiences) (Garnefski, Kraaij, & Spinhoven, 2002). For the purposes of this study, scores on the negative scales (self-blame, rumination, and catastrophizing) were summed to create a negative index score, similarly, the positive strategy subscales (positive refocusing and positive appraisal) were summed, and the remaining subscales (other-blame, acceptance, refocus on planning, putting into perspective) were summed. The three resulting index scores – negative strategy, positive strategy, and

neutral strategy – will be used in analyses. The reliability (Cronbach's alpha) of these three scales, in our sample was .82, .83, and .81 respectively.

The measure has been tested on participants ranging in age from early childhood through adulthood and shows consistent reliability and validity (Garnefski & Kraaij, 2006a; Garnefski, et al., 2005). In addition, it can identify differences due to symptoms of psychopathology in nonclinical samples (Garnefski & Kraaij, 2006b; Jermann, et al., 2006). Specifically, mood disordered participants score more highly on the self-blame, catastrophizing, and rumination scales than healthy controls (Garnefski & Kraaij, 2006b; Garnefski, et al., 2005).

Family Index of Risk for Mood (FIRM). The FIRM (Perez Algorta et al., 2011) is intended to provide a simple method by which information about study participants' family history of mental health issues can be gathered without burdening participants or attempting to collect excessive details that might be of questionable reliability. The FIRM was validated in a pediatric bipolar study of 162 families. Parents were presented with an array of questions about mental health history (suicide, depression, mania, hospitalization, or substance use) for each of several relatives (youth's grandparents, parents, aunts/uncles, siblings, or children), resulting in a total of 25 checkboxes that a respondent could endorse. The brief family history items were well tolerated by families. The data collected showed a strong relation with youth diagnoses of pediatric bipolar disorder and the family history information provided incremental validity when predicting bipolar diagnoses, even after controlling for other information provided by the same informant.

The Brief Young Adult Alcohol Consequences Questionnaire (BYAACQ). The BYAACQ (Kahler, Strong, & Read, 2005) assesses both alcohol consumption and

consequences among young adults. The 24-item scale was derived from a longer version using item response theory to comprehensively an efficiently capture the continuum of alcohol problems without gender bias. The scale includes items about consumption (e.g., "How often in the past 90 days have you been drunk (not just a little high) on alcohol?") and its consequences (e.g., "I have driven a car when I knew I had too much to drink to drive safely"). Response patterns reliably order participants, with a Rasch model person reliability estimate of 0.82. The Rasch model–based index may be interpreted similarly to other methods of internal consistency, like Cronbach's alpha, which was .90 in the present sample.

Mood rating scale. There is an active field of research on the best way in which to measure subjective human experiences, like mood. One method, the visual analog mood scale, strikes a nice balance between ease of use and ability to capture individual differences. It has been used successfully in many previous studies assessing the impact of a mood induction (Roiser, et al., 2009; Standage, Ashwin, & Fox, 2010). In the present study, each participant was presented with a question regarding their current mood, (e.g., "How fearful do you feel right now?") and used a visual measure (a bar that can be completely or not at all filled in) to indicate how much they felt the specified emotion from "not at all" to "very" (Huntsinger, Sinclair, Dunn, & Clore, 2010). Ratings were based on the length (left to right) of the portion of the bar that is filled in, and were quantified automatically by the Qualtrics software

Demographics. Participants were asked about the following demographic characteristics: age, sex, race and ethnicity, and whether or not they smoke (due to the effects of smoking on one's ability to taste).

Procedure

Participants signed-up to participate in the study using the University's Human Participation in Research website. They came to the study lab at a designated time and were told not to eat, drink, chew gum or smoke for at least one hour prior to their session.

Upon arrival at the lab, a trained research assistant guided participants through the informed consent procedure. They were told that their participation was optional, and that they could quit at any time without penalty. Participants were told that their participation would consist of completing a number of questionnaires, watching brief film clips, tasting a test strip, and having their tastebuds counted (by noninvasive method). Participants were told that the test strip was soaked in a chemical compound that has been tested by the FDA and is completely safe. If participants chose not to taste the paper, they were allowed to participate in the rest of the study; no one elected not to taste the paper.

Once informed consent was complete, participants were set-up at a computer and asked to complete the questionnaires on Qualtrics. The BDI and HCL were administered first, the order was random to avoid ordering effects in the results. Following the BDI and HCL, participants answered the appropriate Age of Onset questions, followed by the CERQ and the FIRM.

The mood induction was next. The induction of a particular mood in participants has been the basis for many experiments, and there are many processes that have been used to do so. Some of these methods, and the effectiveness and validity of mood induction in general, have come under criticism. In order to evaluate the effectiveness of different mood induction techniques and to compare their effect sizes, a meta-analysis was conducted (Westermann, Spies, Stahl, & Hesse, 1996). The results of this study found that the most effective mood

induction was achieved with the use of a movie clip, combined with the instruction for participants to *really notice* the intended mood. The average effect size for studies using this method was >.73 for both positive and negative moods (Westermann, et al., 1996).

In keeping with the results of the meta-analysis, a film-based mood induction was used in the study. Participants each watched two film clips; one was intended to induce a dominant / triumphant (positive) response, the other was intended to induce a threat / fear (negative) response. The clips were selected by the author and rated by an independent panel as the most effective for inducing the moods of interest out of a sample of clips.

The clips were presented using online media and were counterbalanced to help ensure that there were no ordering effects. Just prior to the first mood induction, participants rated their mood on the following dimensions:

- Fear ("How fearful do you feel right now?")
- Threat ("How threatened do you feel right now?")
- Anger ("How angry do you feel right now")
- Sadness ("How sad do you feel right now?")
- Happiness ("How happy do you feel right now?")
- Strong (strong will be used in lieu of dominance; previous work by this group suggests that dominance is a socially-unacceptable emotion and, therefore, less likely to be endorsed) ("How strong do you feel right now?")
- Triumph ("How triumphant do you feel right now?")
- Excitement ("How excited do you feel right now?")

Mood induction response was measured by the response indicated on the visual analog mood scale. The mood rating (length of VAMS bar) pre-induction was subtracted

from the post-induction length to determine change for each induction. This mood rating was repeated after each clip. Between inductions, participants were asked to complete a survey of demographic information as a distracter.

Supertasting was tested following the mood induction. The rationale for this sequencing is that supertasters are often reported to have a very strong, visceral reaction to the indicator paper. As such, it is possible that the experience of tasting the bitter compound would affect participants' mood and subsequent mood ratings. Importantly, previous studies have shown that the measurement of sensitivity to bitter tastes is robust across levels of mood, likely due to its importance evolutionarily (Greimel, et al., 2006).

In order to get an accurate measurement of participants' taste of the PROP compound, they were instructed not to eat, drink, chew gum, or smoke for an hour before their appointment. Their adherence to this instruction was asked about and noted (Y/N) in the database. Given that taste sensitivity was measured last, after participants had already been in the lab for nearly an hour, adherence to this rule would be unlikely to have influenced the results.

PROP tasting was determined using paper strips with 50 mmol/l concentration of 6-n-propylthiouracil (Bartoshuk, 2011; Zhao, Kirkmeyer, & Tepper, 2003). Participants were instructed to put the paper on their tongue and to allow it to become saturated with saliva. Rating of the intensity of the taste of the paper was conducted using a labeled magnitude scale (LMS). The LMS is a 100-mm scale with the left side rated "barely detectable" and the right side labeled "strongest imaginable." Previous studies of supertasting have reliably differentiated between taste groups using the LMS with PROP-soaked paper strips. Guidelines for distinguishing between taste groups based on the LMS scale differ somewhat

between studies; ratings of <17 mm are consistently associated with nontaster status, whereas supertaster status has been assigned at ratings of >51mm (Tepper, Christensen, & Cao, 2001), >71 (Zhao, et al., 2003) and >78 mm (Bartoshuk, 2011). Medium tasters fall between nontasters and supertasters (Bartoshuk, 2011; Tepper, et al., 2001; Zhao, et al., 2003). Because group assignment metrics vary, the more conservative estimate for supertaster of >78 mm, which was derived from the largest sample (*N*=5500), and using the same methodology as the present study, was used in analyses. Additionally, further investigation of taste status, including participants' physical reaction to the PROP strip was noted in order to investigate the relation between an instinctual, physical response and supertasting status.

Because some research has indicated that taste threshold is not always reliable as a measure of taste sensitivity, a secondary method of measuring supertasting was used in order to corroborate the PROP strip test result (Bartoshuk et al., 2004). This method involves painting the participant's tongue with blue food coloring. The dye colors the tongue, but not the tastebuds, so they can be easily counted. See Figure 1 (Utermohlen, 2010). Previous research has successfully distinguished between supertasters and nontasters based on a count of ≥25 papillae within a 6mm diameter circle on the anterior region of the tongue (supertasters) or ≤25 tastebuds (nontasters) (Duffy, Davidson, et al., 2004), or a mean of 98 tastebuds per cm^2 (supertasters), 73 per cm^2 (medium tasters), or 54 per cm^2 (nontasters) (Bartoshuk, et al., 1994; Snyder, Duffy, Marino, & Bartoshuk, 2008). In the present study, participants' tongues were swabbed with a Q-tip laden with blue food coloring. The participant was then instructed to place a piece of paper with a small hole (6mm) cut in it against the tip of their tongue. Two research assistants counted and recorded the number of tastebuds independently.

Following the determination of taste sensitivity, participants were debriefed. They were provided with a brief description of the study and its hypotheses and encouraged to ask any questions they might have. Participants were provided with the name and contact information for the principal investigator and faculty advisor, along with the Psychological Services Clinic, should they wish to discuss the study and / or any concerns about their own mental health.

Analytic plan

Scores from the BDI, HCL-32, and CERQ were converted into a percent of the maximum possible score (POMP). Converting summed scores into percents provides a framework by which to interpret differences (Cohen, Cohen, Aiken, & West, 1999). Scores on the mood induction and PROP intensity were already on a 0-100 scale. Detailed descriptions of the other analyses are included in the Results section.

Results

Preliminary Analyses

Eating, drinking, or smoking prior to the assessment of supertasting may affect the result. This was a relatively minor concern in the present study due to the fact that we assessed supertasting status at the end of the experiment. Participants were asked whether or not they smoke regularly, as well as "Have you eaten in the past hour?" "Have you had anything to drink, other than water, in the past hour?" "Have you smoked in the past hour?" Forty-five people reported being regular smokers, 67 reported eating in the past hour, 47 reported drinking, and 3 reported having a cigarette. The number of smokers per taste group was not different ($X^2(4)=3.6$, $p=.47$). All analyses were run excluding these people; the relations between variables did not change. See Appendix A.

Based on the taste group guidelines published by Bartoshuk (2011), 15% of the sample was defined as nontasters, 71% as medium tasters, and 14% as supertasters. The proportions of nontasters and supertasters are somewhat lower than expected (25% nontasters, 25% supertasters). Given the fact that the taste groupings based on PROP intensity ratings are somewhat arbitrary (Bartoshuk, 2011; Tepper, 2008), other methods of grouping were assessed and then compared to the Bartoshuk method on the primary study analyses. The alternatives explored were quarters based on study data (125 nontasters, 249 medium tasters, 125 supertasters), and two groups (209 nontasters, 290 supertasters) based on the observed bimodal distribution of the data (see Figure 2). Neither alternative was associated with different outcomes on the primary analyses, therefore, the Bartoshuk groups,

which have greater empirical support, were used in all subsequent analyses. Additionally, where appropriate, the PROP intensity rating was included as a continuous variable to detect whether or not sensitivity is associated with the outcomes of interest, regardless of group assignment. Though PROP intensity is not typically included as a continuous variable, research has shown that creating groups by using cut-off scores, in lieu of retaining a continuous variable, reduces power and can result in spurious relations (Cohen & Cohen, 1983; MacCallum, Zhang, Preacher, & Rucker, 2002).

As planned, the number of tastebuds counted was used as a test of the validity of taste groupings. Two research assistants counted each participant's tastebuds, the reliability between counts was good (intraclass correlation=.91); the average of the two counts was used. Based on the designation of nontasters having less than 25 tastebuds in a 6mm diameter circle and supertasters having more than 25 (Duffy, Davidson, et al., 2004), 326 people (65%) were designated as supertasters. This is much higher than the prevalence suggested by other studies, in which tastebud count and supertasting group were correlated (*rho*=.58, *p*=.05). Perhaps not surprisingly, the correlation between PROP intensity and number of tastebuds (*r*= -0.04, *p*=.34) was not significant in our sample. Number of tastebuds was only correlated with outcomes related to alcohol use. See Table 1 and Figure 3.

Next, the coding of participants' physical reaction to the PROP strip was compared with their ratings of the flavor intensity. Only 37 people were noted to have a physical reaction; regressing PROP intensity rating on physical reaction indicated that physical reaction was indicative of supertasting (*B*=22.49, *p*<.0001). However, not all people labeled as a supertaster had a physical reaction (14 out of 70) and in preliminary regression analyses, it was not related to outcomes of interest. Furthermore, with only 37 people, using physical

reaction, rather than PROP intensity, was not a viable alternative.

Due to the apparent lack of relation between the outcomes of interest and tastebud count and physical reaction, as well as the fact that most supertasting research – including research focusing on the serotonergic system – relies on reported taste sensitivity, rather than tastebud count or physical reaction, we decided to focus on PROP sensitivity and the groups thereby implied, rather than tastebud count or physical reaction, in the analyses.

There were no sex differences found between taste groups ($X^2(2)=4.38$, $p=.11$). See Table 2. Similarly, there were no age differences ($p=.95$). Some studies have found racial differences in the prevalence of supertasting, with higher estimates of supertasters in China, Japan, and Africa, and lower estimates in India (Tepper, 2008), but recent studies of American populations have suggested that race does not affect supertasting (Mennella, et al., 2010). In our sample, we found that people who identified as Caucasian were significantly less likely to be supertasters ($X^2(8)=18.73$, $p<.05$) than other racial groups.

Primary Analyses

Age of onset. In order to test the hypothesis that supertasters would have an earlier age of onset than medium and non-tasters, age of onset was determined by the younger of the two ages reported in the age of onset questionnaire (age at which concern was expressed or age at which services were sought). Forty percent of the sample ($n=199$) reported having problems related to their mood or emotion at some point. The average age of first concern was 12.76 years. There was no difference in age of onset between taste groups ($F(2,196)=2.04$, $p=.13$). See Table 3.

Twenty-one percent of the sample ($n=107$) reported having seen a mental health professional. Treatment-seeking did not vary between groups ($X^2(2)=1.85$, $p=.40$). See Table

3. Cox regression was used to determine whether or not there was a difference in age of treatment seeking ($X^2(1)=3.19$, $p=.07$). Substituting taste group with PROP intensity as a continuous predictor, the result remained nonsignificant. See Figure 4.

Family history. In order to test the hypothesis that supertasters have a higher rate of familial psychiatric disorder than medium and non-tasters, data from the FIRM were summed to determine the proportion of relatives affected by mental illness. See Table 4. Forty-two percent of the sample ($n=210$) reported a family history of mental illness. Chi-squared analysis was used to determine whether or not there was a difference in the presence (yes/no) of alcoholism, depression, bipolar disorder, psychiatric hospitalization, and suicide in participants' families; there were no differences across the taste groups (ps=.15-.84). Kruskall-Wallis was used to determine whether or not there was a difference in the number of affected relatives across tasting groups; there were no significant differences (ps=.16-.96). Additionally, given previous findings related to family histories of depression and alcoholism among supertasters, a combined score of family members with depression and/or alcoholism was compared across groups; no differences were found. Using PROP intensity as a continuous predictor in a regression analysis to assess whether taste sensitivity predicts overall familial psychiatric disorder, controlling for ethnicity, the result was not significant ($p=.64$). Because familial depression and alcoholism have been shown to relate to supertasting in previous studies, these were also assessed using PROP intensity as a continuous variable in regression analyses; PROP intensity was not a predictor of familial depression ($p=.44$) or alcoholism ($p=.32$).

Mood. Scores on the POMP-scored BDI ranged from 0 to 61.90, with an average of 9.35 (the range of raw scores was 0 to 39, with a mean of 5.89) To assess the hypothesis that

supertasters have higher BDI scores than medium and non-tasters, a one way ANOVA was used to assess differences in mean BDI scores across groups. The result indicated that supertasters had significantly higher scores than the other groups ($F(2,496)=4.41$, $p<.01$). Levene's Test of Homogeneity of Variances was also significant, indicating that the within group variances differed significantly; the nontasters and medium tasters had lower average scores, but also significantly less variability in BDI scores than the supertasters. Using Games-Howell post-hoc test, this result was no longer significant ($p=.08$). Additionally, examination of the distribution of BDI scores revealed seven extreme outliers (more than three times the interquartile range). See Figure 5. Repeating the ANOVA without these data points led to a nonsignificant result ($F(2, 489)=1.11$, $p=.33$). After eliminating the outliers, PROP intensity as a continuous variable predicting BDI scores, controlling for ethnicity, was not significant. See Figure 6.

Examination of the HCL POMP scores did not reveal any outliers. The scores ranged from 0 to 93.75 with an average score of 60.01. ANOVA was used to assess differences in mean HCL scores across taste groups; there were no differences in HCL scores ($F(2,496)=1.26$, $p=.29$). Similarly, regressing HCL scores on PROP intensity, controlling for ethnicity, PROP was not a significant predictor.

Alcohol. The sample was split by age for the analyses on alcohol use to account for the fact that most of the sample was under 21 and access to alcohol may affect their drinking behavior, independent of taste group. Alcohol consequence scores from the BYAACQ were summed and examined across taste groups. For the participants under 21 ($n=453$), there were no differences in the number of consequences ($F(2,450)=1.73$, $p=.18$) across taste groups. Participants 21 and over ($n=46$) also had equivalent consequence scores ($F(2,43)=.90$,

p=.42). Alcohol consumption was assessed using Kruskal-Wallis; for those under 21, supertasters drank less frequently ($p<.05$), consumed more than five drinks on fewer days per week ($p<.05$), and got drunk less often ($p<.05$). Supertasters over 21 also drank less frequently ($p<.05$), but did not differ on the number of days they drink more than five drinks ($p=.15$) or on the frequency they get drunk ($p<.30$). See Table 5.

Regressing alcohol consequences on PROP intensity in the younger age group, was not significant ($B= -.06$, $p=.09$). The same was true for the participants over 21 ($B= -.06$, $p=.66$). In previous studies (Driscoll, Perez, Cukrowicz, Butler, & Joiner, 2006), gender was found to moderate this relationship, such that female supertasters had more alcohol related consequences; in the present study, for participants under 21, being female predicted fewer consequences overall ($B= -4.26$, $p<.05$), but the interaction of PROP intensity and gender was not significant ($p=.76$) (Aiken, West, & Reno, 1991). The same held true for those over 21. Given the theory that people who are supertasters and have symptoms of depression might be more likely than non-depressed supertasters to use alcohol, alcohol use (sum of z-scored consumption scores) was regressed on PROP intensity and BDI scores, and on the interaction of these two terms. PROP intensity was a significant predictor of alcohol use in those under 21 ($B= -.01$, $p<.02$), but not for those over 21 ($B= -.03$, $p<.06$). Depression and the interaction of PROP and BDI were not significant predictors. In the full model, for both age groups, less than 2% of the variance in alcohol consumption was explained by BDI scores, PROP intensity, and the interaction term.

Emotion reaction. Mood induction was examined next. First, we looked at whether or not there were differences in reported baseline moods across groups; there were no differences. Next we examined the change scores (difference between baseline and post-

induction mood) to look for outliers. There were scores in the opposite direction of the intended induction (e.g., increased threat scores following the triumphant mood induction) these 57 responses were eliminated from relevant analyses (Parrott, 1991). Participants' responses to the triumphant clip and the threatening clip were correlated, r=.47, p<.0001, indicating that people who reacted strongly to one induction, tended to react strongly to the other induction as well. ANOVA was used to assess whether or not a participants' tasting status could account for the variance in response to (1) the threat-inducing clip and (2) the triumphant clip. There was no difference between groups on the induced triumph score (F(2,448)=.65, p=.52). Similarly, there was no difference in induced threat response (F(2, 486)=1.71, p=.18). See Table 6.

Next, differences in the overall positive (sum of happy, strong, excitement, triumph) and overall negative (sum of fear, anger, threat, sad) mood induced across taste groups were assessed. ANOVA indicated group differences in response to the negative mood induction (F(2,493)=2.98 p=.05). Post-hoc analyses demonstrated that the supertasters responded more strongly than medium tasters. Interestingly, investigating PROP intensity as a predictor of mood induction using regression, controlling for ethnicity, we found that PROP was related to Triumph, (B=0.13, p<.05), Excitement (B=0.11, p<.05) and overall Positive Mood (B=0.09, p<.05), as well as Threat (B=0.14, p<.05)

Next, to determine whether or not a person's mood reactivity affects their mood symptomatology, regression was used to assess whether one's mood induction response accounts for variance in mood symptomatology. Regressing BDI scores on threat response, triumph response, and overall positive and negative mood induction response, only threat response was a significant predictor (B=0.06, p<.05). After eliminating the BDI outliers

identified earlier, this result was no longer significant. None of the variables of interest were significant predictors of HCL scores.

Emotion regulation. Trait emotion regulation strategies were measured using the CERQ self report. In order to reduce the risk of Type I error, the nine subscales were reduced to three index scores (negative, positive, neutral). Both the positive (r=0.66, p<.0001) and negative scales (r=0.30, p<.0001) were correlated with the neutral scale, but were not correlated with each other. Differences in emotion regulation strategy across taste groups were assessed; no significant differences were detected. See Table 7.

For the hypothesis that the relation between supertasting and mood induction response would be moderated by emotion regulation, such that higher scores on the positive and neutral CERQ scales would predict less intense response to the mood induction, participants' scores on the reduced scales from the CERQ were used to assess the interaction between supertasting and emotion regulation (Aiken, et al., 1991). First, emotion regulation scores and PROP intensity ratings were mean centered. Next, interaction terms for taste sensitivity and emotion regulation were created. Overall positive mood induction was regressed on PROP intensity, emotion regulation scores were added in the next block, followed by interaction terms for emotion regulation and PROP intensity, and the ethnicity covariate. In the final model, the only significant predictors of positive mood induction were PROP intensity (B= .06, p<.05) and the interaction of negative emotion regulation and PROP (B= -.01, p<.05). The interaction indicates that, for people who are very sensitive to the taste of PROP, negative emotion regulation strategies result in lower positive mood reactivity. See Figure 7.

Negative mood induction, regressed on PROP intensity, emotion regulation scores, interaction terms for emotion regulation and PROP rating, and the ethnicity covariate, was predicted by PROP intensity (B=.08, p<.05), negative emotion regulation (B=.16, p<.01), positive emotion regulation (B=.17, p<.01), and the interaction term for negative emotion regulation and PROP rating (B= -.01, p<.05). Similarly to the result found for positive mood induction, people sensitive to PROP, who use negative emotion regulation strategies, show less negative mood reactivity. See Figure 7.

Finally, the hypothesis that the relation between mood symptomatology and mood induction is moderated by emotion regulation was assessed using linear regression. Regressing BDI scores on positive and negative mood induction, positive, neutral, and negative emotion regulation strategies, on each of the interaction terms between mood induction and CERQ scales, only emotion regulation – both positive (B= -.07, p<.05) and negative (B= .26, p<.0001) – was a significant predictor. These relations held after excluding the outlier BDI scores. Interestingly, positive emotion regulation was not a significant predictor of HCL scores, but both negative emotion regulation strategies (B= .11, p<.05) and neutral emotion regulation strategies (B= .18, p<.01) predicted increases in HCL scores. This somewhat surprising result may be related to the "sunny," positive, and the "dark," negative sides of hypomania; related, we found that positive and neutral emotion regulation is correlated with the Energy factor of the HCL (r=.18, p<.001), and negative emotion regulation is correlated with the Problems factor of the HCL (r=.09, p<.05) and the Energy factor (r=.17, p<.001). There were no other significant predictors of HCL scores.

Sensitivity analyses and equivalence testing. Given the lack of significant results in the present study, we conducted a sensitivity power analysis, using G*Power (Faul,

Erdfelder, Lang, & Buchner, 2007), to determine the effect size we were powered to detect. Given our sample size (*N*=499), alpha=.05, and power .80, we should be able to detect correlations as small as *r*=±.09. Though it is possible that there are relations between the variables explored in this study that are smaller than ±09, these would not be likely to make a meaningful difference in the mood outcomes of interest.

Given that the main objective of this study was to determine whether or not supertasting might have utility as an objective measure of risk for mood disorders, we decided to use equivalence testing to determine whether there is potential for any of the indicators of interest to predict either depression (BDI scores) or mania (HCL scores). Equivalence testing, in this case, looks to see whether the relation found between two variables, and its standard error, fall within the zone of indifference (Foody, 2009). For the present study, we decided that the zone of indifference would fall between ±.2; this corresponds with an Area Under the Curve of .614, indicating predictive power of only slightly better than chance. Examining the correlations between BDI scores and PROP intensity, threat mood induction, triumph mood induction, overall positive mood induction, overall negative mood induction, and the positive, negative, and neutral emotion regulation strategies, only the positive and negative emotion regulation strategies broke out of the zone of indifference. Similarly, for HCL scores, negative, positive, and neutral emotion regulation scores may be related to hypomania in a way that has some clinical utility.

Discussion

Overall, the hypotheses related to supertasting as a potential biomarker for mood disorder risk found minimal support. The difference in prevalence of supertasters found in this sample, as compared to the prevalence found in previous studies, may be a factor. However, the grouping of tasters is said to be arbitrary (Bartoshuk, 2011; B. Tepper, 2008), and different criteria have been used to create groups in previous studies (Tepper, et al., 2001; Zhao, et al., 2003). In this sample, the bimodal distribution of the taste intensity data suggests a two, rather than three, group model might better represent the data; however, using a dichotomous grouping did not affect the results. Furthermore, with 60% of the sample qualifying as supertasters under this classification, the value of being designated as a supertaster would be minimal, as it would offer little diagnostic specificity. Using the intensity rating as a continuous variable preserves the variability of taste sensitivity and should capture a relation between taste sensitivity and the other variables of interest, if one exists. Related, this is the largest sample of the extant studies of mood and supertasting: more than double the size (N=200) needed for adequate power to detect an effect. Therefore, the low – or absent – effect size of taste sensitivity in the present analyses casts doubt on the utility of taste sensitivity as a biomarker for mood disorder risk.

We hypothesized that supertasting would be related to earlier age of onset for emotional or mood-related problems because it might be indicative of a biologically-based risk factor. Previous studies have shown that people who have a family history of mood disorder experience mood problems earlier than others (Faraone, et al., 2003). Similarly, in a

study of supertasting and depression, those people who were supertasters had an earlier age of onset than the depressed people who were nontasters (Whittemore, 1990). This result was not supported; supertasters did not report an earlier age of onset and PROP intensity was not correlated with age of onset. This result may be related, in part, to the age of the sample; the average age is 19, which is not past the window of risk for onset of psychiatric problems. Additionally, taste sensitivity was not related to treatment seeking, which again may be due to the age of the sample and the fact that participants may not yet have the need for psychiatric treatment. That said, given that the hypothesis was that there would be an *early* onset – younger than age 18 – it is unlikely that supertasting is related to early risk.

The hypothesis of early onset was predicated on the theory that supertasting might be indicative of familial risk. Specifically, supertasters were hypothesized to have higher rates of mood disorder in their families and to have lower rates of alcoholism. Neither of these results was supported. Similarly, the result from a previous study (Joiner & Perez, 2004) indicating that supertasters are *less* likely to have familial depression was not found in the present study.

Based on a previous study (DiCarlo & Powers, 1998) that found that supertasters were more likely to have a family history of both alcoholism and depression, combined total family history of these two disorders was compared across taste groups; no differences were found. It may be that the reported family history information was not accurate; young adults may be unaware of family history and self-reported information is fallible (Milne et al., 2009). Still, the overall prevalence of psychiatric disorder in this sample was consistent with that found in multiple epidemiological studies (Moffitt et al., 2010). The lack of support for the hypothesis that supertasting may be an indicator for a more heritable form of mood

disorder is disappointing, but perhaps not surprising, given the low levels of mood symptomatology in the present sample (of 499 people, only 22 scored higher than 19 on the BDI, indicating moderate-severe depression) (Beck, et al., 1988; Lasa, Ayuso-Mateos, Vasquez-Barquero, Diez-Manrique, & Dowrick, 2000). It may be that, in a healthy sample, the relation between depression and supertasting is not detectable, or perhaps supertasting is associated with a subtype of mood disorder, just not one characterized by family history of psychiatric illness and early onset of symptomatology. Previous studies have challenged the theory that familial depression is related to supertasting (Joiner & Perez, 2004), pointing out methodological problems in the measurement of familial depression and in the grouping of tasters in studies that found an association (Whittemore, 1986, 1990).

The results related to depressive symptomatology were interesting; the initial relation between the BDI and supertasting (both across groups and using PROP intensity as a continuous variable) was found to hinge on a handful of high BDI scores. Overall, the BDI scores were low, and there may not be enough variability to detect the relation between taste sensitivity and depression. The high scores were not necessarily inaccurate, and it is possible that, in a clinically depressed sample, we would see a stronger relationship. We made the argument that college samples are not less likely than the general population to experience mood disorder, and as an analog for community samples, college students are generally appropriate (Vredenburg, et al., 1993). However, there is a difference between college students and clinical patients; in our aim to identify a subtype of mood disorder, based on a biomarker, we may have been better served focusing on participants who have a mood disorder diagnosis. In addition, given that previous studies – with significant findings – of

depression and supertasting have used clinical samples, it would be worthwhile to follow-up on the hypotheses related to supertasting and mood disorder in a clinical sample.

Hypotheses related to mania were strictly exploratory; no previous examination of mania and supertasting has been published. There was no relation found between reports of elevated mood and supertasting groups or PROP intensity rating. The theory that high rates of impulsivity and pleasure-seeking (Joiner & Perez, 2004) seen among supertasters might be related to similar characteristics of [hypo]mania was not directly supported. However, there may be a confound related to the sample; overall, the scores on the HCL 32 were quite high and 43% of the sample rated their mood as "generally higher" than other people. Items asking about confidence, sociability, energy, sexual activity, and being distractible may tap into characteristics of college students, as much as they do symptoms of mania. Further evidence that the high scores were not due to mania is that items related to impairment were rated in the opposite direction of symptom ratings; participants reported that others were not concerned about their "high" episodes, and that these periods tend to last just 1-3 days, below the duration criteria for hypomania. Similar to the results with the measure of depression, it may be that this sample did not have an adequate level of manic symptoms to relate to supertasting; this hypothesis should be pursued in a clinical sample, in order to reach a more definitive conclusion.

One of the few hypotheses supported in this study was that supertasters would be more likely to avoid alcohol. We found that supertasters drank less frequently than nontasters. Though the sample is not, on average, old enough to drink, frequency of alcohol consumption among supertasters was just over half that of the nontasters, among those under 21, and was only a third of nontasters among those over 21. Interestingly, though there was

no difference in reported alcohol-related problems when assessed by group, PROP intensity was a predictor of fewer alcohol-related problems. This result supports the idea that maintaining taste sensitivity as a continuous variable may be beneficial. Previous research found that male and female supertasters may have different patterns of alcohol-related problems (Driscoll, et al., 2006), this hypothesis was not supported in our sample.

Additionally, past studies have found that, among families of supertasters, depression was related to increased alcohol use. We decided to look at comorbid depression and alcohol use in the proband, in addition to their family. We found that combined scores of PROP intensity (supertasting) and depression did not predict increased alcohol consumption. If, as previous studies suggested, alcohol and food are used by supertasters as coping mechanisms for depression, it may be that, among this under-aged sample, alcohol is not used as a coping mechanism because it is not as easily accessible as other coping mechanisms.

Based on the evolutionary theory that supertasting is a genetic protection against poisonous plants, and the hypothesis that supertasters might be more sensitive to threat in the environment generally, we looked at mood change following a threatening film clip across taste groups. Examining the sum of the negatively-valenced moods following the threatening clip, supertasters did react more strongly. Previous work (Macht & Mueller, 2007) found that supertasters reacted more strongly than others to negative mood induction; replication of this result offers intriguing support for the hypothesis that supertasting represents a global sensitivity to threat. Though the summed positive mood score did not differ across groups, the specificity of the relation between threat and taste sensitivity is called into question by the fact that triumph, excitement, and overall positive mood, as well as threat were predicted by PROP sensitivity in regression analyses. This suggests that people who are sensitive to 6-n-

propylthiouracil may also be more sensitive to their environment, in general, than other people. It would be very interesting to use a clinical sample to see whether supertasting can explain part of the relation between increased mood reactivity and bipolar disorder, as found in other studies (Roiser, et al., 2009).

As hypothesized, we found that threat response was predictive of higher BDI scores. Previous work has found that people with bipolar disorder tend to react more strongly to mood induction than other people (Roiser, et al., 2009), and we hypothesized that chronic emotional overreactivity may contribute to the development of a mood disorder. The fact that even a small effect was observed in a sample with very limited depressive symptomatology, offers support for further exploration of this relation in a clinical sample, especially since the relation appeared to be driven by the high BDI scores. The lack of a relation on the HCL is somewhat surprising, given the association between mania and emotional excitability, but there has not been a previous investigation into this hypothesized relation, and again, the population represented in this sample may be reporting symptoms of hypomania without actually having bipolar disorder – or its underlying diatheses.

We expected to find differences in the emotion regulation strategies across taste groups, corresponding to increased levels of psychopathology among supertasters. Other studies (Garnefski & Kraaij, 2006b; Garnefski, et al., 2005) have found that people with psychopathology tend to use more negative emotion regulation strategies; though we did find a relation between negative emotion regulation strategies and both the BDI and HCL; this relation appeared to be independent of supertasting.

We anticipated that the relation between taste sensitivity and overall sensitivity (as measured by mood induction) might be moderated by emotion regulation. If one is a skilled

emotion regulator, instinctual over-response to emotional stimuli may be dampened. This hypothesis was supported; we found that for positive mood induction, though PROP intensity predicted higher reactivity, the interaction of PROP and negative emotion regulation resulted in lower negative mood induction scores. The effect of the interaction was small (change in R^2=1%), but intriguing, in that it suggests a learned emotion regulation technique may provide a way by which to lessen a biological risk factor toward mood dysregulation. Though dampening positive mood may seem maladaptive, for people at risk for bipolar disorder, mood dysregulation in either direction can be problematic.

Predicting negative mood induction with PROP intensity and emotion regulation produced particularly interesting results: both taste sensitivity and negative emotion regulation strategy predict higher scores on mood induction when entered independently, however, the interaction of the two resulted in a reduction in mood reactivity. It is not clear why emotion regulation strategies, on their own, led to an increase in negative mood. One possibility is that, people who are good at regulating emotion may be sensitive to emotion in general, and equally capable of increasing or decreasing their reactions; the method used for the mood induction, which encouraged participants to *really notice* the specified mood, may have led to more reactivity in people who are generally in control of their emotions.

Regarding the reduced response attributed to the interaction of negative emotion regulation and PROP sensitivity, it may be that the heightened response associated with supertasting results in *overregulation* by the negative emotion regulation system. Negative emotion regulation strategies include self-blame, ruminating, and catastrophisizing; if one reacted strongly to a brief film clip, in a somewhat public situation, embarrassment might follow, it is not hard to imagine that negative regulation strategies might then be triggered,

whereas without the instinctual over-response due to threat sensitivity, the emotion regulation strategies would not be cued. Overall, the variance in negative and positive mood accounted for by the models was only seven and five percent, respectively, and the coefficients for the predictors are small. Still, this is an intriguing result that would be interesting to explore in a sample for whom emotion reactivity and regulation are of clinical significance.

Though the relation between mood symptomatology and mood induction was not significant, we still tested the hypothesis that the relation between mood symptomatology and mood induction would be moderated by emotion regulation. None of the interaction terms between mood induction (positive and negative) and emotion regulation (positive, neutral, and negative) were significant predictors. Emotion regulation related to depressive symptomatology in the expected way; negative emotion regulation strategies were associated with higher depression scores, positive emotion regulation strategies were related to lower depression scores. Interestingly, both negative emotion regulation strategies and neutral emotion regulation strategies predicted increases in HCL scores. This may represent the "sunny" and "dark" aspects of hypomania (Akiskal, Hantouche, & Allilaire, 2003); negative emotion regulation might lead to irritability and substance use, associated with the dark aspects of hypomania, whereas neutral emotion regulation might lead to increased confidence and autonomy, sunny aspects of hypomania.

Limitations and future directions

The method of assessing supertasting is not definitive, which raises questions about the validity of taste groups. This is a valid critique and an area of research that needs to be further elucidated before we can determine the utility of supertasting clinically; however, the observed bimodal distribution of the PROP intensity rating suggests that there is a difference

in the way people experience 6-n-propylthiouracil. Perhaps, rather than relying on arbitrary taste groups, taste sensitivity is better represented across a spectrum. Given the shift toward a spectrum model of mood disorder (Youngstrom, et al., 2010), it makes sense that many risk factors are also better characterized across a range. Though, genetically, we expect three types of tasters, based on the number of dominant and recessive alleles, considering the other factors that may affect our taste – or at least the measurement of taste (concentration of PROP used, tastebud count, gender, smoking) – perhaps retaining more information through a dimensional model makes sense.

Though the current study offers some support for the hypothesis that supertasters may be more reactive to their environment than other people, the potential implications of this reactivity were obscured by the lack of association with clinical symptomatology. It may be that there is no relation between supertasting and mood; this is a larger sample than those previously tested and should be adequately powered to detect an effect. However, previous studies have used clinical samples, whereas we used college students, who were mainly nondepressed. If our hypothesis is correct, and supertasting is indicative of a particular subtype of mood disorder, a more heritable, and perhaps pernicious, variety, it may be that there simply are not enough people in a university setting to support the model. In fact, individuals suffering from an early-onset disorder may be less likely to attend college. Future research in a clinical population could yield results suggestive of a subtype of mood disorder. If such a subtype exists, it may have important implications for diagnosis and treatment.

Conclusion

The literature on supertasting is not extensive, but it is an intriguing concept – one that has undoubtedly appealed to many curious research groups. Why has there been so little

published? With our faint results – a perhaps spurious relation to depression, a weak correlation with negative mood reactivity, and a long-established link to low alcohol use – we are left wondering whether we would have found more significant results in a clinical sample, one with greater levels of mood disorders and poorly regulated emotion, or if perhaps – regardless of sample – studies of supertasting are bound mostly for the null finding filing cabinet. Looking back at other studies that explored supertasting and mood, both the effect sizes and the samples tend to be rather small. Two studies found correlations between PROP/PTC tasting and depression of r=0.14 and r=0.19 in samples of 123 and 98, respectively. These results are consistent with the present study. Two other studies found much larger effects, r=0.52 and r=0.45, in samples of 37 and 41. The results from these studies are statistically different from our own (p<.05). One of these studies was a clinical sample, which may help to account for the difference in relation. However, the other study was a college sample with only one reported BDI score in the clinical range; it is difficult to understand why this study had results so different from our own, but there is an important methodological difference – they incorporated both threshold and magnitude of the PROP tasting, which may offer better results given the methodological issues of measurement mentioned earlier.

It would be interesting to know whether supertasting, in some form, has a relation with one's risk for developing a mood disorder; however, the current state of the literature is too inconsistent to draw conclusions. Though it may be premature to decide whether our taste sensitivity is, or is not, related to our general sensitivity to the environment, given the small effect sizes, it does not seem too soon to suggest that supertasting is likely not a clinically useful tool. If we convert the correlation we found between depression scores and PROP

intensity (*r*=.09) into the number needed to treat (9.8) (Furukawa & Leucht, 2011), we can conclude that, for roughly every ten supertasters, one would be depressed.

Though there is no way to know how many null finding studies of mood and supertasting are tucked away on hard drives and in filing cabinets, we can estimate how many there would need to be for the pooled effect size to be too small to be meaningful. Using a modification of Rosenthal's fail-safe *N* concept (Orwin, 1983), we can combine the effect sizes for the published studies of supertasting and depression, and determine how many there would have to be that fail to reject the null hypothesis, in order to drive the pooled effect size below *d*=.2. For the six known studies (including ours), the average effect size is .518, indicating a fail-safe *N* of 10 null finding studies. Further, pushing the desired critical effect size to just .5, results in a fail-safe *N* of only .2 studies. So, though we may not have access to every abandoned supertasting project, we need only be confident that there are a handful of other null findings out there to bolster our confidence that the effect size of supertasting on depression is not large enough to be clinically meaningful. From an intellectual perspective, the idea of supertasting is interesting and the theory behind it tells a compelling tale, but in a high stakes clinical situation, making a decision about an individual's diagnosis and, consequently, treatment, we need indicators that perform better than chance.

Appendix A: Study results excluding participants who smoke regularly, or smoked, ate or drank prior to participating

Table 1. *Correlations between taste sensitivity and number of tastebuds and outcomes of interest*

	PROP intensity	Tastebuds	BDI	HCL-32	Alcohol Problems	Days per week drink	Days drink >5	Days per week drunk	Family Depression	Any Family Psych	Threat Response	Triumph Response	Positive Mood Response	Negative Mood Response	Negative Emotion Regulation	Positive Emotion Regulation	Neutral Emotion Regulation
PROP Intensity	1	-0.03	0.08§	-0.07	-0.12*	-0.2*	-0.15*	-0.14*	-0.011	-0.044	0.064	0.061	0.046	0.084	-0.01	-0.03	-0.07
Tastebuds	-0.031	1	-0.06§	0.01	-0.07	-0.05	-0.03	-0.01	-0.03	-0.02	0.06	0.07	0.06	0.09	-0.01	0.02	-0.08

*$p<.05$; § without BDI outliers, PROP $r=-.01$, *ns*, tastebuds $r=-0.05$, *ns*

Table 2. *Demographic differences between taste groups*

	Non Taster *n*=59	Medium Taster *n*=276	Super Taster *n*=45
Age	19.1(1.0)	19.1(2.7)	18.8(1.2)
Female	58%	67%	71%
Race			
Caucasian	78%	72%	51%
African American	12%	11%	30%
Asian	3%	13%	14%
Native American	2%	1%	0%
Other	5%	4%	5%
Hispanic	5%	9%	12%

Table 3. *Differences in treatment seeking across taste groups*

	Non taster	Medium Taster	Super Taster
Age of Onset	14.8 (*n*=21)	13.0 (*n*=112)	14.7 (*n*=18)
Psychiatric treatment sought (Y/N)	30% (*n*=18)	19%(*n*=53)	16%(*n*=7)
Age of treatment seeking	15.2 (*n*=18)	13.6 (*n*=53)	16.3(*n*=7)

Table 4. *Differences in familial and self-reported psychiatric disorder*

Family History of:	Non Taster *n*=59	Medium Taster *n*=276	Super Taster *n*=45
Psychiatric Illness	42%	40%	36%
Alcohol or Drug Problems	44%	42%	53%
Mood Disorder	41%	37%	36%
Alcohol and Depression	25%	26%	25%
Depression	39%	35%	31%
Bipolar Disorder	8%	10%	9%
Suicide	12%	9%	7%
Psychiatric Hospitalization	17%	11%	13%
BDI Score POMP	9.3 (8.2)	7.9 (8.2)	13.7 (15.6)
BDI Score POMP without outliers	9.3 (8.2)	7.6 (7.4)	9.8 (9.4)
HCL Score POMP	61.4 (13.9)	58.6 (14.0)	57.6 (14.9)

Table 5. *Differences in alcohol consumption and consequences*

	Non taster		Medium Taster		Super Taster	
	<21 *n*=52	>21 *n*=7	<21 *n*=252	>21 *n*=24	<21 *n*=40	>21 *n*=5
Alcohol Consequences Score POMP	20.75 (19.4)*	36.9 (27.2)	16.2 (17.7)	24.1 (21.8)	10.5 (12.8)	21.7 (30.5)
Days per week drink	1.3 (1.2)*	3.0(2.1)*	1.0(1.1)*	1.7 (1.3)	0.6 (0.8)	0.4 (0.9)
Days per week >5 drinks	0.6 (0.7)*	1.6 (1.3)	0.4 (0.8)	0.6 (0.8)	0.2 (0.5)	0.2 (0.4)
Days per week drunk	0.8 (0.8)*	1.4 (1.4)	0.5 (0.9)	0.6 (0.7)	0.3(0.6)	0.2 (0.4)

*results different from super tasters, $p<.01$

Table 6. *Differences in mood induction across taste groups*

	Non taster *n*=57	Medium Taster *n*=272	Super Taster *n*=45
Triumph Response	29.1 (26.6)	28.6(23.9)	28.5 (32.4)
Threat Response	35.0 (33.2)	33.8 (29.8)	36.8 (34.5)
Positive Mood POMP	17.9 (17.3)	17.3 (15.0)	17.4 (18.8)
Negative Mood POMP	21.6 (19.4)	21.5 (18.4)	25.9 (19.8)

Table 7. *Differences in emotion regulation strategies across taste groups*

	Non taster *n*=59	Medium Taster *n*=276	Super Taster *n*=45
Negative ER POMP	38.1 (14.7)	34.2 (14.8)	36.3 (18.5)
Positive ER POMP	49.6 (20.1)	49.3 (17.9)	50.8 (20.9)
Neutral ER POMP	51.1 (13.6)	48.6 (12.7)	48.0 (12.9)

Table 1. *Correlations between taste sensitivity, number of tastebuds and outcomes of interest*

	PROP intensity	Tastebuds	BDI	HCL-32	Alcohol Problems	Days per week drink	Days drink >5	Days per week drunk	Family Depression	Any Family Psych	Threat Response	Triumph Response	Positive Mood Response	Negative Mood Response	Negative Emotion Regulation	Positive Emotion Regulation	Neutral Emotion Regulation
PROP intensity	1	-0.04	0.09§	-0.08	-0.08	-0.16*	-0.10*	-0.11*	-0.03	-0.05	0.09	0.08	0.06	0.10*	0.01	-0.01	-0.03
Tastebuds	-.040	1.00	-0.03§	-0.03	-0.13*	-0.09*	-0.09*	-0.06	-0.05	-0.04	0.04	-0.01	-0.01	0.05	0.05	0.03	-0.02

*$p<.05$

§ without BDI outliers, PROP $r=.04$, *ns*, tastebuds $r=-0.03$, *ns*

Table 2. *Demographic differences between taste groups*

	Non Taster *n*=76	Medium Taster *n*=353	Super Taster *n*=70
Age	19.0 (1.0)	19.1(2.5)	19.0 (1.2)
Female	40 (53%)	228 (65%)	43 (61%)
Race			
Caucasian	82%	72%	54%*
African American	9%	12%	24%
Asian	4%	12%	15%
Native American	1%	0%	1%
Other	4%	4%	6%
Hispanic	7%	9%	15%

*less likely to be a supertaster, $p<.01$

Table 3. *Differences in treatment seeking across taste groups*

	Non taster	Medium Taster	Super Taster
Age of Onset	14.8 (*n*=23)	13.1 (*n*=147)	14.5 (*n*=29)
Psychiatric treatment sought (Y/N)	26% (*n*=20)	21%(*n*=75)	17%(*n*=12)
Age of treatment seeking	15.3 (*n*=20)	13.8 (*n*=75)	16.2(*n*=13)

Table 4. *Differences in familial and self-reported psychiatric disorder*

Family History of:	Non Taster *n*=76	Medium Taster *n*=353	Super Taster *n*=70
Psychiatric Illness	46%	43%	34%
Alcohol or Drug Problems	50%	44%	53%
Mood Disorder	45%	39%	33%
Alcohol and Depression	30%	28%	28%
Depression	41%	37%	30%
Bipolar Disorder	18%	10%	11%
Suicide	11%	11%	10%
Psychiatric Hospitalization	12%	10%	10%
BDI Score POMP	9.1 (8.0)	8.8 (8.9)	12.5 (13.8)
BDI Score POMP without outliers	9.1 (8.0)	8.4(8.1)	10.0 (9.4)
HCL Score POMP	62.4 (14.3)	59.6 (14.1)	59.4 (16.1)

Table 5. *Differences in alcohol consumption and consequences*

	Non taster		Medium Taster		Super Taster	
	<21 *n*=67	>21 *n*=9	<21 *n*=325	>21 *n*=28	<21 *n*=61	>21 *n*=9
Alcohol Consequences Score POMP	22.1 (20.2)	38.9 (27.2)	18.1 (19.4)	26.4 (23.0)	13.6 (16.0)	24.3 (28.1)
Days per week drink	1.5(1.3	2.9(1.8)	1.1 (1.2)	1.8 (1.3)	0.9 (1.0)*	1.0 (1.4)*
Days per week >5 drinks	0.7 (0.9)	1.6 (1.3)	0.5 (0.9)	0.7 (0.9)	0.4 (0.8)*	0.7 (0.3)
Days per week drunk	0.9 (1.0)	1.3 (1.3)	0.6 (0.9)	0.6 (0.7)	0.5 (0.7)*	0.7 (1.3)

*results different from non tasters, $p<.05$

Table 6. *Differences in mood induction across taste groups*

	Non taster *n*=69	Medium Taster *n*=321	Super Taster *n*=61
Triumph Response	29.1 (26.2)	28.3(23.8)	32.3 (32.2)
Threat Response	32.8 (31.9)	32.9 (29.2)	40.2 (33.6)
Positive Mood POMP	17.9 (16.4)	17.0 (15.0)	19.9 (20.1)
Negative Mood POMP	19.9(18.4)	21.0(17.8)	26.5 (20.1)*

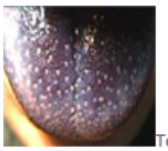
*results different from medium tasters, $p<.05$

Table 7. *Differences in emotion regulation strategies across taste groups*

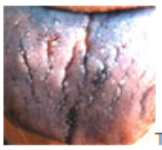
	Non taster *n*=76	Medium Taster *n*=353	Super Taster *n*=70
Negative ER POMP	36.1 (15.4)	34.6 (14.9)	37.0 (17.8)
Positive ER POMP	48.4 (19.6)	48.3 (18.1)	49.3 (21.1)
Neutral ER POMP	49.5 (15.0)	48.3 (13.1)	48.0 (14.1)

Figure 1. *Counting of tastebuds using food coloring* (Utermohlen, 2010)

Tongue of a mildly sensitive taster.
The fungiform papillae are the small pinkish dots on this tongue. Note that they are small and seed-shaped.



Tongue of a moderately sensitive taster.
The fungiform papillae on this tongue are larger and more rounded than those on the tongue above.



Tongue of a highly sensitive taster.
This tongue is covered with fungiform papillae. Note that this tongue does not have any blue areas like those in the tongues shown above.

Figure 2. *Distribution of PROP intensity ratings with taste grouping alternatives considered*

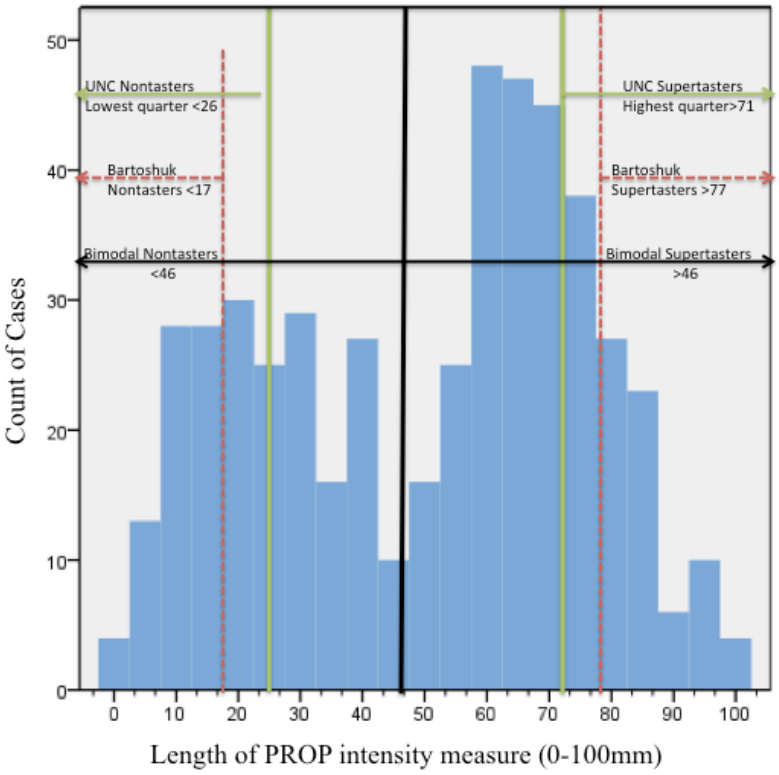


Figure 3. Scatterplot of PROP intensity ratings and number of tastebuds

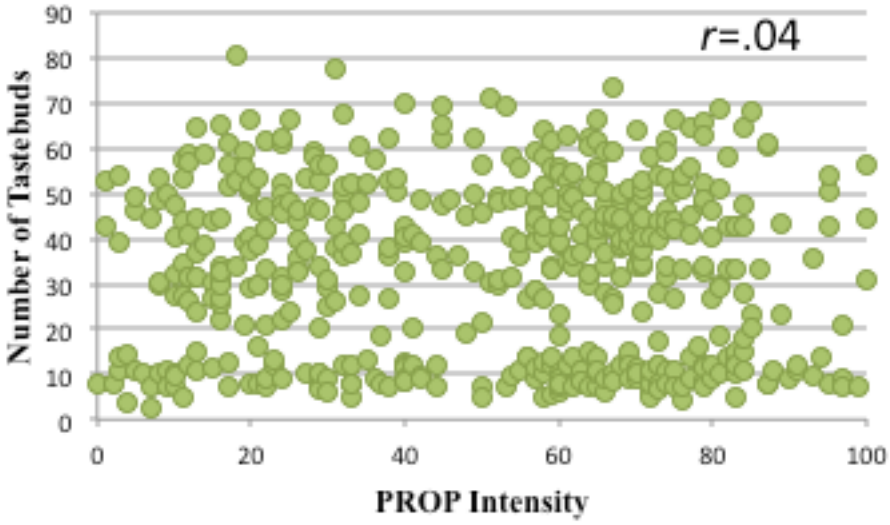


Figure 4. *Cox regression indicating age of onset of emotional or mood problems*

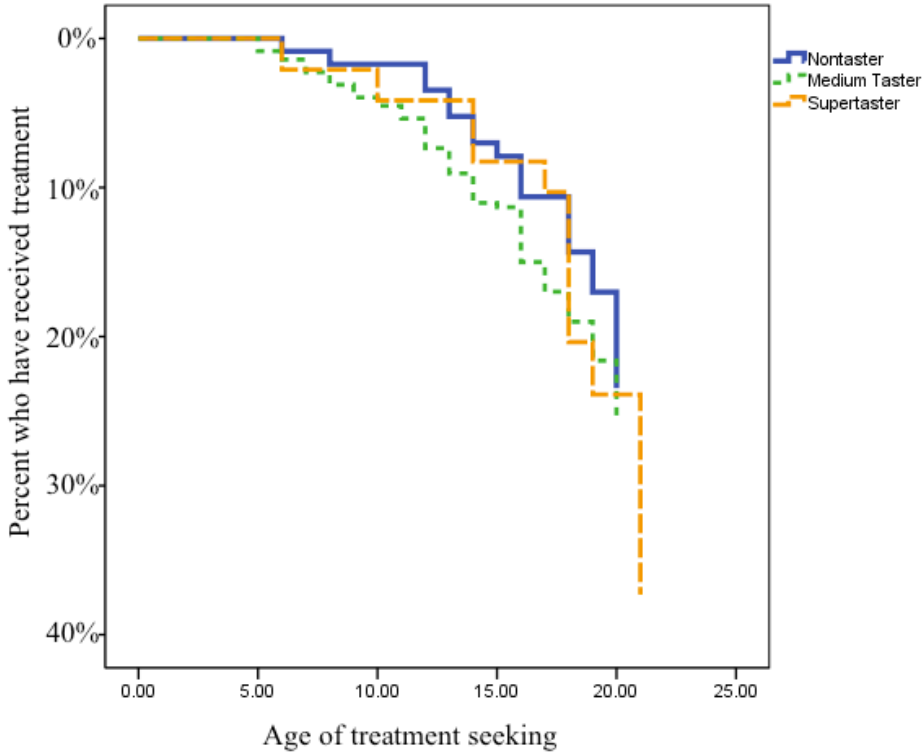


Figure 5. *Illustration of BDI POMP score distribution*

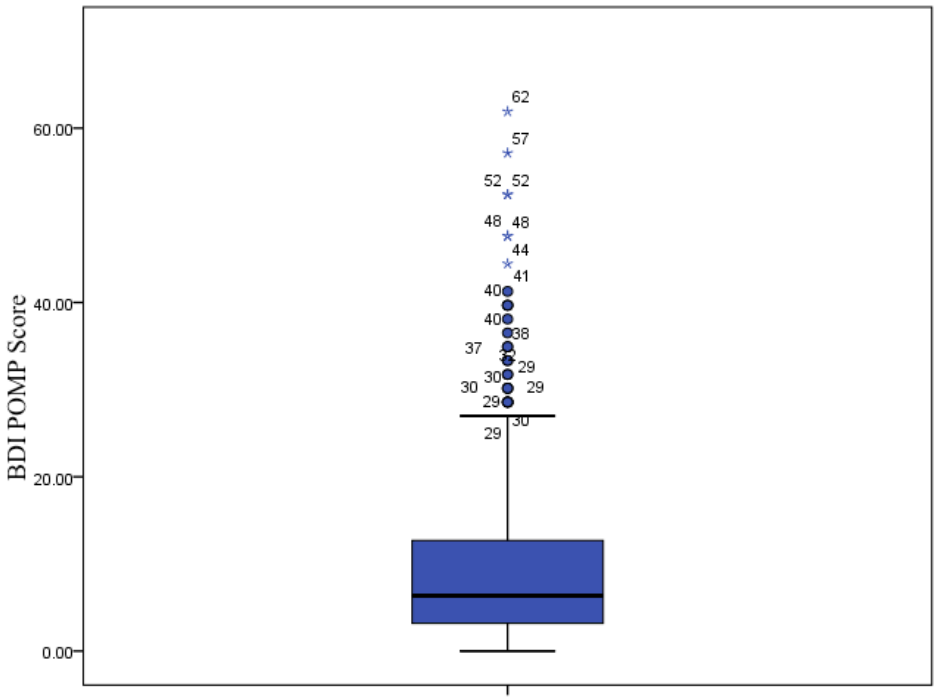


Figure 6. *Comparison of regressing BDI POMP scores on PROP intensity with and without outliers*

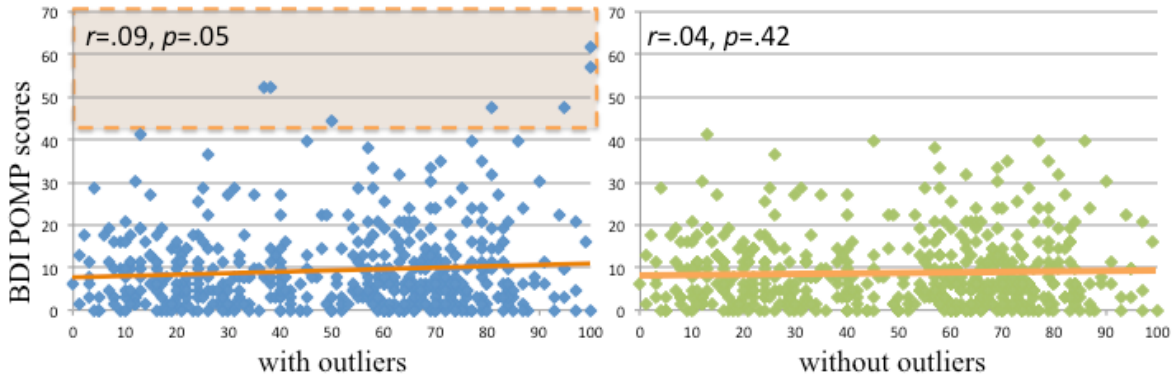


Figure 7. *Interaction of negative emotion regulation and PROP intensity rating predicting both negative and positive mood*

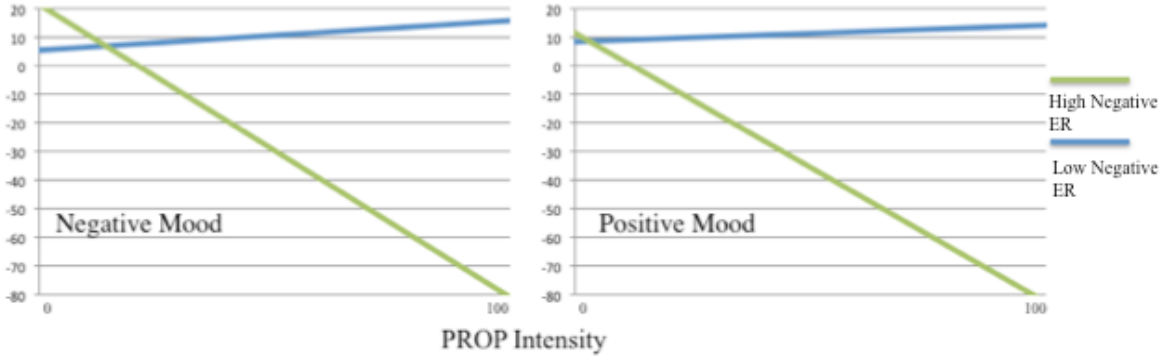
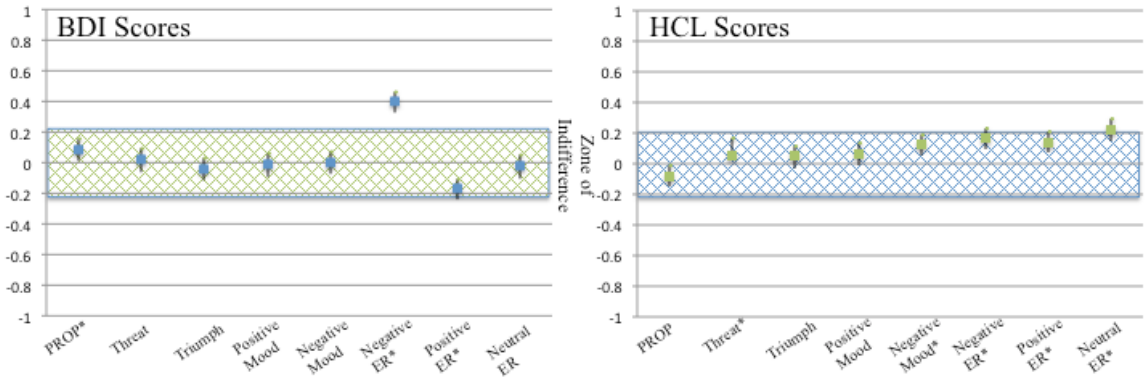


Figure 8. *Equivalence testing of the indicators of interest*



*correlation between mood and predictor, $p<.05$

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