High intelligence: A risk factor for psychological and physiological overexcitabilities

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ABSTRACT

High intelligence is touted as being predictive of positive outcomes including educational success and income level. However, little is known about the difficulties experienced among this population. Specifically, those with a high intellectual capacity (hyper brain) possess overexcitabilities in various domains that may predispose them to certain psychological disorders as well as physiological conditions involving elevated sensory, and altered immune and inflammatory responses (hyper body). The present study surveyed members of American Mensa, Ltd. ($n = 3715$) in order to explore psychoneuroimmunological (PNI) processes among those at or above the 98th percentile of intelligence. Participants were asked to self-report prevalence of both diagnosed and/or suspected mood and anxiety disorders, attention deficit hyperactivity disorder (ADHD), autism spectrum disorder (ASD), and physiological diseases that include environmental and food allergies, asthma, and autoimmune disease. High statistical significance and a remarkably high relative risk ratio of diagnoses for all examined conditions were confirmed among the Mensa group 2015 data when compared to the national average statistics. This implicates high IQ as being a potential risk factor for affective disorders, ADHD, ASD, and for increased incidence of disease related to immune dysregulation. Preliminary findings strongly support a hyper brain/hyper body association which may have substantial individual and societal implications and warrants further investigation to best identify and serve this at-risk population.

Intelligence quotient (IQ) is generally touted as a gift predicting exceptional outcomes in many domains including educational attainment and income level (Bergman, Corovic, Ferrer-Wreder, & Modig, 2014) and is a positive indicator of high system integrity (Gale, Hatch, Batty, & Deary, 2009; Gale, Batty, Tynelius, Deary, & Rasmussen, 2010; Gottfredson, 2004; Lubinski & Humphreys, 1992; Wraw, Deary, Gale, & Der, 2015; Wrulich et al., 2013). However, there are conflicting studies in the literature which point to an association between gifted IQ, particularly high verbal ability, and various mental and immunological outcomes such as depression (Jackson & Peterson, 2003; Wraw, Deary, Der, & Gale, 2016); bipolar disorder (Gale et al., 2013; MacCabe et al., 2010; Smith et al., 2015); anxiety disorders (Lancon et al., 2015); ADHD (Rommelse et al., 2016); allergies, asthma, and immune disorders (Benbow, 1985, 1986); and autism spectrum disorder (ASD) (Clark et al., 2016). These seemingly contradictory outcomes may be partially reconciled by taking a closer look at the field of psychoneuroimmunology (PNI) which examines the way in which the stress response to the environment, particularly that which is chronic and

sustained, influences the communication between the brain and immune system (Ader, 2001).

This article extends the current literature on PNI and a variety of physiological and psychological disorders by assessing whether high intelligence may be linked to higher prevalence rates. This is a critical contribution, as research on intelligence and many disorders tends to focus on those with low intelligence to normal IQ scores, rather than extending to the upper ranges of the spectrum. To investigate these links, we assessed whether the prevalence rates in 2015 for those with high intelligence were higher than the reported national averages for mood disorders, anxiety disorders, ADHD, allergies, asthma, autoimmune diseases, and ASD. The connections between intellectual overexcitabilities and each of these conditions are discussed next.

1. Literature review

"A broader and deeper capacity to comprehend their surroundings" is a hallmark in one definition of intelligence agreed upon by fifty-two

notable researchers within the field of cognitive ability (Gottfredson, 1997). Those who are highly intelligent possess unique intensities and overexcitabilities which can be at once remarkable and disabling. For example, the same heightened awareness that inspires an intellectually gifted artist to create (Jauk, Benedek, Dunst, & Neubauer, 2013; Karwowski et al., 2016; Pässler, Beinicke, & Benedikt, 2015) can also potentially drive that same individual to withdraw into a deep depression (Jamison, 1993; Kyaga et al., 2011; Ludwig, 1992, 1995; Simonton & Song, 2009). It is hardly a new notion that unusually high rates of adult psychopathology are displayed among some of the most eminent geniuses (Jamison, 1993; Ludwig, 1992, 1995) with the poorest in mental health being among imaginative writers such as poets, novelists, and dramatists. The intensities leading to these psychological disorders were found to have manifested at a young age (Simonton & Song, 2009). Gere, Capps, Mitchell, and Grubbs (2009) gathered data on 6 to 11-year-old gifted students attending a public school gifted program and found that gifted children reacted with significantly heightened emotional and behavioral responses to their environment than did those of average intelligence. Due in part to this increased awareness, those with an overexcitable cognitive ability tend toward hyper-reactivity of the central nervous system (Chang & Kuo, 2013), which can lead to various other psychological and physiological consequences.

1.1. Intellectual overexcitabilities

Overexcitability (OE) is a term first introduced by Polish psychiatrist and psychologist, Kazimierz Dabrowski. He is most known for his theory of positive disintegration which came about by studying individuals with a high cognitive ability across their lifespans to understand their higher levels of emotional development (Dabrowski, 1964a, 1964b, 1966; Dąbrowski, 1976). His coined term is the English translation of the Polish word 'nadpobudliwosc' that originally means 'superstimulatability.' Dabrowski found these hyper-reactions and intensities to occur with greater frequency and of greater strength in the intellectually gifted compared to those with a normal or lower IQ. According to his clinical observations, bright individuals tended to be "neurotically allergic or nervous," a condition which he observed to be relatively absent in the intellectually delayed. They demonstrated a uniquely heightened way of experiencing and responding to their environment within five specific areas: psychomotor, sensory, intellectual, imaginational, and emotional domains. He found these overexcitabilities to be associated with personality development, and observed symptoms of slight neuroses among them as well, such as depression, mild anxiety, and tics (Mendaglio, 2008; Miller, Falk, & Huang, 2009).

1.2. Psychological overexcitabilities: Affective disorders and ADHD

Recent research agrees with Dabrowski and finds that an intense emotional response of individuals to their environment can lead to increased rumination and worry, both which have been associated with higher cognitive ability (Penney, Miedema, & Mazmanian, 2015). Rumination predicts chronicity of depressive disorders and anxiety symptoms including the new onsets of episodes (Nolen-Hoeksema, 2000). A highly ruminative cognitive style has been shown to be associated with increased vulnerability to major depression (Marchetti, Koster, Sonuga-Barke, & De Raedt, 2012; Nolen-Hoeksema, 2000) and contributes to symptom severity (Coplan et al., 2006, 2012; Kuehner & Weber, 1999)). Worry is the proposed cognitive process underlying general anxiety disorder (GAD) (American Psychiatric Association, 2013; Clark & Wells, 1995; Nolen-Hoeksema, 2000) and as with rumination, those who tend to worry more often and more severely, score higher on tests of intelligence. Penney et al. (2015) demonstrated that verbal intelligence in particular is a positive predictor of worry and rumination as well as being predictive of severity of both processes.

The presence of mood and anxiety disorders was found among other psychological conditions such as attention deficit/hyperactivity disorder (ADHD). In a longitudinal study by Michielsen et al. (2013), adults were assessed over a 6-year period for symptoms of ADHD, depression, and anxiety. The authors found that those with ADHD were at higher risk for both of these affective disorders. Further, Kessler et al. (2006) found that adults with ADHD often present co-occurring psychiatric conditions, including anxiety (47%) and mood (38%) disorders. Of note, those with high intelligence who experience psychomotor overexcitabilities in particular are those often diagnosed with ADHD due to the lack of general knowledge about this particular OE. This diagnosis oftentimes prevents individuals from being properly identified as having a gifted intellectual ability (Rommelse et al., 2016).

1.3. Physiological overexcitabilities: Psychoneuroimmunology

As the field of PNI grows and develops, many discrete pathways connecting psychology and immunity are being discovered (Ader, 2001). For highly intelligent individuals with overexcitabilities, even normal stimuli such as a clothing tag or a common but unnatural sound can become physically painful. Continuous seemingly minor insults such as these may mimic a low level, chronic stress which can eventually launch an inappropriate immune response. As with other environmental threats, like an infection or toxin, the body believes it is in danger. When the sympathetic nervous system becomes chronically activated, it finds itself in a continuous fight, flight, or freeze state, which triggers a series of changes in the brain and the body that can dysregulate immune function (Glaser et al., 1992; Kiecolt-Glaser, Glaser, Gravenstein, Malarkey, & Sheridan, 1996; Padgett & Glaser, 2003). We are learning that stress has a significant effect on the ability of the immune system to protect us and consequences can take many forms including allergies, asthma, and autoimmune disease (Nasr, Altman, & Meltzer, 1981). While there is empirical evidence that mood disorders are associated with immune dysregulation, researchers have yielded conflicting results as to whether this dysregulation contributes to the pathophysiology of depressive disorders (Postal & Appenzeller, 2015; Young, Bruno, & Pomara, 2014) or whether depressive disorders increase susceptibility to immune-related disorders and health conditions such as infection, allergy and autoimmune diseases (Kronfol, 2002; Sansone & Sansone, 2011). Evidence for the latter is compelling given that altered immune function has been shown to be induced by chronically stressful stimuli in both human and animal models (Padgett & Glaser, 2003).

1.3.1. Allergies, asthma, and autoimmune disease

A combination of high intelligence and various allergies that begin in early childhood is not only a common stereotype, it is also verified in the scientific literature. In 1966, a significantly increased rate of allergies and asthma were reported at a school for gifted children (Hildreth, 1966). In a study of allergies and asthma in such children, 44% of those with an IQ over 160 suffered from allergies compared to 20% of age matched peers and 10% report having asthma (Rogers & Silverman, 1997; Silverman, 2002). Benbow (1986) conducted a study of over 400 highly mathematically and verbally talented students who were tested by the Scholastic Aptitude Test (SAT) to be in top 0.01% in reasoning ability. Among the students, she found that about half reported allergies, asthma, and other immune disorders compared to the expected prevalence of each disorder. She also found that parents and siblings of the students were more likely to suffer from these conditions than average-ability individuals (Benbow, 1985, 1986). Further, increased evidence of allergies, autoimmune disease, sensory sensitivity and high IQ has been found in a subset of individuals with ADHD (Chen et al., 2013; Cordeiro et al., 2011) and among those with autism spectrum disorder (ASD) (Gottfried, Bambini-Junior, Francis, Riesgo, & Savino, 2015; Lyall, Van de Water, Ashwood, & Hertz-Picciotto, 2015).

1.3.2. Autism spectrum disorder

The association between highly intelligent individuals and ASD has been recently reexamined but it is certainly not new. In 1943, the original description of classic autism was reported by its pioneer, Dr. Leo Kanner, as children who were highly intelligent and able to remember and reproduce complex patterns but who also displayed "a powerful desire for aloneness" and "an obsessive insistence on persistent sameness" (Kanner, 1943, p. 249). Kanner found a common denominator in the backgrounds of the children he studied: they all came from highly intelligent families. Of the 11 families, four of the fathers were psychiatrists, two had law degrees, and the others included a chemist, a plant pathologist, a professor, an engineer, and a successful business entrepreneur. Nine of the eleven mothers were college graduates. Kanner observed both parents and grandparents as being "obsessive," and "strongly occupied with abstractions of a scientific, literary, or artistic nature" (p. 250). Shortly thereafter in 1944, a German scientist named Hans Asperger described a milder, higher functioning form of the condition described by Kanner, which became known as Asperger's syndrome. Again, the cases he reported were all boys who were highly intelligent but had trouble with social interactions and specific obsessive interests. Asperger called his young patients "little professors" and felt they would be capable of exceptional achievement and original thought later in life (Asperger, 1944/1991; Frith, 1991).

Roughly seventy years later, we are beginning to see evidence that agrees with the early observations of Kanner and Asperger suggesting that enhanced brain functioning may lie at the heart of ASD. In a recent study, by the Psychiatric Genetics Consortium, the polygenic risk for ASD was calculated from genome-wide association studies in a cohort of 9863 individuals in the general population and found polygenic risk for ASD was positively correlated with higher intellectual ability. The specific areas in which they were intellectually superior were logical memory, verbal fluency, and vocabulary. The authors found that even among those who never developed ASD, carrying the complex genetic traits associated with the disorder was linked to scoring better on cognitive tests than controls. This was replicated in the same study in another cohort of 1522 individuals where, again, polygenic risk for ASD had higher full-scale IQ (Clark et al., 2016). Notably, and as reported by Kanner initially, there is a greater incidence of ASD in offspring whose parent(s) are in professions typically associated with a high cognitive ability, particularly those in engineering or the hard sciences (Wheelwright & Baron-Cohen, 2001). This makes a heritable, high intellectual capacity a critical variable to investigate when researching the genetic contributions to ASD, especially given that high intelligence was a consistent variable originally observed by both Kanner and Asperger.

1.4. Hyper brain/hyper body theory

To our knowledge, no studies have examined the potential psychoneuroimmunological interplay between each of the variables herein. Our goal for this exploratory study was to cast a wide net in order to investigate possible associations. We sought to directly address the question, "Is there a relationship between a heightened cognitive capacity (hyper brain) and heightened psychological and subsequent physiological immune responses (hyper body)?" We examined the prevalence of mood and anxiety disorders, ADHD, food and environmental allergies, asthma, autoimmune disease, and ASD in those with high intelligence compared to the national average. We introduce evidence to support our hypothesis that high intellectual capacity is a risk factor for each of the above psychological and physiological conditions and propose the present findings to be in alignment with a novel, hyper brain / hyper body theory.

2. Method

2.1. Participants

Participants were members of American Mensa, Ltd., a society open to persons who have at some point attained a verified score within the upper 2% of the general population on an approved intelligence test that has been professionally administered and supervised. Since there are a large number of tests with different scales, American Mensa, Ltd. has set a percentile rather than a score as the cutoff in order to avoid confusion. Although the American division of Mensa consists of 55,000 members, only 20,000 members belong to the pool of available research subjects. These members have previously agreed to be available to participate in research studies in general and were not directly asked for this study in particular. Since over 5000 members entered the Qualtrics site (4328 members consented to the study), the response rate was between 20 and 25%. For an email survey request with no follow-up, a 25–30% response rate is expected (Kittleson, 1997). Of those recruited, 4931 (approximately 10% of the American Mensan, Ltd. population) responded to the invitation which was sent directly to each individual by American Mensa, Ltd., via an email with a direct link to the survey.

We excluded those who (a) did not provide consent, (b) did not finish the survey, and (c) did not completely view the allergies and/or psychological conditions sections. This left 3715 remaining participants who sufficiently completed the survey for our analysis.

Participants included 2213 that identified as male (60%), 1472 that identified as female (40%), 22 who identified as non-cisgender, and 8 who declined to state. Regarding race, 87.9% of participants reported being European American, 2.7% as Asian American, 2.6% as Hispanic or Latino, 1.6% as African American, and 5.2% as other, mixed race, or who did not respond. The age range of participants was from 18 to 91 years ($M = 53$, $SD = 15.18$). Thus, the majority of participants were older, male, and European American.

Incomes were reported as their highest annual salaries earned rather than their current income to demonstrate potential earnings. Though 92 did not respond, of those that did, 41.7% earned over $100,000; 16.9% earned between $76,000 and $100,000; 20.1% earned between $51,000 and $75,000; 14.9% earned between $26,000 and $50,000; and 3.9% earned less than $25,000. For highest degree earned, 116 did not respond; of those that did, 3.7% earned a doctoral or post-doctoral degree, 38.7% earned a master's degree, 29.2% earned a bachelor's degree, 12.8% earned an associate's degree or completed a trade school or certificate program, 12.4% finished high school with no further education, and 0.1% did not complete any degree. Overall, our sample reported higher education levels and higher income than national averages which supports prior literature citing a relationship between high cognitive ability and positive educational and socioeconomic outcomes (Bergman et al., 2014).

2.1.1. Control group

In order to compare the prevalence of each condition between those with a high cognitive ability against those with normal intelligence, we needed a sample of the latter to act as a control group. Because we were not able to survey a comparative number of participants with a reliably tested intelligence in the average range, national survey data for each condition were used instead. Although the national surveys would likely capture a broad range of intelligence, statistically only 2% of the national data would include those with a gifted cognitive ability such those who qualify for membership in American Mensa, Ltd. For each diagnosis, data from the most recent year was used to compare to the 2015 data collected from Mensa.

2.2. Materials and procedure

After receiving IRB approval from Pitzer College and American Mensa, Ltd., members received an email inviting them to participate in the present study. Participants were provided a link to an online questionnaire. Upon providing consent, participants voluntarily responded to the survey questions, followed by a debriefing page.

The survey consisted of two sections: one for the participant, and another for the participant's children and the child's other biological

Table 1
Prevalence and risk ratios in high intelligence sample compared to the national average.

Conditions	National average		High intelligence		Confidence intervals		*p* value	Risk ratio
	Percent	Frequency	Percent	Frequency	Lower	Upper		
Mood disorders								
Diagnosed	9.5%	352	26.8%	995	0.25	0.28	< 0.001	2.82
Combined			36.6%	1361	0.35	0.38	< 0.001	3.85
Anxiety disorders								
Diagnosed	10.9%	405	20.0%	743	0.19	0.21	< 0.001	1.83
Combined			37.3%	1387	0.36	0.39	< 0.001	3.42
ADHD/ADD								
Diagnosed	4.1%	152	7.4%	275	0.07	0.08	< 0.001	1.80
Combined			13.9%	517	0.13	0.15	< 0.001	3.39
ASD								
Diagnosed	1.0%	037	1.2%	44	0.01	0.02	< 0.001	1.20
Combined			6.3%	235	0.06	0.07	< 0.001	6.30
Food allergies								
Diagnosed	3.7%	137	9.6%	357	0.09	0.11	< 0.001	2.59
Combined			15.0%	556	0.14	0.16	< 0.001	4.05
Environmental allergies								
Diagnosed	10.6%	394	33.2%	1232	0.32	0.35	< 0.001	3.13
Combined			45.9%	1706	0.44	0.48	< 0.001	4.33
Asthma								
Diagnosed	7.4%	275	15.4%	573	0.14	0.17	< 0.001	2.08
Combined			17.3%	641	0.16	0.19	< 0.001	2.34
Autoimmune disease								
Diagnosed	8.0%	297	14.7%	546	0.14	0.16	< 0.001	1.84
Combined			16.0%	594	0.15	0.17	< 0.001	2.00

Note: Mood disorders include depressive disorder, dysthymic disorder, and bipolar disorder. Anxiety disorder includes generalized anxiety, social anxiety, and obsessive compulsive disorder. Autism spectrum disorders (ASD) include the DSM-IV diagnoses of autism, Asperger's syndrome, and other pervasive developmental disorders. The "combined" category includes individuals that suspect they have the disorder (self-diagnoses) in addition to those who were diagnosed by a medical professional. Confidence intervals are for the percent of diagnosed individuals with high intelligence (and those with combined diagnoses). Risk ratio indicates the risk for those with high intelligence in having each disorder compared to the national averages.

parent. Only the data specifically related to the participants themselves were used for this study. Within each section, participants first answered demographic questions about their age, gender, educational and occupational background, and IQ scores. They also answered questions about other factors, such as average stress levels and sleep habits, on a 5-point scale of *very unhealthy* to *very healthy* (analysis of factors unrelated to overexcitabilities were not included in this analysis).

Participants were then asked to indicate whether they have been either (a) *diagnosed* or (b) *suspected* they should be diagnosed with a variety of diseases, syndromes, and dysregulations with the item: *"For each syndrome, disease, or disorder you have, please indicate if it was formally diagnosed by a medical professional or only suspected to exist."* Distinguishing between diagnosed and suspected conditions was important because many do not seek medical help for certain conditions, preferring instead to self-diagnose and manage them independently. For example, only about half of people with depressive symptoms seek medical treatment (González et al., 2010). Therefore, we felt it was pertinent to include these individuals in our analysis to provide a more complete representation of prevalence. Further, survey items asked about those conditions they *currently* had, which ensures *point* prevalence rather than *lifetime* prevalence of each examined condition.

Examined conditions included a wide variety of disorders, from allergies to sleep apnea. This study included analysis on those disorders for which we had evidence suggesting a relationship between them and high cognitive ability. Unrelated conditions, such as sleep apnea, were ultimately not included in the final analysis. Overall, two broad categories of disorders emerged: psychological and physiological conditions. Psychological disorders included affective disorders (mood and anxiety), ADHD, and ASD. Mood disorders included major depressive disorder, dysthymic disorder, and bipolar disorder. Anxiety disorders included generalized anxiety, social anxiety, and obsessive-compulsive

disorder. These affective disorder subcategories correspond with those included in our control prevalence data (NIMH). All psychological disorders aligned with DSM-IV diagnoses (American Psychiatric Association, 2000) which was the current manual at the time of the survey development as well as for the comparative national surveys. Importantly, ASD diagnoses rely on the DSM-IV. The changes in the DSM-5 (American Psychiatric Association, 2013) radically impacted the diagnosis of ASD, which formerly differentiated between autism, Asperger's syndrome, and other pervasive developmental disorders (PDD). Currently Asperger's is now labeled *high functioning autism.* However, since this survey and the national surveys used for comparison use the older term, this study uses *Asperger's syndrome* rather than the DSM-IV terminology. Because ASD is listed in the DSM-IV, we have listed it as a psychological overexcitability. However, it is also included herein as a physiological overexcitability given the immunological processes at play that have been demonstrated in the scientific literature.

Physiological diseases included allergies (food and environmental), asthma, and autoimmune dysregulations including those found in ASD. For both food and environmental allergy categories, we asked participants to differentiate whether they have an allergy versus a sensitivity or intolerance. We focused on those who reported a true allergy in order to more accurately compare with the national average. As a collection of diseases, those that were autoimmune in nature were the most difficult to define and compare, as there are several agencies that report the prevalence of autoimmune disease. The NIH is one of the more conservative organizations; they point to approximately 80 diseases that occur as a result of the immune system attacking the organs, tissues, and cells of the body, however their statistics only included 24 diseases for which good epidemiology studies were available (U.S. Department of Health and Human Services, 2002), compared to over 100 diseases included by the AARDA. However, since not all scientists

agree with the classification of these other diseases as autoimmune conditions, we chose to use the NIH standards as a more conservative and reliable estimate.

3. Results

This study examines the prevalence of several disorders in those with higher intelligence compared to those with average abilities. For this analysis, we conducted a series of binomial tests which compares observed proportions in the study sample (those with tested high intelligence) to hypothesized values (national averages). For example, according to the NIH (Kessler, Chiu, Demler, & Walters, 2005) the prevalence rate for ADHD is 4.1%. Given the total sample size of 3715, we would expect 152 participants to have been diagnosed with this disorder. However, the actual frequency of ADHD diagnoses within this study's participant sample was 239 diagnoses (7.4%), which is significantly greater (exact binomial $p < 0.001$). See Table 1 for a comparison of prevalence between those of high intelligence and the national averages across all conditions.

Significance for this test indicates that there is a higher proportion of individuals with high intelligence with the specified disorder compared to what one would expect, given the national averages, suggesting that high cognitive ability may be a risk factor for the specified disorder. Due to the number of the tests being performed, there is an increased probability of committing at least one Type I error. Thus, we used Bonferroni's correction ($\alpha = 0.05/16$), resulting in a corrected $\alpha = 0.0031$. As can be seen in Table 1, the prevalence of all eight disorders were significantly greater in the high intelligence sample compared to the national averages. These results held true for both diagnosed disorders and for the combination of diagnosed and suspected disorders.

The 95% confidence intervals (CI) provide further support for these significant findings, as none of the interval ranges include zero. In addition, all of the CI ranges are narrow, indicating low error rates. As shown in Table 1, CI ranges are > 3%, indicating that the reported prevalence for those with high intelligence in each category is accurate within approximately ± 1.5%.

However, significance and confidence intervals are impacted by sample size, and this study's large sample makes finding significance unsurprising, even after correcting for the number of calculations. Therefore, effect size is particularly useful to explore. For effect sizes, we reported the relative risk, or risk ratio (RR), which indicates the probability of being diagnosed with the specified disorder for an individual with high cognitive ability (observed) compared to an individual with average intelligence (hypothesized). This is calculated as

Relative Risk (RR) = $P_{observed}$ / $P_{hypothesized}$

An RR of 1 indicates that there is no difference between groups; an RR > 1 indicates that there is a higher risk for those with higher intelligence. For example, an RR of 2.5 indicates that an individual with high cognitive ability has 2.5 times the risk of developing a particular disorder as compared to the norm. This represents an increased risk of 150% of developing the disorder above those representing the national average. As can be seen in Table 1, all disorders held increased risks of both diagnoses and self-diagnoses among those of high cognitive ability compared with national averages.

3.1. Psychological disorders

In this study, mood disorders included depressive disorder, dysthymic disorder, and bipolar disorder. Estimates of mood disorders within the adult population range from 8.4% to 12% (Olfson, Blanco, & Marcus, 2016; Pinto Pereira, Geoffoy, & Power, 2014). As expected, there was a higher incidence of mood disorders in the high intelligence sample; 17.3% more than the national average as reported by the NIMH (Kessler et al., 2005). The associated RR is 2.82, which indicates an

increase of risk by 182% for those of high intelligence in developing at least one of these mood disorders. For the combination of diagnosed and suspected diagnoses, there was 3.85 times the risk (an increase of 285%).

Anxiety disorders in this study included generalized anxiety, social anxiety, and obsessive compulsive disorder (OCD). As hypothesized, there was a greater prevalence of anxiety disorders among those with high intelligence: 9.1% more than the national average when limited to these three categories (Kessler et al., 2005). Overall, there was 1.83 times the risk of being formally diagnosed with anxiety (an increase of 83%). When including those who suspected that they should be diagnosed, there was a risk of 3.42, an increase of 242% as compared to the national average.

Prevalence of ADHD (and the older diagnosis of ADD) was significantly greater in the high intelligence sample—an increased prevalence of 3.3% compared to the national average (U.S. Department of Health and Human Services, 2002). Overall, those of high cognitive ability are 1.8 times as likely to be diagnosed with ADHD compared to the national average (an 80% increase). Further, when combined with those who suspected diagnoses, there was 3.39 times the risk of developing ADHD symptoms, a 239% increase compared to the national average.

Autism spectrum disorder (ASD) includes the DSM-IV diagnoses of autism, Asperger's syndrome, and other PDDs. ASD is typically only studied in children, thus rendering comparisons between prior studies and this sample difficult.

Our sample consisted of adults born in the years 1924 to 1997 ($M = 1962$). Blaxill (2004) found that most diagnosis occurred around ages 5 and 6, suggesting that the rates for the 2000s would be the highest appropriate estimate for our sample. Thus, the highest estimates for prevalence rates for our participants, as children, would be no higher than .57%. However, in recent years these rates have increased. For example, Blumberg et al. (2013) report a rate of 2% for diagnoses in 2007 to 2011, and Christensen et al. (2016) report rates of 1.46% in 2012. As a compromise between these diverse rates, and in the event the lower diagnoses rates from earlier years included errors caused by lower diagnosis rates rather than a lower prevalence, this study used a conservative 1% (100 per 10,000) as a comparison rate rather than the lower estimate of .57%. This figure corresponds with the only known study to investigate ASD in adults which came out of England and found the weighted prevalence of ASD in adults was estimated to be 9.8 per 1000 or just under 1% (Brugha et al., 2011). We were not able to specifically use this resource as an official comparison due to the fact that we were measuring our data against national averages in the United States.

Although only Asperger's is characterized by high intelligence in the DSM-IV, this study's combined ASD sample included 0.2% more than expected, using this conservative estimate. Though the increase was small, it was still significant (see Table 1). When comparing diagnosed patients, there was 1.2 times the risk within the high intelligence group compared with national averages (a 20% increase). However, an additional 5.1% suspected that they should be diagnosed with ASD, for a total combined risk of 6.3 times the national average, an increase of 530%.

3.2. Physiological diseases

Physiological overexcitabilities include diseases and dysregulations that are primarily physical in nature—allergies, asthma, and autoimmune diseases. The data on food allergies in adults is limited; most studies center around rates in childhood. Liu et al. (2010) estimates rates of 2.5% for adults in 2005–2006, but here we used the higher, more conservative rate of 3.7% from the U.S. Department of Health and Human Services (2002) since childhood rates have been increasing over time (CDC, 2013–2015). Food allergies had an increase of 5.9% in the high cognitive ability sample, for an RR of 2.59, indicating an increased

risk of 159% compared to the national average. For those who self-diagnosed food allergies, there was an increased risk of 159% (RR = 2.59).

Studies of environmental allergies are likewise often on children. Estimates for adults in recent studies range from 7.3–14.7% (Blackwell, Lucas, & Clarke, 2014; CDC, 2015; Han, Forno, Gogna, & Celedón, 2016; Schiller, Lucas, Ward, & Peregoy, 2012). Using the moderate rate of 10.6%, we found that the rate of environmental allergies increased by 22.6% in the high intelligence sample compared to the national average. Overall, there was 3.13 times the risk of being diagnosed with an environmental allergy, an increase of 213%, for those with high cognitive ability. When included self-diagnoses, there was 4.33 times the risk, for an increase of 333%.

For asthma, there was an 8% increase in the number of diagnoses in those with high cognitive ability compared to national averages, as reported by the CDC (U.S. Department of Health and Human Services, 2014). Rates have remained steady in recent years, between 7.4 and 8% (Blackwell et al., 2014; CDC, 2015; Schiller et al., 2012). There was 2.08 times the risk of diagnoses, for an increase of 108%. When including self-diagnoses, there was 2.34 times the risk, for an increase of 134%.

Lastly, for autoimmune diseases, there was an increase of 6.7% diagnoses in the high intelligence sample compared with the estimates provided by the NIH (U.S. Department of Health and Human Services, 2002). Those with higher cognitive ability were 1.84 times as likely to be diagnosed with at least one of the two dozen disorders included this category, for an increased risk of 84%. When combined with self-diagnoses, there was 2 times the risk for this in the sample, a 100% increase of symptomatology compared with the national average.

3.3. Auxiliary analyses

Given that several of the original categories of conditions were comprised of multiple disorders (e.g., anxiety disorders), where possible we decided to conduct auxiliary analyses to see if the above pattern of significance held true for these specific disorders. For these additional six analyses, we applied a second Bonferroni's correction ($\alpha = 0.05/6$), resulting in a corrected $\alpha = 0.008$. For all analyses, individuals with co-morbid, or overlapping, diagnoses within the same
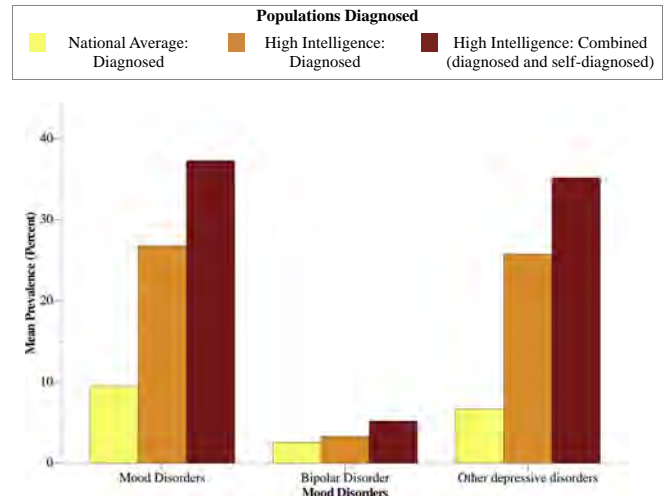

Fig. 1. Prevalence comparisons between the high intelligence sample and the national average for all mood disorders, bipolar disorder, and other depressive disorders including major depression and dysthymic disorder. For all mood disorders, there was a significant ($p < .001$) increase in prevalence in individuals with high intelligence.

category (e.g., mood disorders) were counted once within the category but multiple times for each specific disorder. For example, if an individual indicated a diagnosis of major depression and a suspicion of bipolar disorder, they would be counted as having been diagnosed with a mood disorder, diagnosed with major depression, and included in the combined category of bipolar disorder. See Table 2 for the comparative prevalence, 95% CIs, significance values, and RRs.

3.3.1. Mood disorders

In order to examine these results in more detail, as recommended by the NIMH, we separated those with bipolar disorder from those with other depressive disorders (see Fig. 1). This separation is important because bipolar disorder includes depressive episodes, but is markedly different from other depressive disorders, given the additional of manic episodes. Additionally, while rates for major depression have remained relatively stable in recent years (Baxter et al., 2014), rates for bipolar

Table 2
Auxiliary analysis measuring the prevalence and risk ratios for mood, anxiety and social disorders comparing the high intelligence population and national averages.

Conditions	National average		High intelligence		Confidence intervals		p value	Risk ratio
	Percent	Frequency	Percent	Frequency	Lower	Upper		
Bipolar Disorder								
Diagnosed	2.6%	97	3.3%	124	0.03	0.04	0.004	1.28
Combined			5.2%	194	0.04	0.06	< 0.001	2.01
Other depressive disorders								
Diagnosed	6.7%	249	25.8%	957	0.24	0.27	< 0.001	3.85
Combined			35.2%	1308	0.34	0.37	< 0.001	5.25
Generalized anxiety								
Diagnosed	3.1%	115	17.8%	662	0.17	0.18	< 0.001	5.74
Combined			27.7%	1030	0.26	0.29	< 0.001	8.94
Social anxiety								
Diagnosed	6.8%	253	6.1%	226	0.05	0.07	0.043	0.90
Combined			19.5%	498	0.18	0.21	< 0.001	2.87
OCD								
Diagnosed	1.0%	37	3.3%	121	0.03	0.04	< 0.001	3.30
Combined			10.9%	404	0.10	0.12	< 0.001	10.90
Asperger's syndrome								
Diagnosed	0.026%	1	1.1%	41	0.01	0.01	< 0.001	42.31
Combined			5.8%	216	0.05	0.07	< 0.001	223.08

Note: The "combined" category includes individuals that suspect they may have the disorder (self-diagnoses) in addition to individuals that were diagnosed by a medical professional. Confidence intervals are for the percent of diagnosed individuals with high intelligence (and those with combined diagnoses). The risk ratio indicates the risk for individuals with high intelligence possessing the disorder compared to the national averages. With Bonferroni's correction, significance assessed at $\alpha = 0.008$.
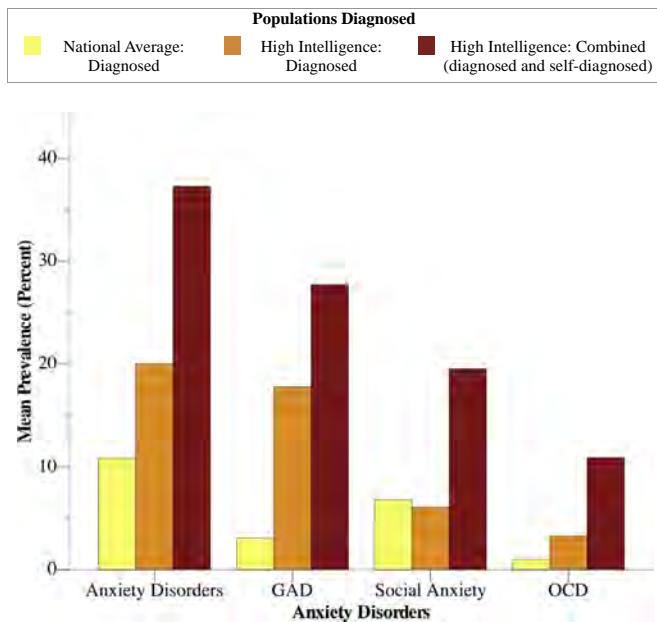
Fig. 2. Prevalence comparisons between the high intelligence sample and the national average for selected anxiety disorders: generalized anxiety disorder (GAD), social anxiety, and obsessive compulsive disorder (OCD). For GAD and OCD there were significant ($p < 0.001$) increases in prevalence for those with high intelligence. For social anxiety, there were no significant differences in clinical diagnoses between those of high intelligence and the national average. However, there was a significant increase of prevalence when including those who suspected diagnoses.

have been increasing (Harpaz-Rotem, Leslie, Martin, & Rosenheck, 2005; Harpaz-Rotem & Rosenheck, 2004; Moreno et al., 2007). Individuals with bipolar disorder as a diagnosis comprised of 3.3% of the sample (124 individuals), and increased to 194 (5.2%) when combined with self-diagnoses. In comparison, the NIMH reports a prevalence rate of 2.6% (Kessler et al., 2005). For diagnoses of bipolar disorder, the difference between those with high cognitive ability and the national average was significant (95% CI [0.03, 0.04], RR = 1.28, $p = 0.004$). When including those who had suspected diagnoses, the relationship was also significant (95% CI [0.04, 0.06], $p < 0.001$), such those with high intelligence are more likely to have bipolar symptoms. Thus, when including those who suspected they had bipolar disorder, there was 2.01 times the risk for those of high intelligence (an increase of 101%).

Other depressive disorders had a diagnosis rate of 25.8% (957 individuals) among the high intelligence sample, which increased to 1308 (35.2%) when combined with self-diagnoses. The NIMH (Kessler et al., 2005) reports that 6.7% of the national population experiences at least one major depressive episode each year. This is consistent with the 2015 data from the NIMH, suggesting that rates of major depression have remained steady for the last decade (Substance Abuse and Mental Health Services Administration, 2015). For diagnoses of other depressive disorder, the binomial was significant ($p < 0.001$), indicating a higher prevalence for those of high intelligence in comparison to national average, 95% CI [0.24, 0.27]. There was 3.85 times the risk (an increase of 285%) for those of high intelligence of being diagnosed. When including those who suspected depression, there was also a significant increase (95% CI [0.34, 0.37], $p < 0.001$). There was 5.25 times the risk (and increase of 425%) for those of high intelligence of experiencing symptoms of major depression to the extent of suspecting either major depressive disorder or the longer-lasting dysthymic disorder.

3.3.2. Anxiety disorders

Like the mood disorders, we separated the anxiety disorders in order to see if there were differences among the three types (see Fig. 2). For

generalized anxiety disorder (GAD), there were 662 diagnoses (17.8%), which increased to 1030 (27.7%) when combined with self-diagnoses. According to the NIMH (Kessler et al., 2005), there is a 3.1% national prevalence rate, though only 52.3% of these are receiving treatment. For diagnoses of GAD, the binomial was significant ($p < 0.001$), indicating a higher prevalence of GAD in the high intelligence sample, 95% CI [0.17, 0.19]. For the study participants, there was 5.74 times the risk (an increase of 474%) of being diagnosed with GAD compared to the national average. When combined with self-diagnoses, the binomial was also significant in the same direction (95% CI [0.26, 0.29], $p < 0.001$). There was 8.94 times the risk (an increase of 794%) of experience GAD symptoms for those of high intelligence compared to the national average.

For social anxiety, there were 226 diagnoses (6.1%), which increased to 498 (19.5%) when combined with self-diagnoses. According to the NIMH (Kessler et al., 2005), there is a 6.8% national prevalence rate, though only 45.6% are seeking treatment. For diagnoses of social anxiety, the binomial was not significant when taking into account Bonferroni's corrections (95% CI [0.05, 0.07], $p = 0.043$). There was 0.9 times the risk of being diagnosed with social anxiety, a decrease of 10% compared to the national average. However, when including self-diagnoses, the binomial was significant (95% CI [0.18, 0.21], $p < 0.001$), such that there was a higher prevalence of those who also self-diagnosed social anxiety compared to the national average. There was 2.87 times the risk (an increase of 187%) for those of high cognitive ability to experience symptoms of social anxiety compared to the national average.

For OCD, there were 121 diagnoses (3.3%), which increased to 404 (10.9%) when combined with self-diagnoses. According to the NIMH (Kessler et al., 2005), there is a 1.0% national prevalence rate. For diagnoses of OCD, the binomial was significant ($p < 0.001$), such that there was a higher incidence in those with high intelligence, 95% CI [0.03, 0.04]. There was 3.3 times the risk for those with high intelligence (an increase of 230%) of being diagnosed with OCD compared to the national average. When combined with self-diagnoses, the binomial was also significant, ($p < 0.001$), such that there was a higher incidence in those with high intelligence, 95% CI [0.10, 0.12]. There was 10.9 times the risk for those of high intelligence (an increase of 990%) of developing OCD symptomatology compared to the national average.

3.3.3. Autism spectrum disorders

ASD as a combination of disorders deserves special attention, as it uniquely contains those who are often miscategorized as low functioning or low intelligence. For example, Christensen et al. (2016) found that of children with symptoms of ASD, only 43.9% were classified as having an IQ score above 85. Of our participants, 10 (0.3%) identified as diagnosed with autism (37 or 1% when combined with self-diagnoses) and 1 as PDD (5 or 0.1% when combined with self-diagnoses). In contrast, 41 (1.1%) identified themselves as having been diagnosed with Asperger's syndrome, and when combined with self-diagnoses, this number rose to 216 (5.8%). The traditional diagnosis of autism occurs approximately 4–10 times as frequently as Asperger's (Christensen et al., 2016; Fombonne, 2005), and thus the proportion of those with autism, Asperger's, and other PDDs are markedly different between the national averages and this study's sample.

When isolating the 2005 rates of Asperger's (0.026%; Fombonne, 2015), there were significant increases in diagnoses rates among those in this study ($p < 0.001$), with 42.31 times the risk (an increase of 4131% for those of high intelligence). When combined with self-diagnoses, these numbers increase impressively (95% CI [0.05, 0.07], $p < 0.001$). There is 223.08 times the risk for those of tested high intelligence of self-diagnosing Asperger's syndrome, an increased risk of 22,208% compared to the national average (see Fig. 3).

Overall, we found there to be increased risks for every examined condition in those with high intelligence compared to those among the
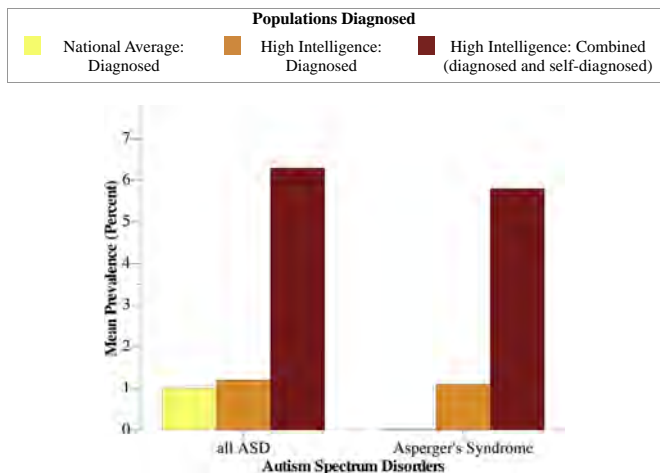
Fig. 3. Comparisons of the prevalence of Autism Spectrum Disorder were significant for both ASD as a category (as described by the DSM-5) and when isolating for Asperger's Syndrome (as described by the DSM-IV).

national data (see Fig. 4). This pattern holds true for both self-diagnoses and clinical diagnoses. Thus, our hyper brain/hyper body hypothesis was supported.

4. Discussion

Fig. 5 is a novel, visual representation of our hyper brain/hyper body theory of integration. The model posits a unique psychoneur-oimmunological process such that those with a hyper brain in the form of very superior (at or above 130) cognitive ability lend themselves to a greater tendency to respond to environmental stressors by ruminating and worrying which are positive predictors of risk for psychological overexcitabilities leading to affective disorders. These disorders are closely associated with a hyper body which manifests in physiological overexcitabilities which take the form of immune and inflammatory dysregulation, which can also bi-directionally instigate psychological effects. In order to test our hyper brain/hyper body theory, we looked at between-group differences by comparing those with verified intelligence falling at or above the 98th percentile against the national average prevalence of the disorders and conditions under investigation. High statistical significance and a remarkably high relative risk ratio of diagnoses for all examined conditions were confirmed in the Mensa

group. The present results strongly support our theoretical framework, demonstrating that highly intelligent individuals are at a significantly greater risk for mood and anxiety disorders, ADHD, and conditions involving inflammation and dysregulation of the immune system such as allergies, asthma, autoimmune disease, and ASD when compared to national averages. Many of these conditions tend to co-occur/overlap making it challenging to discuss them solely on an individual basis. With this in mind, we suggest several possible explanations for our findings.

4.1. Intellectual overexcitabilities

The examined conditions may be, in part, a result of the over-excitabilities found among the intellectually gifted as first introduced by Dabrowski (1964a, 1964b, 1966, 1976). These exist within five specific domains: psychomotor, sensory, intellectual, imaginational, and emotional. According to his research, highly intelligent individuals may or may not possess all of them and may find themselves to be considerably stronger in one area over another. Some excitabilities within high IQ can even be diagnosed as medical conditions (eg. someone with a psychomotor OE being misidentified as having ADHD). Thus, it is beneficial to consider OEs as part of a comprehensive assessment especially for gifted children who will grow up to be gifted adults. Since Dabrowski first developed his theory, there have been several scales created to measure these domains (Rost, Wirthwein, & Steinmayr, 2014) and there is evidence to suggest that a reliable OE scale, such as the ElemenOE, can assist in effectively screening gifted students who were not identified by traditional methods such as an IQ test (Bouchard, 2004). The scale can also be used to complement other methods in providing effective counseling for children and adults with gifted intelligence (Mendaglio & Tillier, 2006). Other theories involving the role of overexcitabilities in both human and animal models have been proposed, including that of the intense world theory (IWT) in those with ASD (Markram & Markram, 2010).

4.2. Psychological overexcitabilities: affective disorders and ADHD

As shown in Figs. 1 and 2, participants with very superior (130 and above) intelligence were significantly prone to rumination leading to mood disorders, and worry leading to anxiety disorders. However, the relationship between genius and madness is not a new notion. It is well known that many historical intellectuals such as Leonardo da Vinci, Sigmund Freud, Albert Einstein, and Pablo Picasso were plagued by overexcitabilities leading to documented pervasive affective and mood
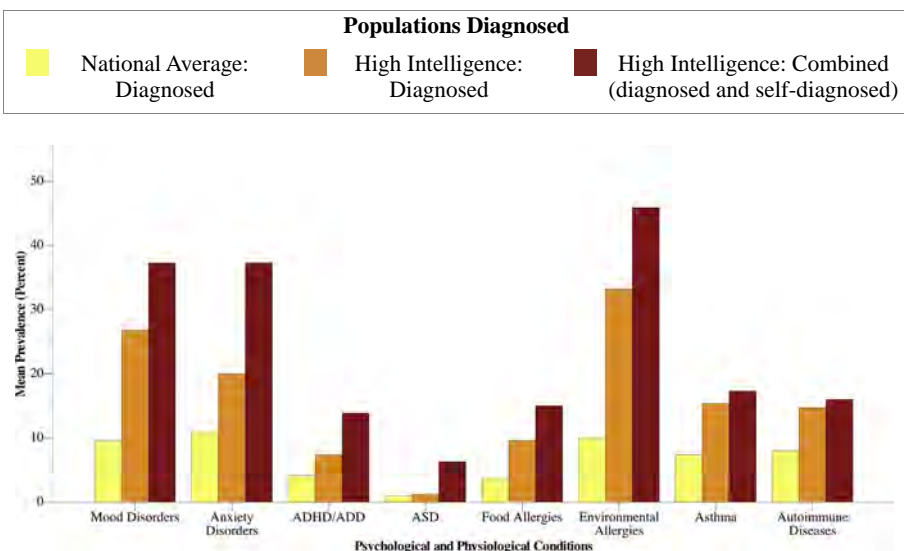

Fig. 4. Prevalence of diagnoses for those of high intelligence compared to the national average. For all measures including mood disorders, anxiety disorders, ADHD, ASD, food allergies, environmental allergies, asthma, and autoimmune disease, there are significant ($p < 0.001$) increases in the number of diagnoses in individuals possessing high cognitive ability.
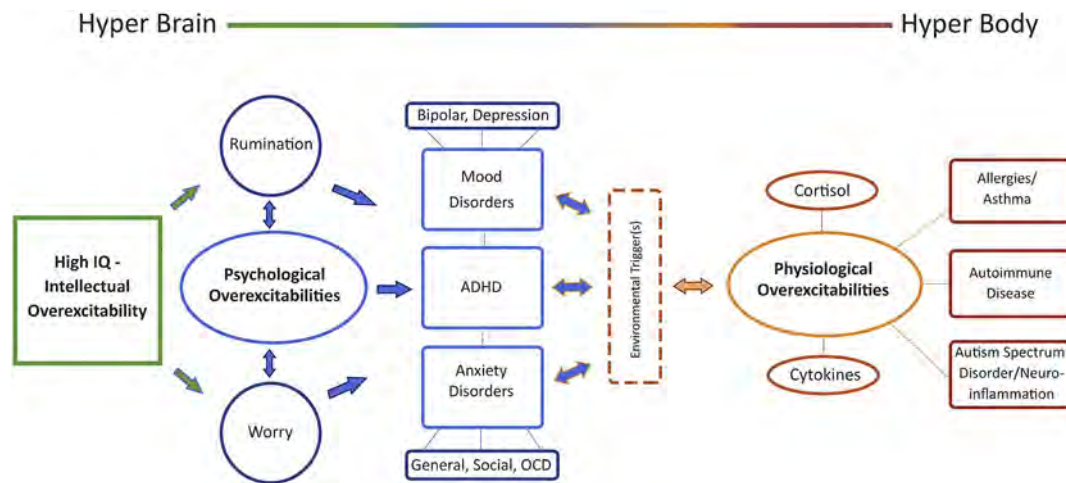
Fig. 5. Hyper Brain/Hyper Body: a theoretical framework.

disturbances (Ehrenwald, 1984). Sir Isaac Newton viewed his gifted creativity as a product of intense, prolonged rumination. He brooded over past mistakes and worried excessively which eventually led him to suffer a nervous breakdown in 1693 (Westfall, 1981). Those who surround such individuals tend to be overwhelmed by their intense way of seeing and feeling "too much." This can lead to increased familial and relational conflicts (Morawska & Sanders, 2009) and painful isolation from peers (Gallagher, 1958), causing a crisis of self as they attempt to minimize their responses to better adapt and fit in with, the vast statistical majority around them who possess an average IQ. This struggle begins from a young age and, given that IQ remains relatively constant can continue for a lifetime.

There are various theories about which neurological mechanisms and processes are involved in developing mood disorders in those with hyper brains. The field of PNI points us to one of the better-understood and most likely networks that may be at play. That being the impact that environmental and psychological stress has on the hypothalamic-pituitary-adrenal (HPA) axis, particularly stress that is chronic as opposed to brief. The HPA readies the body for a "fight, flight, or freeze" response in times of stress. It does not distinguish between real or perceived threats. If there is chronic activation of this system, it contributes to wear and tear on the body and organs (McEwen, 2000). It is possible that the overexcitabilities present in those with a high cognitive ability and the chronic mental activation which they experience in response to their environment may continually activate the HPA axis. These chronic stress-induced brain-immune interactions reduce the ability of the immune system to do its vital work. The brain (nervous system) regularly cross talks with, and has a measurable effect on, the strength of the immune system. It stands to reason that a hyper brain (high IQ), with its overexcitabilities, could be miscommunicating these perceived stressors/threats more often and more intensely than the general population. The studies that point to the overexcitabilities experienced by the intellectually gifted, as well as those that find high IQ to be associated with negative mental and immunological health outcomes, lend credibility to this relatively new line of research and to our hyper brain/hyper body theory.

It has been demonstrated in the literature that an overconnectivity of local brain networks facilitates rumination. For example, Leuchter, Cook, Hunter, Cai, and Horvath (2012) used weighted network analysis to measure connectivity in depressed participants and found that individuals with major depressive disorder showed increased synchronization across all frequencies of neural electrical activity. They were observed to have significantly enhanced connections between most areas of the brain with electrical signals that do not seem to shut off. Interestingly, the greatest degree of heightened connectivity was found to be in the seat of cognitive ability, the prefrontal cortex. Another theory points to the default network as an example of a highly interconnected brain system. This network takes what is going on inside of us and then uses that information to assess what is going on around us, allowing us to envision the future and react according to our judgments about our experiences (Hamilton, Farmer, Fogelman, & Gotlib, 2015; Raichle et al., 2001). It is normally active when the mind wanders off and then it shuts down when an individual starts concentrating on a task. Those who have mood disorders seem to have connections and networks that stay excitable instead of shutting down, rendering their nervous system unable to fully relax (Anticevic et al., 2012).

Depression and anxiety are found to be associated with a hyper-inflammatory response via pro-inflammatory cytokines (Tian, Hou, Li, & Yuan, 2014) indicating that intense and/or prolonged stress can influence immunity. For example, Glaser et al. (1992) found among medical students taking exams, that extreme stress and their perception of social support affected their immune responses to the hepatitis B vaccine. If medical students, who are extremely likely to possess a high cognitive ability, produced weaker immune responses to a vaccine then we might conclude these same individuals who are chronically stressed would be slower to develop a proper immune response to various other insults and pathogens as well (Padgett & Glaser, 2003). A similar result was observed among those who were under chronic stress caring for a spouse with Alzheimer's disease such that they displayed a poorer antibody response to an influenza virus vaccine (Kiecolt-Glaser et al., 1996). A potential role for brain cytokine elevations in depression has been further confirmed in studies using rodent models of the disorder showing that psychosocial stressors up-regulated expression of cytokines in the prefrontal cortex and hippocampus (Audet, Jacobson-Pick, Wann, & Anisman, 2011; Audet, Mangano, & Anisman, 2010). Among the few studies examining brain cytokine variations associated with depressive illness in humans, several were reported to be up-regulated in the postmortem PFC of patients with major depression (Dean, Tawadros, Scarr, & Gibbons, 2010; Shelton et al., 2011) and in postmortem PFC of teens that died by suicide (Pandey et al., 2012). Although not all tissue had come from those who had a prior diagnosis of major depression, we might strongly suppose that they more than qualified as being depressed at the point in time when the suicide occurred.

It has been demonstrated that mood and anxiety disorders precede immune responses including allergies and/or asthma. In one such study, a longitudinal cohort was followed for eight years to discover potential associations between allergies and major depression. Findings supported major depression as being associated with an increased risk of developing non-food allergies, but results in the opposite direction could not be confirmed. Thus the results of the study did not support the hypothesis that a psychosocial impact of allergies increases the risk of

depression but instead concluded that the observed effect may be due to shared genetic, epigenetic, or immunological responses that occur during major depression (Patten & Williams, 2007; Patten, Williams, Lavorato, & Eliasziw, 2009). The results of the present study point to one genetic component to consider being that of high IQ.

Prevalence of ADHD was significantly greater in our high intelligence sample. Overall, for those with high cognitive ability there is 1.8 times the risk for being diagnosed with ADHD compared to the national average. The psychomotor OE is especially important to highlight in regards to the significant ADHD prevalence within our sample. Our finding lends support to the potential problem of children and adults possessing this particular overexcitability who may be likely to be given an ADHD diagnosis which may interfere with also being properly identified as having high intelligence (Nelson, Rinn, & Hartnett, 2006; Rinn & Reynolds, 2012).

4.3. Physiological overexcitabilities: allergies, asthma, and autoimmune disease

Our findings show a significant increase of risk for allergies, asthma, and autoimmune disease in high cognitive capacity individuals which lends weight to the hyper body component of the presently proposed theory. Researchers have implicated inflammation as being at the forefront of many of the above conditions and also of psychiatric disorders (Couzin-Frankel, 2010; Slavich, 2015). It is known to contribute to the onset and progression of immune disease, including allergic and autoimmune diseases. Low-grade inflammation is distinct from acute inflammation such as fever, infection, and swelling. It is a systematic and long-term phenomenon that is more sensitive and susceptible to stress (Känel et al., 2007; Rohleder, 2014). Alterations in the pro-inflammatory cytokines IL-6 and TNFα in rodents may indicate a pathophysiological pathway from acute and chronic stress to the development of depression. IL-1 and TNFα in the lungs can cause inflammation and allergic and asthmatic reaction, and changes in IL-4 and IL-10 may link acute and chronic stress to allergies and autoimmune diseases (Himmerich et al., 2013). These atopic consequences have been observed in those with affective disorders and also in those diagnosed with ADHD. In a study of 8201 participants identified as having ADHD, there was an increased prevalence of allergic diseases, including asthma, allergic rhinitis, atopic dermatitis, and urticaria (hives), compared with the control group. Lind, Nordin, Palmquist, and Nordin (2014) looked at levels of stress, exhaustion, and anxiety of participants and found that they reported higher prevalence of allergic asthma and atopic dermatitis. Kovács, Stauder, and Szedmák (2003) found that 32.2% of those with allergies scored above normal levels of depression with 12.5% reporting clinically significant depressive symptomatology. Also found was that the higher the level of depression, the higher the reported allergic symptomatology.

Prior research suggests an interplay between genetics and the environment in disorders involving immune dysregulation (Goines & Ashwood, 2013; Nevison, 2014). We know that identical twins are more likely to suffer from the same autoimmune disease than fraternal twins (Wang, Wang, & Gershwin, 2015). We also know that relatives of patients with autoimmune diseases are at higher risk for development of the same or another autoimmune disease which supports a genetic basis (Cárdenas-Roldán, Rojas-Villarraga, & Anaya, 2013). That said, the concordance of autoimmune disease in identical twins is often in the 25 to 40% range. This supports the idea that the etiology of autoimmune disease likely involves both genetic and environmental factors (Campbell, 2014; Mazmanian, Round, & Kasper, 2008).

4.3.1. Immune and inflammatory dysregulation in ASD

The significant prevalence of ASD, especially of Asperger's, in our sample with high intelligence, agrees with prior literature that has already demonstrated an association between high IQ and ASD individuals (Clark et al., 2016). Presently, the CDC estimates that one in

every 68 children (approximately 1%) are diagnosed with ASD (Christensen et al., 2016). This is up exponentially from the 2–5 in 10,000 children recorded in the 1980s (Smalley, Asarnow, & Spence, 1988). Younger ages at diagnosis, differential migration, changes in diagnostic criteria, and inclusion of milder cases can not fully explain these observed increases (Hertz-Picciotto & Delwiche, 2009). We feel it is relevant to consider this data, as it indicates the strong possibility of one or more environmental factor(s) being introduced in the middle to late 1980s when the ASD prevalence began to dramatically rise (Nevison, 2014). This relates to the present study because it is known that certain environmental triggers combined with a unique genetic predisposition may launch immunological/inflammatory responses in certain susceptible individuals (Rossignol & Frye, 2012). Researchers are still trying to understand what that genetic component might be which sets certain at-risk individuals apart from those who do not respond in the same way to the same environmental insult(s). The results of the present study suggest that one such plausible genetic component to consider is that of high IQ which may be responsible for a hyper physiological response to these insults and a resulting development of ASD.

4.3.2. The intense world theory of overexcitability in ASD

In order to explain the overexcitabilities found in those with ASD, Markram and Markram (2010) proposed the intense world theory (IWT), a hyper-functioning of local neural microcircuits, characterized by hyper-reactivity and hyper-plasticity. Much of the traditional autism research has posited an intellectual disability in the form of mental retardation or under-reactivity. However, Markham and his colleagues could not find malfunctions in the inhibitory synapses, but did find too many excitatory connections were being formed in the neocortex of autistic animal models (Markram, Rinaldi, & Markram, 2007). In the same study they discovered the circuit responded excessively when a stimulus was introduced and that the synapses learned about the stimulus much faster and more fluidly than normal. This excessive reactivity and rapid memory formation of experiences are boosted by an amplified emotional component. Further behavioral studies on autistic animal models found that they developed excessive fear memories that lasted longer than normal and were difficult to extinguish. These studies lend evidence pointing to an obsessive and detailed, overexcitable processing of their world until the environment becomes so painfully intense that they are forced to withdraw (Markram & Markram, 2010). Like others with sensory overexcitabilities, stimuli that is undetectable to most (e.g., clothing tags, common but unnatural sounds) may be unbearable to an individual with autism. Velázquez and Galán (2013) found that the brains of autistic children actually produce, on average, 42% more information when in a resting state than non-autistic children, further suggesting a mental overload. Thus, according to the IWT, and given what we know about high IQ having strong heritable/genetic components which contribute to ASD (Asperger, 1944/1991; Clark et al., 2016; Kanner, 1943, p. 249; Wheelwright & Baron-Cohen, 2001), those with autism could actually be intellectually gifted individuals who see and feel so intensely that they must engage in avoidance and lock down behaviors in order to for them to escape.

Further research is needed to further confirm or discount the possibility of an association between high IQ, environmental insults, and ASD. Researcher and pediatrician Dr. Michael J. Goldberg has suggested that what we call 'autism' today is not the original Kanner's autism at all (Kanner, 1943); rather it is Neuro-Immune Dysfunction Syndromes (NIDS) which is a medical classification for illnesses or disorders that may have psychiatric or developmental labels caused by a complex neuro-immune illness, affecting cognitive and body functions to which those with certain genetic profiles, such as high intelligence, are more prone (Vargas, Nascimbene, Krishnan, Zimmerman, & Pardo, 2005). Ideally, immune regulation is an optimal balance of pro- and anti-inflammatory response. It should zero in on inflammation with force and then immediately return to a calm state. In those with the

overexcitabilities previously discussed, including in those with ASD, this system appears to fail to achieve a balance and thus inflammatory signals create a state of chronic activation. We can see consequences of this dysregulation in the autistic brain that shows astroglial and microglial cells which are enlarged from chronic activation producing an influx of pro-inflammatory signals (Morgan et al., 2010; Tetreault et al., 2012). The genes involved appear to be switched on in response to an antigen or trigger early in development, typically between one and three years of age (Hallmayer et al., 2011). The autoimmune theory of autism was investigated by surveying both families with autistic children and those with healthy children regarding the prevalence of autoimmune (AI) disease in first-and second-degree relatives (Comi, Zimmerman, Frye, Law, & Peeden, 1999). The authors found that the frequency of disorders in the families with autism was higher than controls, particularly among parents, and especially mothers, of autistic children. Sweeten, Bowyer, Posey, Halberstadt, and McDougle (2003) extended these findings by determining the frequency of AI disorders in families that have probands with pervasive developmental disorders (PDDs), including autism, and compared them with families who have a child with an autoimmune disease, and those with a healthy child. They found that autoimmunity was significantly increased in families with PDD compared to those with AI disease and healthy control subjects which is suggestive of a possible association between autism and AI disease. An interesting finding of the study was the increase of autoimmunity in grandmothers and uncles, and mothers and brothers of PDD probands which points to a possible transmission of susceptibility to AI disease from mother to son in the PDD families. This could prove meaningful given that there is a significantly disproportionate prevalence of ASD in boys.

Maternal immune dysregulation in the form of an inflammatory response to infection or an immune disorder, has been shown to be a risk factor for neuropsychiatric disorders in addition to correlating with a later diagnosis of autism (Estes & McAllister, 2016). A Danish study, which included nearly 700,000 births over a decade, concluded that a mother's rheumatoid arthritis elevated a child's risk of developing ASD by 80% and the presence of Celiac disease increased the risk 350% (Abdallah et al., 2012; Atladóttir et al., 2009). Similarly, maternal infection in the first and second trimester and the subsequent significant immune activation, increases autism risk (Atladóttir et al., 2010) and maternal immune activation is an environmental trigger, which has been demonstrated in the lab to induce autism-like behavioral defects in a mouse model (Malkova, Collin, Hsiao, Moore, & Patterson, 2012).

A recent magnetic resonance spectroscopy (MRS) study revealed that certain autoimmune processes and neuronal network instability can result in cingulate overactivation which induces regulatory inhibitory neuronal processes, resulting in autistic symptoms (Van Elst et al., 2014). Findings support a potential role for dysregulated immunoregulatory process and neuroinflammation in the central nervous system. Interestingly, the brain tissue of people with autism showed signs of chronic inflammation in the same areas that exhibited excessive growth. The brain areas that showed hyperproliferation in white matter also revealed inflammation and increased production of proinflammatory and anti-inflammatory cytokines by neuroglia was found (Morgan et al., 2010). Vargas et al. (2005) investigated the presence of immune activation in postmortem brain specimens and CSF from subjects with autism. The authors found marked active neuroinflammation in the cerebral cortex and cerebellum of brain tissue of those with autism. This pro-inflammatory process was characterized by a marked cellular activation of microglial and astroglial cells and displayed an altered cytokine pattern. These results suggest that an aberrant immune response in the neuroglia of autistic patients may influence neural function and neural development that may contribute to the onset of autism (Onore, Careaga, & Ashwood, 2012; Tetreault et al., 2012).

Like Kanner, recent research suggests a relationship between high IQ and ASD and there are studies that have demonstrated that autism and autoimmune dysfunction are closely related, however there have been few to no studies that have looked at high cognitive ability as a genetic risk factor for autoimmune dysregulation and resulting autistic symptomatology. We have added to the literature by demonstrating high cognitive ability as a significant and viable variable to be further explored within the field of ASD research. Our present theory of high intelligence as a risk factor for ASD could potentially be disproved if the rate of national prevalence rises significantly beyond the percentage of the population who possess a high cognitive ability. On the other hand, if the rate of ASD levels off and remains at a prevalence comparable to the number of individuals with high cognitive ability (2–3% of the population) then it could feasibly lend further support to the HBHB theory as it pertains to ASD.

4.4. Discrepancies among intelligence/health research

There are distinct patterns woven throughout the present results. Based on our demographics, our findings do support prior research that a higher cognitive capacity is predictive of positive outcomes such as higher levels of both educational and financial attainment. However, the following points are essential to consider when evaluating the substantial amount of literature that links increased cognitive ability to a decreased risk of negative physical and mental health outcomes and may serve to offer explanations for the results of our study that seem to contradict those of the popular high intelligence/high system integrity research.

First and foremost, many of the often-cited studies only examine IQ one standard deviation above or below the mean and thus, stop short of including those with very superior (130 and above) intelligence (at or above the 98th percentile) in their analysis. They report a higher risk for negative mental and physical health outcomes in lower IQ and decreased risk with each increase in standard deviation. It is assumed that this trend will continue upward and into the highly-gifted ranges as well (a linear relationship). This is important to note because without including those in the uppermost percentiles of intelligence (at or above two standard deviations above the mean), results cannot be reliably compared against those of the present study. Those we did find which specifically looked at the upper percentiles of intelligence did find associations between mood and anxiety disorders, mania, low self esteem, and the highest levels of IQ, specifically in those with high verbal reasoning (Gale et al., 2013; Lancon et al., 2015; Smith et al., 2015). By researchers not including those with gifted IQ in their samples, they may be stopping short of discovering a possible curvilinear relationship or a SLODR-like effect (Deary & Pagliari, 1991) such as ours seems to suggest. If a curvilinear relationship exists, it is likely to manifest closer to the second standard deviation above the mean and upward. Thus, these often-cited studies would fail to observe such a relationship. In comparison, MacCabe et al. (2010) found a non-linear association among 900,000 Swedish students who received the lowest, but also those who received the highest grades. Each was at greater risk of developing bipolar disorder as adults. Similarly, Gale et al. (2013) reported that a 'reversed-J' shaped association was found in men with the lowest intelligence and the men among the highest intelligence, primarily those with the highest verbal or technical ability, being at the greatest risk of being admitted with pure bipolar disorder. This suggests that more studies should be conducted that specifically include those among the top 2% of intelligence to verify if there is a curvilinear relationship of intelligence to system integrity. Why might IQ and health have a nonlinear relationship? As mentioned, our hyper brain / hyper body theory posits that the overexcitabilities specific to those with high intelligence may put these individuals at risk for hypersensitivity to internal and/or external environmental events. The rumination and worry that accompanies this heightened awareness may contribute to a chronic pattern of fight, flight, or freeze responses which then launch a cascade of immunological events.

Secondly, much of intelligence/psychiatric health studies are based on psychiatric hospital admission records. While this can be a more

objective measure than self-report, it is only likely to include patients with more severe forms of mental illness. Therefore, the relationship found between intelligence and mental disorders in these datasets may overlook those with milder cases of mental illness found among the general population. Further, more severe forms of pathology can cause cognitive impairment and actually mask true intellectual ability (Bourne et al., 2013; Hammar & Ardal, 2009). Therefore, these studies may be under reporting or missing numbers of those who also have an unidentified, gifted intelligence.

Thirdly, in many studies the physical health disorders examined are vastly different than those we analyzed in the present study which focused most specifically on those that are immunological in nature. For example, most intelligence/health research includes important questions about conditions such as high blood pressure/hypertension, heart disease, cancer and lung disease, general health such as sleep habits and number of doctors visits, and overall fitness health including questions about energy levels and mobility (Gottfredson, 2004; Lubinski, 1992). The present study focused specifically on psychoneuroimmunological processes among those with high intelligence. This makes a direct comparison between much of the current intelligence/health studies and ours impossible.

Finally, the subjective and inconsistent ways in which gifted IQ or achievement is defined and/or quantified across studies must be considered. For example, those which use academic achievement measures in lieu of cognitive tests may exclude those who are twice exceptional which are those who are both highly gifted intellectually while having a concurrent disability (e.g., executive function deficits such as working memory difficulties, etc.) that masks their ability and which may prevent them from achieving to their cognitive potential (Baum, 1989; Belgan, 2005). Students such as these would likely not be included in the studies looking at high intelligence. For example, it has been shown that as many as 9% of those who receive special education services (Barnard-Brak et al., 2009) and as many as 20% of those who drop out of high school (Renzulli & Park, 2000) are intellectually gifted.

Of note, prior studies suggest a greater risk for some of the presently examined conditions among those with higher verbal IQ specifically (Clark et al., 2016; Coplan et al., 2006, 2012; Gale et al., 2013; MacCabe et al., 2010; Penney et al., 2015; Smith et al., 2015) and for those who lean toward creative fields such as art, poetry, music, and theater (Simonton & Song, 2009) versus those who are gifted in quantitative reasoning (Brody & Benbow, 1986; Lubinski, 1992). Therefore, any study that includes high intelligence but looks only at those gifted in quantitative reasoning may miss a subset of those with high intelligence who may be at the greatest risk. Our study did not distinguish between these subcategories but future research would likely benefit from doing so.

4.5. Limitations

The present study enjoyed several strengths, such as reliable confirmation of intelligence scores at or above two standard deviations above the mean, and substantial sample size. However, a few limitations should be considered before strong conclusions may be drawn. With the self-report, survey method there is often a risk of selective recollection of data as well as potential over- or under-reporting. However, there are numerous published, peer reviewed articles in highly reputable journals that use a self-report method, including those that use data sets which rely heavily on survey data (e.g., Der, Batty, & Deary, 2009; Wraw et al., 2015; Wrulich et al., 2013). Additionally, empirical research has confirmed that self-reported health ratings are reliable and valid measures of health (Haapanen, Miilunpalo, Pasanen, Oja, & Vuori, 1997; Liang, 1986). It is possible that our participants perceived our interest in the various disorders and responded positively to its presence more or less frequently. However, given their higher than average educational and socioeconomic levels, it is likely that our participants had more than adequate cognitive, financial, and

educational resources to properly assess and research their symptoms in order to reliably self-report both diagnosed and suspected medical conditions.

Although biases can creep into survey data, consistent and relevant patterns can typically be observed, especially among large samples. Our study benefited from a robust sample provided by American Mensa, Ltd. that would be difficult to obtain had the design required us to individually test each participant ourselves. To be considered for inclusion into Mensa, over 200 approved IQ tests are allowed to be submitted in lieu of being tested by Mensa directly. They also allow applicants to provide prior, professionally administered test scores for consideration. Ideally, our sample would include participants of both high and average intelligence who have taken the same intelligence test in order to get more comparative results, however doing so would have limited the sample size significantly.

Lastly, prevalence type and age criteria within national average figures tended to vary somewhat among various agencies and published studies, making exact comparisons across studies more challenging. We closely matched our prevalence type with those used by our national comparison groups and are confident that the strong results herein would likely absorb any minor discrepancies.

4.6. Future research

While the ideal would be to individually test all participants for IQ we found no scientific data that would give reason to discredit Mensa members in any country from being representative of those with high cognitive ability. If, however, the present findings were to be exclusive to the American Mensa population, it would be a worthwhile pursuit to take a closer look at what may be unique about this group that would cause them to report strikingly higher prevalence of the presently discussed disorders. Future work might also compare Mensa groups from other countries to determine if there is consistency of results internationally. This study and the field of PNI as a whole would benefit from research that examines the present variables in other high IQ sample groups where reliable tests scores to differentiate are required, such as in a prestigious university setting or the military, each which would have their own uniquenesses to contribute.

Secondly, due to the strict guidelines set by Mensa for inclusion in the Society, we were able to reliably confirm that participants fell within the top 2% of intelligence. However, because exact scores were not verified, it was difficult to look at within-group differences. A possible way to further explore a curvilinear association is to distinguish between individuals at the second percentile from those who are reliably tested to be in the top 1% of intelligence such as are found in Intertel (the 99th percentile), or Triple Nine Society (99.9th percentile). The literature would benefit from future studies that seek to examine these unique within-group dynamics and differences.

Thirdly, consistent low-grade inflammation is considered a risk factor for neurodegenerative diseases such as Parkinson's and Alzheimer's. Some recent work has shown that connectivity changes (including hyperconnectivity) correlates with amyloid deposition (Yi et al., 2015). Our initial data set includes information to be explored in the future regarding reported prevalence of both of these diseases.

Finally, there is great promise in further investigating high IQ as a risk factor for the present variables using well-designed animal studies. A viable mouse model of high cognitive ability already exists. A team of scientists led by Princeton neurobiologist Joe Z. Tsien set out to better understand human cognition by creating a strain of highly intelligent mice they aptly named, "Doogie" after the teenage genius on the TV show, Doogie Howser, MD (Tang et al., 1999). They designed these mice by manipulating them to make more of the gene NR2B which encodes the NMDA (*N*-methyl-ᴅ-aspartate) nerve cell receptor, sometimes referred to as the brain's "coincidence detector" (Tabone & Ramaswami, 2012). This receptor is also said to play a key role in how memories are made (Li & Tsien, 2009). The researchers were able to demonstrate that these

transgenic mice consistently made stronger connections, leading them to learn and remember things significantly faster than normal mice (Han et al., 2013). They also exhibited an unusual alertness and excitability, more intense emotional memories of fearful experiences and they remembered them for a longer period of time (Wei, Wang, & Kerchner, 2001). Making associations and possessing a superior memory are each reflected in the subtests of most measures of human intelligence and are considered among the hallmarks of having a high cognitive ability. In depth psychological and immunological studies using these mice would likely yield promising results that may help further our understanding of PNI processes specifically in those with high intelligence.

5. Conclusion

Tsien (2016) defines intelligence as, "the ability to self-discover knowledge and patterns from a world full of uncertainties and infinite possibilities," whose mission it is to "solve various problems in their natural and social environments in order to survive and thrive" (p. 2). The highly intelligent individual has a remarkable capacity for seeing and internalizing these vast uncertainties, possibilities, and problems. This gift can either be a catalyst for empowerment and self-actualization or it can be a predictor of dysregulation and debilitation as the present results suggest. If these individuals take in their world in such an over-excitable manner intellectually (hyper brain), then the potential exists for an intense level of physiological processing as well (hyper body).

The hyper brain/hyper body theory is new and as such a number of studies will need to be carried out to better understand its strengths and limitations. Understanding the relationship between high intelligence and illness could have a significant personal and societal impact. In this study, we have presented a plausible, highly testable, theoretical framework that hopes to serve as a springboard for future experimental designs across disciplines. We have provided evidence to demonstrate that those with high intelligence are at significantly greater risk for the examined psychological disorders and physiological diseases; however, more work needs to be done to demonstrate causation. With the recent advancement of the study of intelligence using neuroimaging techniques and full-scale attempts to map the genome combined with the newer research being conducted to better understand psychoneuroimmunological processes, it is possible that we will continue to see vital growth of our understanding in this understudied area. Intelligence research most often focuses on the flashes of lightning seen in this rare population, however in order to serve this group of individuals fully, we must not neglect to acknowledge the rumbles of thunder that follow in the wake of their brilliance.

Declaration of conflict of interest

The authors declare no potential conflicts of interest with respect to the research, authorship, and/or publication of this article. None of the authors are associated with the organizations which published the National averages, nor are they members of American Mensa, Ltd. The interpretations and conclusions contained herein do not represent those of American Mensa, Ltd.

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