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The relationship between cavum septum pellucidum and psychopathic traits in a large forensic sample

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ABSTRACT

Cavum septum pellucidum (CSP) is a neuroanatomical variant of the septum pellucidum that is considered a marker for disrupted brain development. Several small sample studies have reported CSP to be related to disruptive behavior, persistent antisocial traits, and even psychopathy. However, no large-scale samples have comprehensively examined the relationship between CSP, psychopathic traits, and antisocial behavior in forensic samples. Here we test hypotheses about the presence of CSP and its relationship to psychopathic traits in incarcerated males (N = 1432). We also examined the incidence of CSP in two non-incarcerated male control samples for comparison (N = 208 and 125). Ethnic and racial composition was varied with a mean age of 33.1, and an average IQ of 96.96. CSP was evaluated via structural magnetic resonance imaging. CSP was measured by length (number of 1.0 mm slices) in continuous analyses, and classified as absent (0) or present (1 + mm), as well as by size (absent (0), small (1–3), medium (4–5), or large (6 + mm)) for comparison with prior work. The Wechsler Adult Intelligence Scale (WAIS-III), Structured Clinical Interview (SCID-I/P), and Hare Psychopathy Checklist-Revised (PCL-R) were used to assess IQ, substance dependence, and psychopathy, respectively. CSP length was positively associated with PCL-R total, Factor 1 (interpersonal/affective) and Facets 1 (interpersonal) and 2 (affective). CSP was no more prevalent among inmates than among non-incarcerated controls, with similar distributions of size. These results support the hypotheses that abnormal septal/limbic development may contribute to dimensional affective/interpersonal traits of psychopathy, but CSP is not closely associated with antisocial behavior, per se.

1. Introduction

Cavum septum pellucidum (CSP) is a relatively common neuroanatomical variant of the septum pellucidum, the thin triangular membrane between the right and left lateral ventricles in the medial frontal lobe of the human brain (Tubbs et al., 2011). The septum is composed of two thin layers of tissue. The *cavum* is a fluid-filled space between these two leaflets. During normal human neural development of the septum, this space forms between the two laminae, but the cavity usually closes around the 20th week of gestation. In some cases, however, the gap does not close and CSP persists (Rakic and Yakovlev, 1968; Sarwar, 1989; Shaw and Alvord, 1969).

The septum pellucidum is a component of the septo-hippocampal and limbic system (Pansky et al., 1988), which regulates instinct, affect, mood and behavior. Its glia and fiber tracts act as a relay station to communicate between the hippocampus, hypothalamus, and corpus callosum (Sarwar, 1989), and it serves a functional role integrating signals between these structures (Raybaud, 2010). CSP is bounded by the genu and body of the corpus callosum, the anterior limb and pillars of the fornix, the anterior commissure and the rostrum of the corpus callosum, and the leaflets of the septum pellucidum (Born et al., 2004). This cavity is sometimes referred to as the “fifth ventricle”, but this term has fallen out of favor as CSP is typically of the non-communicating type and therefore not part of the ventricular system (Shaw and Alvord, 1969). In most cases, enlarged CSP and persistence of a CSP beyond infancy is considered a marker for fetal neural maldevelopment as it is associated with cerebral dysgenesis (Bodensteiner and Schaefer, 1990) and neuropsychiatric disturbances (Jou et al., 1998; Sherer et al., 2004; Winter et al., 2010), and is uncommon postnatally (Griffiths et al., 2009). Little is known about the causes of maldevelopment of midline limbic structures that lead to CSP, though there is speculation on the teratogenic roles of prenatal alcohol exposure (Swayze et al.,

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1997). While primarily a reflection of abnormal growth of the limbic structure, in other instances, CSP has formed following head trauma or traumatic brain injury in boxers (McCrory, 2002; Aviv et al., 2010), football players (Gardner et al., 2016), or as a surgical complication (Sherman and Aygun, 2006).

While its clinical significance is non-specific, CSP is considered an abnormal variation and a marker for disrupted brain development (Bodensteiner and Schaefer, 1990). Several reports have indicated associations between CSP and a long list of psychologically and behaviorally relevant traits and conditions. These have included psychosis and schizophrenia (Choi et al., 2008; Filipović et al., 2004; Kwon et al., 1998; Nopoulos et al., 1998, 2000; Rajarethinam et al., 2001; Takahashi et al., 2008; Trzesniak et al., 2011; Liu et al., 2017), schizotypal personality disorder (Dickey et al., 2007; Kwon et al., 1998), Tourette's syndrome (Kim and Peterson, 2003), post-traumatic stress disorder (May et al., 2004), obsessive compulsive disorder (Chon et al., 2010), bipolar and other mood disorders (Kim et al., 2007; Landin-Romero et al., 2016), substance abuse (Filipović et al., 2004; Hwang et al., 2013), a history of head injury (Filipović et al., 2004), and particularly relevant to this report, antisocial personality and psychopathic traits (Raine et al., 2010; White et al., 2013).

The general incidence of CSP has been a long-enduring topic of investigation as prior estimates of the rates of CSP in healthy normal adults have been highly variable. CSP was estimated to be present in approximately 12–20% of the general population (Sarwar, 1989), but later estimates have suggested a prevalence of up to 80% of healthy individuals (Born et al., 2004). Prevalence rates vary widely depending on definitions and classification of CSP. Born et al. (2004) reported that depending on age, 66–80% of healthy individuals had *variant* occurrences of CSP (1–3 slices or 1.5–4.5 mm in length), 11.9% had *borderline* occurrences of CSP (4 slices or 6 mm in length), and 3–11% had *enlarged* occurrences of CSP (> 4 slices or > 6 mm in length). Using similar classifications, Nopoulos et al. (1997) originally observed variant CSP in 58% of both schizophrenia and control groups, but significantly more instances of enlarged CSP in 20.7% of patients with schizophrenia. Other studies considered among these reports had a wide range of estimates from 2% to 80% of their samples. It has been argued that incidences of small CSP, 1–2 mm longitudinally, are common in healthy individuals, and are therefore considered a normal variant in brain anatomy (Nopoulos et al., 1997). However, methodological variability is, indeed, a major issue contributing to inconsistent findings in prior reports. For example, the threshold of CSP as absent or present, as well as classification of CSP by size differs across studies.

1.1. CSP, antisocial behavior, and psychopathy

There is a growing body of literature indicating that a number of neurodevelopmental abnormalities may promote some instances of disruptive behavior and persistent antisocial traits (Anderson and Kiehl, 2012; Blair, 2013; Van Goozen et al., 2007; Raine, 2018). A number of studies have specifically identified abnormalities in the brain's limbic system as particularly influential in promoting psychopathic traits (Anderson and Kiehl, 2013, 2014; Ermer et al., 2012, 2013; Glenn and Raine, 2008; Weber et al., 2008; Ling and Raine, 2017). The limbic system and other closely-related structures in the brain are important for basic emotional processing (e.g. reward and punishment) and integrating these neural responses to guide behavior (Floresco et al., 2008). Among the various traits constitutive of psychopathy, the core affective/interpersonal characteristics (e.g. callousness, shallow affect, grandiosity) are considered essential for differentiating psychopathy from other instances of persistent antisocial behavior (Anderson and Kiehl, 2014; Hare and Neumann, 2005). Evidence that disruptions in the septal region leads to significant behavioral dysregulation (e.g. perseveration, unrestrained approach) has also fundamentally contributed to prominent etiological models of psychopathy (Gorenstein and Newman, 1980; Smith and Lilienfeld, 2015). It is reasonable to suspect that among instances of antisocial behavior, CSP may be more prevalent among those who exhibit the core elements distinctive of psychopathy.

Since the presence of CSP in adulthood may indicate disrupted neural development during critical formation of limbic structures, many have considered it a possible neural marker indicating proneness to aggressive and violent behavior (Raine et al., 2010; Toivonen et al., 2013; White et al., 2013). However, limited investigations of this neurodevelopmental hypothesis have directly explored the link between CSP, antisocial characteristics, and psychopathy, and the studies that do exist have conflicting results. Raine et al. (2010) used data from males and females from temporary employment agencies, and found that those with CSP had higher scores of antisocial personality disorder and psychopathy, as well as more criminal charges and convictions when compared to controls. Toivonen et al. (2013) found no significant differences between violent male offenders and non-incarcerated healthy controls in the incidence of CSP. White et al. (2013) found that youth with large CSP have a higher risk for aggression, psychopathic traits, and a disruptive behavior disorder (DBD) diagnosis, including conduct disorder (CD) and oppositional defiant disorder (ODD).

The variation in findings could be due to relatively small sample sizes, sampling variability, as well as variations in defining and classifying CSP. Raine et al. (2010) examined 87 non-incarcerated community participants (primarily male) ages 21–46. White et al. (2013) examined 59 adolescents, 25 males and 19 females. Toivonen et al. (2013) examined 51 male participants: 26 violent offenders (age: $M = 34$, $SD = 10$) and 25 age-matched healthy controls ($M = 35$, $SD = 8$), and there were only two cases of CSP in each of the control and violent groups. Such studies with low base rates of CSP speak to the need for larger-scale study. In addition, researchers have been inconsistent in their definition and classification of CSP. While Raine et al. (2010) and Toivonen et al. (2013) defined CSP as present when visible in six or more 1.0 mm thick coronal slices, White et al. (2013) classified CSP of 4 mm or greater in length. Some of the other studies discussed above recorded the presence of CSP in at least one 1.0 mm coronal slice, but also measured grade (length/width/size) coded as absent, questionable, mild, moderate, severe (Chon et al., 2010; Gardner et al., 2016). Others proposed the dichotomous organization or normal (1–4 slices) and abnormal (6 or more slices) CSP (Kwon et al., 1998; Nopoulos et al., 1998; Choi et al., 2008; Dickey et al., 2007).

1.2. The current study

The rationale for the current study was to clarify the specific relationship between CSP and psychopathic traits within a very large forensic sample (N = 1432). We further aimed to compare the general incidence of CSP among incarcerated (antisocial) and healthy, non-incarcerated groups. The results of this study allow for a better understanding of the role of CSP as a possible neurobiological marker of psychopathy and antisocial traits. We report the overall incidence of CSP, and its relationship with several outcome variables such as incarceration status, psychopathic traits, age, IQ, and substance use disorders. Based on the previous findings, we hypothesized that small CSP would be a relatively common occurrence in both the incarcerated and non-incarcerated populations. We further hypothesized that, due to its relationship with limbic development, the size of CSP would be positively correlated with psychopathic traits among inmates, particularly the interpersonal and affective features that are considered fundamental to the construct of psychopathy and are distinguishing from antisocial behavior overall (Hare, 2003).

2. Material and methods

2.1. Participants

Data were collected from adult male volunteers incarcerated in

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