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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

Table 1
Demographic information for adult male inmates and MRN controls.

Variables	*Adult male inmates*	*% N*	*MRN controls*
N	1432	–	125
Age	33.1 (SD = 10.33)	–	27.5(*SD* = 9.99)
IQ	96.96 (SD = 13.41)	–	121.35(SD = 12.91).
Hispanic	420	29.32	–
Not Hispanic	811	56.63	–
No ethnicity identified	210	14.65	–
American Indian/Alaskan Native	86	6.01	–
Asian	4	0.28	–
Black or African American	231	16.13	–
Native Hawaiian or Other Pacific Islander	6	0.42	–
White/Caucasian	625	43.65	–
One or more race	16	1.11	–
No race identified	464	32.40	–

prisons and forensic institutions in New Mexico and Wisconsin. Participants were recruited through brochures and voluntary enrollment. The 1432 participants had a mean age of 33.1 (SD = 10.33) and an average IQ of 96.96 (SD = 13.41). The ethnic composition of the sample was 420 Hispanic and 811 Non-Hispanic. There were 210 participants that chose not identify ethnicity. The racial composition of the sample was 86 American Indian/Alaska natives, 4 Asians, 231 Black or African Americans, 6 Native Hawaiian or Other Pacific Islanders, 625 White/Caucasian, and 16 identified themselves as more than one race. The 464 remaining participants chose not to identify their race. These data can be found in Table 1. We excluded participants reporting major head injury resulting in greater than 10 min loss of consciousness. The majority of our sample comprises participants from studies which exclude for major psychiatric illness (psychotic disorders, bipolar, major depression; see Section 2.4 below). For a supplementary analysis, we separated out individuals detained in forensic psychiatric treatment ($N = 215$) who exhibit other significant diagnoses. This forensic psychiatric sample includes inmates at a facility specific to sex-offenders and others in treatment at a forensic hospital housing individuals found not guilty by reason of insanity, some of whom exhibit psychotic features. These analyses are summarized briefly below, but they did not appreciably change the main findings reported here.

For comparison against forensic samples, we also evaluated 208 adult male participants in a publicly available database from the Human Connectome Project (HCP). These data were obtained from the MGH-USC Human Connectome Project (HCP) database (https://ida.loni.usc.edu/login.jsp). The HCP project (Principal Investigators: Bruce Rosen, M.D., Ph.D., Martinos Center at Massachusetts General Hospital; Arthur W. Toga, Ph.D., University of California, Los Angeles, Van J. Weeden, MD, Martinos Center at Massachusetts General Hospital) is supported by the National Institute of Dental and Craniofacial Research (NIDCR), the National Institute of Mental Health (NIMH) and the National Institute of Neurological Disorders and Stroke (NINDS). Collectively, the HCP is the result of efforts of co-investigators from the University of California, Los Angeles, Martinos Center for Biomedical Imaging at Massachusetts General Hospital (MGH), Washington University, and the University of Minnesota. (http://www.humanconnectomeproject.org/).

For the HCP control sample, the distribution of age was as follows: 41 were 22–25 years, 88 were 26–30 years, 77 were 31–35 years, and 2 were 36 or more years. These data can be found in Table 2. For another comparison, we evaluated 125 adult male healthy controls collected by the Mind Research Network (MRN) with a mean age of 27.5(SD = 9.99) and an average IQ of 121.35(SD = 12.91). These data can be found in Table 1. We included these healthy control samples to address the general incidence of CSP among non-incarcerated samples and more closely evaluate suggestions that CSP may be related to antisocial behavior more generally (Raine et al., 2010; White et al., 2013).

Table 2
Demographic age information for HCP controls N = 208.

Age range	*N*	*% N*
Under 22	0	0
22–25	41	19.71
26–30	88	42.30
31–35	77	37.00
36 and older	2	0.09

2.2. MRI acquisition

High-resolution T1-weighted structural MRI scans were acquired on a Siemens 1.5 T Avanto mobile scanner, which was stationed at the correctional facility so participants could be escorted to the machine. The scanner used a multiecho MPRAGE pulse sequence (repetition time = 2530 ms, echo times = 1.64 ms, 3.50 ms, 5.36 ms, 7.22 ms, inversion time = 1100 ms, flip angle = 7°, slice thickness = 1 .3 mm, matrix size = 256 × 256) yielding 128 sagittal slices with an in-plane resolution of 1.0 mm × 1.0 mm. Data were preprocessed and analyzed using Statistical Parametric Mapping software (SPM12; Wellcome Department of Cognitive Neurology, London, U.K.; http://www.fil.ion.ucl.ac.uk/spm). T1 images were manually inspected by an operator blind to subject identity and realigned to ensure proper spatial normalization, and segmented into gray matter, white matter, and cerebrospinal fluid, and modulated to preserve total volume (Ashburner and Friston, 2000, 2005). To assess total intracranial volume (TIV) as a control variable, it was calculated as the total gray matter GM, white matter (WM), and cerebrospinal fluid (CSF).

Structural data gathered as part of the HCP protocol were acquired on a Siemens 3 T Prisma scanner using a T1-weighted MPRAGE sequence (repetition time = 2400 ms, echo time = 2.14 ms, 1000 ms inversion, flip angle = 8°, yielding 0.7 mm isotropic voxel size). For detailed protocol information: http://www.humanconnectome.org/documentation/Q1/imaging-protocols.html. For quantification purposes, simple conversions were applied for comparing HCP data directly to data gathered on our scanners (see Section 2.3 below).

2.3. Measures of the CSP

MPRAGE images were used to determine the longitudinal size of CSP. Examining 1 mm coronal slices in anterior-to-posterior order, the first image where the CSP was clearly visible was used as the first frame for CSP quantification. The number of slices in which CSP appeared was counted manually by individuals blind to group membership and psychological assessment data. The anterior portion of the mid-thalamus was determined to be the maximum, posterior end-point for CSP quantification, so as not to conflate CSP with incidence of Cavum Vergae (Tubbs et al., 2011). Slice counts for HCP data (0.7 mm isotropic) were divided by 1.429, then rounded to the nearest whole count to make comparisons with data from incarcerated samples (1 mm slice resolution). Two independent raters evaluated the size of CSP in all participants. Pearson's correlation ($r = .97$), Intraclass Correlation Coefficient (ICC = 0.983, 95% CI [.981, .984]). In instances where the slice counts differed between raters, the mean of the two ratings was used for final analysis.

There is substantial variability in how CSP is defined and classified in prior reports. In order to compare results with prior work, we employed several different categorization methods. We examined CSP length as determined by the number of 1.0 mm slices counted for all continuous analyses (Nopoulos et al., 1997). We further dichotomously

classified CSP as absent (0) or present (1 + mm) (Chon et al., 2010; Gardner et al., 2016). Lastly, following Born et al. (2004), we categorized individuals' CSP based on the number of 1.0 mm slices as absent (0), small (1–3), medium (4–5), or large (6 +).

2.4. Clinical assessments

All assessments were collected by research staff. Psychopathy was assessed using the PCL-R (Hare, 2003). The PCL-R is an expert-administered rating scale based on details collected during a semi-structured interview and an extensive collateral file review. Twenty PCL-R items are rated on a three-point scale: zero indicating no evidence, one indicating some evidence, and two indicating pervasive evidence in many domains of an individual's life. Out of a maximum of 40 points, a score of 30 or higher is the recommended cutoff for an operational definition of psychopathy. The PCL-R also provides a dimensional assessment of psychopathic traits (Hare and Neumann, 2005). These traits have traditionally been divided into two major factors: (1) selfish, callous and remorseless use of others, the so-called core personality traits of psychopathy, and (2) chronically unstable, antisocial and socially deviant lifestyle (Harpur et al., 1989). More recently traits have been separated into four facets: (1) interpersonal, (2) affective, (3) lifestyle, and (4) antisocial behavior (Hare, 2003; Hare and Neumann, 2005). The PCL-R is internally reliable across male offenders, male forensic psychiatric patients, and female offenders with ICC ranging from .86 to .97, alpha ranging from .81 to .85, and mean interitem correlation ranging from .19 to .23 across samples (Hare and Neumann, 2006). Within our lab, ICC is approximately .96 for double rated PCL-Rs. In addition, the PCL-R is valid when compared to other measures of related personality disorders such as the DSM-IV diagnosis of APD (r = .73) and the ICD-10 category of dissocial personality disorder (r = .79) (Hare, 2003).

Participants were also evaluated for other psychiatric diagnoses using the Structured Clinical Interview for DSM-IV-TR, Axis I disorders (SCID I/ P; First et al., 2002). Participants were not excluded for substance abuse or dependence, as substance use is a common feature observed in psychopathic samples (Smith and Newman, 1990). IQ estimates were calculated using the vocabulary and matrix reasoning subtests of the Wechsler Adult Intelligence Scale III (WAIS-III; Wechsler, 1997). Silva (2008) calculated WAIS-III reliability scores with Fisher's z transformation. The WASI-III was reliable across age (ranging from .70 to .93), IQ (scores range from .94 to .98), index scores (ranging from .88 to .96), and raters (ranging from .91 to .95). The WASI-III is concurrently valid when compared to other measures of intelligence such as Standard Progressive Matrices (r ranging from .49 to .79) and Stanford – Binet Intelligence Scale – Fourth Edition (SB-IV) (r ranging from .78 to .89).

2.5. Statistical analysis

Structural MRI data was available for 1765 participants. The full sample of 1432 inmates was used for comparison with non-incarcerated samples (N = 333). For 1180 of the inmates, MRI data was paired with at least one additional measure of the following: IQ, PCL-R total, PCL-R Factor 1, PCL-R Factor 2, PCL-R Facet 1, PCL-R Facet 2, PCL-R Facet 3, PCL-R Facet 4, and substance dependence. This sample's characteristics and the number of participants with data in each category are provided in Table 3. For these 1180 participants, we calculated Spearman's correlations between all variables to assess the relationship between CSP, PCL-R scores, Age, IQ, and substance dependence. The distribution of CSP is expectedly skewed as larger instances are increasingly rare. Spearman's correlations, which are robust to the effects of skew and outliers, were used to investigate effects Table 4. We also provide 95% confidence intervals for the correlation coefficients and direct comparisons between Spearman's correlations are provided using z-transformed tests (Myers and Sirois, 2006). To address possible covariate effects we performed partial correlations controlling for total intracranial volume, Age, IQ, and substance dependence. These were followed by multiple regression analyses examining the stability of the associations between CSP size and PCL-R scores in the presence of all covariates. CSP size was set as the dependent variable with three separate models including PCL-R total score, PCL_R Factors 1 and 2 simultaneously, or PCL-R Facets 1–4 simultaneously, all with Age, IQ, and substance dependence as covariates.

Table 3
Descriptive statistics for adult male inmates.

Variables	*N*	*M*	*SD*
CSP (1.0 mm slices)	1432	2.61	2.94
IQ	1345	96.96	13.41
PCL-R Total	1180	21.86	7.01
Factor_1	1180	7.17	3.63
Factor_2	1154	12.50	4.01
Facet_1	1180	2.54	2.07
Facet_2	1180	4.63	2.18
Facet_3	1143	6.00	2.25
Facet_4	1162	6.47	2.63
SUD	1200	1.51	1.56

Note. CSP: cavum septum pellucidum length in millimeters. Factors and facets are derived from the PCL-R criteria (see methods). SUD: substance use dependence diagnoses as evaluated by SCID criteria (see methods).

Finally, we compared the distributions of the HCP healthy control sample, the MRN healthy control sample, and the incarcerated sample. Independent samples t-tests and chi-square tests were performed to examine the relationships between sample (inmate, HCP control, MRN control), CSP presence, and CSP size categories described above.

2.6. Supplementary analyses

We also compared our forensic psychiatric sample (N = 215) of participants from a sex-offender-specific facility and an adult forensic mental health center (housing offenders found not guilty by reason of insanity) to the remainder of the inmate sample (N = 1217). We compared the length of CSP in these two groups using independent samples t-tests, and examined distribution of categorical CSP designations using Chi-Squared tests. We also carried out all primary analyses using both the full all-inclusive sample (N = 1432), and the reduced (N = 1217) sample without the forensic psychiatric participants for comparisons.

An independent samples t-test was used to examine to differences in CSP length between individuals with PCL-R scores 30 and above (high group and operational cut-off for psychopathy) (N = 214) with those with scores of 20 or below (low group) (N = 496).

3. Results

We accounted for CSP in several ways: continuously as the number of 1.0 mm slices, categorically as absent or present, and further by breaking categories into size (absent, small, medium, or large; following Born et al., 2004). Figs. 1–3 show the distributions of the incidence of CSP among N = 208 non-incarcerated HCP controls, N = 125 non-incarcerated MRN controls, N = 1432 total adult male inmates, and the reduced N = 1217 adult male inmates sample after excluding forensics psychiatric participants from forensic mental health centers described above (N = 215).

As expected, PCL-R score was significantly correlated with the number of 1.0 mm slices showing CSP (PCL-R total (r_s (1180) = 0.09, p = .002). This effect was limited to the interpersonal and affective components of psychopathy (PCL-R Factor 1 (r_s (1180) = .12, p < .001); Facet 1 (r_s (1180) = .11, p < .001), and Facet 2 (r_s (1180) = .09, p = .002)). Scatterplots of these significant effects can be found in Fig. 4. CSP length was not significantly associated with Age, IQ, PCL-R Factor 2, Facet 3 and 4, or substance dependence. These and other

Table 4
Spearman's Rho correlations between CSP, IQ, PCL-R scores, and substance dependency for adult male inmates.

Variables	CSP	Age	IQ	PCLR total	Factor 1	Factor 2	Facet 1	Facet 2	Facet 3	Facet 4	SUD
CSP	–										
Age	.015	–									
IQ	.023	.045	–								
PCL-R total	.091**	−.107**	.002	–							
Factor 1	.116**	−.100**	.017	.812**	–						
Factor 2	.038	−.154**	−.008	.852**	.452**	–					
Facet 1	.112**	−.013	.087**	.709**	.833**	.404**	–				
Facet 2	.089**	−.145**	−.043	.681**	.866**	.382**	.464**	–			
Facet 3	.044	−.162**	−.002	.742**	.491**	.782**	.407**	.441**	–		
Facet 4	.025	−.091**	−.028	.650**	.249**	.831**	.250**	.187**	.329**	–	
SUD	−.012	−.116**	.017	.204**	.003	.319**	.026	−.012	.250**	.277**	–

Note. CSP: cavum septum pellucidum length in millimeters. Factors and facets are derived from the PCL-R criteria (see methods). SUD: substance use dependence diagnoses as evaluated by SCID criteria (see methods).
** Indicates p < .01.

correlations are provided in Table 4. Confidence intervals for the correlations can be found in Table 5. Differences in CSP correlations between Factor 1 and Factor 2 were marginally significant ($z = 1.89$, $p = .058$). Differences in CSP correlations between Facet 1 and 4 were significant ($z = 1.65$, $p = .035$). All other comparisons of CSP correlations between Facets were not significant ($z's < 1.09$, $p's > .09$).

IQ scores were available for 105 of the 125 MRN control sample. As with the incarcerated sample, IQ was not significantly correlated with

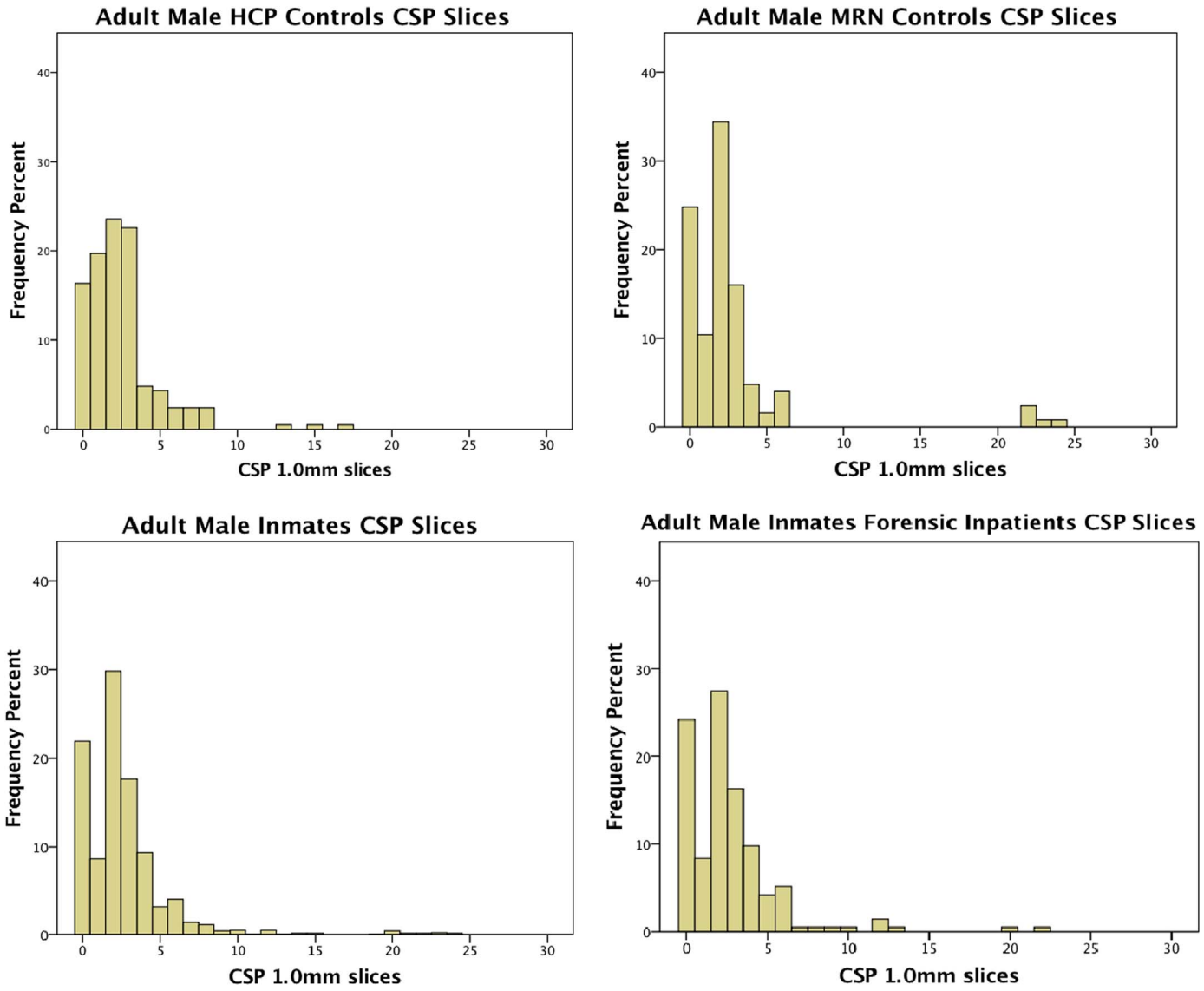

Fig. 1. Relative frequencies of cavum septum pellucidum (CSP) among healthy controls (HCP; $N = 208$) (MRN; $N = 125$) and inmates: full sample ($N = 1432$) and forensic inpatients ($N = 215$). CSP is defined by length in mm.

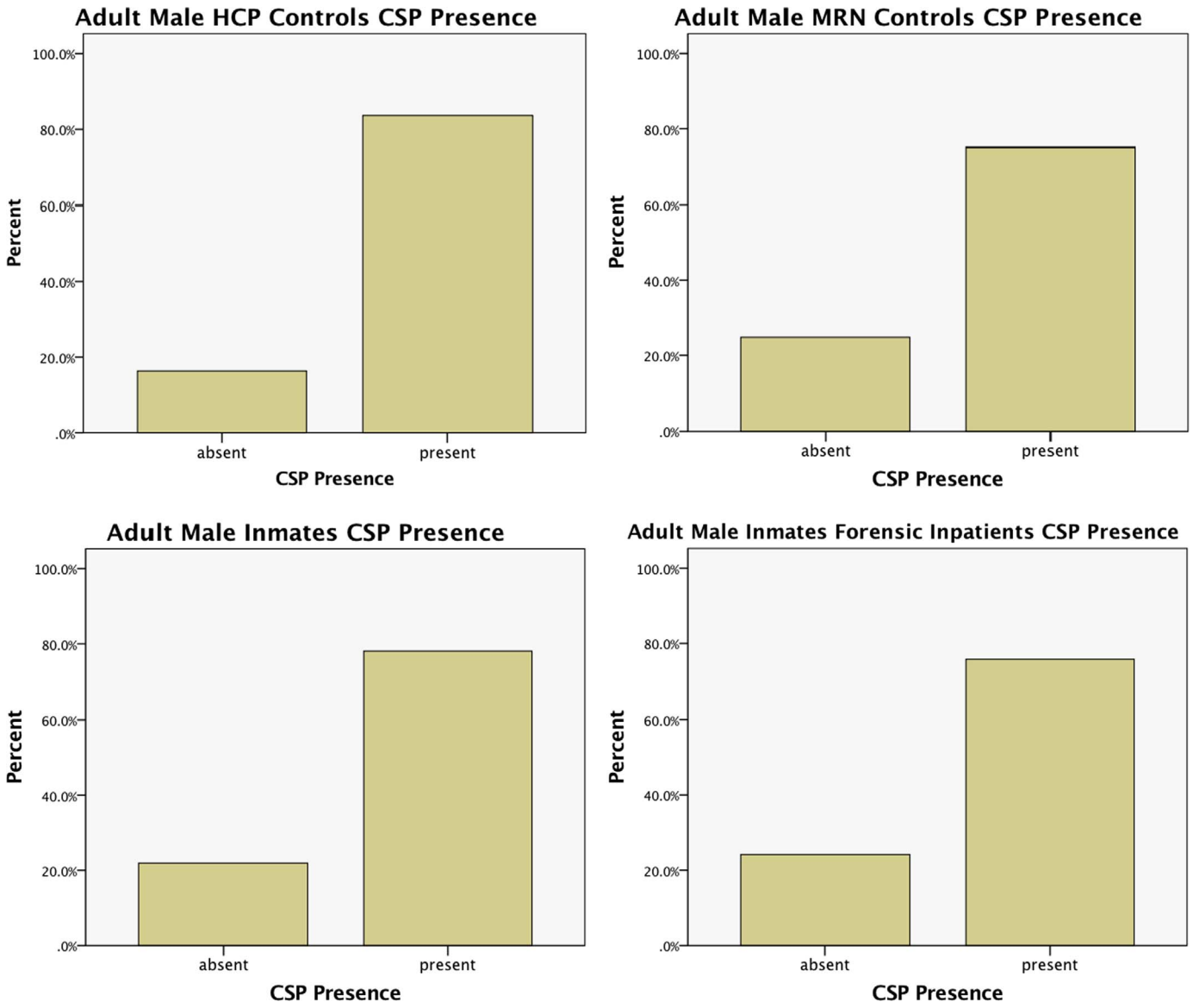

Fig. 2. Relative frequencies of cavum septum pellucidum (CSP) among healthy controls (HCP; $N = 208$) (MRN; $N = 125$) and inmates: full sample ($N = 1432$) and forensic inpatients ($N = 215$). CSP is defined by presence or absence.

CSP size ($r_s < .03$, $p > .3$). Partial correlations controlling for brain volume (TIV) did not substantially affect the relationships. Likewise, partial correlations controlling for Age, IQ, and substance dependence did not substantially change these effects (see Table 5).

In regression analyses examining the unique effects of PCL-R dimensions while accounting for all covariates, PCL-R Total scores remained a significant predictor of CSP size, $\beta = .085$, $t(1105) = 2.786$, $p = .005$. When factors 1 and 2 were included together in model, Factor 1 emerged as driving this relationship, significantly predicting CSP size, $\beta = .079$, $t(1077) = 2.32$, $p = .02$. Factor 2 was never a significant CSP size. When the four facets were all included in the model, Facet 1 emerged as driving this relationship, significantly predicting CSP size, $\beta = .073$, $t(1049) = 2.032$, $p = .042$. Facet 2 was not significant in this model, though it should be noted that these facet constructs overlap ($r = .464$). In a model examining associations between CSP size and PCL-R Facet 2 alone in the presence of these covariates, Facet 2 was a marginally significant predictor of CSP size, $\beta = .057$, $t(1104) = 1.881$, $p = .06$. Facets 3 and 4 were never significant predictors of CSP size. Among the covariates, TIV emerged as a significant predictor of increased CSP size in all three models. Age, IQ, and substance dependence were never significant predictors of CSP size.

There were no significant differences between samples (HCP or MRN controls, HCP control or inmate, and MRN control or inmate) in the rate of CSP or the length (in mm) (F (2, 1762) = .252; $p > .77$). Likewise, t-tests examining pairwise comparisons revealed no differences in CSP size (t's $< .7$, p's $> .5$) between groups. Further, when examining the incidence of CSP by categorical divisions used in prior work, CSP was found to be independent of group membership. The proportion of CSP presence was slightly higher among HCP controls (83.7%) compared to MRN controls (76.0%) and inmates (80.4%). However, the relationship between sample (HCP control, MRN control, or inmate) and CSP presence were not significant, χ^2 (3, $N = 1765$) = 9.937, $p > .2$. In addition, inmates did have a slightly lower proportion of small CSP (58.4%) compared to HCP controls (65.9%) and MRN controls (61.6%) as well as a slightly higher proportion of medium CSP (13.3%) compared to HCP controls (9.1%) or MRN controls (6.4%). However, the relationship between sample (HCP control, MRN control, or inmate) and CSP size categories (absent, small, medium, large) was not significant, χ^2 (3, $N = 1765$) = 10.52, $p > .1$). These data can be found in Fig. 3. Testing additional classification schemes that have been previously used in the literature, CSP was also found to be unrelated to incarceration status when categorized as at least 4 mm (White et al.,

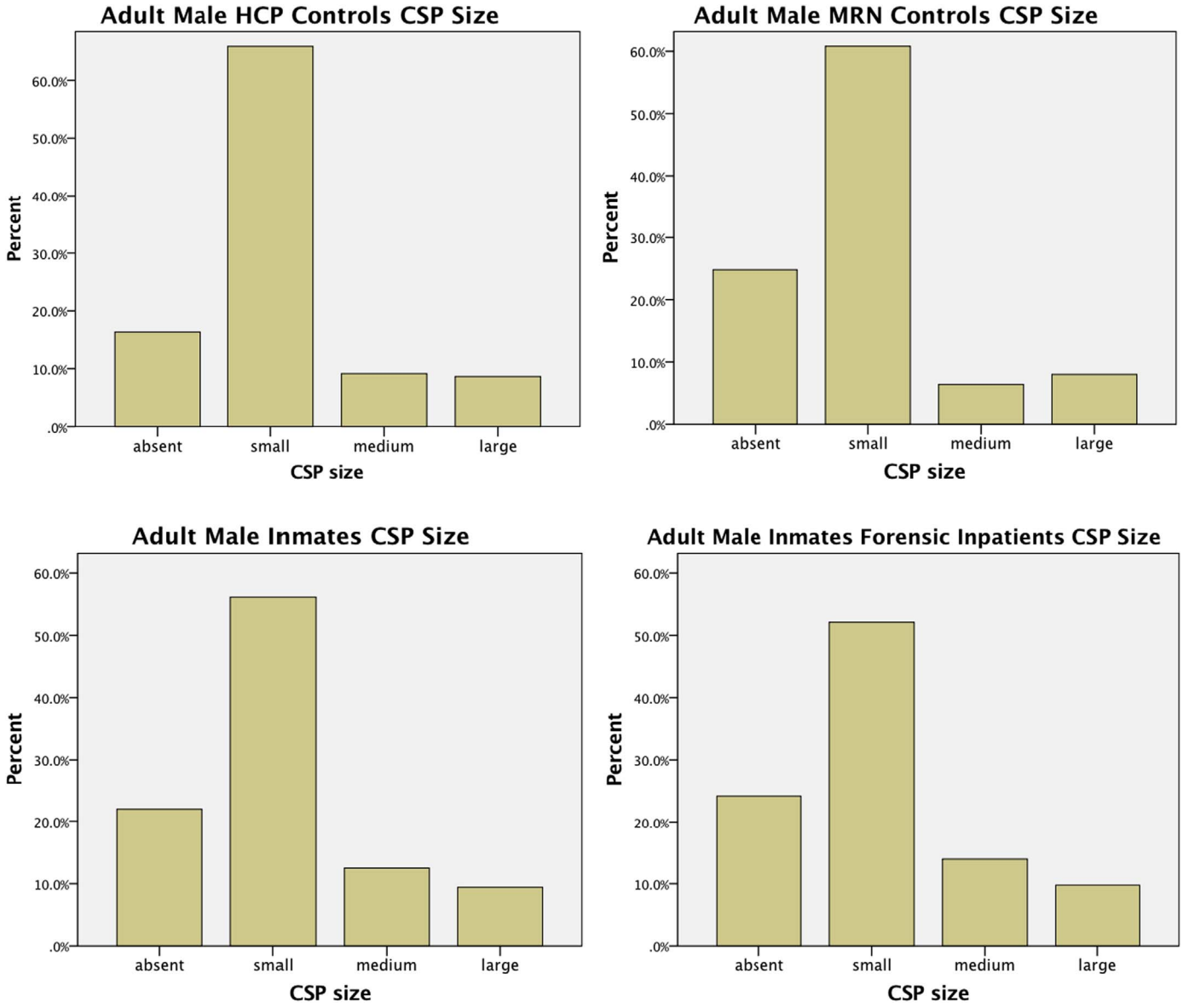

Fig. 3. Relative frequencies of cavum septum pellucidum (CSP) among healthy controls (HCP; $N = 208$) (MRN; $N = 125$) and inmates: full sample ($N = 1432$) and forensic inpatients ($N = 215$). CSP is defined by absent, small, medium, and large categories.

2013) (χ^2 (3, N = 1765) = 5.326, p = .07) or at least 6 mm (Raine et al., 2010; Toivonen et al., 2013) (χ^2 (3, N = 1765) = .374, p = .829).

3.1. Supplementary results

We found the length of CSP (in mm) to be similar across the forensic psychiatric patients ($N = 215$, $M = 2.61$, $SD = 2.933$) and the larger inmate sample ($N = 1217$, $M = 2.61$, $SD = 2.939$), $t(1430) = .013$, $p = .990$). In addition, individuals from these institutions showed similar distributions of CSP presence, χ^2 (1, $N = 1432$) = .754, $p = .385$, and CSP size, χ^2 (3, $N = 1432$) = 1.787, $p = .618$, compared to the larger inmate sample. Excluding these individuals did not appreciably change the results of other statistical relationships examined, thus the remaining comparisons consider all inmates together in a single, large sample. These data can be found in Figs. 1–3.

The group with high PCL-R scores (i.e., 30 and above and the operational definition of psychopathy) had significantly larger CSP ($M = 3.09$, $SD = 3.19$) than the group with low PCL-R scores (i.e., 20 and below; $M = 2.45$, $SD = 2.86$), ($t(708) = -2.65$, $p = .008$).

4. Discussion

The purpose of this study was to test for specific relationships between CSP and psychopathic traits in a large incarcerated sample and to better understand the role of CSP as a possible neurobiological marker promoting psychopathy. To our knowledge, this study reports on the largest sample, to date, investigating CSP and its relationship with incarceration, antisocial behavior, and psychopathic traits.

As expected, small CSP was relatively common among both incarcerated and non-incarcerated individuals. Among inmates, however, those meeting diagnostic criteria for psychopathy exhibit larger CSP than inmates who scored low on the PCL-R. Further, the size of CSP was positively correlated with PCL-R total score as well as individual factor and facet scores. In line with our hypotheses, CSP size was positively correlated with the affective-interpersonal dimension of psychopathy (represented by Factor 1 of the PCL-R), and also with the interpersonal (Facet 1) and affective (Facet 2) features considered separately. CSP size was not significantly correlated with the lifestyle and antisocial features of psychopathy represented by PCL-R Factor 2 and Facets 2 and 3. In addition, CSP was not significantly related to Age, IQ, or substance

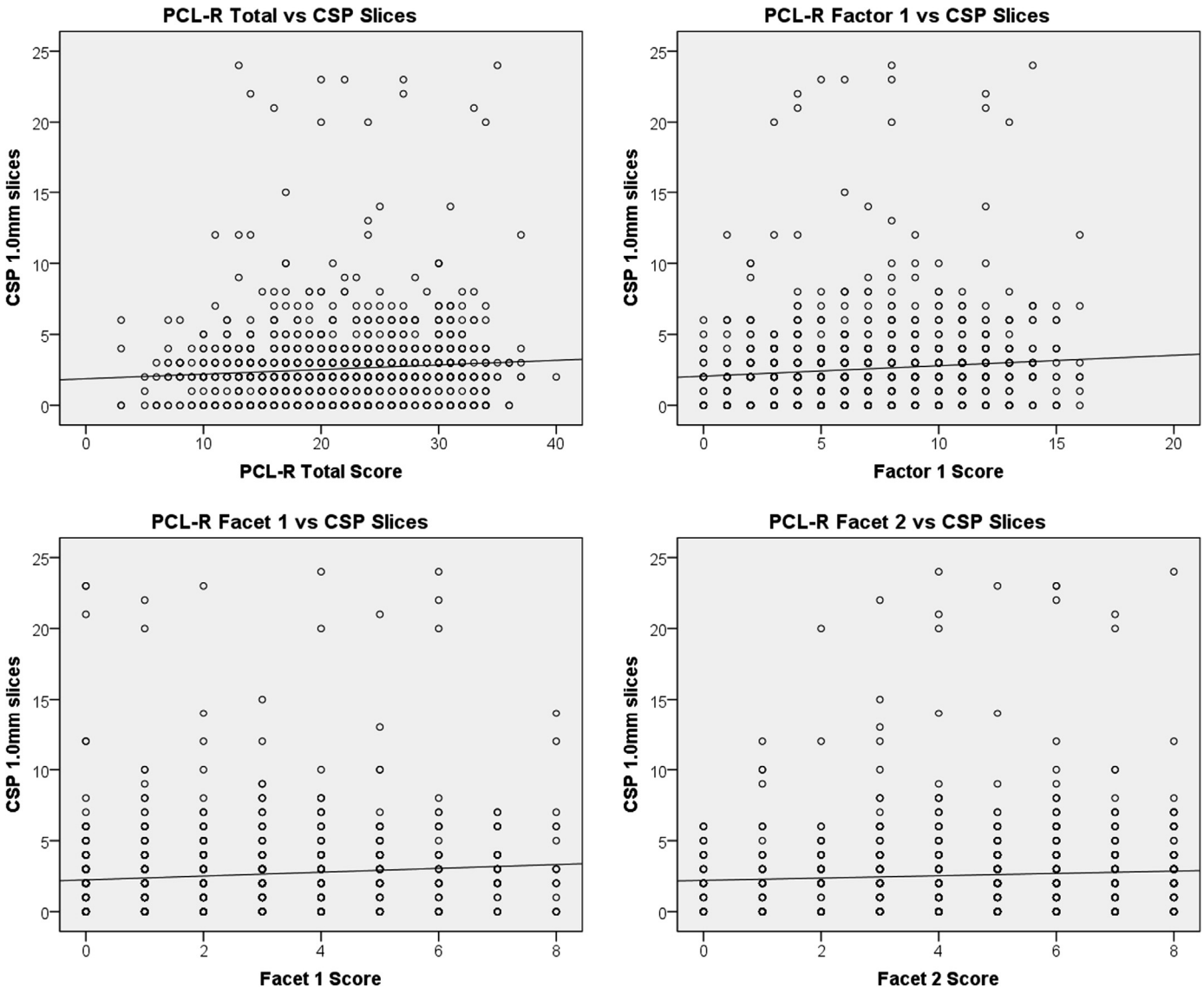

Fig. 4. Scatterplots for significant effects (PCLR Total vs. CSP, Factor 1 vs. CSP, Facet 1 vs. CSP, and Facet 2 vs. CSP).

Table 5
95% Confidence intervals for Spearman's correlations to CSP and partial correlations.

Variables	r_s	*Lower bound*	*Upper bound*	*Partial r_s controlling for TIV*	*Partial r_s controlling for age*	*Partial r_s controlling for IQ*	*Partial r_s controlling for SUD*
Age	.015	−.037	.067	.016	–	.017	
IQ	.023	−.030	.077	.011	.022	–	.023
PCL-R Total	.091**	.034	.148	.093**	.094**	.091**	.096**
Factor_1	.116**	.059	.173	.110**	.117**	.115**	.115**
Factor_2	.038	−.020	.096	.044	.041	.038	.044
Facet_1	.112**	.055	.168	.108**	.110**	.108**	.110**
Facet_2	.089**	.031	.144	.084**	.092**	.090**	.089**
Facet_3	.044	−.014	.102	.050	.052	.049	.053
Facet_4	.025	−.033	.082	.029	.023	.022	.025
SUD	−.012	−.069	.044	−.003	−.009	−.011	–

Note. CSP: cavum septum pellucidum length in millimeters. Factors and facets are derived from the PCL-R criteria (see methods). SUD: substance use dependence diagnoses as evaluated by SCID criteria (see methods).

** Indicates $p < .01$.

dependence. Direct comparison of the correlations between CSP and the factors of the PCL-R found that Factor 1 was marginally stronger than was the effect for Factor 2. Relationships between facet-level items were more distinct. Correlations between CSP and Facet 1 were significantly different from Facet 4. Regression analyses confirmed the relative importance of Factor 1 and Facet 1 when considering all covariates simultaneously. The relative importance of Facet 2 may have been limited by the close association between Facets 1 and 2 (r_s = .464). Though the effect sizes remain small (r's < .1), these comparisons support the hypothesis that CSP is more closely associated with

interpersonal and affective elements of psychopathy than to antisocial behavior, per se. This is an interesting finding as Factor 1 elements help to distinguish psychopathy from other instances of persistent antisocial behavior (Hare, 2003), commonly represented by Factor 2 elements alone. It is reasonable that signs of neurodevelopmental abnormalities giving rise to psychopathy are tied to these distinguishing elements of psychopathy in universally antisocial samples.

Turning to categorical relationships, in contrast to Raine et al. (2010), CSP size was found to be independent of incarceration status, and was not more prevalent among incarcerated individuals than among the non-incarcerated healthy control groups. Further, results showed no significant correlations with measures of antisocial traits among the incarcerated sample (PCL-R Factor 2). It may be concluded that CSP is not particularly discriminating or unique to those exhibiting antisocial behavior, or those likely to be incarcerated. These results also support prior findings by Toivonen et al. (2013) who reported no differences in CSP between violent offenders and non-incarcerated males. In addition, CSP is not specific to any particular Age, IQ, or number of substance dependencies.

The present findings suggest that CSP is slightly more related to affective-interpersonal psychopathic traits, though the effects are modest. These results support neurobiological models of psychopathy that emphasize dysfunction in paralimbic regions of the brain and disruption in septo-hippocampal development (Kiehl, 2006; Blair, 2006; Gorenstein and Newman, 1980; Smith and Lilienfeld, 2015; Anderson and Kiehl, 2013, 2014; Glenn and Raine, 2008; Weber et al., 2008). White et al. (2013) similarly found that youth with large CSP had a higher risk for psychopathic traits. Likewise, Raine et al. (2010) found that non-incarcerated individuals with CSP, who were recruited from temporary employment agencies, had higher scores of psychopathy than controls. Our results agree with Raine et al. (2010) pertaining to psychopathic traits, but conflict with the findings of a stronger relationship between CSP and antisocial traits.

4.1. Strengths and limitations

This study has a number of strengths including the large sample, diverse sampling pools (multiple comparison groups), and the implementation of several methods for quantifying CSP. To address the variability in prior CSP definition and categorization, we included several measures of CSP including dimensional and categorical approaches in our analysis. These strengths contribute to a more comprehensive assessment of these relationships than has been provided in extant literature, and ultimately suggests that CSP is more associated with the distinctive affective/interpersonal features of psychopathic traits among those characterized by antisocial behavior more generally.

Although our study has a number of unique merits, there are also limitations to consider. First, we can only make conclusions about the relationship between CSP and psychopathic traits within incarcerated populations because assessment of psychopathic traits was not available for non-incarcerated, healthy controls. It should be noted, however, that the PCL-R was developed to measure psychopathic traits among institutional samples, and estimates of PCL-R scores from healthy, non-incarcerated populations are very low (i.e., limited variance in healthy samples; see Hare, 2003). Different relationships between CSP and psychopathic traits might emerge by examining non-incarcerated samples using different assessment measures (i.e., self-report).

CSP has historically been associated with a number of other psychiatric conditions (Kim et al., 2007; May et al., 2004; Nopoulos et al., 2000). It's important to note that the effects observed in psychopathy are not sensitive or specific enough to consider CSP a distinguishing feature of psychopathy. It remains possible that CSP is a non-specific indicator of limbic mal-development that accompanies many psychiatric disorders. That is, there are likely many non-exclusive etiological influences over these outcomes, none of which are necessary or sufficient for determining pathology alone. However, these conclusions are speculative and specific relationships will need to be examined more closely in future studies.

Other important considerations from this report include notable differences in IQ between the MRN control sample and the incarcerated sample. However, analysis revealed that IQ was not significantly related to CSP size in either the inmates or the MRN controls examined separately. Finally, this study focuses on a relatively limited segment of the population—adult males. Future research should explore these relationships among females, youth, and further among non-incarcerated individuals.

4.2. Summary and conclusion

The data presented here provide evidence that CSP is more related to the interpersonal and affective traits of psychopathy than to impulsive/antisocial behavior. It is reasonable to expect that these relationships stem from functional consequences of abnormal neurodevelopment in the septo-hippocampal limbic system, which has been a regular focus in the study of etiological routes of psychopathic traits. The main effect sizes are quite small (r's $< .1$), but the benefit to utilizing such a large sample is that we can characterize the reliability of such small effects with greater certainty. Though the phenomena focused on here do not appear to be large, the results of this study support prevailing neurobiological models of psychopathy. Nevertheless, we should be careful not to conclude that CSP is an essential feature of psychopathy or that all those with CSP will exhibit elevated psychopathic traits. The evidence here merely suggests that as a neurological feature of abnormal limbic development, large instances of CSP may systematically coincide with an elevated risk for developing certain psychopathic personality features. We recognize its influence to be fairly limited and do not consider CSP to be a sensitive or specific indicator of psychopathy. We hope that these data encourage additional research examining this relationship in greater detail.

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