Supplement 1 – Literature review of the genetic coherence of higher-order spectra

Internalizing spectrum encompasses five subfactors: fear, distress, eating pathology, mania and sexual problems. The intergenerational transmission of anxiety and depressive disorders is largely non-specific (Kendler, Davis, & Kessler, 1997; Starr, Conway, Hammen, & Brennan, 2013), and twin studies support a common genetic factor underlying emotional disorders (Cosgrove et al., 2011; Hettema, Neale, Myers, Prescott, & Kendler, 2006; Kendler, Aggen, et al., 2011; Kendler & Myers, 2014; Lahey, Van Hulle, Singh, Waldman, & Rathouz, 2011; Mikolajewski, Allan, Hart, Lonigan, & Taylor, 2013; Mosing et al., 2009; Silberg, Rutter, & Eaves, 2001), although separate genetic influences on distress and fear subfactors have also been identified (Hettema, Prescott, Myers, Neale, & Kendler, 2005; Kendler, Prescott, Myers, & Neale, 2003; Kendler et al., 1995; Waszczuk, Zavos, Gregory, & Eley, 2014). Furthermore, multiple forms of eating pathology aggregate in families (Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004; Strober, Freeman, Lampert, Diamond, & Kaye, 2000; Thornton, Mazzeo, & Bulik, 2010) and share genetic overlap (Bulik et al., 2010; O'Connor et al., 2016; Waszczuk, Waaktaar, Eley, & Torgersen, 2019), with a number of structural twin studies finding a common genetic factor influencing eating pathology and emotional disorders (Kendler, Aggen, et al., 2011; Kendler et al., 1995; Silberg & Bulik, 2005; Thornton, Welch, Munn-Chernoff, Lichtenstein, & Bulik, 2016; Wade, Fairweather-Schmidt, Zhu, & Martin, 2015). For example, Silberg and Bulik (2005) identified a single genetic factor influencing eating disorder, depression, overanxious and separation anxiety symptoms in children, and this genetic factor also contributed to the continuity of symptoms into adolescence. Finally, twin and family studies indicate a partial genetic overlap between mania and unipolar depression (McGuffin et al., 2003; Smoller & Finn, 2003; Song et al., 2015). Overall, although the genetic overlap between different disorders and subfactors within the internalizing spectrum is prominent, genetic evidence linking certain disorders,

such as mania and eating pathology, or sexual dysfunction and other subfactors (Forbes, Baillie, Eaton, & Krueger, 2017), is currently lacking.

Externalizing spectra: disinhibited and antagonistic, jointly harbor antisocial behavior subfactor, which contains syndromes such as conduct disorder (CD) and antisocial PD. Additionally, disinhibited externalizing spectrum consists of the substance abuse subfactor, and antagonistic externalizing harbors four PDs: narcissistic, histrionic, paranoid and borderline. Developmental studies focused on disorders within the antisocial behavior subfactor identified common genetic influences (Bornovalova, Hicks, Iacono, & McGue, 2010; Cosgrove et al., 2011; Hink et al., 2013; Lahey et al., 2011; Mikolajewski et al., 2013; Tuvblad, Zheng, Raine, & Baker, 2009), with parent-child resemblance accounted for by the transmission of general liability to these behaviors (Bornovalova et al., 2010). The substance abuse subfactor was also found to be characterized by a highly heritable single latent factor (Hicks, Schalet, Malone, Iacono, & McGue, 2011). Investigating disorders within antisocial behavior and substance abuse subfactors together, twin studies consistently identify a higherorder heritable disinhibited externalizing factor (Hicks, Foster, Iacono, & McGue, 2013; Hicks, Krueger, Iacono, McGue, & Patrick, 2004; Kendler, Aggen, et al., 2011; Kendler & Myers, 2014; Kendler et al., 2003; Krueger et al., 2002; Wolf et al., 2010; Young, Stallings, Corley, Krauter, & Hewitt, 2000), with non-specific intergenerational transmission of a wide range of disinhibited externalizing disorders. Notably, the substance use subfactor may have a significant proportion of unique genetic etiology, independent of the influences shared with the disinhibited externalizing spectrum (Kendler et al., 2003). Within the antagonistic externalizing spectrum, twin data generally support genetic commonality among PDs with antagonistic properties(Kendler et al., 2008; Livesley, Jang, & Vernon, 1998; Torgersen et al., 2008), and their genetic links to disorder within the antisocial behavior subfactor (Kendler, Aggen, et al., 2011). Overall, disinhibited and antagonistic externalizing spectra have shown genetic coherence.

Thought disorder spectrum encompasses schizophrenia spectrum disorders, mood disorders with psychosis, three PDs: schizotypal, schizoid and paranoid, and the mania subfactor. Family and twin studies have found that schizophrenia and the three PDs aggregate in families (Calkins, Curtis, Grove, & Iacono, 2004; Ettinger, Meyhöfer, Steffens, Wagner, & Koutsouleris, 2014; Kendler, Czajkowski, Tambs, Torgersen, Aggen, Neal, et al., 2006; Kendler & Gardner, 1997; Kendler et al., 1993; Tarbox & Pogue-Geile, 2011) and share genetic influences (Kläning et al., 2016). One twin study identified a single genetic factor that explained all variance in schizotypal PD and also contributed to paranoid PD and schizoid PD (Kendler, Czajkowski, Tambs, Torgersen, Aggen, Neale, et al., 2006). Another analysis of this cohort that included the full range of PDs also assigned avoidant PD and dependent PD to this factor (Kendler et al., 2008), a result confirmed in another analysis (Kendler, Aggen, et al., 2011), but these two studies did not include schizophrenia or bipolar disorders, which may explain why factor content broadened beyond thought disorders. Importantly, family and twin modelling results also indicate genetic overlap among psychotic and bipolar disorders (i.e., between the thought disorder spectrum and mania subfactor), providing support for the placement of the mania subfactor under the thought disorder spectrum (Bramon & Sham, 2001; Cardno, Rijsdijk, Sham, Murray, & McGuffin, 2002; Cardno et al., 2012; Lichtenstein et al., 2009; Pettersson, Larsson, & Lichtenstein, 2016; Song et al., 2015). Overall, existing genetic evidence supports the coherence of the thought disorder spectrum, however structural studies that examine full range of thought disorders in context of diverse psychopathology are needed.

<u>Detachment spectrum</u> contains four PDs: schizoid, avoidant, dependent and (negatively loading) histrionic. To date only three twin studies have addressed the issue of distinctiveness

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of the detachment spectrum from the rest of psychopathology. First, a twin study encompassing a broad range of maladaptive personality traits found a genetic factor influencing intimacy problems and restricted expression traits, which are key features of detachment (Livesley et al., 1998). Second, analysis of all PDs found a unique genetic factor shared by schizoid and avoidant PDs, and with a smaller loading on dependent PD (Kendler et al., 2008), which was also confirm in a study looking across a broad range of PDs and clinical disorders (Kendler, Aggen, et al., 2011). Thus, twin studies to date suggest that the detachment spectrum shares a degree of common genetic etiology, but its distinctiveness from thought disorder is not certain.

Somatoform spectrum includes somatic symptom disorder and illness anxiety disorder. Relatively little is known about the genetic structure of this spectrum. One twin study to date suggested that a common genetic factor contributes to four somatic symptoms: recurrent headache, irritable bowel syndrome, chronic impairing fatigue, and chronic widespread pain (Kato, Sullivan, Evengård, & Pedersen, 2009), independent of genetic influences shared with major depression and generalized anxiety disorder. Other twin studies suggest that a significant proportion of genetic influences on somatoform spectrum symptoms are independent from internalizing problems (Gillespie, Zhu, Heath, Hickie, & Martin, 2000; Hansell et al., 2012), however somatoform and internalizing spectrum may share genetic underpinnings at a higher level of generality (Ask, Waaktaar, Seglem, & Torgersen, 2016; Ball et al., 2011; Gillespie et al., 2000; Hansell et al., 2012; Kato et al., 2009; Kendler, Aggen, et al., 2011). Structural studies often have poor somatic symptom coverage that prevents modelling the separate genetic factor and future work should consider a wider symptom coverage.

Supplement 2 – Structure of environmental influences

Although non-shared environment typically contributes to the distinction among psychiatric conditions, a proportion of environmental influences contributes to the coherence of HiTOP spectra, with higher-order non-shared environmental factors found for internalizing (Hettema et al., 2006; Hettema et al., 2005; Mosing et al., 2009; Thornton et al., 2016), externalizing (Bornovalova et al., 2010; Krueger et al., 2002; Seglem, Torgersen, Ask, & Waaktaar, 2015; Tuvblad et al., 2009; Young et al., 2000), thought disorder (Cardno et al., 2012) and somatoform (Kato et al., 2009) spectra, although they accounted for considerably less variance in the phenotypes than higher-order genetic factors. Nonetheless, some studies found non-shared environmental factors that do not align with the HiTOP (Ørstavik et al., 2012; Torgersen et al., 2008). For example, in addition to a common set of non-shared environmental influences on four externalizing PDs: antisocial, borderline, narcissistic and histrionic, the first two PDs shared an additional set of higher-order environmental influences, indicating closer etiology that is not in line with the current HiTOP classification (Torgersen et al., 2008). Furthermore, some studies in youth reported that shared environmental influences parallel the externalizing (Bornovalova et al., 2010; Burt, Krueger, McGue, & Iacono, 2001; Hicks et al., 2013; Hicks et al., 2011; Seglem et al., 2015; Tuvblad et al., 2009) and internalizing (Eley et al., 2003; Silberg & Bulik, 2005; Silberg et al., 2001) spectra. Future research should identify specific environmental influences that contribute to the coherence of and distinctions between major psychopathologic dimensions. Studies that have started identifying transdiagnostic environmental risk factors found that child maltreatment and discrimination may operate at the level of higher-order spectra (Anda et al., 2006; Eaton, 2014; Kendler, Eaves, et al., 2011; Keyes et al., 2012; Lahey et al., 2012; Vachon, Krueger, Rogosch, & Cicchetti, 2015). Finally, HiTOP spectra are sensitive to the interplay between genetic and environmental vulnerabilities. For instance, heritability

estimates for the internalizing spectrum vary across environmental circumstances (e.g. socioeconomic status, school environment) (Lamb, Middeldorp, Van Beijsterveldt, & Boomsma, 2012; South & Krueger, 2011).

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