How related are hair pulling disorder (trichotillomania) and skin picking disorder?
A review of evidence for comorbidity, similarities and shared etiology

Ivar Snorrason⁎, Emily L. Belleau, Douglas W. Woods

Department of Psychology, University of Wisconsin-Milwaukee, Garland Hall, 2441 Harford Avenue, Milwaukee, WI, USA

HIGHLIGHTS
► Hair pulling disorder (HPD) and Skin picking disorder (SPD) co-occur more often than chance would predict.
► HPD and SPD have substantial similarities in clinical characteristics and have overlapping risk factors.
► The two disorders likely share etiology and should be categorized together in the DSM-5.

ABSTRACT
Hair pulling disorder (HPD; trichotillomania) and skin picking disorder (SPD) are relatively common and potentially severe psychiatric conditions that have received limited empirical attention. Researchers are increasingly recognizing the similarities and co-occurrence of HPD and SPD, and several authors have suggested that the two disorders should be categorized together in the DSM-5. In the present article, we critically examined the evidence for comorbidity of HPD and SPD, and reviewed a diverse literature pertaining to shared risk factors and similarities in clinical characteristics. Evidence suggests that the two disorders co-occur more often than can be expected by chance, have substantial similarities in a variety of clinical characteristics (e.g., symptom presentation and course of illness) and may have some distal risk factors in common (e.g., genetic vulnerabilities). Implications for classification in the DSM-5, clinical management and research on etiology were discussed.

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⁎ Corresponding author at: University of Wisconsin-Milwaukee, Department of Psychology, 2441 East Hartford Avenue, Milwaukee, WI 53201, USA. Tel.: + 1 414 335 5919.
E-mail address: ivarsnorrason@gmail.com (I. Snorrason).

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1. Introduction

Hair pulling disorder (HPD; trichotillomania) and skin picking disorder (SPD; also known as neurotic/psychogenic excoriation and pathologic skin picking) are characterized by recurrent and excessive hair pulling and skin picking, respectively. Both conditions have been described in the medical literature for more than a century (Hallopeau, 1889; Wilson, 1875) but have received limited empirical attention. Mounting evidence shows significant morbidity associated with HPD and SPD; including functional impairment, severe emotional distress, disfigurement and medical complications (Arnold et al., 1998; Diefenbach, Tolin, Hannan, Crocetto, & Worhunsky, 2005; Flessner & Woods, 2006; Franklin et al., 2008; Lewin et al., 2009; Morales-Fuentes, Camacho-Maya, Coll-Clemente, & Vázquez-Minero, 2010; O'Sullivan, Phillips, Keuthen, & Wilhelm, 1999; Odlaug & Grant, 2008a; Odlaug, Kim, & Grant, 2010; Soriano et al., 1996; Tucker, Woods, Flessner, Franklin, & Franklin, 2011; Wetterneck, Woods, Norberg, & Begotka, 2006; Wilhelm et al., 1999; Woods, Flessner, et al., 2006). Nonetheless, little is known about the etiology of these disorders, and there is a lack of consensus about how to conceptualize or categorize them.

Currently, HPD (trichotillomania) is classified as an impulse control disorder in the fourth edition of the Diagnostic and Statistical Manual for Mental Disorders (DSM-IV), but SPD lacks its own diagnostic category. In the last two decades, researchers and clinicians increasingly have recognized the similarities and co-occurrence of HPD and SPD (Penzel, 2003), and several authors have suggested that the two disorders should be classified in the same class of disorders in the next edition of the DSM (Bohne, Keuthen, & Wilhelm, 2005; Hoppe, Ipser, Lochner, Thomas, & Stein, 2010; Stein, Chamberlain, & Fineberg, 2006; Stein et al., 2010; Teng, Woods, Twohig, & Marcks, 2002). Moreover, an APA work group (Stein et al., 2010) has provided a preliminary recommendation that SPD should have its own diagnostic category in the DSM-5, and that the two disorders should optimally be classified under the overall category of body focused repetitive behavior disorders (BFBRD). It is thus timely to review the existing literature with respect to the nature and the extent of the relationship between HPD and SPD. Greater understanding of the relationship not only informs nosological decisions, but can also have implications for clinical management and guide research on the etiology of both disorders.

The aim of the present review was to investigate the relationship between HPD and SPD through three lines of inquiry. First, we reviewed studies examining whether co-occurrence of the disorders exceeds chance levels (i.e., whether the two disorders are comorbid). Because co-occurrence of psychiatric conditions can result from various methodological reasons (e.g., Klein & Riso, 1993; Neale & Kendler, 1995), we critically examined existing studies with the aim of ruling out methodological artifacts. Second, we reviewed a diverse literature pertaining to similarities and differences in clinical characteristics. The current conceptualization of psychiatric disorders (APA, 2000) is based on the assumption that a group of individuals demonstrating similar clinical characteristics (e.g., similar symptom presentation and course of illness) represent a distinct diagnostic entity that reflects common etiology (Robins & Guze, 1970). Likewise, if two syndromes present a highly similar clinical picture, they could share etiological mechanisms. Thus, our second aim was to compare HPD and SPD on a variety of clinical characteristics including symptom presentation, phenomenology, general demographics, age of onset, course of illness, response to treatment and relation with other psychopathology. In our third line of inquiry we examined evidence for shared underlying risk factors including familiality, genetic vulnerabilities, neural abnormalities, neurocognitive deficits, temperamental traits and environmental stressors. Throughout the review we tried to identify methodological limitations and gaps in the literature.

2. Definitions

2.1. Hair pulling disorder (trichotillomania)

HPD involves excessive and recurrent pulling out hairs from the scalp, eyebrows, eyelids, pubic area, legs or other parts of the body. In DSM-IV (APA, 2000), HPD is defined as recurrent hair pulling that is not better accounted for by another mental disorder (e.g., delusions) or medical condition (e.g., dermatological problem), and results in noticeable hair loss and significant distress or functional impairment. Additional criteria include increased arousal before pulling or when the behavior is resisted, and pleasure, gratification or relief when hair is pulled out. Recently, a work group formed by the APA recommended these additional criteria be deleted because many sufferers of clinically significant hair pulling fail to report these experiences (Stein et al., 2010).

2.2. Skin picking disorder

SPD is characterized by excessive and recurrent picking or scratching of the skin on the face, extremities or other parts of the body. People with SPD tend to pick at scabs, acne or other irregularities on the skin. The APA work group (Stein et al., 2010) suggested the following definition of SPD for DSM-5: recurrent skin picking that results in skin lesion(s) and clinically significant distress or functional impairment, but is not restricted to the symptoms of another mental
disorder (e.g., body dysmorphic disorder or delusional disorder) or due to a medical condition or the effects of drugs. Other similar criteria have been offered (e.g., Keuthen et al., 2010; Odlaug & Grant, 2012), some of which also included a requirement of mounting tension, urge or intrusive preoccupation before picking and gratification or relief during the act (Arnold, Auchenbach, & McElroy, 2001).

3. Comorbidity

Comorbidity can be defined as a co-occurrence of two disorders in a population that exceeds chance levels. The likelihood of an individual having two disorders by chance can be calculated by multiplying the prevalence of each disorder. Thus, co-occurrence exceeding this number indicates comorbidity between the disorders (Klein & Riso, 1993). Unfortunately, there are no data available on the co-occurrence of HPD and SPD in the general population. However, several studies have assessed prevalence of HPD in SPD samples, and prevalence of SPD in HPD samples. It is therefore possible to compare the frequency of co-occurrence in these samples with base-rates in clinical populations. The estimated lifetime prevalence of HPD in inpatient psychiatric samples ranges from 1.3 to 4.4% (Grant, Levine, Kim, & Potenza, 2005; Müller et al., 2011; Tamam, Zengin, Karakus, & Ozturk, 2008) and the lifetime prevalence of SPD in inpatient samples ranges from 7.3 to 11.8% (Grant, Williams, & Potenza, 2007; Müller et al., 2011). According to these base-rates (i.e., average of 9.6% for SPD and 2.7% for HPD), the prevalence of co-occurrence by chance in inpatient settings should be around 0.26% (i.e., 9.6 × 2.7 = 0.26), and presumably somewhat lower in outpatient settings (data on base-rates in outpatient samples are not available for both HPD and SPD). Our review of the literature showed that the prevalence of SPD in HPD outpatient samples ranged from 10 to 34% (average of 20.8% across studies, see Table 1) and the prevalence of HPD in SPD outpatient samples ranged from 5 to 29.2% (average of 15.5% across studies, see Table 2). Thus, the co-occurrence of SPD and HPD appears to be much higher than expected by chance.

However, several methodological limitations need to be considered before confidence in this conclusion can be expressed. For instance, many of the studies (e.g., Christenson, 1995; Simeon, Cohen, et al., 1997; Tay, Levy, & Metry, 2004) defined SPD quite loosely (e.g., failed to exclude SPD due to dermatologic problems), and most of the studies did not include a comparison group (e.g., psychiatric controls). Nonetheless, the few studies that did use strict diagnostic criteria showed high co-occurrence rates (e.g., Grant, Odlaug, & Kim, 2007; Lochner, Seedat, & Stein, 2010; Snorrason, Smari, & Olafsson, 2011) and one study found higher rates of HPD in a SPD sample than in an OCD sample (Grant, Odlaug, & Kim, 2010). Another limitation is the use of treatment seeking samples that are known to artificially inflate co-occurrence rates (e.g., because of a Berkson’s bias or a clinical selection bias). Reassuringly, non-treatment seeking samples have produced similar findings as those conducted in treatment seeking samples (see Tables 1 and 2). Additionally, Hajcak et al. (2006) found a moderate correlation (r = 0.30) between severity of hair pulling and skin picking in a large sample of non-selected college students. Thus, even though population based epidemiological studies are warranted, we tentatively conclude that the co-occurrence of HPD and SPD is higher than expected by chance.

4. Similarities and differences in clinical characteristics

4.1. Symptom presentation

HPD and SPD have strikingly similar symptom presentations (Bohne et al., 2005; Lochner, Simeon, Niehaus, & Stein, 2002; Odlaug & Grant, 2008b; Teng et al., 2002). At a general level, both disorders involve recurrent behavior aimed at removing parts of the body (i.e., hair or skin). People with HPD often seek to pull hairs that are different from other hairs (e.g., coarse or gray hairs) and people with SPD target certain kinds of imperfections on the skin. In both groups, the sight or feel of these preferred features often triggers episodes (Arnold et al., 1998; Odlaug & Grant, 2008b). Individuals with HPD usually pluck out hair one by one, often targeting specific areas on the body (Christenson, Mackenzie, & Mitchell, 1991), and individuals with SPD typically pick methodically at specific areas (Arnold et al., 1998). Patients in both groups ordinarily use the fingers to pick or pull but some may also use implements (e.g., tweezers) (Arnold et al., 1998; Christenson et al., 1991; Snorrason et al., 2011; Wilhelm et al., 1999).

Rituals before and after pulling or picking are common and frequently involve tactile stimulation (e.g., stroking hair against the lips or rolling the skin between the fingers). Studies show that 48 to 72% of HPD patients report at least one oral habit associated with hair pulling (Christenson et al., 1991; Lochner et al., 2010) and 10 to 30% sometimes eat the hair afterwards (Christenson et al., 1991; Odlaug & Grant, 2008b; Schlosser, Black, Blum, & Goldstein, 1994). Likewise,

<p>| Table 1 | Prevalence of skin picking disorder (SPD) in hair pulling disorder (HPD) samples. |</p>
<table>
<thead>
<tr>
<th>Reference</th>
<th>Sample</th>
<th>Settings</th>
<th>Assessment</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Treatment seeking samples</strong></td>
<td></td>
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</tr>
<tr>
<td>Reeves, Bernstein, and Christenson (1992)</td>
<td>Ten children/adolescents with HPD</td>
<td>Outpatient child psychiatric clinic</td>
<td>Interview with patients and their parent. Definition of SPD not reported</td>
<td>One participant (10%) had a current “face picking habit”.</td>
</tr>
<tr>
<td>Christenson, 1995*</td>
<td>164 adults with HPD</td>
<td>Outpatient psychiatric clinic</td>
<td>Interview. Definition of SPD not reported</td>
<td>34% reported “scab picking” and 34% “acne picking”.</td>
</tr>
<tr>
<td>Tay et al. (2004)</td>
<td>Ten consecutive children/adolescents with HPD</td>
<td>Outpatient dermatology clinic in children’s hospital</td>
<td>Retrospective chart review. Definition of SPD not reported</td>
<td>One participant (10%) had SPD in the chart. (The authors did not report whether SPD was systematically screened for)</td>
</tr>
<tr>
<td>Lochner et al. (2010)</td>
<td>58 adults with HPD</td>
<td>Outpatient psychiatric clinic</td>
<td>Interview. SPD defined according to proposed DSM-5 criteria</td>
<td>17 participants (29.3%) had SPD</td>
</tr>
<tr>
<td><strong>Non-treatment seeking samples</strong></td>
<td></td>
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</tr>
<tr>
<td>Simeon, Cohen, et al. (1997)</td>
<td>71 adolescents and adults with HPD</td>
<td>Participants were responders to a national magazine article</td>
<td>Mail self-report survey. Definition of SPD not reported</td>
<td>20 participants (28%) reported lifetime history of at least some “skin picking” and ten (14%) lifetime history of at least some “skin scratching”.</td>
</tr>
<tr>
<td>Stein et al. (2008)</td>
<td>1697 adults with hair pulling problem. Thereof, 990 met strict DSM-IV criteria for HPD</td>
<td>Survey posted on a website of a patient advocacy organization</td>
<td>Self-report Internet survey. Definition of SPD not reported</td>
<td>51% of participants with hair pulling problem and 53% of participants with HPD reported problematic skin picking habit.</td>
</tr>
</tbody>
</table>

people with SPD often engage in post picking behaviors, and studies show that 32 to 35% eat the skin afterward (Snorrason et al., 2011; Wilhelm et al., 1999). Pulling the hair or picking the skin on other people is reported in a minority of both groups (Snorrason et al., 2011; Wilhelm et al., 1999; Woods, Flessner, et al., 2006) but may be more common among SPD patients (Odlaug & Grant, 2008b).

Both hair pulling and skin pulling tend to be highly habitual behaviors and sufferers usually pick or pull every day. People in both groups commonly pick or pull in intermittent episodes throughout the day, and some individuals spend hours each day engrossed in the activity. Odlaug and Grant (2008b) found that individuals with SPD spent significantly more time per day engaged in the behavior than individuals with HPD (105 vs. 56 min on average per day). Many patients in both groups report general worsening of the behavior in the evening (Lochner, Simeon, et al., 2002; Wilhelm et al., 1999), and it is interesting that both hair pulling (Murphy, Redenius, O'Neill, & Zallek, 2007) and skin picking (Singareddy, Moin, Spurlock, Merritt-Davis, & Uhde, 2003) have been found to occur during sleep.

4.2. Phenomenology

4.2.1. Affective experiences

Individuals with HPD and SPD commonly report affective experiences in relation to pulling/picking episodes. It has been proposed that these behaviors function to produce stimulation (i.e., pleasure, gratification or relief) or regulate states of high or low arousal, such as anxiety or boredom (Mansueto, Townsley-Stemberger, Thomas, & Golomb, 1997; Penzel, 2003; Stein et al., 2006). Existing evidence supports these hypotheses. Several studies in clinical samples, as well as large Internet surveys, indicate that a similar proportion of adults in both diagnostic groups (around 80%) report increased tension before picking/pulling and relief or gratification during the act (Arnold et al., 1998; Christenson et al., 1991; Cohen et al., 1993; Odlaug & Grant, 2008a; Tucker et al., 2011; Wilhelm et al., 1999; Woods, Flessner, et al., 2006).

Also, a series of studies have asked people with HPD and SPD to retrospectively rate the intensity of various affective states before, during and after skin picking or hair pulling episodes (Diefenbach, Mouton-Odum, & Stanley, 2002; Diefenbach, Tolin, Meunier, & Worhunsky, 2008; Duke, Bodzin, Tavares, Geffen, & Storch, 2009; Duke, Kelley, Ricketts, Geffen, & Storch, 2010; Meunier, Tolin, & Franklin, 2009; Neal-Barnett & Stadulis, 2006; Neziroglu, Rabinowitz, Breymyan, & Jacofsky, 2008; Shusterman, Feld, Baer, & Keuther, 2009; Snorrason, Smari, & Olafsson, 2010; Wilhelm et al., 1999). Results show that the majority of patients in both groups report a rise in gratification or relief during pulling/picking. The results also show that many patients report negative affect (e.g., boredom, tension or anxiety) just before pulling/picking and a marked reduction in these states after the behavior. Taken together, the evidence show that affective experiences associated with hair pulling and skin picking are remarkably similar and consistent with the notion that these behaviors serve to regulate arousal/affect and produce pleasurable feelings.

4.2.2. Dissociation

Approximately one-third of people with SPD report being in a trance or feeling mesmerized while picking (Snorrason et al., 2010; Wilhelm et al., 1999) and one study showed that 21.3% of individuals with HPD sometimes experience depersonalization during hair pulling episodes (du Toit, van Kradenburg, Niehaus, & Stein, 2001).

4.2.3. Automaticity

An interesting feature of both hair pulling and skin picking is that many sufferers report little or no reflective awareness of the act as it occurs. A substantial proportion of patients in both groups reports being unaware of the act at least some of the time, although very few exclusively pick or pull without awareness (Christenson, Ristvedt, & Mackenzie, 1993; Flessner, Conelea, et al., 2008; Tucker et al., 2011; Walther, Flessner, Conelea, & Woods, 2009; Woods, Flessner, et al., 2006). Some authors have suggested that both HPD and SPD may be meaningfully divided into two subtypes or styles of engaging in the behavior. One style involves “automatic pulling/picking” that occurs out of reflective awareness in sedentary situations and the other, called “focused pulling/picking”, happens in full awareness in response to urges or negative affective states. Factor analytic studies have identified distinct factors representing both automatic and focused styles in HPD (Flessner, Conelea, et al., 2008) and SPD (Walther et al., 2009) samples, however, further research is needed to establish the predictive validity and clinical utility of these subtypes (Lochner et al., 2010).

4.3. General demographics

Three studies directly compared HPD and SPD samples and found very similar educational level, marital status, work status, mean age and racial/ethnic distribution (Lochner, Simeon, et al., 2002; Odlaug & Grant, 2008b; Odlaug, Kim, & Grant, 2010). McCrery, Spira, and Ceminsky (2002) surveyed African American and non-African

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**Table 2**

Prevalence of hair pulling disorder (HPD) in skin picking disorder (SPD) samples.

<table>
<thead>
<tr>
<th>Reference</th>
<th>Sample</th>
<th>Settings</th>
<th>Assessment</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Simeone, Stein, et al. (1997)</td>
<td>21 adult with SPD</td>
<td>Outpatient psychiatric clinic</td>
<td>Interview. HPD defined according to DSM-III-R</td>
<td>Two (9.5%) had HPD.</td>
</tr>
<tr>
<td>Arnold et al. (1998)</td>
<td>34 adults with SPD</td>
<td>Outpatient dermatology clinic</td>
<td>Interview. HPD defined according to DSM-IV</td>
<td>Five (14.7%) had HPD.</td>
</tr>
<tr>
<td>Wilhelm et al. (1999)</td>
<td>31 adult with SPD</td>
<td>Outpatient psychiatric clinic</td>
<td>Interview. HPD defined according to DSM-IV</td>
<td>Seven (23%) had HPD.</td>
</tr>
<tr>
<td>Grant, Odlaug, &amp; Kim (2007)</td>
<td>24 adults with SPD</td>
<td>Outpatient psychiatric clinic</td>
<td>Interview. HPD defined according to DSM-IV</td>
<td>Seven (29.2%) had HPD.</td>
</tr>
<tr>
<td>Neziroglu et al. (2008)</td>
<td>40 adults with SPD</td>
<td>Outpatient psychiatric clinic</td>
<td>Interview. HPD defined according to DSM-IV</td>
<td>Two (5%) had HPD.</td>
</tr>
<tr>
<td>Grant, Odlaug, &amp; Kim (2010)</td>
<td>Adults with SPD (n=53) and OCD (n=51)</td>
<td></td>
<td>Interview. HPD defined according to DSM-IV</td>
<td>Nine (17.3%) SPD and two (3.9%) OCD participants had current HPD.</td>
</tr>
<tr>
<td>Non-treatment seeking samples</td>
<td></td>
<td>University settings</td>
<td>University settings</td>
<td></td>
</tr>
<tr>
<td>Snorrason et al. (2011)</td>
<td>University students with (n=55) and</td>
<td></td>
<td>Interview. HPD defined according to DSM-IV</td>
<td>Six (10.9%) SPD participants had HPD, and additional two (3.6%) had problematic hair pulling. No participant in the control group had HPD.</td>
</tr>
<tr>
<td></td>
<td>without SPD (n=55)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Walther et al. (2009)</td>
<td>92 adults with SPD</td>
<td>Survey posted on a website of a patient advocacy organization</td>
<td>Self-report Internet survey</td>
<td>25% had HPD.</td>
</tr>
</tbody>
</table>

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American college students and did not find differences in HPD frequency. Skin picking was not assessed.

Existing data also suggest that the gender ratio in HPD and SPD is very similar, with high female preponderance in both groups. Findings from adult clinical samples and surveys in patient advocacy organizations show that 88 to 94% of HPD patients are female (Cohen et al., 1995; Christenson et al., 1991; Diefenbach et al., 2008; Gershuny et al., 2006; Orlaugh et al., 2010; Woods, Flessner, et al., 2006) and 75 and 94% of SPD patients are female (Arnold et al., 1998; Flessner & Woods, 2006; Neziroglu et al., 2008; Orlaugh & Grant, 2008a; Snorrason et al., 2010; Schuck, Kejers, & Rinck, 2011; Wilhelm et al., 1999; Tucker et al., 2011). The reason for this large gender bias is not clear. It could be that more women than men suffer from SPD and HPD, or perhaps women are more likely than men to seek help or support when dealing with these problems and are thus overrepresented in clinical populations.

4.4. Age at onset

Both HPD and SPD can occur in any age range, from infancy to old age. However, studies in adult clinical psychiatric samples, as well as surveys in a patient advocacy organization, have consistently shown that adolescence is the most common age at onset for both SPD and HPD. The average age at onset for SPD reported in these samples has ranged from 12.3 to 16.1 years, with an average of 13.5 years across studies (Flessner & Woods, 2006; Grant, Orlaugh, et al., 2007; Grant, Orlaugh, & Kim, 2010; Lochner, Simeon, et al., 2002; Orlaugh et al., 2010; Orlaugh & Grant, 2008a, 2008b; Snorrason et al., 2010; Wilhelm et al., 1999). The average age at onset for HPD has ranged from 11.8 to 14.2 years, with an average of 12.9 years across studies (Cohen et al., 1995; Christenson et al., 1991; Christenson, Mackenzie, Mitchell, & Callies, 1991; Christenson, Chernoff-Clementz, & Clementz, 1992; Grant, Orlaugh, & Kim, 2010; Lochner et al., 2010; Norberg, Wetterneck, Woods, & Conelea, 2007; Orlaugh & Grant, 2008b; Orlaugh et al., 2010). It is worth noting, however, that some studies in dermatology samples have found that the most common age at SPD onset is later in adulthood (Arnold et al., 1998; Fruensgaard, 1984; Gupta, Gupta, & Haberman, 1986). The reason for the discrepancy between dermatology samples and other samples is not clear. It is possible that individuals with later age at onset are for some reason more likely to seek help from a dermatologist compared to individuals with earlier onset.

4.5. Course of illness

There are no data available on the long-term natural course of either disorder. However, cross-sectional studies, mostly conducted in adult clinical samples, suggest that the typical course of illness in both disorders is chronic with waxing and waning symptom severity (Christenson et al., 1991; Gershuny et al., 2006; Snorrason et al., 2011; Swedo, Leonard, Lenane, & Rettwe, 1992; Wilhelm et al., 1999). It has been suggested that HPD occurring at a preschool age is often a benign form of the disorder that has a more episodic course and may resolve without intervention (Santhanan, Fairley, & Rogers, 2008; Swedo et al., 1992; Swedo & Leonard, 1992; Tay et al., 2004). However, this notion has not been subject to empirical scrutiny with longitudinal data. Even though childhood SPD onset is commonly reported in adult SPD samples, a parallel benign childhood subtype of SPD has not been discussed in the literature, to the best of our knowledge. It is conceivable that skin picking in childhood is less likely than hair pulling to come to clinical attention, as the behavior (e.g., picking at scabs) may be considered less abnormal and more socially acceptable than recurrent hair pulling.

4.6. Response to treatment

Empirical evidence has consistently shown that habit reversal is effective in eliminating or reducing both hair pulling (Bloch et al., 2007; Keuthen et al., 2010; van Minnen et al., 2003; Woods, Wetterneck, et al., 2006) and skin picking (Schuck et al., 2011; Teng, Woods, & Twohig, 2006), at least in the short term (for review see Woods, Snorrason, & Espil, 2012). Preliminary evidence also shows that acceptance and commitment therapy may be effective for both conditions (Flessner, Busch, et al., 2008; Twohig, Hayes, & Masuda, 2006; Woods, Wetterneck, et al., 2006).

Several drugs have been shown to benefit individuals with HPD, including clomipramine (Bloch et al., 2007) and olanzapine (van Ameringen, Mancini, Patterson, Bennett, & Oakman, 2010), but no study has examined these drugs in SPD samples. A large randomized controlled trial showed that N-Acetylcysteine, a glutamate modulator, was superior to placebo in reducing hair pulling (Grant, Orlaugh, & Kim, 2009), and case reports indicate that individuals with SPD might benefit from this drug (Orlaugh & Grant, 2007). Similarly, a randomized controlled trial (Grant, Orlaugh, Chamberlain, & Kim, 2010) showed that lamotrigine, a drug that also acts on glutamate, reduced skin picking in a subgroup of SPD patients that performed poorly on a cognitive set-shifting task (the efficacy of lamotrigine for SPD was also demonstrated in an uncontrolled trial, see Grant, Orlaugh, et al., 2007). Taken together, these studies suggest a possible glutamnergic dysfunction in a subgroup of both SPD and HPD patients.

SSRs are the only medications that have been tested in randomized trials in both HPD and SPD samples. Case studies and uncontrolled trials have shown some efficacy of SSRIs in treating HPD (e.g., Lancu, Weizman, Kindler, Sasson, & Zohar, 1996). However, a meta-analysis of the four randomized controlled trials conducted so far indicated that SSRIs are no more effective than a placebo in reducing hair pulling (Bloch et al., 2007). Uncontrolled trials have demonstrated some post treatment reduction in skin picking (e.g., Keuthen et al., 2007), but the three controlled trials published so far have produced mixed findings (Arab, Ellis, Thompson, & Koren, 2001; Simeon, Stein, et al., 1997).

4.7. Relation with other psychopathology

4.7.1. Common axis I disorders

Studies in clinical samples show that people with SPD (Arnold et al., 1998; Calikusu et al., 2003; Orlaugh & Grant, 2008a; Neziroglu et al., 2008; Snorrason et al., 2011; Wilhelm et al., 1999) and HPD (Cohen et al., 1995; Schlosser et al., 1994) are frequently afflicted with other psychiatric conditions, in particular anxiety- and depressive disorders. Lochner, Simeon, et al. (2002) directly compared the frequency of common Axis I disorders in SPD and HPD samples and found very similar comorbidity patterns. People in both samples reported a high incidence of lifetime major depression and anxiety disorders. The only significant difference between the groups was higher prevalence of dysthymia in the SPD sample (the authors noted that this difference might be due to the different cultures the samples came from). In another study, no differences in Axis I comorbidity patterns were found between SPD and HPD patients (Orlaugh & Grant, 2008b).

4.7.2. Axis II disorders

Studies show that personality disorders are common among individuals who seek treatment for HPD and SPD (Christenson et al., 1992; Cohen et al., 1995; Schlosser et al., 1994; Swedo & Leonard, 1992; Wilhelm et al., 1999). Lochner, Simeon, et al. (2002) compared HPD and SPD samples and found no difference in the prevalence of the personality disorders that were assessed (i.e., obsessive–compulsive personality disorder, avoidant personality disorder, schizotypal personality disorder and borderline personality disorder).

4.7.3. Obsessive compulsive disorder (OCD)

It has been suggested that both HPD and SPD are related to OCD or belong to the putative OCD spectrum (Stanley & Cohen, 1999; Stein, Hunt, Spitz, & Hollander, 1993). Given the substantial phenomenological
similarities between SPD and HPD, it is not surprising that differences and similarities between these disorders and OCD are alike. Both HPD and SPD are similar to OCD in that they involve repetitive motor behavior, but are dissimilar in that the behavior is typically not preceded by harm-avoidant cognitions (Arnold et al., 1998; Woods, Flessner, et al., 2006) and is ego-syntonic rather than ego-dystonic (Grant, Odlaug, & Kim, 2010; Stanley & Cohen, 1999).

Studies in OCD samples show that prevalence of HPD (4 to 36%) and SPD (10 to 26%) is similar (Bienvenu et al., 2000; Cullen et al., 2001; Grant, Odlaug, & Kim, 2010; Grant, Mancebo, Eisen, & Rasmussen, 1999; Haller, Steketee, & Morey, 2000; Hasler et al., 2007; LaSalle et al., 2004; Lovato et al., 2012; Miguel et al., 2008), although one might expect somewhat higher prevalence of SPD given that it has higher base-rate in clinical samples. Prevalence of OCD is similar across HPD (8.3 to 30.4%) and SPD (6 to 45%) samples (Arnold et al., 1998; Calikusu, Yucel, Polat, & Baykal, 2003; Christenson et al., 1991; Lochner, Simeon, et al., 2002; Odlaug & Grant, 2008b; Schlosser et al., 1994), however, two studies conducted in OCD clinics have obtained even higher rates of OCD in SPD samples (Neziroglu et al., 2008; Wilhelm et al., 1999). Finally, mixed findings have been reported in studies investigating the familial relation between HPD/SPD and OCD (Bienvenu et al., 2000; Cullen et al., 2001; Grant, Odlaug, & Kim, 2010; Lenane et al., 1992), making it difficult to determine whether HPD and SPD have different familial associations with OCD.

4.7.4. Body dysmorphic disorder (BDD)

Studies show that individuals with BDD often pick their skin in order to correct minor or imagined flaws or imperfections on the skin (Phillips & McElroy, 1992; Phillips & Taub, 1995; Tanguery, Lynch & Masand, 1992). Few case studies have also described BDD patients that pull hair in order to correct appearance (Fontenelle, Mendelowicz, Mussi, Marques & Versiani, 2002; Tanguery, 1994). Typically, definitions of HPD and SPD exclude hair pulling and skin picking that is solely due to BDD. Nonetheless, BDD patients who pick skin in response to appearance concerns have similar affective experiences related to skin picking (i.e., gratification, tension reduction etc.) as pure SPD cases (Phillips & Taub, 1995), and it is not clear whether or in what way SPD (or HPD) secondary to BDD is different from primary forms of the disorders.

It may be that BDD is more related to SPD than HPD. Grant, Menard, and Phillips (2006) interviewed 176 BDD patients and found that 45% had a lifetime history of SPD secondary to BDD. Only 2.3% had primary HPD, but the authors did not examine primary SPD, possibly because all the skin pickers in the sample were defined as secondary SPD. Nonetheless, these findings indicate that BDD is possibly more related to SPD than HPD. Studies in SPD samples have found prevalence of BDD unrelated to the SPD ranging from 5 to 32% (Arnold et al., 1998; Neziroglu et al., 2008; Snorrason et al., 2011; Wilhelm et al., 1999). Only two studies have documented BDD in SPD samples. One study found a 22% prevalence of BDD in a HPD sample (Soriano et al., 1996), however BDD was assessed with a self-report questionnaire which may have resulted in overestimation. In contrast, Christenson and Mackinzie (1995) reported that only 5 out of 169 HPD patients (3%) had comorbid BDD. However, the authors noted that BDD was not systematically assessed in the sample, so this might be an underestimation. Interestingly, all the comorbid BDD/HPD cases in the Grant et al. sample and most of the comorbid BDD/HPD cases in the Christenson and Mackinzie sample suffered from SPD as well. Thus, it may be that HPD is only associated with BDD through its comorbidity with SPD. Taken together, the overall data suggest that SPD may be more strongly related to BDD than is HPD.

4.7.5. Body focused repetitive behaviors (BFRB)

Several authors have noted associations between HPD/SPD and other repetitive behaviors aimed at the body, such as nail biting and cheek/lip biting (Sarkhel, Praharaaj, & Akhtar, 2011; Stein et al., 2008; Stein et al., 2010; Teng et al., 2002). Collectively these behaviors have been called BFRBs, and it has been argued that all of these problems should be categorized together in the DSM-5 (Stein et al., 2010). In support of this notion, evidence suggests comorbidity between HPD/SPD and other BFRBs (Christenson et al., 1991; du Toit et al., 2001; Odlaug & Grant, 2008b; Grant, Odlaug, & Kim, 2010; Reeve, Bernstein & Christenson, 1992; Stein et al., 2008; Teng et al., 2002). Studies also show a high incidence of nail biting in family members of HPD and SPD patients (Grant, Odlaug, & Kim, 2010; Odlaug & Grant, 2008b), and a genetic association between pathological hair pulling, skin picking and nail biting (Bienvenu et al., 2009). Finally, studies show that nail biting serves to regulate arousal/afflict just like HPD and SPD (Teng, Woods, Markcs, & Twohig, 2004; Wells, Haines, Williams, & Brain, 1999).

5. Shared risk factors

5.1. Familiality

Several studies have investigated the prevalence of HPD and SPD in first-degree relatives of patients with either or both disorders. Unfortunately, most of the studies relied on the family history method (i.e., asked patients about problems in their relatives without interviewing relatives directly). Also, most of the studies failed to report how many relatives were inquired about. Therefore, we reviewed the findings in terms of how many patients reported at least one relative with the disorders (rather than examining percentage of afflicted individuals in samples of first-degree relatives). Findings show that 5.3 to 17.8% of HPD patients report having a first-degree relative with HPD, and one study (Cohen et al., 1995) found that 3% of HPD patients reported having a relative with a formal diagnosis of HPD (Table 3). Arnold et al. (1998) interviewed 34 individuals with SPD and only one participant (2.9%) reported having a first-degree relative with SPD. In contrast, other studies in SPD samples have shown that 19 to 45% of participants report having first-degree relatives with SPD (see Table 3). Familiality across the two disorders has also been examined in a few studies. No participant in the Arnold et al. (1998) SPD sample reported a family history of HPD, but other studies have shown that 3.8 to 9.5% of SPD patients report a family history of HPD (see Table 4). Likewise, two studies have shown a 6.6 to 8.3% prevalence rate of SPD in first-degree relatives of individuals with HPD (see Table 4).

Most of the studies examining familiality of SPD or HPD have methodological limitations that need to be considered. First, given that only a few studies reported how many relatives were inquired about, it is impossible to compare disorder prevalence in first-degree relative samples with prevalence in the general population. Moreover, most of the studies failed to include comparison groups, making it difficult to compare prevalence of HPD/SPD patients reporting afflicted relatives with prevalence in general (or clinical) populations reporting afflicted relatives. However, one study (Grant, Odlaug, & Kim, 2010) included a clinical comparison group and found a significantly higher percentage of SPD patients reporting relatives with SPD compared to a percentage of OCD patients reporting relatives with SPD (36.5% vs. 0). The results also showed a non-significant trend toward higher rates of family history of HPD in the SPD group compared to the OCD group (3.8% vs. 0).

Second, all the studies with the exception of one (Swedo & Leonard, 1992) asked the patients about their relatives rather than interviewing relatives directly. This method can be inaccurate and lead to either under-reporting or over-reporting. However, it is interesting that the only study that did interview relatives of HPD clients directly reported high rates of afflicted relatives (Swedo & Leonard, 1992). Perhaps this is because HPD and SPD are very secretive disorders and many sufferers keep their problem hidden even from family members (Penzel, 2003; Wilhelm et al., 1999).

A third limitation involves not taking into account comorbidity in the patients themselves when asking about their relatives. For example, a high incidence of relatives with HPD in a SPD sample might simply be...
because many of the patients themselves have HPD. Only one study reported findings from pure HPD and SPD samples (Odlaug & Grant, 2008b). The results showed high prevalence of HPD familiality in SPD patients without HPD (6.1%) as well as high prevalence of SPD familiality in HPD patients without SPD (8.3%) (see Table 4). Finally, no study reported how many relatives had both SPD and HPD, and many studies failed to use specific diagnostic criteria when asking patients about HPD or SPD in their relatives (i.e., only asking a single question about whether any relative had either disorder). In conclusion, preliminary evidence suggests that both HPD and SPD run in families, however, given the underdeveloped literature it is difficult to determine whether the two disorder share familiality.

5.2. Genetic risk factors

A few studies have investigated the genetic underpinnings of HPD and SPD. Novak et al. (2009) examined concordance rates for HPD in a group of monozygotic (MZ) (n = 24) and dizygotic (DZ) twins (n = 10). Results showed that concordance rates for MZ twins (38.1%) were greater than the concordance rates of DZ twins (0%), suggesting that HPD has a significant genetic component (heritability estimate of 76%). The authors also assessed concordance rates for SPD in the sample, and found a trend toward a significant difference between MZ and DZ twins (p = 0.06). The failure to find statistical difference was possibly due to lack of power.

In addition to twin study methodology, other researchers have begun to examine specific genes associated with HPD and SPD. On the bases of leads from animal models (Welch et al., 2007) demonstrating that a deletion of the gene encoding SAP90/PSD95-associated protein 3 (Sapap3) resulted in pathological hair and skin removal, researchers have attempted to link the Sapap3 gene to HPD and SPD in humans. Zuchner et al. (2009) found an association between Sapap3 and HPD, but Boardman et al. (2011) failed to find an association after correction for multiple testing. Bienvenu et al. (2009) examined the link in a large OCD family study and discovered that both SPD and HPD were associated with single nucleotide polymorphism (SNP) 3, rs6662980 in the Sapap3 region of chromosome 1p35. Each disorder also had unique associations with other SNPs, where HPD was associated with SNP6 (rs4652867) and SPD with SNP4 (rs4652867). This suggests that HPD and SPD may have both common and unique genetic associations. Interestingly, neuroimaging studies (Chamberlain et al., 2008; Lee et al., 2010) have shown that individuals with HPD have abnormalities in the striatum, a brain region where Sapap3 is highly expressed.

5.3. Neural substrates

To date, there has been no neuroimaging study conducted in an SPD sample, and the few studies conducted in HPD samples have produced somewhat mixed findings. Several studies have found evidence for abnormalities in multiple brain areas, including regions that are involved in habit learning (e.g., striatum), emotion regulation (e.g., amygdalo-hippocampal complex) and the ability to generate and suppress motor responses (e.g., several cortical areas) (Chamberlain et al., 2008; Chamberlain et al., 2010; Grachev, 1997; Keuthen et al., 2007; Lee et

Table 3

<table>
<thead>
<tr>
<th>Reference</th>
<th>Sample Description</th>
<th>Settings</th>
<th>Assessment</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Swedo and Leonard (1992)</td>
<td>28 consecutive HPD patients (children, adolescents, and adults) and 18 healthy controls</td>
<td>Outpatient psychiatric clinic</td>
<td>Relatives of HPD cases (n = 106) and relatives of healthy controls (n = 65) were interviewed by raters blinded for diagnostic status.</td>
<td>Five (17.8%) relatives of the HPD participants and no relative of the controls had HPD.</td>
</tr>
<tr>
<td>Schlosser et al. (1994)</td>
<td>22 adults with HPD</td>
<td>Outpatient psychiatric clinic</td>
<td>Participants interviewed about their relatives</td>
<td>Five participants (22.7%) had relatives with HPD.</td>
</tr>
<tr>
<td>King et al. (1995)</td>
<td>15 children/adolescents with HPD</td>
<td>Outpatient psychiatric clinic</td>
<td>Parents interviewed (n = 30)</td>
<td>One participant (6.7%) had a parent with HPD.</td>
</tr>
<tr>
<td>Cohen et al. (1995)</td>
<td>123 children, adolescents and adults with HPD</td>
<td>Participants were respondents to a national magazine article.</td>
<td>Mail self-report survey. Respondents asked about formal diagnosis of HPD in their family members</td>
<td>Four (3%) reported having at least one family member formally diagnosed with HPD.</td>
</tr>
<tr>
<td>du Toit et al. (2001)</td>
<td>47 adults with HPD</td>
<td>Outpatient psychiatric clinic</td>
<td>Participants interviewed about their relatives</td>
<td>Twelve (25.5%) had a family history of HPD.</td>
</tr>
<tr>
<td>Malhorta, Grover, Baweja, and Bhateja (2008)</td>
<td>20 consecutive children/adolescents with HPD</td>
<td>Child/adolescent psychiatric outpatient clinic</td>
<td>Retrospective chart review of HPD cases (it is not clear whether family history was systematically screened for)</td>
<td>Charts of two (10%) cases indicated a family history of HPD.</td>
</tr>
<tr>
<td>Santhanam et al. (2008)</td>
<td>38 consecutive children/adolescents with HPD</td>
<td>Outpatient psychodermatology clinic</td>
<td>Participants and their parents interviewed</td>
<td>Two (5.3%) children had relatives with SPD.</td>
</tr>
<tr>
<td>Odlaug and Grant (2008b)</td>
<td>Adults with HPD (n = 24), SPD (n = 33) and comorbid HPD/SPD (n = 20)</td>
<td>Outpatient psychiatric clinic</td>
<td>Participants interviewed about their relatives</td>
<td>12.5% of the HPD individuals and 20% of comorbid HPD/SPD individuals reported having first degree relative with HPD.</td>
</tr>
<tr>
<td>Simeon, Stein, et al. (1997)</td>
<td>21 adult with SPD</td>
<td>Outpatient psychiatric clinic</td>
<td>Participants interviewed about their relatives</td>
<td>Four (19%) had first degree relative with SPD.</td>
</tr>
<tr>
<td>Arnold et al. (1998)</td>
<td>34 adults with SPD</td>
<td>Outpatient dermatology clinic</td>
<td>Participants interviewed about their relatives</td>
<td>One (2.9%) reported family history of SPD.</td>
</tr>
<tr>
<td>Wilhelm et al. (1999)</td>
<td>31 adult with SPD</td>
<td>Outpatient psychiatric clinic</td>
<td>Participants interviewed about their relatives</td>
<td>Fourteen (45%) reported having first degree relative with SPD.</td>
</tr>
<tr>
<td>Neziroglu et al. (2008)</td>
<td>37 adults with SPD</td>
<td>Outpatient psychiatric clinic</td>
<td>Participants interviewed about their relatives</td>
<td>Sixteen (43%) reported having first-degree relative with SPD.</td>
</tr>
<tr>
<td>Odlaug and Grant (2008b)</td>
<td>Adults with SPD (n = 33) and comorbid HPD/SPD (n = 20)</td>
<td>Outpatient psychiatric clinic</td>
<td>Participants interviewed about their relatives</td>
<td>19 SPD (36.5%) and no OCD participant had first degree relative with SPD.</td>
</tr>
<tr>
<td>Grant, Odlaug, &amp; Kim (2010)</td>
<td>Adults with SPD (n = 53) and OCD (n = 51)</td>
<td>Outpatient psychiatric clinic</td>
<td>Participants interviewed about their relatives</td>
<td>Four (7.8%) reported family history of SPD.</td>
</tr>
</tbody>
</table>
al., 2010; Swedo et al., 1991; O’Sullivan et al., 1997). In contrast, other researchers have failed to find brain abnormalities in individuals with HPD (Rauch et al., 2007; Stein, Coetzter, Lee, Davids, & Bouwer, 1997). Thus, further studies are needed to examine brain abnormalities in these disorders, and complete lack of data for SPD is especially noteworthy.

5.4. Neurocognitive deficits

Only a handful of studies have investigated neurocognitive deficits in SPD and HPD. Chamberlain, Blackwell, Fineberg, Robbins, and Sahakian (2005) theorized that deficits in inhibitory control underlie both HPD and SPD. However, mixed findings have been reported from studies comparing individuals with HPD or SPD to healthy controls on neurocognitive tests designed to assess motor inhibition (i.e., the Stop Signal Task and the Go/No-go task; Bohne, Savage, Deckersbach, Keuthen, & Wilhelm, 2008; Chamberlain, Fineberg, Blackwell, Robbins, & Sahakian, 2006; Martin et al., 1993; Grant, Odlaug, & Chamberlain, 2011; Odlaug, Chamberlain, & Grant, 2010; Snorrason et al., 2011). A range of other neurocognitive deficits have been found in HPD samples including problems in spatial working memory (Chamberlain et al., 2007), divided attention (Stanley, Hannay, & Breckenridge, 1997), and visuo spatial learning (Chamberlain, Odlaug, Boulougouris, Fineberg, & Grant, 2009). However, no data are available on these deficits in SPD samples. Studies using the Intra-dimensional/Extra-dimensional set shifting task have consistently shown intact cognitive flexibility in both HPD and SPD participants (Chamberlain et al., 2006; Grant et al., 2011; Odlaug et al., 2010).

5.5. Temperamental antecedents

There have been no longitudinal studies conducted on temperamental antecedents of SPD or HPD. However, preliminary cross sectional data indicate that both disorders deviate in similar ways from normal populations with respect to some temperamental traits. First, people with SPD and HPD both score higher than normal controls on questionnaires assessing emotionality or emotion regulation (Lochner, Simeon et al., 2002) found that HPD and SPD patients differed in similar ways from population norms on subscales of the Temperament and Character Inventory. Both groups scored high on harm avoidance (worry, fearfulness, pessimism), very high on reward dependence (sociality, warmth, dependence) but in the average range on novelty seeking. Another study using a different version of this scale in a community sample found that hair pulling was associated with harm avoidance, but not reward dependence, and skin picking was related to neither dimension (Favaro, Ferrara, & Santonastaso, 2007). However, this study is limited due to inadequate diagnosis of HPD and SPD and few diagnosed individuals. In sum, preliminary data suggest that both HPD and SPD are characterized by emotional temperament, and possibly harm avoidance and reward dependence. The extent to which these traits represent causal agents in the development of these problems (as opposed to e.g., being a result of the behavior itself or comorbid conditions) remains to be investigated.

5.6. Environmental risk factors

5.6.1. Under-stimulated environment

Lack of stimulation is a well documented risk factor for compulsive behaviors, including over-grooming, in animals (Moon-Fanelli, Dodman, & O’Sullivan, 1999), but this issue has received scant research attention in the human literature. Nonetheless, there are some clues suggesting that this type of environmental stressor may have a role in HPD and SPD. First, experimental studies (Teng et al., 2004) and self-report surveys (Shusterman et al., 2009; Snorrason et al., 2010) show that boredom (i.e., lack of stimulation) often triggers skin picking and hair pulling episodes. Secondly, case reports (Evans, 1976; Gupta et al., 1986) suggest that a period of severe activity restriction sometimes precedes the development of HPD and SPD. Further studies are warranted to investigate this interesting hypothesis.

5.6.2. Trauma

A number of case studies have reported an association between HPD and history of trauma (Bordnick, Thyer, & Ritchie, 1994) and a few uncontrolled studies suggest high incidence of trauma in clinical samples of HPD (Boughn & Holdom, 2003; Gershuny et al., 2006) and SPD (Neziroglu et al., 2008) patients. However, it is unclear whether history of trauma is more prevalent in these samples compared to other psychiatric samples (Lochner, du Toit et al., 2002). Favaro et al. (2007) interviewed a large community sample (n = 934) of young females

| Table 4 |
| Shared Familiality of hair pulling disorder (HPD) and skin picking disorder (SPD). |

<table>
<thead>
<tr>
<th>Reference</th>
<th>Sample</th>
<th>Settings</th>
<th>Assessment</th>
<th>Results</th>
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</thead>
<tbody>
<tr>
<td>HPD in relatives of individuals with SPD</td>
<td>Simeon, Stein, et al. (1997)</td>
<td>21 adult with SPD</td>
<td>Outpatients psychiatric clinic</td>
<td>Participants interviewed about their relatives</td>
</tr>
<tr>
<td></td>
<td>Arnold et al. (1998)</td>
<td>34 adults with SPD</td>
<td>Outpatient dermatology clinic</td>
<td>Participants interviewed about their relatives (n = 279)</td>
</tr>
<tr>
<td></td>
<td>Odlaug and Grant (2008b)</td>
<td>Adults with HPD (n = 24), SPD (n = 33) and comorbid HPD/SPD (n = 20)</td>
<td>Outpatients psychiatric clinic</td>
<td>Participants interviewed about their relatives</td>
</tr>
<tr>
<td></td>
<td>Grant, Odlaug, &amp; Kim (2010)</td>
<td>Adults with SPD (n = 53) and OCD (n = 51)</td>
<td>Outpatient psychiatric clinic</td>
<td>Participants interviewed about their relatives</td>
</tr>
<tr>
<td>SPD in relatives of individuals with HPD</td>
<td>King et al. (1995)</td>
<td>15 children/adolescents with HPD</td>
<td>Outpatient child/adolescent psychiatric clinic</td>
<td>Parents interviewed (n = 30)</td>
</tr>
<tr>
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<td>Odlaug and Grant (2008b)</td>
<td>Adults with HPD (n = 24), SPD (n = 33) and comorbid HPD/SPD (n = 20)</td>
<td>Outpatient psychiatric clinic</td>
<td>Participants interviewed about their relatives</td>
</tr>
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</table>
6. Discussion

6.1. What is the nature of the relationship between HPD and SPD?

Overall, our review revealed strong evidence for relatedness between HPD and SPD. Existing data suggest that the disorders have substantial similarities in a variety of clinical features and have significant comorbidity that is likely not due to methodological artifacts. Nonetheless, there are many possible explanations for the relationship between HPD and SPD. For instance, it is conceivable that the two disorders do not share etiology and that one disorder for some reason simply enhances the risk for the other, thus explaining the high co-occurrence rates. However, empirical data showing a consistent pattern of one disorder preceding the other are lacking. Also, the substantial similarities in clinical characteristics and shared risk factors suggest that the relationship is more inherent than that. Another possibility is that comorbid cases represent a distinct entity with independent etiology from pure cases. Again, this explanation is unsatisfactory because it does not account for the similarities in clinical features. Moreover, Orlaugh and Grant (2008b) did not find meaningful differences between comorbid cases and pure forms of the disorders.

The most parsimonious explanation for the overall literature is that the two disorders share etiology. Given evidence for both unique and shared genetic underpinning, and some unique risk factors across HPD and SPD (see below), it is perhaps inaccurate to conclude that the two disorders are simply different expressions of the same underlying condition. They likely have both shared and unique etiological mechanisms. The state of the literature does not allow strong statements regarding the nature or the extent of the shared etiology, but some broad conclusions can be made. First, evidence suggests that the two disorders have a common genetic underpinning possibly explaining, at least in part, the comorbidity and similarities in clinical features. Second, it seems plausible that the two disorders share some proximal etiological mechanisms. The similarities in phenomenology may provide clues as to what these mechanisms might be. Both hair pulling and skin picking modulate affective states as well as providing pleasurable stimulation, and it appears that affective experiences play an important role in maintaining and possibly causing these problems (Shusterman et al., 2009; Snorrason et al., 2010).

Interestingly, healthy individuals report similar affective experiences associated with normal body grooming behaviors (e.g., picking at acne or plucking eyebrows for cosmetic reasons), although the intensity is much less than reported by patients (Bohne, Wilhelm, Keuthen, Baer, & Jenike, 2002; Keuthen et al., 2000; Snorrason et al., 2010). It may thus be hypothesized that some of the underlying etiological mechanisms shared by HPD and SPD have to do with a propensity for intense affective experiences in relation to body-grooming behaviors.

It should be noted that both SPD and HPD may be heterogeneous conditions (Arnold et al., 2001; Grant, Orlaugh, et al., 2010; Lochner et al., 2010), and it is possible that only subgroups of each disorder share etiological mechanism. For instance, Stein et al. (2008) found that HPD patients with a comorbid BFRB, including skin picking, were more likely to be “focused pullers” (i.e., pull with full awareness in response to an urge or negative affect) than HPD patients without such comorbidity.

6.2. Future directions

Even though significant progress has been made in describing symptom presentation of HPD and SPD, further research is warranted. For example, clinical lore suggests that perfectionistic experiences are common in SPD and HPD (Arnold et al., 1998; Fruensgaard, 1984; Gupta, Gupta, & Schork, 1996; Swedo et al., 1992), but empirical data are lacking (although some data concerning picking/pulling to obtain symmetry have been collected, Tucker et al., 2011; Woods, Flessner, et al., 2006). In addition to investigating the commonality in symptoms, a potentially fruitful approach is to identify shared underlying psychological, neurocognitive or psychobiological processes. For example, investigating psychophysiological or psychocutaneous processes involved in affective experience surrounding body grooming may prove fruitful. Other researchers have suggested that HPD and SPD may have overlapping deficits in affective regulation, inhibitory control and processes involving reward/addiction (Stein et al., 2006). Given preliminary evidence for a shared genetic risk between SPD and HPD, it may be useful to investigate whether deficits in some underlying processes represent a shared endophenotype (Chamberlain et al., 2005).

Another useful approach to shed light on the relationship between SPD and HPD is to investigate different explanatory models of comorbidity (e.g., partly or fully shared etiology, one disorder causes the other, etc.). By quantifying data from epidemiological studies, longitudinal family studies or twin studies, researchers are increasingly able to test hypotheses concerning causes of comorbidity (Klein & Riso, 1993; Neale & Kendler, 1995). These methods typically require large sample sizes but can be helpful in understanding the relationship between SPD and HPD.

Finally, understanding the differences between HPD and SPD may provide unique insights into the etiology of either or both conditions. There are at least four lines of research suggested by the current literature. First, one study found that dermatological problems often preceded the development of SPD (Wilhelmi et al., 1995), and it is possible that these types of problems serve as a higher risk for SPD than HPD. Second, in a preliminary study involving few participants, it was found that SPD patients, but not HPD patients, showed enhanced response to an opiate sensitivity test, suggesting a difference between the disorders with regard to the role of endogenous opiates (Frecska & Arato, 2002). Third, given data showing a higher prevalence of SPD secondary to BDD, than HPD secondary to BDD (Grant et al., 2006), it may be that appearance concerns are more of a risk for SPD than HPD. Fourth, Bienvenu et al. (2009) discovered independent as well as shared genetic underpinning associated with HPD and SPD. Likewise, the family history studies showed that relatives of HPD patients were more likely to have HPD than SPD, and relatives of SPD patients were more likely to have SPD than HPD. Thus, future studies may want to investigate familial environment or a genetic underpinning that differently affects the risk for the two disorders.

6.3. Implications for research and practice

The findings from the current review have nosological implications. Given the evidence for co-occurrence of HPD and SPD in clinical samples along with data showing relatedness in clinical characteristics and response to the same treatment (e.g., habit reversal), it seems appropriate to categorize the two disorders in the same overall category in DSM and ICD. The possibility that HPD and SPD share etiological mechanisms provides further support for these nosological arrangements. As to the question of what general category the two disorders fit best, our review concurs with the preliminary recommendation of the APA work group (Stein et al., 2010) that the two disorders should optimally be classified in an overall category of BFRB. Consistent with this view are data showing high comorbidity between different BFRBs, similarities in phenomenology (e.g., arousal regulation) and genetic associations.

Other classification schemes may also have validity. Some authors have argued that SPD and HPD are heterogeneous conditions that can be conceptualized as part of the putative obsessive–compulsive spectrum (Ferrão, Miguel, & Stein, 2009; Stein et al., 1995), and others have suggested classifying SPD patients according how much their symptoms involve impulsive and compulsive features (Arnold et al., 2001). Our review showed that the majority of patients in both groups endorse...
impulsive symptoms [i.e., preceding arousal and subsequent gratification], but data concerning compulsivity is less clear (Ferrão, Almeida, Bedin, Rosa, & Busnello, 2006). Nonetheless, there appears to be some affinity between HPD/SPD and compulsive disorders (OCD and BDD) that warrants further research attention (Ferrão et al., 2009). Similarly, despite evidence for impulsive features, further studies are needed to investigate the relationship between SPD/HPD and other disorders involving impulsivity (Grant, Odlaug, & Potenza, 2007; Odlaug & Grant, 2010; Snorrason et al., 2011).

Our review also has implications for research. Shared etiology would suggest that researchers investigating the etiology of either disorder might benefit from considering the other one too. Comparing and contrasting findings from studies on SPD and HPD may provide unique insights into the etiology of either or both disorders, and information concerning one disorder could guide research on the other. Moreover, a failure to consider the other disorder when studying etiological factors in either one may result in loss of power. For example, if the disorders share etiology, then failure to screen for both disorders when selecting healthy controls may obscure group differences. Finally, given that HPD and SPD serve similar functions (e.g., affect/avoidance regulation), clinicians and researchers investigating treatment may want to consider the possibility of symptom substitution and monitor changes in both behaviors when treating comorbid cases.

References


Arnold, L. M., Auchenbach, M. B., & McElroy, S. L. (2001). Psychogenic excoriation: the possibility of symptom substitution and monitor changes in both share etiology, then failure to screen for both disorders when selecting contrasting might benefit from considering the other. Moreover, a failure to consider the other disorder when studying etiological factors in either one may result in loss of power. For example, if the disorders share etiology, then failure to screen for both disorders when selecting healthy controls may obscure group differences. Finally, given that HPD and SPD serve similar functions (e.g., affect/avoidance regulation), clinicians and researchers investigating treatment may want to consider the possibility of symptom substitution and monitor changes in both behaviors when treating comorbid cases.

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