Olfaction and Social Drive in Schizophrenia

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Background: The neurobiology of social dysfunction in schizophrenia is unknown, but smell identification deficits (SIDs) exist in schizophrenia, and olfaction is related to social affiliation in other mammals. The SIDs have been linked with negative symptoms and the deficit syndrome, but any specificity of SIDs for social dysfunction is unstudied. Low intelligence might explain this relationship, if it is associated with both negative symptoms and SIDs. We examined whether SIDs in schizophrenia were related broadly to negative symptoms, as are a number of other neuropsychological measures, or whether they might show a more specific relationship with social drive.

Methods: Smell Identification Test scores, Wechsler Adult Intelligence Scale-Revised IQ, symptomatology assessed with the Positive and Negative Syndrome Scale, and the deficit syndrome were determined in 70 patients with DSM-IV schizophrenia.

Results: The SIDs were related to negative symptoms and the deficit syndrome, but the association of SIDs with diminished social drive explained both relationships. Smell identification was also related to Wechsler Adult Intelligence Scale-Revised IQ, but intelligence was independent of the relationship of SID and social drive. The worse Smell Identification Test scores in male patients were attributable to a greater preponderance of men with the deficit syndrome.

Conclusions: These analyses demonstrated independent relationships of Smell Identification Test scores to social drive and intelligence that together accounted for almost 50% of the variance in Smell Identification Test scores. There may be common neural substrates for the low social drive and SIDs in schizophrenia.

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SOCIAL DEFICITS represent a significant component of disease expression in schizophrenia. They are often present from early life, preceding the onset of psychosis and further deterioration in the disease, and they constitute the most treatment-refractory negative symptoms in patients with familial disease. Little is known about the neurobiological substrates of this social impairment. However, social affiliation is related to olfactory processing in most mammals, and olfactory dysfunction is well described in schizophrenia, hinting that olfaction and social function may be interconnected in schizophrenia. Smell identification deficits (SIDs), in particular, exist irrespective of clinical state and medication treatment and, like social dysfunction, occur across the disorders of the schizophrenia spectrum, including schizoaffective disorder and schizotypal personality disorder, and in high-risk individuals. A role for SIDs as an endophenotype for this social incapacity might significantly benefit genetic research in schizophrenia.

The relationship of SIDs to phenomenology in schizophrenia has been relatively unstudied. Disorganized and depressive symptoms were previously considered in relationship to SIDs, but a few recent reports have linked SIDs with negative symptoms and social behavior deficits on the Life Skills Profile, flattened affect, and the deficit syndrome (DS). The DS indicates the presence of enduring primary negative symptoms, and it is associated with premorbid social dysfunction and poor long-term social and occupational outcome. These findings enhance the plausibility that SID and social incapacity could be specifically associated in schizophrenia, although there may be other explanations for these findings. For example, SID could be one of the many neurobehavioral abnormalities that have been non-specifically associated with negative symptoms. Endemic deficits in attention might also underlie SIDs, although this seems unlikely because SIDs are unrelated to vigilance or sustained attention on neuropsychological tests. The intellectual impairments that are the source...
of other cognitive deficits in schizophrenia could cause SIDs. Indeed, SIDs have been related to IQ estimated from abbreviated forms of the Wechsler Adult Intelligence Scale-Revised (WAIS-R) and the National Adult Reading Test. However, the verbal information subtest of the WAIS-R and reading comprehension tests, which are commonly used to estimate intelligence, also depend heavily on educational and cultural opportunities, limiting the interpretation that low IQ explains SIDs. No previous study has administered the complete WAIS-R to more fully address the relationship of intelligence to SIDs in schizophrenia. Nonetheless, patients with enduring negative symptoms have lower intelligence, so an association between SIDs and negative symptoms could be explained by the relationship of both measures with intelligence. To the contrary, if SIDs show a specific relationship to social motivation after controlling for intelligence, a parsimonious hypothesis is that dysfunction in the circuitry of olfactory processing and impaired social performance are more directly related.

It is unclear whether negative symptoms cause social dysfunction, whether social dysfunction is simply one of several negative symptoms, or whether negative symptoms and social deficits reflect separate processes. Under the impairment criteria in the DSM-IV, social performance is considered inability to work, study, socialize, or provide self-care. It is merely alluded to in the negative symptom criteria, which exemplify negative symptoms as flat affect, reduced speech, and lack of volition. It is arguable whether poor social function suffices to fulfill the negative symptom criteria in the absence of other negative symptoms, since these are separate criteria in the DSM-IV. If not, then social impairments can be present without prominent negative symptoms, and these may reflect independent domains.

We examined the relationship between SIDs, social function, negative symptoms, and other phenomenology in a large sample of patients with well-characterized schizophrenia. Aspects of social function are captured by the enduring symptoms of the DS and by cross-sectional rating of negative symptoms. We also considered the relationship of SIDs to intelligence and the extent to which intelligence might mediate any relationship between symptoms and SIDs.

**METHODS**

**SUBJECTS**

Subjects included 70 patients with DSM-IV schizophrenia or schizoaffective disorder and 68 community comparison subjects who had no personal or family history of psychosis and no Axis I disorder in the past 2 years. Patients were interviewed with the Diagnostic Interview for Genetic Studies, and their diagnosis represented a consensus among clinical and research staff. All subjects provided written informed consent for this institutional review board–approved study.

**METHODS**

Smell identification was assessed with the University of Pennsylvania Smell Identification Test (SIT) (Sensometrics, Inc, Haddon Heights, NJ), a standardized multiple-choice scratch-and-sniff test of 40 common microencapsulated odors, each accompanied by 4 presented words (eg, chocolate, pizza, smoke, and lilac), one of which is the odor name.

The DS was assessed with the Schedule for the Deficit Syndrome using information from patient and family interviews, medical chart reviews, and discussions with the clinical staff. Restricted affect, diminished emotional range, poverty of speech, curbing of interests, diminished sense of purpose, and diminished social drive were rated from 0 (normal) to 4 (severe). The DS was rated as present if 2 of these symptoms were determined to be severe, primary, and stable.

Cross-sectional symptoms were rated using the Positive and Negative Syndrome Scale (PANSS) and the Hamilton Rating Scale for Depression (HRSD). The PANSS data were summarized into the original positive, negative, and general psychopathology scales and into the empirically derived factors of the pentagonal model that separates PANSS data into positive, dysphoric mood, negative, activation, and autistic preoccupation factors. The latter 3 factors consider negative types of symptoms but differ from the original PANSS negative scale by excluding difficulty in abstraction and stereotyped thinking items and by including motor retardation, mannerisms, uncooperativeness, impaired volition, and impulsivity. The PANSS dysphoric mood factor separates depression from negative symptoms better than the HRSD, evidenced by its greater sensitivity to the lower levels of depression in patients with the DS.

Level of function in the previous month was rated with the Global Assessment of Function scale. Intellectual ability full-scale IQ score was based on a complete WAIS-R administration. Verbal and performance IQ scores were separately determined for verbal comprehension and perceptual organization.

The Quick IQ Test modified Mini-Mental State Examination (MMSE) were also administered to some patients, including those who could not complete the full WAIS-R.

**STATISTICAL METHODS**

We compared SIT scores, age, and sex distributions between the patient and comparison samples. In the patients, associations between SIT scores and years of education, intelligence, and symptoms were examined by means of Pearson correlations. We used t tests to compare DS groups on SIT scores and demographic measures and to compare SIT scores for male and female patients. The DS domain was operationalized into dimensional variables of global severity (0-4), severity for each item (0-4), and total deficit item severity (0-24 for the sum of the 6 items), and these were examined with respect to SIT scores.

Logistic regression analyses evaluated the likelihood that the DS was related to SIDs. We expected from our earlier study of 50 of these 70 patients that the DS category would be a strong significant predictor of SIT scores. We herein examined the suitability of using the severity of diminished social drive to index the entire DS. Preliminary analyses selected which variables were included on step 1 of a series of hierarchical regression equations, the DS was entered on step 2, and SIT score was specified as the outcome variable. We alternatively included specific PANSS symptom ratings that were significantly associated with SIT scores in regression analyses of the main hypotheses. The PANSS factors were precluded from simultaneous entry into regression equations due to shared items. We examined whether the enduring symptoms rated with the DS were more sensitive to SIDs than cross-sectional PANSS ratings of negative symptoms by constructing hierarchical regression equations with the PANSS negative scale on step 1 and the deficit domain variable on step 2, specifying SIT score as the outcome. The DS/non-DS categorization was coded as a dummy
RESULTS

SIT SCORES IN THE SAMPLES

The patients had significantly lower SIT scores than comparison subjects (mean scores, 31.8±4.7 vs 36.1±3.0; t110,7 =−6.5; P < .001; 95% confidence interval [CI], −5.7 to −3.0). These group differences could not be explained by sex (the groups were approximately 63% male) or by the deterioration of olfactory ability with age, as the patients were significantly younger than the comparison subjects (mean age, 32.9±4.8 vs 33.3±4.3; t68=4.16; P < .001). Lower mean SIT scores for male than female patients could be completely explained by the larger proportion of male patients (16/19 [84%] vs 28/51 [55%]; χ²1 = 5.09; P = .02).

In contrast, mean Quick IQ Test (90.1±17.3; n = 36) and MMSE scores (30.9±3.3; n = 57) were not associated with the SIT scores (r = −.30; 95% CI, 0.59-2.82) and had a greater proportion of male patients (16/19 [84%] vs 28/51 [55%]; χ²1 = 5.09; P = .02). Male patients (30.9±3.3; n = 57) had significantly less education than the non-DS group (mean years of education, 11.1±1.7 vs 12.8±2.8; t62=3.07; P = .003; 95% CI, 0.59-2.82) and had a greater proportion of male patients (16/19 [84%] vs 28/51 [55%]; χ²1 = 5.09; P = .02).

As we have previously reported,16 SIDs in these patients were not related to age, ethnicity, smoking, socioeconomic status, or clinical characteristics such as diagnostic subtype, age of onset, number of psychiatric hospitalizations, or duration of illness. Mean SIT scores also did not differ between the 54 patients receiving stable doses of antipsychotic medications and the 16 patients who were not taking antipsychotic medications (31.5±4.7 and 32.9±4.8, respectively; t68 = 1.05; P = .30). Male patients had significantly lower mean SIT scores than female patients (30.9±4.7 vs 33.3±4.3; t68=−2.17; P = .03).

Educational attainment (mean, 12.3±2.6 years) was significantly correlated with SIT scores in the patients (r62=0.28; P = .021), as were mean WAIS-R full-scale (82.2±13.7), verbal (85.7±14.3), and performance (79.7±13.5) IQ scores (r42=−.43; P<.003). In contrast, mean Quick IQ Test (90.1±17.3; n = 36) and MMSE scores (30.9±3.3; n = 57) were not associated with the SIT scores (r = −.30; 95% CI, 0.59-2.82) and had a greater proportion of male patients (16/19 [84%] vs 28/51 [55%]; χ²1 = 5.09; P = .02).

The DS group had significantly lower mean SIT scores than the non-DS group (28.3±4.7 vs 33.1±4.1; t62=4.16; P < .001; 95% CI, 2.5-7.0). The SIT scores expressed as percentile scores based on published age- and sex-specific norms36 revealed that 17 (89%) of 19 in the DS group had a clinically significant impairment in olfaction likely to have an impact on daily function, compared with only 6 (12%) of the 51 patients in the non-DS group (odds ratio [OR], 7.6; 95% CI, 1.6-36.1). The mean SIT score for the DS group was only at the ninth (±7.8) SIT percentile.

Increasing DS severity was associated with lower SIT scores (Table 1), irrespective of whether DS severity was indexed as global severity, deficit domain severity, or severity of diminished social drive. Post hoc analyses showed that 5 of the 6 DS items were significantly inversely associated with SIT scores and that the sixth, poverty of speech, showed a similar trend.

LOWER SIT SCORES IN MALE PATIENTS ATTRIBUTABLE TO THEIR DS DIAGNOSIS

Lower mean SIT scores for male than female patients could be completely explained by the larger proportion of male patients categorized as having the DS. Female patients in the DS group scored as poorly as male patients in the
DS group. In analyses that controlled for sex, the DS explained a significant unique amount of SIT score variance and was associated with a 4.3-point decrement in SIT scores. Conversely, after controlling for the DS, sex and SIT scores were no longer related.

SIT SCORES AND SYMPTOMS

The PANSS positive symptoms, general psychopathology, and depressive symptoms were unrelated to the SIT scores, but SIT performance was significantly negatively correlated with the negative scale (r$_{61}$ = −0.32; P = .01), negative factor (r$_{61}$ = −0.28; P = .03), and autistic preoccupation factor (r$_{61}$ = −0.26; P = .04). Post hoc analyses of the 15 PANSS symptoms included in any of these negative symptom indexes showed that only 2, lack of spontaneity and flow of conversation and also impaired volition, were significantly related with SIT scores (Table 2). Lack of spontaneity is included in the traditional PANSS negative subscale and the negative factor, and impaired volition contributes to the negative and autistic preoccupation factors, although it is not included in the traditional PANSS negative scale. The PANSS impaired volition was worse in the DS than non-DS groups (r$_{22.18}$ = −2.79; P < .01), and it was strongly related to the overall severity (r$_{21}$ = 0.47; P < .001) and the severity of the item for diminished sense of purpose (r$_{61}$ = 0.54; P < .001) in the Schedule for the Deficit Syndrome.

DIMINISHED SOCIAL DRIVE AS AN INDEX FOR DS

The correlations among the severity ratings for the 6 DS items, which are considered to be a cohesive set of trait-related phenomena, were high and homogeneous (r$_{70}$ = 0.50-0.83; P < .001). Regression analyses showed a strong significant association between diminished social drive and the sum of the other 5 deficit symptoms (r$_{70}$ = 0.77; P < .001), indicating that diminished social drive could account for the entire Schedule for the Deficit Syndrome score. Similarly, regression analyses showed it was the strongest predictor of SIT scores. It was selected by means of stepwise criteria in a simultaneous regression and accounted for 23% of the SIT score variance (F$_{1.68}$ = 20.6; P < .001). With diminished social drive entered on step 1 in hierarchical regression equations predicting SIT scores, none of the other 5 DS symptoms, as a group or individually, increased the amount of explained SIT score variance.

Next, all demographic variables with significant zero-order correlations to SIT scores were included on step 1 of a hierarchical regression equation. The DS was entered on step 2, and the SIT score was specified as the outcome variable (Table 3). Sex, years of education, and WAIS-R full-scale IQ score together explained 30% of the variance in SIT scores (F$_{3.38}$ = 5.4; P = .003), but only WAIS-R full-scale IQ score remained a significant predictor of SIT score, with the other 2 variables held constant. The addition of DS category to step 2 increased the amount of predicted variance in SIT scores by 15% (F$_{1.38}$ = 11.7; P = .001), resulting in a complete equation that accounted for 47% of the variance in SIT scores in the patients with schizophrenia (F$_{1.38}$ = 8.1; P < .001). The DS category then predicted a 4.7-point reduction in SIT score (95% CI, −7.5 to −1.9), with the other variables held constant. To check for suppression effects, an attempt was made to enter each demographic and clinical variable on step 3 of the equation using a stepwise method. No additional variable entered the equation.

DS EXPLAINS GREATER AMOUNT OF VARIANCE IN SIT SCORES THAN PANSS NEGATIVE SYMPTOM RATINGS

A hierarchical regression equation with the PANSS negative symptom scale on step 1, the DS category on step 2, and SIT score specified as the outcome showed that adding the DS category on step 2 resulted in a significant 11% increment in explaining the variance in SIT scores (F$_{1.38}$ = 8.1; P = .006) (Table 3). The DS category pre-

Table 2. PANSS Symptom Items and Post Hoc Correlations With SIT Scores

<table>
<thead>
<tr>
<th>PANSS negative symptoms</th>
<th>Mean (SD)</th>
<th>Pearson r</th>
<th>No. of Patients</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Blunted affect</td>
<td>2.7 (1.5)</td>
<td>−0.13</td>
<td>61</td>
<td>.34</td>
</tr>
<tr>
<td>2 Emotional withdrawal</td>
<td>2.6 (1.4)</td>
<td>−0.20</td>
<td>61</td>
<td>.12</td>
</tr>
<tr>
<td>3 Poor rapport</td>
<td>2.3 (1.5)</td>
<td>−0.19</td>
<td>61</td>
<td>.14</td>
</tr>
<tr>
<td>4 Passive/apathetic social withdrawal</td>
<td>2.5 (1.4)</td>
<td>−0.18</td>
<td>61</td>
<td>.17</td>
</tr>
<tr>
<td>5 Difficulty in abstract thinking</td>
<td>3.2 (1.5)</td>
<td>−0.21</td>
<td>60</td>
<td>.11</td>
</tr>
<tr>
<td>6 Lack of spontaneity</td>
<td>2.5 (1.4)</td>
<td>−0.35</td>
<td>61</td>
<td>.005</td>
</tr>
<tr>
<td>7 Stereotyped thinking</td>
<td>1.7 (1.0)</td>
<td>−0.18</td>
<td>61</td>
<td>.16</td>
</tr>
</tbody>
</table>

Additional symptoms from negative factor

<table>
<thead>
<tr>
<th></th>
<th>Mean (SD)</th>
<th>Pearson r</th>
<th>No. of Patients</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>5 Mannerisms and posturing</td>
<td>1.3 (0.9)</td>
<td>−0.01</td>
<td>61</td>
<td>.93</td>
</tr>
<tr>
<td>8 Uncooperativeness</td>
<td>1.9 (1.4)</td>
<td>−0.05</td>
<td>61</td>
<td>.68</td>
</tr>
<tr>
<td>13 Disturbance of volition</td>
<td>1.5 (1.0)</td>
<td>−0.50</td>
<td>61</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>14 Poor impulse control</td>
<td>1.4 (0.9)</td>
<td>0.06</td>
<td>61</td>
<td>.63</td>
</tr>
</tbody>
</table>

Additional symptoms from autistic factor

<table>
<thead>
<tr>
<th></th>
<th>Mean (SD)</th>
<th>Pearson r</th>
<th>No. of Patients</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>11 Poor attention</td>
<td>2.0 (1.4)</td>
<td>−0.06</td>
<td>61</td>
<td>.66</td>
</tr>
<tr>
<td>15 Preoccupation</td>
<td>2.2 (1.5)</td>
<td>−0.01</td>
<td>61</td>
<td>.94</td>
</tr>
<tr>
<td>3 Hallucinatory behavior</td>
<td>2.6 (1.7)</td>
<td>−0.13</td>
<td>61</td>
<td>.31</td>
</tr>
</tbody>
</table>

Abbreviations: PANSS, Positive and Negative Syndrome Scale; SIT, Smell Identification Test.
dicted a 4.1-point reduction in SIT score (95% CI, −6.9 to −1.2) when the negative symptom scale was held constant. Each of the 2 negative-type PANSS factors with zero-order correlations to the SIT scores were then substituted on step 1 of the equation. When the DS category was entered after the PANSS negative factor, it resulted in an additional 13% of the explained variance in the SIT scores ($F_{1,58}=9.1; P=.004$), and when the DS was entered after the PANSS autistic factor, it increased the explained variance in SIT scores by 17% ($F_{1,58}=12.8; P=.001$). However, when the DS category was entered onto the first step of a hierarchical regression equation predicting the SIT scores, no subsequently entered PANSS state rating served as a significant predictor of the SIT scores.

Irrespective of the specific PANSS or DS variable used, regression analyses showed that the primary and enduring symptom of diminished social drive, captured by the DS designation, explained a significant amount of variance in the SIT scores beyond that indexed by the cross-sectional negative symptoms rated with the PANSS.

### SEPARATE STRONG LINKS OF INTELLIGENCE AND THE DS WITH SIT SCORES

Higher intelligence was associated with better SIT scores in the DS and non-DS groups, with 25% of the variance in SIT score accounted for by the full-scale IQ score alone. Verbal and performance IQ scores had large, independent, positive correlations with SIT scores. This is consistent with a strong link between higher general intelligence and smell identification ability, rather than a more circumscribed connection explanation that verbal comprehension or perceptual organization accounted for smell identification ability.

This link between the DS and SIDs was independent of this strong relationship between IQ and SIT score. The WAIS-R IQ score and DS category each accounted for a statistically significant unique portion of variance in SIT scores. When the WAIS-R full-scale IQ score was held constant, DS category accounted for an additional 20% of the variance in SIT scores and was associated with approximately a 4.9-decrement in SIT score. When the DS category was held constant, WAIS-R IQ accounted for an additional 12% of the variance in SIT scores, with 10 IQ points associated with approximately 1.2 SIT points.

### COMMENT

This study showed a specific relationship between SIDs and diminished social drive in schizophrenia. In accord with every other published study, we found significantly lower SIT scores in patients with schizophrenia than in healthy comparison subjects. These SIDs were not associated with positive symptoms, general psychopathology, or depressive symptoms, but were strongly associated with the PANSS negative symptoms. However, the only PANSS negative symptoms associated with SIDs were lack of spontaneity and impaired volition. Moreover, after accounting for the primary and enduring symptom of diminished social drive captured by the DS designation, these state-related negative symptoms were no longer associated with SIDs. Diminished social drive alone accounted for 23% of the total variance in smell identification ability, and it completely explained the relationship between the DS and SIT scores.
Although WAIS-R full-scale IQ score also explained a quarter of the SIT score variance, this relationship was independent of the association between social drive and smell identification ability. The independent relationships of intelligence and social drive with SIT scores together accounted for almost 50% of the SIT score variance. The WAIS-R verbal, performance, and full-scale IQ scores each had similar strong links to the SIT scores. This finding is consistent with a strong link between higher general intelligence and smell identification ability, rather than a more circumscribed connection explanation that verbal comprehension or perceptual organization accounted for smell identification ability. Furthermore, MMSE and Quick IQ Test scores were not related to SIT scores in this sample. The MMSE assesses orientation, concentration, naming of common objects, and comprehension of simple directions, which corresponds to the nonolfactory task demands of the SIT. The Quick IQ Test, also called the Picture Vocabulary Test, requires matching words to common objects, which is also analogous to the SIT except that the stimulus is an object rather than an odor. Since impairments in these abilities did not detectably lower SIT scores, the correlation between WAIS-R IQ and SIT scores is also unlikely to be explained by impaired attention, concentration, or vocabulary. Rather, it may be useful to consider that higher intelligence may augment SIT performance, although it does not protect against the SIDs that are associated with diminished social drive. No previous SIT schizophrenia study has administered the complete WAIS-R rather than abbreviated forms to so fully address these issues.

The DS and non-DS groups did not differ in intelligence, although the DS group had significantly fewer years of education. We propose that the lower social drive in the DS group leads to lack of persistence in school, which may especially impede the transition from high school to college. The observed association of SIT scores with education resulted, in part, from the fewer years of education and the worse SIT scores achieved by the DS group. Education and SIT scores were also associated through the mediating variable of WAIS-R IQ scores. Although education and SIT scores were moderately associated in the DS group, the lower level of education in the DS group did not explain their SIDs, because education was not related to SIT scores after controlling for the DS category. Thus, although each additional year of education was associated with a half-point increase in SIT score, this could be explained by an increased attrition of subjects with low social drive from formal schooling.

We also replicated reports showing significantly more SIDs in male than female patients with schizophrenia, but we conducted additional analyses that established sex difference as entirely explained by the higher proportion of male patients in the DS group. Although the excess of male patients with the DS is well described, the reason that male patients with schizophrenia are more likely to have a low social drive remains to be determined. If deficient social drive and SIDs have a common etiopathology, then sex differences in the biological underpinnings of olfactory and social function, which favor female patients, may be relevant for consideration.

We were particularly interested in social drive because, at face value, it is most closely analogous to social affiliation in other mammals. Lack of engagement in social relationships may provide the context in which emotional experience and expression is dampened, communication is impoverished, sense of purpose in life is attenuated, and interests are stunted. Lack of social interest may be central to the lower education level and poor vocational and social adaptation of some patients with schizophrenia throughout their lives. Diminished social drive from the DS and impaired volition and lack of spontaneity from the PANSS all concern impaired volition. Volition is the extent to which someone posits, initiates, sustains, and/or completes goal-directed activity. Avolition is a contemporary conceptualization of the Kraepelin avolition syndrome, defined as “a weakening of those emotional activities [that] permanently form the mainsprings of volition,” resulting in “emotional dullness, failure of mental activity, loss of mastery over volition, of endeavor and of ability for independent action.” It is one of 3 core negative symptoms in the DSM-IV diagnostic criteria for schizophrenia. This definition was operationalized in the DS, and the PANSS avolition item considers the overarching definition of the Kraepelin pathological process as well as a specific manifest symptom.

Although our results replicate other reports associating cross-sectional (state) ratings of negative symptoms with SIT scores, future investigations may find only weak or nonsignificant associations between global negative symptoms and SIT scores, particularly if the negative symptoms assessed do not consider impairments in volition or social drive. We found no relationship between SIT scores and depression using the HRSD or the PANSS dysthymia factor. Earlier studies reporting such a relationship could have misclassified deficit symptoms as depressive symptoms. Loss of interest in work or loss of libido can occur in the DS and depression and would be scored on the HRSD even if it were a primary manifestation of the DS.

Decrements in social drive and SIDs could result from a common neurodevelopmental etiopathology. During childhood and adolescence, social affiliative behavior and smell identification ability follow a maturational course that depends on appropriate neural circuitry laid down during fetal life. In the adult brain, neurogenesis continues to occur exclusively in 2 privileged regions, ie, the olfactory bulb and entorhinal-hippocampal cortex. Thus, schizophrenia-linked abnormalities in neural development and regeneration would be expected to affect primary olfactory cortices preferentially and consequently result in behavioral deficits in olfaction and social affiliation. Anatomical disarray at the level of synapses or neurons or deficient neurotransmitter levels could account for the random, rather than circumscribed, misidentification of specific odors that occur on the SIT in patients with schizophrenia. Determining the etiology of social impairments could be key to identifying the pathophysiology of a subgroup of schizophrenia cases, or to gene identification relevant to the social incapacity in schizophrenia. Such knowledge could also augment research on the biology of human social affiliation, in general.
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