Neurocognitive predictors of confabulation in Schizophrenia: a systematic and quantitative review

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Confabulations, or false memories, are observed in various disorders, including schizophrenia. In forensic psychiatric assessment, this is problematic, particularly when garnering a clinical history and detailed account of the index offense(s) from the individual being charged. This study sought to quantitatively synthesize the existing literature regarding the frequency of confabulations in schizophrenia and its neurocognitive correlates. The findings suggest that patients with schizophrenia confabulated more than healthy controls for new information if it was related to old information. The relationship between confabulations and neurocognitive variables was inconsistent. Together, the results from this quantitative review has important implications for interviewing techniques in forensic psychiatric assessment. Additionally, identifying predictors of confabulations in schizophrenia may allow for a greater understanding of what subgroup of patients is more likely to confabulate. This quantitative review will aim to synthesize the findings on the frequency of confabulations in these studies, as well as the neurocognitive predictors of confabulation. To the best of our knowledge, no quantitative review employing meta-analytic methods has been undertaken on the neurocognitive predictors of confabulations in this patient population to date.

Confabulations

Variations of the Deese–Roediger–McDermott paradigm [8,9] tend to be used most frequently to measure confabulations. Participants are shown a list of words. They are then shown these words again, along with new words that either are or are not semantically related to the first set of words. Patients are asked to identify whether they have seen the word before, as well as how confident they are in their answers. An intrusion, whereby participants state a new word was previously presented, is considered to be evidence of confabulation. Research suggests that patients tend to confabulate for new, semantically related words more than do controls [10]. In contrast, several studies have found that both healthy controls and patients made comparable levels of intrusions when the new words were semantically related to the old words. Further, as the semantic relationship decreased, the number of intrusions decreased across both groups [5,11-13]. This suggests that anyone is susceptible to making confabulations when the new and old words are related semantically.

Introduction

Confabulations, or false memories, are observed in various disorders, including schizophrenia. In forensic psychiatric assessment, this is problematic, particularly when garnering a clinical history and detailed account of the index offense(s) from the individual being charged. Numerous studies have identified that patients with schizophrenia confabulate more frequently than do healthy controls [1-4], though other studies have found no differences between these groups [5-7]. Confabulations in schizophrenia have important implications in forensic assessments with respect to the use of interviewing techniques. Additionally, identifying predictors of confabulations in schizophrenia may allow for a greater understanding of what subgroup of patients is more likely to confabulate. This quantitative review will aim to synthesize the findings on the frequency of confabulations in these studies, as well as the neurocognitive predictors of confabulation. To the best of our knowledge, no quantitative review employing meta-analytic methods has been undertaken on the neurocognitive predictors of confabulations in this patient population to date.

Key words

Schizophrenia, confabulation, false memories, intrusions, neurocognition, source monitoring

A common variation to this paradigm involves both the participant and experimenter generating words. Participants are later presented with the generated words and new words and are asked to identify whether the word is old or new. If they identify it as old, they are asked whether it was said by the experimenter or themselves. Patients were more likely than were controls to attribute new words to both the experimenter and the self [10,14] but tended to display a bias in labelling words as being said by the experimenter [14]. Thus, this may simply be a source monitoring deficit, rather than evidence of confabulations. Indeed, other research suggests that patients were more likely to misattribute self-presented words as being said by the experimenter and vice versa [13].

If patients with schizophrenia confabulate more than healthy individuals, this effect should be observed using other stimuli as well. Mammarella et al. [15] asked participants to either imagine an action or perform an action. Twenty-four hours later, they were presented with several actions and asked if they imagined it, performed it, or neither (i.e. a new action). Patients incorrectly attributed new actions as previously performed actions more than did controls, suggesting that patients did in fact confabulate that they had previously performed an action.

Other studies have used pictures and videos, which may tap into the visual aspect of confabulations (i.e. being able to visually represent a memory in one’s mind). Several studies employing the DRM paradigm using pictures found that like when using words, both patients and controls confabulated at a similar frequency when new images were highly related to previously presented images. In contrast, however, patients confabulated more frequently than did controls on new images that were moderately related to previously presented images [16,17]. This suggests that while both patients and controls have a tendency to misattribute new stimuli as previously presented when they are related, patients with the illness have a greater tendency than do healthy controls to do this when the stimuli are not related.

Peters, Hauschildt, Moritz, and Jelinek [18] employed a similar methodology using videos but varied their valence: positive, negative, neutral, and delusional. Patients with schizophrenia confabulated more frequently than healthy controls for positive videos only. That is, for negative, neutral, and delusional videos, no differences in confabulation were found between patients with schizophrenia and healthy controls. This suggests that emotionality may play a role in confabulation, which makes sense, given that patients’ real-world confabulations tend to have an emotional valence. This further speaks to the multitude of factors that likely play a role in predicting the likelihood of confabulations.

**Neurocognition**

Given that confabulations are related to one’s ability to form memories, it is necessary to examine the neurocognitive predictors associated with this phenomenon. For instance, Nienow and Docherty [20] found that patients with schizophrenia were more likely than controls to confabulate, but this effect disappeared when intellectual ability and verbal working memory was taken into account. Because confabulations are associated with other disorders as well, it is possible that it is linked to specific neurocognitive deficits, rather than being associated with schizophrenia more broadly.

Various studies have identified that both semantic and working memory differentiated confabulators from non-confabulators but not episodic memory [5,6,21,22]. While Nienow & Docherty [20] found that verbal working memory was associated with confabulations, thought disorder accounted for variance above and beyond verbal working memory. Indeed, several studies have found that thought disorder is uniquely associated with the tendency to confabulate [12-14,23,24]. Thus, it appears that thought disorder plays a specific role in confabulations but that semantic and working memory may be uniquely associated with this phenomenon above and beyond symptomatology.

In general, previous findings suggest a relationship between deficits in executive functioning and confabulations. These findings have been consistent for both
source monitoring paradigms [5,10,24] and story-recall [22]. On the other hand, other studies have yielded contradictory findings [14,21]. Thus, these differences need to be further understood.

Other neurocognitive domains have received less attention to date than have memory and executive functioning. Attentional abilities do not appear to play a role in confabulations [25], but this has only been examined in a limited number of studies. Moreover, Brebion et al. [25] examined processing speed and found this to be unrelated to intrusions. It is possible that while processing speed did not play a role in the source discrimination task overall, it may still have played a role in the production of confabulations as suggested by the finding that patients were slower to reject new words than were controls [26]. Further, even though verbal fluency is suggested to be a possible neurocognitive endophenotype in schizophrenia [27,28], it was examined in only one study in its relation to confabulation. Deficits in category fluency were related to a tendency to confabulate new, semantically unrelated words [6].

**Purpose**

The purpose of this study was to undertake a quantitative review of the research literature accumulated to date on the neurocognitive predictors of confabulations in schizophrenia. It was hypothesized that (1) patients would confabulate more than would healthy controls and (2) semantic memory, working memory, and executive functioning would be associated with confabulations.

**Methods**

**Literature Search**

A computerized search was performed on Pubmed, PsycInfo, and Scopus to locate potential primary studies to include our quantitative review. The search terms “schizophrenia” or “psychosis” in combination with “false memory,” “false memories,” and “confabulation” were used. Of the identified primary studies that met inclusion criteria, references were examined for additional studies to include in the quantitative review. No remote date limit in searching the literature was set. Hence, the research literature was canvased up to 2016. Studies included ranged in publication date from 1995 to 2007.

**Inclusion Criteria**

The search yielded 250 candidate research papers. After duplicates were removed, five primary studies met inclusion criteria. The following inclusion criteria was utilized: (1) Participant samples that included patients with schizophrenia and healthy controls; (2) commercially available neuropsychological test measures were employed (i.e., no experimental paradigms were considered); (3) quantitative data (i.e., means and standard deviations) were available so that an effect size could be computed; (4) published findings in peer-reviewed academic journals and written in English. Hence, no dissertations were included, nor studies published in non-English, academic journals.

**Exclusion Criteria**

Primary studies that combined healthy and psychiatric controls were excluded. Studies examining patients at clinically high risk or those with first-episode psychosis were excluded from our quantitative synthesis because previous work has demonstrated that the neurocognitive profile of these populations is different from that of patients with a diagnosis of schizophrenia [29,30]. Studies that looked at only the moderating effect of IQ on confabulation were not included. Due to the problematic nature of IQ being a composite score of multiple neurocognitive domains, it provides little information as to the cognitive processes responsible [31]. Studies using a source monitoring paradigm that did not include data for intrusions (that is, attributing a new word as a previously stated word) were not included, as this was the variable of interest. The derived effect size from anything but raw data is never exact, but rather an estimate of effect size. Hence, to be precise in our overall estimate of effects, studies that only provided test statistics (e.g., \( F, t, p \)-values) but not means and standard deviations were not included. The primary reasons for exclusion was that the study did not examine confabulations, neuropsychological test
measures were not employed, or means and standard deviations were not available to compute an effect size.

**Moderating Variables**

Recorded demographic variables included age, gender, education, and IQ. Clinical variables examined included duration of illness and symptomatology (i.e., positive symptoms, negative symptoms, and thought disorder). The demographic and study characteristics for the studies that met inclusion criteria are outlined in Table 1. Due to the various measures of symptomatology used across studies, this data was not recorded in the tables.

**Results**

Of the 250 results, five primary studies met inclusion criteria, resulting in a total sample size of 292 (144 healthy controls, 148 patients with schizophrenia).

Due to the limited number of studies and the wide range of methodology and neuropsychological test measures employed, an effect size analysis, rather than a meta-analysis, was deemed most appropriate and hence undertaken.

**Statistical Analyses**

For each of the studies, the mean and standard deviation (SD) for both patients and controls were extracted for the assessment of confabulations. In addition, the sample size for both groups were extracted. This information was used to calculate Cohen’s d [32] for confabulations for each study. Cohen’s d was chosen because it accounts for the differing variance in control and patient samples [33]. When effect sizes were reported for the correlation between neurocognitive functioning and confabulation, this data was extracted as well. All effect sizes were converted to Cohen’s d. This was done because Pearson’s r is influenced by sample size and the purpose of using effect sizes is to provide meaningful information about an effect, above and beyond what can be provided by significance testing, which, incidentally, is also influenced by sample size [33]. Lastly, the magnitude of effect was not interpreted in keeping with Cohen’s [32] heuristic framework but rather that of its clinical meaningfulness in the context of forensic psychiatric assessment [33].

**Confabulations**

The effect sizes for confabulations for each study can be found in Table 2. Overall, the first hypothesis appears to be supported: in six of the eleven computations, patients had a greater tendency to confabulate than did controls. Here, patients were more likely to confabulate for new words that were semantically related to previously stated words. It appears that patients were more likely than controls to attribute new, related words to both the experimenter and themselves. That being said, one study [13] used an index of bias, rather than the number of confabulations, and found that controls actually demonstrated a greater bias toward attributing new words as old words. This was found for both related and unrelated words. It should be noted that there was significant heterogeneity amongst studies, as demonstrated by the wide confidence intervals for each effect (Figure 1).

**Neurocognitive Variables**

Correlations between confabulation scores and neurocognitive scores can be found in Table 3. Overall, the findings were mixed for both executive functioning and working memory. No other neurocognitive variables were examined in the included studies. Further, very few studies reported usable quantitative data on these variables. Three studies demonstrated that executive functioning had a large association with participants’ abilities to discriminate between old and new words, but three studies reported no association.
### Table 1. Summary of Demographic and Clinical Variables

Note. All values rounded to one decimal place; Duration of illness = average number of years; data not available (-); a = assessed by WAIS-R; b = assessed by Shipley Institute of Living Scale; c = assessed by NART-R

<table>
<thead>
<tr>
<th>Study</th>
<th>Year</th>
<th>n</th>
<th>Age M(SD)</th>
<th>% male</th>
<th>Years of Education M(SD)</th>
<th>IQ M(SD)</th>
<th>n</th>
<th>Age M(SD)</th>
<th>% male</th>
<th>Years of Education M(SD)</th>
<th>Duration of Illness M(SD)</th>
<th>IQ M(SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Huron et al. [34]</td>
<td>1995</td>
<td>30</td>
<td>29.3(6.7)</td>
<td>66.6</td>
<td>11.5(3.5)</td>
<td>102.2(13.7)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>30</td>
<td>29.0(6.9)</td>
<td>66.6</td>
<td>10.7(2.6)</td>
<td>7.8(5.3)</td>
<td>85.1(13.9)&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Moritz et al. [14]</td>
<td>2003</td>
<td>21</td>
<td>27.0(10.7)</td>
<td>52.4</td>
<td>11.5(1.7)</td>
<td>-</td>
<td>30</td>
<td>31.1(8.3)</td>
<td>70.0</td>
<td>12.0(1.8)</td>
<td>4.5(6)</td>
<td>-</td>
</tr>
<tr>
<td>Neinow &amp; Docherty [19]</td>
<td>2004</td>
<td>52</td>
<td>37.5(7.2)</td>
<td>48.1</td>
<td>14.6(1.7)</td>
<td>105.6(8.4)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>39</td>
<td>35.9(8.9)</td>
<td>53.8</td>
<td>12.4(1.6)</td>
<td>-</td>
<td>88.3(12.6)&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Peters et al. [13]</td>
<td>2007</td>
<td>20</td>
<td>35.2(9.7)</td>
<td>90.0</td>
<td>-</td>
<td>110.8(10.3)&lt;sup&gt;c&lt;/sup&gt;</td>
<td>23</td>
<td>36.3(13.1)</td>
<td>78.3</td>
<td>-</td>
<td>7.0(7.4)</td>
<td>104.5(13.8)&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Vinogradov et al. [10]</td>
<td>1997</td>
<td>21</td>
<td>38.5(7.6)</td>
<td>42.8</td>
<td>14.9(1.4)</td>
<td>111.1(5.9)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>26</td>
<td>40.2(9.6)</td>
<td>53.8</td>
<td>13.9(1.7)</td>
<td>-</td>
<td>98.9(12.8)&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

### Table 2. Means, Standard Deviations, and Effect Sizes for Frequency of Confabulations

Note. All means are the mean number of intrusions made for that outcome variable unless otherwise noted; a = attribution bias index; d = Cohen's d

<table>
<thead>
<tr>
<th>Study</th>
<th>Outcome Variable</th>
<th>Controls</th>
<th>n</th>
<th>M(SD)</th>
<th>d</th>
<th>Cases</th>
<th>n</th>
<th>M(SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Huron et al. [34]</td>
<td>Intrusions</td>
<td>30</td>
<td>1.90(1.90)</td>
<td></td>
<td>0.04</td>
<td>30</td>
<td>2.00(2.70)</td>
<td></td>
</tr>
<tr>
<td>Moritz et al. [14]</td>
<td>Unrelated word, attributed to experimenter</td>
<td>21</td>
<td>0.05(0.20)</td>
<td>0.03</td>
<td></td>
<td>30</td>
<td>0.03(0.20)</td>
<td>0.03</td>
</tr>
<tr>
<td></td>
<td>Unrelated word, attributed to self</td>
<td>21</td>
<td>0.10(0.30)</td>
<td>-0.28</td>
<td></td>
<td>30</td>
<td>0.03(0.20)</td>
<td>-0.28</td>
</tr>
<tr>
<td></td>
<td>Related word, attributed to experimenter</td>
<td>21</td>
<td>0.24(0.40)</td>
<td>0.83</td>
<td></td>
<td>30</td>
<td>0.97(1.10)</td>
<td>0.83</td>
</tr>
<tr>
<td></td>
<td>Related word, attributed to self</td>
<td>21</td>
<td>0.14(0.40)</td>
<td>-0.03</td>
<td></td>
<td>30</td>
<td>0.13(0.40)</td>
<td>-0.03</td>
</tr>
<tr>
<td>Neinow &amp; Docherty [19]</td>
<td>New word reported as thought&lt;sup&gt;a&lt;/sup&gt;</td>
<td>52</td>
<td>0.31(0.19)</td>
<td>0.33</td>
<td></td>
<td>39</td>
<td>0.38(0.24)</td>
<td>0.33</td>
</tr>
<tr>
<td></td>
<td>New word reported as said&lt;sup&gt;a&lt;/sup&gt;</td>
<td>52</td>
<td>0.19(0.13)</td>
<td>-0.15</td>
<td></td>
<td>39</td>
<td>0.17(0.13)</td>
<td>-0.15</td>
</tr>
<tr>
<td>Peters et al. [13]</td>
<td>hits vs false alarm critical lures&lt;sup&gt;a&lt;/sup&gt;</td>
<td>20</td>
<td>0.86(0.12)</td>
<td>-1.17</td>
<td></td>
<td>23</td>
<td>0.72(0.12)</td>
<td>-1.17</td>
</tr>
<tr>
<td></td>
<td>hits vs false alarm new&lt;sup&gt;a&lt;/sup&gt;</td>
<td>20</td>
<td>0.35(0.20)</td>
<td>-0.4</td>
<td></td>
<td>23</td>
<td>0.27(0.20)</td>
<td>-0.4</td>
</tr>
<tr>
<td>Vinogradov et al. [10]</td>
<td>Related word, attributed to experimenter</td>
<td>21</td>
<td>2.10(1.70)</td>
<td>0.38</td>
<td></td>
<td>26</td>
<td>3.30(4.00)</td>
<td>0.38</td>
</tr>
<tr>
<td></td>
<td>Related word, attributed to self</td>
<td>21</td>
<td>0.9(1.1)</td>
<td>0.54</td>
<td></td>
<td>26</td>
<td>2.6(4.1)</td>
<td>0.54</td>
</tr>
</tbody>
</table>
Table 3. Correlations between Confabulations and Neurocognitive Variables Reported in Studies

<table>
<thead>
<tr>
<th>Study</th>
<th>Outcome Variable</th>
<th>Neurocognitive Variable</th>
<th>Cognitive Domain</th>
<th>d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Huron et al. [34]</td>
<td>Intrusions</td>
<td>Wechsler Memory Test&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Memory</td>
<td>-</td>
</tr>
<tr>
<td>Moritz et al. [14]</td>
<td>Recognition deficits</td>
<td>RAVLT long-term recall</td>
<td>Memory</td>
<td>1.07</td>
</tr>
<tr>
<td></td>
<td>Source monitoring</td>
<td>WCST&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Executive functions</td>
<td>-</td>
</tr>
<tr>
<td>Nienow &amp; Docherty [19]</td>
<td>-</td>
<td>Digit Span Backwards&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Working Memory</td>
<td>-</td>
</tr>
<tr>
<td>Peters et al. [19]</td>
<td>Attribution bias</td>
<td>WCST&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Executive functions</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>Discrimination index</td>
<td>BADS&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Executive functions</td>
<td>1.81</td>
</tr>
<tr>
<td>Vinogradov et al. [10]</td>
<td>Source discrimination</td>
<td>WCST and NSI&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Response disinhibition</td>
<td>0.98</td>
</tr>
<tr>
<td></td>
<td>Source discrimination</td>
<td>WCST and NSI&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Executive dysfunction</td>
<td>0.90</td>
</tr>
</tbody>
</table>

Note. d = Cohen’s d; RAVLT = Rey Auditory Verbal Learning Test; WCST = Wisconsin Card Sorting Test; BADS = Behavioural Assessment of Dysexecutive Syndrome; NSI = Neurological Signs Inventory; a = no relationship was found but statistic not reported; b = statistic not reported or commented on in the results; c = factor score.

Frequency of Confabulations in Patients vs. Controls

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Figure 1. Forest plot demonstrating effect size (Cohen’s d) and 95% confidence interval
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With respect to memory, long-term memory demonstrated a large association with confabulation, while a composite measure of memory did not. This is not surprising given that a composite score provides little information as to the specific aspects of memory involved. Given the conflicting findings and relatively few reported statistics, the second hypothesis could not be answered.

Discussion

The purpose of this study was to systematically and quantitatively assess the frequency of confabulations in patients with schizophrenia compared to healthy controls, as well as the neurocognitive variables associated with confabulations. It appears that in general patients with schizophrenia are more likely to confabulate for new, related words than are healthy controls, which is congruent with our first hypothesis. It should be noted, however, that not all studies supported this conclusion. The findings regarding new, unrelated words were mixed and typically produced less meaningful effects sizes.

It appears that patients with schizophrenia may confabulate more than healthy individuals when new information is related to previous memories. Thus, when an old memory is triggered, patients may be more likely to integrate new information into that memory that did not actually happen.

In contrast, if the new information is not related to an old memory, patients do not seem to differ from healthy individuals in their likelihood to confabulate. This finding has important implications for interviewing techniques in forensic psychiatric assessment. Specifically, leading questions may be particularly problematic when questioning the index offence, as it may introduce new, but obviously related information that was not part of the original memory. Additionally, care should be taken not to introduce unverified information from the file into the interview, as this may result in a confabulation rather than a more accurate account of the event.

It was further hypothesized that executive functioning, semantic memory, and working memory would predict confabulations. Due to the limited number of cognitive domains measured and quantitative data reported, this hypothesis was not testable. Qualitatively, however, long-term memory appeared to have a large association with confabulation. Individuals who have difficulty remembering information over a long delay, may also have difficulty remembering events that occurred in the distance past. Thus, patients with schizophrenia who have deficits in long-term memory may be more prone to confabulate about previous events, particularly when presented with information that is somewhat related to their memories. Findings pertaining to executive functions were mixed and as such, it is unclear what role this may play in confabulations.

Given the inconsistent findings pertaining to neurocognition, it is possible that symptomatology is more predictive of confabulations than are deficits in neurocognition. While findings related to positive and negative symptoms are inconsistent, thought disorder appears to play a prominent role in confabulations [12,14,23,24]. Moritz et al. [23] hypothesized that this may be related to greater semantic activation in patients with thought disorder, resulting in additional, less related associations, compared to those without thought disorder. This leads patients to believe that new, semantically-related words have been shown before because those schemas were previously activated in memory. This theory is consistent with findings from several other studies [11,34,35]. Accordingly, it is possible that there are neurocognitive correlates underlying the relationship between thought disorder and confabulation specifically, but additional research is required.

There are several limitations that we are mindful of as it pertains to our findings. It should be highlighted that only studies examining both confabulation and neurocognition were included. Due to the limited number of studies that have explicitly examined the relationship between confabulation and neurocognition, any conclusions that are drawn from this study should be interpreted with caution. Yet, our quantitative synthesis of this literature
is inherently more robust than a single primary study. In light of our collective findings, this review should serve as a starting point for further research in this area. Secondly, it should be noted that the DRM paradigm often asks patients to discriminate the source of the information (i.e., the experiment vs the self; thought about an action vs. performing an action). This means that an inherent limitation to this methodology is that instead of assessing confabulations, these studies may in fact be tapping into source monitoring deficits. Nevertheless, patients were more likely than controls to attribute new, related words to both the experimenter and themselves, which suggests that these differences may not simply be a source monitoring deficit. Greater research is needed to improve the methodology used to measure confabulations, however.

Conclusion

In conclusion, this study suggests that patients with schizophrenia may be more likely to confabulate than are healthy individuals when new information is presented that is related to an old memory. These findings are particularly important for interview techniques in forensic psychiatric assessment. The assessor should take great care not to ask leading questions or introduce unverified, contextual information into the interview, as it may increase the likelihood of confabulation. With respect to specific predictors, deficits in long-term memory appear to be related with an increased likelihood of confabulating, though thought disorder may be a more reliable predictor. Future research should examine the neurocognitive correlates that underlie this relationship.

Conflict of Interest: none

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