

Network Model of Decreased Context Utilization in Autism Spectrum Disorder

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Abstract Individuals with autism spectrum disorders (ASD) demonstrate impaired utilization of context, which allows for superior performance on the “false memory” task. We report the application of a simplified parallel distributed processing model of context utilization to the false memory task. For individuals without ASD, experiments support a model wherein presentation of one word, e.g., “apple,” strongly activates the neighboring nodes of closely related words such as “fruit,” “tree,” whereas in ASD these neighboring nodes are relatively less activated. We demonstrate this model to be consistent with the superior performance on recognition testing on the false memory test, but not on free recall. This may have an anatomic basis in diminished hippocampal neuronal arborization and the abnormal minicolumnar pathology in ASD.

Keywords Autism · Neural network · False memory · Semantic · Memory · Hippocampus

Introduction

Autism is a developmental condition leading to atypical social and language development as well as resulting in restricted interests and repetitive behaviors. Many theories have been postulated to explain the pattern of cognitive strengths and weaknesses among those with an autism spectrum disorder (ASD). Frith and Happé (1994) proposed a cognitive theory attempting to explain why those with autism have difficulty in utilizing context when processing information. They suggested that individuals with autism have a cognitive style that reflects weak central coherence. Individuals with autism have difficulties integrating information from the environment in order to form a coherent “whole” picture and instead engage in a more piecemeal processing style. Perhaps some “savant” skills commonly associated with autism could be explained in this way. Supporting evidence shows those with autism perform well in tasks where attention to local information is important, such as the Block Design task (Shah & Frith, 1993), the embedded figures test (Shah & Frith, 1983), and resisting visual illusions (Happé, 1996), but poorly at those requiring global processing such as reading homographs in text: “she has a tear in a dress” versus “she has a tear in her eye” (Happé, 1997). A local bias in the auditory domain has also been demonstrated in music (Mottron, Peretz, & Ménard, 2001) as well as in other language tasks (Goldstein, Minshew, & Siegel, 1994). This is also substantiated by studies showing that autistic children have more difficulty when asked to recall short meaningful sentences as compared to children without neurodevelopmental diagnoses despite similar performance on random words (O’Connor & Hermelin, 1967).

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An impaired ability to utilize context could be related to restricted semantic and associative networks. Beversdorf et al. (2000) found support for this proposal using the “false memory” paradigm first demonstrated by Roediger and McDermott (1995). High-functioning participants with ASD (normal performance on intelligence testing) were presented with a list of semantically and associatively related words (e.g., “sharp,” “pin,” “thread,” “sewing,” “eye,” “point,” “prick,” “thimble,” “haystack,” “thorn,” “hurt,” “injection,” “syringe,” “cloth,” and “knitting”) and when tested for recognition, were presented with an index item, or critical lure, which is closely related to the words on the list but was not presented (e.g., “needle”). Beversdorf et al. (2000) found participants with ASD to be less susceptible than a matched comparison group in reporting they heard the critical lure. Therefore, they were less likely to experience a “false memory” effect. This indicates less susceptibility to the influence of associatively and semantically related words in inducing illusory recognition suggesting restricted semantic associative networks, at least among high functioning individuals with ASD. Our purpose is to utilize the findings of this recent study in developing the concept of a rudimentary neural network model of memory in ASD, which we propose may explain some of the difficulties experienced by those with ASD in the utilization of context.

Neural network models attempt to model neural and cognitive functioning, typically by running computer simulations of groups of neurons, synaptic connections and changes in synaptic strength due to learning (Cohen, 1994; Rumelhart & McClelland, 1986). Artificial groups of neurons (or nodes) are typically organized in interconnected layers, commonly three types (input layer, interconnecting layers, output layer), and each interconnection between layers has an associated “weight” which can be adjusted to produce the desired output. The network is “trained” through numerous presentations of the data with feedback resulting in adjustments to weights until it computes the desired output at its output layer. The weights in our simplified model however, are not learned (changing), but are static through the analysis, since they are explicitly derived from experimental data.

Cohen (1994) attempts to explain difficulties in generalization among those with autism by presenting a neural network model rooted in neuroanatomical findings (e.g., Bauman & Kemper, 1986; Kemper & Bauman, 1993). A back-propagation method was used to investigate the consequences of having an abnormal number of neurons or neuronal connections in proposed brain regions such as the cerebellum, amygdala

and hippocampus, which may cause the reduction in susceptibility to “false memory.” Cohen (1994) found that the number of nodes in the interconnecting layers is inversely proportional to the extent of abstraction and integration of the information represented by those nodes.

McClelland (2000) presents a neural network model to explain why individuals with autism tend to represent things in a highly specific way such that items within a particular category are not necessarily generalized. This hyperspecificity could explain certain patterns seen in the memories of people with autism such as superior skills in tasks requiring rote memory and their lack of utilization of meaning. For example, as described above, individuals without neurodevelopmental diagnoses show a strong advantage for words presented in a meaningful sentence as compared to random strings of words, whereas individuals with ASD show less of an advantage (Hermelin & O’Connor, 1967). McClelland (2000) proposes that due to a slight difference in their neural networks those with autism may be predisposed to detect less overlap between inputs and assign distinct representations to items, subsequently affecting the ability to generalize to other similar situations. McClelland further suggests that parts of the brain dealing with semantic and conceptual representations may be particularly affected.

We propose to design a model derived from the results of a contextual memory study (O’Connor & Hermelin, 1967) applied to “false memory” study (Beversdorf et al., 2000) utilizing a simplified version of the principles of McClelland (2000). The model is developed from the concept that individuals with ASD have restricted semantic associative networks, which impacts their ability to generalize experiences to other similar situations, and to utilize context when processing information. Our neural network model therefore, would be derived from the assumption that when a word is presented, e.g., APPLE, semantically and associatively related network nodes activated for APPLE would be much less for those with ASD compared to matched control groups as shown in Fig. 1.

Within the “false memory” task, semantic and associative relations can also be illustrated as is demonstrated for one of the word lists in Fig. 2. Our purpose is to generate a model based on these interactions between the 13 semantically and associatively related nodes, with weights for the two groups derived from previous research on ASD (O’Connor & Hermelin, 1967).

The input to this model would be in the form of the auditory presentation of each word to the subject and

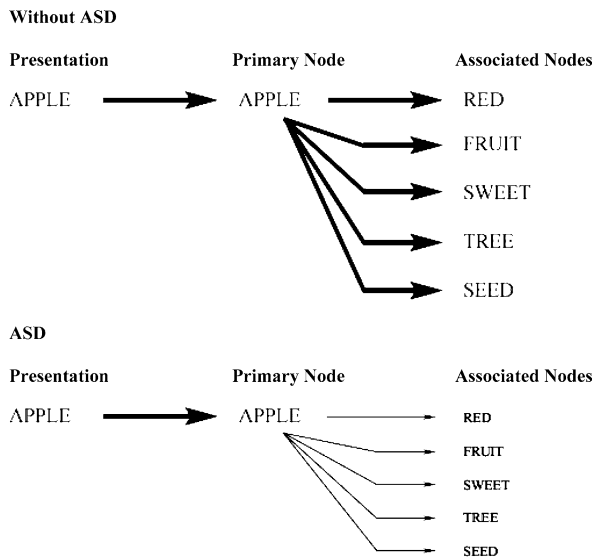
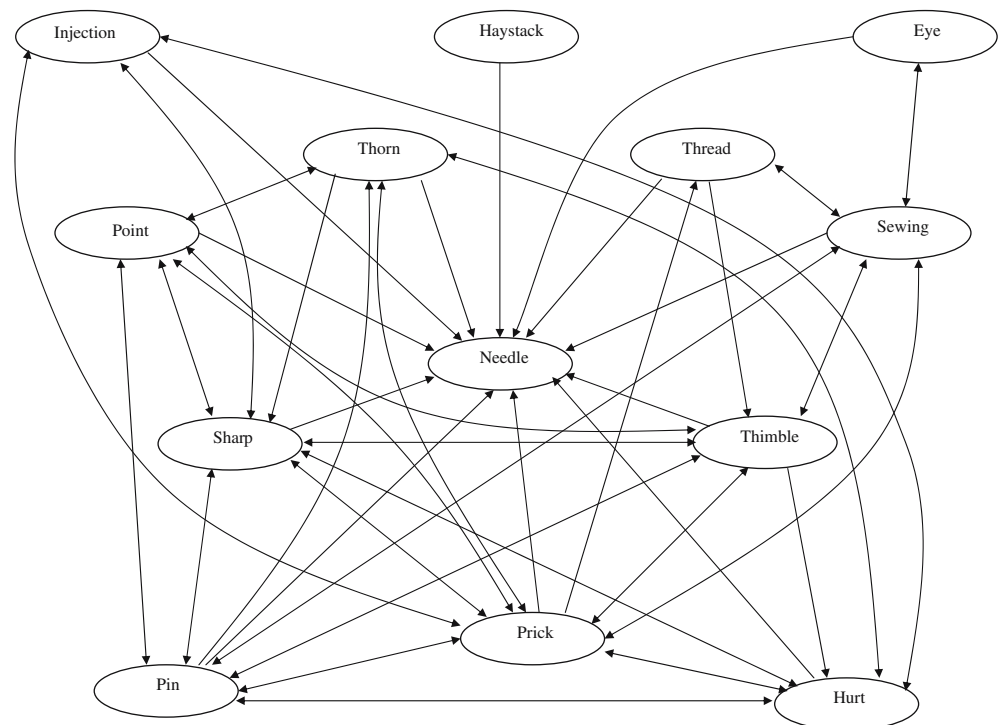


Fig. 1 Exemplar associative node activation in ASD and in individuals without ASD. In individuals without ASD, the nodes associated with “APPLE” are activated strongly (top). In individuals with ASD however, there is weaker activation of the nodes associated with “APPLE” due to restriction of semantic networks (bottom). The thickness of the lines represents the strength of activation

the output is the ability of the subject to discern which words were presented and which words were not. As we are deriving our weights explicitly from data in the literature, at this stage we will develop a simplified model in a controlled manner based upon these nodes directly without hidden nodes.

Fig. 2 The interconnected activation network for the “false memory” word list, as is applied to our model. “Needle” is partially activated by every word. However, since it is not presented, it does not activate any other words. For the sake of simplicity, the activation of each word by itself is not shown



The hippocampus may be essential in the development of semantic networks (Gabrieli, Cohen, & Corkin, 1988), and neuroanatomical research has revealed decreased dendritic arborization and increased neuronal cell-packing density in hippocampus CA1 and CA4 subfields among those with ASD (Bauman & Kemper, 1994). Associative memories are thought to be formed by *N*-methyl-D-aspartate-mediated associative long-term potentiation in the CA1 subfield from diverse cortical inputs. This may serve as a potential anatomical mechanism by which individuals with ASD have less utilization of context in memory due to less diverse cortical inputs. With this hippocampal finding as a guide, we aim to examine this restricted network theory by utilizing the recognition memory findings in false memory and considering whether we can develop a model which can predict other findings regarding memory in ASD, including testing memory tasks not utilized in the development of the model such as free recall. Due to the divergent results of the “false memory” task for the recognition and free recall (Beversdorf et al., 2000; Bowler, Gardiner, Grice, & Saavalainen, 2000), we will wish to examine both recognition and free recall with our model.

Method

An exemplar from the work of Roediger and McDermott (1995) utilized by Beversdorf et al. (2000) was

selected as input in order to construct the network model. In this study a series of 24 word lists each with 12 words were presented auditorily to participants at a rate of one per second. Subsequently participants were given a recognition test consisting of seven items including a critical lure, which was closely related to all of the words on the list but was not presented. In our model, different levels of semantically and associatively related node activation would occur between those with ASD and matched controls in response to the presentation of a word. Those with ASD will have more restricted semantic and associative activation.

The particular activation scores for normal subjects and ASD participants, as well as the scores for their corresponding primacy and recency effects, are derived from the work of O'Connor and Hermelin (1967), in a test of auditory and visual memory. Adjusted scores for individuals with ASD demonstrated that they recalled 16 words placed in context as compared to 24 for individuals without neurodevelopmental diagnoses, despite a similar performance on words not placed in context. This ratio was utilized to derive the differential degree of semantic and associative cross-activation for individuals with ASD (0.13) and individuals without ASD (0.2) in our model. Similarly, individuals without ASD showed an activation score of 17 for recency effect, whereas the corresponding score for ASD was shown to be 21, yielding recency cross-activation factors of 0.12 and 0.16 for individuals without ASD and individuals with ASD, respectively, for the final word in our model, with the next to last word at two-thirds of this factor, and the third from last word at a third of this factor. The data also showed that the primacy effect for individuals without ASD was nearly twice as strong as that for individuals with ASD (scores of 14 and 8, respectively) yielding a primacy cross-activation factors 0.12 and 0.07, respectively for the first word, and the 2nd and 3rd words were adjusted in a similar manner as with the recency effect.

For example, if participants were presented with the word “thread” the model would yield the pattern of node activation for individuals without ASD shown in Table 1. The network node for “thread” is fully activated and therefore given a score of 1. Nodes for semantically and associatively related words are partially activated according to the cross-activation factors described above.

The model is then presented with subsequent words in the Roediger and McDermott exemplar (e.g., “pin,” “eye,” “sewing” etc.), and summing the resulting nodal activation would yield the pattern of node activation in individuals without ASD shown in Table 2.

The word “needle” is not presented.

Table 1 Node activation following presentation of the word “thread” among those without ASD. The presented word fully activates the node for that word, while associated words receive a lesser degree of activation as derived from the data of O'Connor and Hermelin (1967)

	Thread
<i>Without ASD</i>	
Thread	1
Pin	0
Eye	0
Sewing	0.2
Sharp	0
Point	0
Prick	0
Thimble	0.2
Haystack	0
Thorn	0
Hurt	0
Injection	0
Needle	0.2

When asked to recognize words from the presented lists it would be expected that participants would show a primacy and recency effect. This is reflected in Table 3 by allocating increased node activation to words at the beginning and end of the list according to the primacy and recency factors described above.

Correspondingly, those with ASD would be expected to have lesser activation of the nodes for related words. Therefore, these words are assigned a cross-activation score less than the corresponding cross-activation score shown for non-ASD controls as described above and as shown in Table 4. In addition, it is also expected that the participants with ASD would show primacy and recency effects, though different from those shown by the participants without ASD, as is shown in Table 5.

Results

The rank order of node activation results for the two groups are shown in Table 6.

When asked whether a word was heard (recognition), participants establish a criterion of certainty that may be represented by the degree of node activation above which the word is reported as recognized. For individuals without ASD, total activation ranges from 1 to 2.8, and the activation for “needle” is 2.4 (Table 6). If selection of one criterion for recognition within this range is random, there is a 78% chance of recognizing the critical lure (e.g., “needle”) (probability of recognizing critical lure = $\left(\frac{CL-MIN}{MAX-MIN}\right) \times 100$) where CL = total activation for the critical lure, MIN = smallest value of the total activation for any

Table 2 Node activation following presentation of all items in the word list among those without ASD

	Thread	Pin	Eye	Sewing	Sharp	Point	Prick	Thimble	Haystack	Thorn	Hurt	Injection
<i>Without ASD</i>												
Thread	1	0	0	0.2	0	0	0	0.2	0	0	0	0
Pin	0	1	0	0.2	0.2	0.2	0.2	0.2	0	0.2	0.2	0
Eye	0	0	1	0.2	0	0	0	0	0	0	0	0
Sewing	0.2	0.2	0.2	1	0	0	0.2	0.2	0	0	0	0
Sharp	0	0.2	0	0	1	0.2	0.2	0.2	0	0.2	0.2	0.2
Point	0	0.2	0	0	0.2	1	0.2	0.2	0	0.2	0	0
Prick	0.2	0.2	0	0.2	0.2	0.2	1	0.2	0	0.2	0.2	0.2
Thimble	0	0.2	0	0.2	0.2	0.2	0.2	1	0	0	0.2	0
Haystack	0	0	0	0	0	0	0	0	1	0	0	0
Thorn	0	0	0	0	0.2	0.2	0.2	0	0	1	0.2	0
Hurt	0	0.2	0	0	0.2	0	0.2	0	0	0.2	1	0.2
Injection	0	0	0	0	0.2	0	0.2	0	0	0	0.2	1
Needle	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2

node and MAX = largest value of the total activation for any node). For individuals with ASD, activation ranges from 1 to 2.04, and the activation for “needle” is 1.56 (Table 6). If selection of one criterion for recognition within this range is random, there is a 54% chance of recognizing the critical lure (e.g., “needle”). This result is expected since the model is generated from the assumption of restricted semantic associative networks in ASD.

However, in free recall, access to the network is dependent on the search strategy, which in turn is dependent on executive function, known to be impaired in ASD (Russell, 1997). Therefore, in an effort to generate 10 of the words in free recall, individuals without ASD would be expected to have a higher hit rate in the search through activated nodes than

individuals with ASD due to a more efficient search strategy. For example, if individuals without ASD can yield an efficiency rate of four out of five activated nodes through their search strategy, and individuals with ASD can yield one out of two activated nodes, the search strategy may be represented as shown in Table 7.

As can be observed, if these efficiency ratings are assumed, then individuals without ASD have an 80% chance of generating the critical lure (“needle”), whereas individuals with ASD have a 50% chance. It can also be observed that individuals with ASD would not generate as many words from the lists with free recall as compared to individuals without ASD, and they also would recall more words that were not on the list as they attempt to retrieve the expected number of words.

Table 3 Overall pattern of node activation among those without ASD. The “Sum activation” column represents the sum of the corresponding rows in Table 2. The activation caused by the primacy and recency effects was also determined from the work of O’Connor and Hermelin (1967). “Total activation” represents the extent of activation including the primacy and recency effects for test subjects without ASD

	Sum activation	Primacy	Recency	Total activation
<i>Without ASD</i>				
Thread	1.4	0.12	0	1.52
Pin	2.4	0.08	0	2.48
Eye	1.2	0.04	0	1.24
Sewing	2	0	0	2
Sharp	2.4	0	0	2.4
Point	2	0	0	2
Prick	2.8	0	0	2.8
Thimble	2.2	0	0	2.2
Haystack	1	0	0	1
Thorn	1.8	0	0.04	1.84
Hurt	2	0	0.08	2.08
Injection	1.6	0	0.12	1.72
Needle	2.4	0	0	2.4

Discussion

The findings from this model of false memory recognition in ASD, based on cognitive theories of ASD and derived from experimental data (O’Connor & Hermelin, 1967), reveals the same expected pattern of performance as the research of Beversdorf et al. (2000) on the false memory paradigm among high-functioning individuals with ASD for recognition testing. Those with ASD are less susceptible to believing the critical lure had been presented due to less widespread node activation than those without ASD. Restricted semantic networks is in accord with explanations for a number of common findings among those with ASD such as a local bias in processing information, and difficulties in both generalization and utilizing meaning in context. Further support has also been demonstrated by the recent finding of decreased functional connectivity during

Table 4 Node activation following presentation of all items among those with ASD. The presented word fully activates the node for that word, while associated words receive a lesser degree of activation as derived from the data of O'Connor and Hermelin (1967)

	Thread	Pin	Eye	Sewing	Sharp	Point	Prick	Thimble	Haystack	Thorn	Hurt	Injection
<i>ASD</i>												
Thread	1	0	0	0.13	0	0	0	0.13	0	0	0	0
Pin	0	1	0	0.13	0.13	0.13	0.13	0.13	0	0.13	0.13	0
Eye	0	0	1	0.13	0	0	0	0	0	0	0	0
Sewing	0.13	0.13	0.13	1	0	0	0.13	0.13	0	0	0	0
Sharp	0	0.13	0	0	1	0.13	0.13	0.13	0	0.13	0.13	0.13
Point	0	0.13	0	0	0.13	1	0.13	0.13	0	0.13	0	0
Prick	0	0.13	0	0.13	0.13	0.13	1	0.13	0	0.13	0.13	0.13
Thimble	0.13	0.13	0	0.13	0.13	0.13	0.13	1	0	0	0.13	0
Haystack	0	0	0	0	0	0	0	0	1	0	0	0
Thorn	0	0	0	0	0.13	0.13	0.13	0	0	1	0.13	0
Hurt	0	0.13	0	0	0.13	0	0.13	0	0	0.13	1	0.13
Injection	0	0	0	0	0.13	0	0.13	0	0	0	0.13	1
Needle	0.13	0.13	0.13	0.13	0.13	0.13	0.13	0.13	0.13	0.13	0.13	0.13

Table 5 Overall pattern of node activation among those with ASD. The “Sum activation” column represents the sum of the corresponding rows in Table 4. The activation caused by the primacy and recency effects was also determined from the work of O'Connor and Hermelin (1967). “Total activation” represents the extent of activation including the primacy and recency effects for test subjects with ASD

	Sum activation	Primacy	Recency	Total activation
<i>ASD</i>				
Thread	1.26	0.07	0	1.33
Pin	1.91	0.05	0	1.96
Eye	1.13	0.02	0	1.15
Sewing	1.65	0	0	1.65
Sharp	1.91	0	0	1.91
Point	1.65	0	0	1.65
Prick	2.04	0	0	2.04
Thimble	1.91	0	0	1.91
Haystack	1	0	0	1
Thorn	1.52	0	0.05	1.57
Hurt	1.65	0	0.1	1.75
Injection	1.39	0	0.16	1.55
Needle	1.56	0	0	1.56

Table 6 Rank order of node activation results for those without ASD and those with ASD

Without ASD		ASD	
1	Haystack	1	Haystack
1.24	Eye	1.15	Eye
1.52	Thread	1.33	Thread
1.72	Injection	1.55	Injection
1.84	Thorn	1.56	Needle
2	Sewing, point	1.57	Thorn
2.08	Hurt	1.65	Sewing, point
2.2	Thimble	1.75	Hurt
2.4	Sharp, needle	1.91	Sharp, thimble
2.48	Pin	1.96	Pin
2.8	Prick	2.04	Prick
Was there a _____?		Was there a _____?	
Criteria <1 to 2.8		Criteria <1 to 2.04	

sentence comprehension in ASD using functional MRI (Just, Cherkassky, Keller, & Minshew, 2004).

In individuals with ASD, the connection strength between the words is reduced in our simplified model, leading to a reduction in the activation of the critical lure. Figure 2 graphically describes the association of each word used in the “false memory” task with each other word and with the critical lure. This model would explain the higher discrimination of “false memory” in autistic individuals because the critical lure would more often not be sufficiently activated to account for a “false memory.”

Cohen (1994) proposed that in individuals with ASD, the number of hidden nodes might differ from the corresponding number for individuals without neurodevelopmental diagnoses. For example, individuals with ASD may not make the association between *pin* and *sewing*. This might either be due to the presence of additional hidden nodes that impair association, or due to the lack of sufficient number of hidden nodes that reduce the ability of an individual with ASD

Table 7 Free recall data. Every fifth word is not activated (underlined) in normal individuals, as opposed to every other word not being activated for individuals with ASD

Without ASD		ASD	
2.8	Prick	2.04	Prick
2.48	Pin	1.96	<u>Pin</u>
2.4	Needle, sharp	1.91	<u>Sharp</u> , <u>thimble</u>
2.2	<u>Thimble</u>	1.75	Hurt
2.08	<u>Hurt</u>	1.65	<u>Sewing</u> , Point
2	Sewing, point	1.57	<u>Thorn</u>
1.84	Thorn	1.56	Needle
1.72	<u>Injection</u>	1.55	<u>Injection</u>
Pick 10		Pick 10	

to generalize. As the number of hidden nodes changes from normal in either direction, there is a reduction in the generalizing ability of the autistic individuals, causing the symptoms of hyperspecificity. This hyperspecificity in turn, leads to lesser activation of the critical lure causing a reduction in susceptibility to false memories observed in individuals with ASD. Both perturbations would thus lead to a decreased use of context, which would lead to decreased activation of the critical lure that causes the results observed in the false memory task.

Our findings are also in agreement with the hyperspecificity predicted by McClelland's (2000) model. However, as described above, our simplified model differs from these models by the lack of hidden nodes,

since we are deriving our weights explicitly from data in the literature. Therefore, we also do not incorporate into this model the hidden layers that could be designed to represent too many or too few neuronal connections in the manner discussed by Cohen (1994). For the same reason, the weights are static throughout, and so learning is not built into this model. However, even this simplified model can yield results consistent with the decreased utilization of context in ASD. Figure 3 describes the inputs and the outputs, and the pattern of activation of each node (word) in the output, for a putative model that does include hidden layers reflective of the complex mental representation of related words, which could also be examined with sufficient behavioral data.

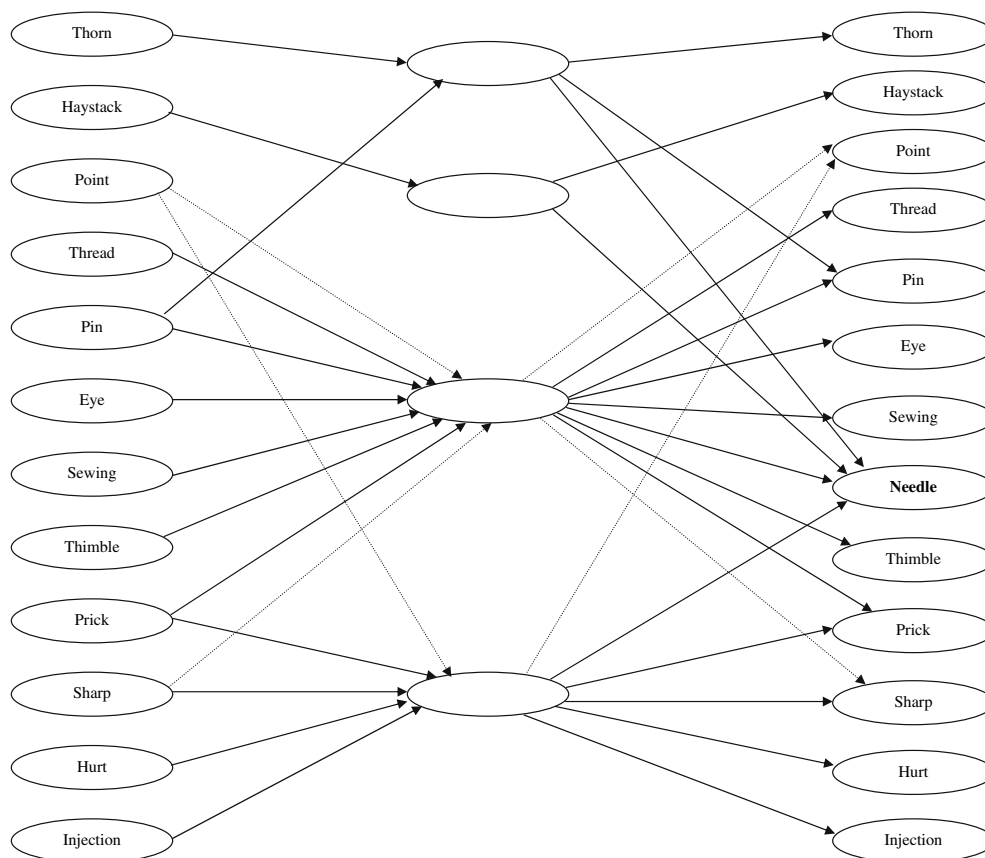


Fig. 3 Illustration of a putative model for the same “false memory” word list including hidden nodes. Here, we suggest that the activation received by each word occurs through our perception of different modalities. These would represent the hidden nodes for the model. For example, the word *haystack* may activate *needle* due to the phrase “needle in a haystack.” Similarly, the words *thread*, *pin*, *eye*, *sewing* and *thimble* may activate *needle* through the participants’ perception of what is involved in the act of sewing, and the words *sharp*, *hurt* and *injection* may activate *needle* through the participants’ perception of what is involved giving or receiving an injection. The word *prick* may activate *needle* through both the sewing and

injection representations. Words that may only activate the representational internodes indirectly through other nodes are denoted by dotted lines. For example, *sharp* would activate the representational internode for the sewing group only indirectly through the word *pin*, and would therefore induce a weaker excitation of that internode. Note that *needle* is the only word that receives activation through all the activating representations, resulting in its high activation level. Due to restriction of semantic networks in ASD, some representations may activate *needle* to a lesser extent. This could result in their better performance observed in “false memory” tests. This model could also be tested with sufficient behavioral data

The free recall findings also reveal the same results as the research of Bowler et al. (2000) on false memory in ASD. Executive function skills play a large role in the use of search strategies, and individuals with ASD have impairments in executive function (e.g., Russell, 1997). Bowler et al. (2000) found that participants with Asperger's syndrome recalled significantly fewer studied words than the matched comparison group, included significantly more words that had not been presented on the lists, and showed a trend towards lower levels of false recall of the critical lure, which matches our results derived from the model. Therefore, we propose that impaired search strategies due to executive dysfunction may relate to the discordant findings on recall and recognition on the "false memory" task, since application of decreased efficiency in search strategies is sufficient to result in decreased performance on free recall in the same network that yields superior recognition performance in ASD.

Overall results derived from the model presented are consistent with the findings from the false memory data and the theory that those with ASD have restricted semantic-associative networks revealed through decreased use of context. The neuroanatomical basis for this could be rooted in the abnormalities located in the hippocampus. Bailey et al. (1998) did not find abnormalities in a study examining other regions that also included an examination of the hippocampus. However, in a systematic examination of the hippocampus, Bauman and Kemper (1994) found decreased dendritic arborization and increased neuronal cell-packing density. Other abnormalities observed in the limbic system and cerebellum may also be responsible for the decreased use of context demonstrated. Interestingly, a recent post-mortem study by Casanova, Buxhoeveden, Switala, and Roy (2002) found abnormalities in the cortical minicolumns of individuals with autism. In area nine of the prefrontal cortex and areas 21 and 22 of the temporal lobe, minicolumns were smaller, less compact and more numerous as compared with nine control subjects. It is proposed that this leads to extra processing units, which are not assimilated into the connective patterns in the brain. This may also be related to the restricted semantic networks among those with ASD. The findings of Just et al. (2004) seem to support the hypothesis of a more global alteration in processing causing decreased context utilization. However, one limitation is that this model is derived from data on verbal patients with ASD, which may limit ability to generalize the model to lower functioning ASD. Further work will be needed to advance our anatomical understanding of decreased context

utilization as well as the presumably executive function-related impairments found in free recall.

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