Repetition improves memory by strengthening existing traces: Evidence from paired-associate learning under midazolam

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Abstract

Here, we examined how repetition under midazolam, a benzodiazepine that prevents the storage of novel associations, affects cued-recall performance of paired-associates. We contrasted word pairs that were initially studied and tested repeatedly without any successful recall prior to the midazolam injection, with other pairs that were studied for the first time after the injection of midazolam. According to our SAC (Source of Activation Confusion) memory model, repetition leads to strengthening existing memory traces rather than creating multiple traces for each repetition. As such, it predicts that repetition under midazolam should benefit only pairs that were originally studied prior to the midazolam injection. This prediction was confirmed. The results suggest that memory traces for pairs studied prior to the midazolam injection were strengthened under midazolam. However, word pairs that had not been studied prior to the injection were not bound in long-term memory because midazolam prevents the formation of new associations.

Keywords: memory strength; paired associate learning; episodic memory; practice; midazolam;

Repetition improves memory performance across the board. Beneficial effects of repetition have been found on most measures of explicit memory such as single-item and paired-associates recognition (e.g., Challis & Sidhu, 1993; Reder et al., 2000), free recall (e.g., Challis & Sidhu, 1993; Underwood, 1969), and cued recall tasks (Meltzer & Constable, 2005; Reder et al., 2007; Reder, Liu, Keinath, & Popov, 2015). However, despite more than a century of research on repetition effects, there is no consensus about the mechanism through which practice affects memory (Criss & Koop, 2015; Hintzman, 2010, 2011; Osth & Dennis, 2015; Pavlik & Anderson, 2005).

Two major types of theories have been proposed to explain repetition effects. *Cumulative-strength models (CSMs)* suggest that memory traces differ in strength or familiarity, and this strength increases with repetition and decays with time (e.g., Murdock, Smith, & Bai, 2001; Pavlik & Anderson, 2005; Reder et al., 2000; Wickelgren, 1972). In these models, recognition and recall are a function of strength and greater strength leads to better memory performance. In contrast, *multiple-trace models (MTMs)*, usually equated with *global matching models* (*GMMs*, Criss & Koop, 2015), state that each repetition of an item is encoded separately in memory (Bower, 1967; Brown, Neath, & Chater, 2007; Hintzman, 1984; Lansdale & Baguley, 2008; Osth & Dennis, 2015). This leads to redundant memory traces, each of which has some probability of being retrieved during test. Interestingly, while both CSMs and MTMs co-exist in the current literature, several proponents of each class believe that certain

empirical findings have conclusively ruled-out the alternative models (Criss & Koop, 2015; Hintzman, 2011).

When it comes to CSMs, some researchers have argued that they are incompatible with findings from judgments of frequency (JOF) and judgments of recency (JOR) tasks (Flexser & Bower, 1974; Hintzman, 2010, 2011 Hintzman & Block, 1971). Many CSMs in the past have assumed that the estimation of frequency, recency and duration of events is based on a single strength dimension (Hintzman, 2011). As a result, these models predict that, for example, if an event is repeated it should also appear to be more recent and to have lasted longer. That is not the case – studies have shown that participants can easily discriminate the frequency, recency and duration of repeatedly studied items (Flexser & Bower, 1974; Hintzman, 2010, 2010; Hintzman & Block, 1971).

This line of work seems to provide strong evidence against CSMs, and yet, they are still popular in modeling recognition and recall. Hintzman (2011) refers to this as "the fallacy of cumulative strength", and suggests that the CSMs are still popular because most theorists focus on recognition memory and recall, while ignoring tasks such as JOR and JOF. However, the same criticism can be directed at conclusions from JOR and JOF tasks – the fact that a single strength dimension cannot explain behavioral patterns in such tasks does not mean that repetition effects on recognition and recall memory are not due in part to cumulative strengthening of existing memory traces. It only indicates that memory representations also include rich contextual information, which is an assumption shared by most current dual-process CSMs.

Similarly, when it comes to MTMs and GMMs, other researchers maintain that they cannot account for the divergent patterns of the *list-length effect* (LLE) and the *list-strength effect* (LSE) on free recall and recognition (Criss & Koop, 2015; Shiffrin, Ratcliff, & Clark, 1990). The LLE shows that increasing the *number of different items* on a study list decreases free recall, cued recall and recognition performance. Similarly, the LSE shows that increasing the *number of repetitions* on some items leads to worse *free recall* for the non-repeated items. However, the LSE generally has no effects on overall performance in *recognition* tasks. Critics of GMMs have argued that they are fundamentally incompatible with this pattern of results (Criss & Koop, 2015). This is because GMMs assume that the same mechanism is involved when the number of different items increases and when some items on a list are repeated. Specifically, they both lead to the creation of additional memory traces and to increased global signal variance, which causes interference during retrieval. As such, GMMs supposedly predict that LSE and LLE should always occur together.

However, multiple proponents of GMMs have questioned both the reliability of the pattern of LSE and LLE effects on recognition (Dennis & Humphreys, 2001; Murdock & Kahana, 1993) and the inability of GMMs to account for it (Murdock & Kahana, 1993; Osth & Dennis, 2014, 2015). Furthermore, despite the fact that most MTMs are also GMMs, a multiple-trace model does not have to depend on global matching for memory decisions, which would make it easier to fit the pattern of LSE and LLE results. For example, even though our Source of Activation Confusion model (SAC; Reder et al., 2000) is a cumulative-strength model, the dual processes that allow it to account for the divergent pattern of LLE and LSE results would allow it to do the same even if each item repetition created a novel episodic trace (Cary & Reder, 2003; Diana & Reder, 2005).

In summary, the major problem with contrasting CSMs and MTMs has been that they make similar predictions when it comes to most memory tasks. One way to overcome this would be to attempt to disrupt the mechanism that is responsible for repetition effects in a specific model such as SAC, to make predictions how that will affect behavioral performance and to evaluate how well the model fits the data. To achieve that, we examined how repetition affects cued-recall under midazolam. Midazolam is a benzodiazepine that creates temporary anterograde amnesia by preventing the storage of new associations in LTM (Ghoneim, 2004; Reder et al., 2006), but it does not impair pre-existing memory traces (Ghoneim, 2004) or their strengthening, as evidenced by its limited effect on repetition priming (Hirshman, Passannante, & Arndt, 2001; Hirshman, Passannante, & Henzler, 1999).

We compared cued-recall performance for paired-associates that were studied for the first time (*control pairs)* under midazolam and a subset of the pairs that were studied both before the midazolam injection and re-studied after the injection *(practice pairs)*. The subset of interest were those pairs that had not been recalled on any of the tests that preceded the re-study session under midazolam. Given that midazolam prevents the storage of new associations in LTM, SAC predicts that the recall of control pairs should be at floor levels; performance on practice pairs that were never recalled correctly was an open question. SAC assumes that pairs that had never been correctly recalled might still have sub-threshold episodic traces in LTM. If repetition of the pair leads to the strengthening of this sub-threshold episodic trace, as SAC originally assumes, then we would expect greater recall of practice pairs compared to control pairs. If, however, repetition leads only to the creation of additional memory traces, then no advantage should be observed for practice compared to control pairs under midazolam, because midazolam will prevent the storage of the new traces in LTM.

Method

The data of interest involve a subset of conditions from a larger study previously reported in Reder et al. (2007) and Reder et al. (2006; study 2). For clarity, we will describe the full design.

Participants

Thirty-one healthy individuals from the Pittsburgh community participated in this experiment. Each participant was screened by a doctor and received $150 upon completion.

Procedure, materials and design

The study took place in two sessions on two separate days. We used a within-subject double-blind cross-over design where the drug condition (saline vs midazolam) was randomly assigned to one of the two days for each participant. Each session consisted of three separate study-test list cycles. The saline/midazolam injection was administered over a 2-min period between Lists 1 and 2. Participants began the study phase of List 2 immediately after the injection.

During each list, participants saw all of the 45 high-frequency word pairs in the following sequence: Study – Test1 and Restudy1 – Test2 and Restudy2. During the initial study phase, each word pair was presented for 3 seconds preceded by a fixation cross for 1 second. After all 45 pairs were studied, participants completed a self-paced cued-recall test for all 45 pairs in a different random presentation order. Test trials began with a 500 ms fixation cross, followed by the presentation of the first word in a pair and a question mark prompting participants to respond. Participants were asked to recall the correct word and type it on a laptop keyboard or press the return key to move to the next trial. Regardless of the accuracy of their response, participants saw the correct answer for 2.5 seconds after each response, which gave them an opportunity to restudy the pairs again. When all 45 pairs were tested and restudied the test-restudy phase was repeated one more time, which concluded the procedure for the first list. This study-testandrestudy1-testand-restudy2 procedure was repeated for two more lists, each of which took approximately 17 min. to complete. On each list, the 45 study pairs were split into 3 conditions with 15 pairs per condition – *control pairs*, which were unique for each list, *practice pairs,* which were the same 15 pairs on all 3 lists, and *interference pairs*, which had the same words on all 3 lists, but the cue words were assigned to different response words on each new list. The order of word pairs in each list, study and test sessions was randomly determined.

Data analysis and logic for the current study

Only a small subset of conditions was relevant for this study (see Figure 1). Specifically, we looked at cued recall performance on List 2 Test 2 for those control and practice pairs for which participants had failed to recall the response word on all previous tests. The control pairs were unique to each list, and as such the ones we selected were previously studied and tested only once at the beginning of List 2. The practice pairs were previously studied and tested twice on List 1 as well. We analyzed only those practice pairs which participants failed to recall on all three occasions (L1T1, L1T2 and L2T1).

We focused on the second test of List 2 (L2T2), rather than on the first test on List 2 for the following reason. Even though the injection was administered before the beginning of the second list, practice pairs were restudied immediately after their second test on List 1 (L1T2). Thus, if we observed improved recall for practice pairs on L2T1, it might have been due to the restudy
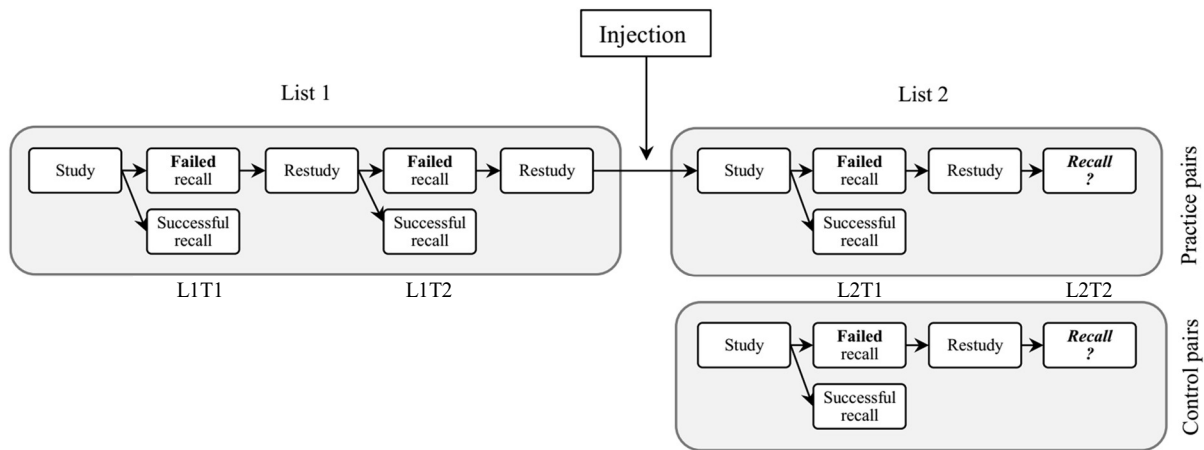
Figure 1. Visualization of the procedure for the subset of conditions relevant for this study. Small boxes represent the phases of each list (study, cued recall, or restudy), and whether the test was successful. Shown are the subsequent phases only for the relevant trials. The first row shows the procedure for practice pairs; the second shows the procedure for the control pairs. List 1&2 are shown in separate columns.

session that occurred prior to the injection rather than due to strengthening during the study session of List 2 under midazolam.

In summary, we selected control and practice pairs that showed no evidence of being learned up until L2T1 in either drug condition (midazolam or saline). These pairs were then restudied immediately following that test, and were then tested on L2T2. For control pairs, 75% qualified for analysis in the saline condition and 99% in the midazolam condition. For practice pairs, 32% qualified under saline and 41% under midaz. We analyzed accuracy on L2T2 as function of pair type (control vs practice) and drug condition (midazolam vs saline) using a logistic mixed effects regression with participants and items as random intercept effects. We compared alternative models with and without each of the main effects and interactions. A Bonferroni correction was applied to all post-hoc tests (n=4).

Results and discussion

The overall recall for each list collapsed over the two tests in a list are presented in Figure 2 and the results for the subset of trials of interest are presented in Figure 3. Control pairs were recalled less accurately than practice pairs, $\Delta AIC = -22$, $\chi^2(1) = 24.12$, $p < .001$. Word pairs were recalled less accurately under midazolam compared to saline, $\Delta AIC = -172$, $\chi^2(1) = 174.32$, $p < .001$. There was a significant interaction between drug condition and type of pair, $\Delta AIC = -17$, $\chi^2(1) = 19.27$, $p < .001$. Post-hoc comparisons revealed that practice pairs were recalled significantly more accurately than control pairs in the midazolam condition ($z = 6.72$, $p < .001$), but not in the saline condition ($z = 2.30$, $p = .09$). Finally, both practice and control pairs were recalled more accurately in the saline compared to the midazolam condition ($z = 9.97$, $p < .001$ and $z = 4.56$, $p < .001$, respectively for practice and control pairs). These results are consistent with the view that repetition can strengthen existing memory traces, because only pairs that were initially studied *prior* to a midazolam injection benefited from additional study under midazolam. These practice pairs were recalled more often than control pairs, which were studied for the first time after the midazolam injection, even though both showed no evidence of learning prior to the final test.

Despite the fact that practice pairs had not been recalled on any of the 3 previous tests, it is possible that an initial association for them was stored during List 1. It seems reasonable to conclude that even though these associations were inaccessible, they must have been registered in LTM since they were strengthened under midazolam while the control pairs were not. Midazolam is known to block the formation of new associations but there is no evidence that it inhibits strengthening of existing traces. On the contrary, implicit memory is spared under midazolam (Hirshman et al., 2001), and the model presented below already assumes that implicit memory is based on strengthening the same representations involved in familiarity-based recognition (Reder, Park & Kieffaber, 2009).

An alternative explanation of these data consistent with multiple-trace theories might be that even if no traces were strengthened under midazolam for practice pairs, the pre-existing sub-threshold traces might be spontaneously recovered in a probabilistic way (Brown, Neath & Chater, 2007). We believe this is unlikely, given that each practice pair analyzed here failed to be recalled on all 3 previous tests. Additionally, we can directly estimate what is the probability of recovery with a multinomial processing tree model (Erdfelder et al., 2009), where at each test there is one of the following possibilities: 1) successful recall of the target due to study/restudy, $r = P$(success on test n | fail on test n-1, or when n=1), 2) failing to recall a previously recalled target, $f = P$(failed recall on test n | success on test n-1), 3) spontaneous recall of a previously unrecalled target regardless of restudy benefit, $u = P'$(success on test n | fail on test n-1). We estimated these probabilities from performance on the control pairs on the two tests on List 1 (data not used in the previous analyses):

$$P(success\ T1\ \&\ success\ T2) = r * (1 - f) = 0.42$$
$$P(success\ T1\ \&\ fail\ T2) = r * f = 0.04$$
$$P(fail\ T1\ \&\ success\ T2) = (1 - r) * (r + u) = 0.29$$
$$P(fai\ T1\ \&\ fail\ T2) = (1 - r) * (1 - r - u) = 0.25$$

Here, successful recall following a failed recall is a combination of reencoding benefit r and a spontaneous recovery u. This analysis showed that the probability of successful recall due to (re)encoding was $r = 0.46$, the probability of forgetting a previous encoding was $f = 0.09$, and the probability of spontaneous

recovery of a forgotten encoding was $u = 0.07$ (similar values were obtained if we consider all sequential pairs tests on all three lists for practice pairs in the saline condition, $u = 0.089$). One-tailed t-tests showed that the benefit of restudy under midazolam (Fig 3), was significantly higher for practice pairs, but not for control pairs. Thus, spontaneous recovery of previously forgotten items cannot account for our results.
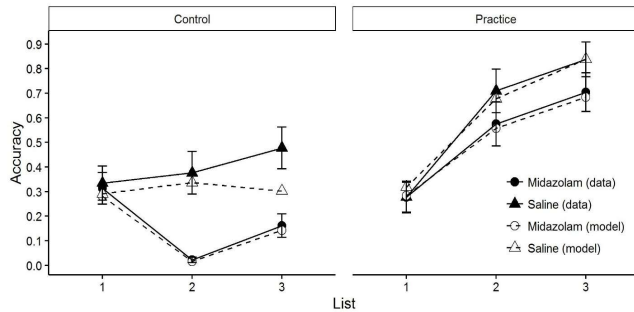

Figure 2. Cued-recall performance during acquisition, collapsed over the two tests (black points and solid lines) and model fits (white points and dashed lines) in all lists for control and practice pairs as a function of drug condition.
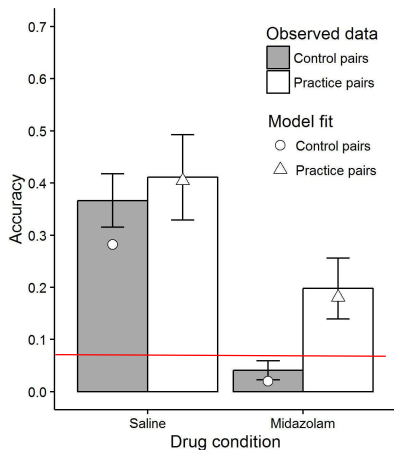

Figure 3. Accuracy on L2T2 for control and practice pairs that were not recalled correctly on any of the previous tests as a function of whether the L2 items were studied under saline or under midazolam. Horizontal red line shows the probability of spontaneous recovery of a forgotten item without strengthening.

To demonstrate that CSMs can fit the data not only verbally, but quantitatively as well, we fit a SAC model (Reder et al., 2000) on a trial-by-trial basis separately for each participant. In general, SAC posits that semantic, episodic and contextual information is represented as a network of interconnected concepts, event and context nodes varying in strength. Each node has an activation value that increases when a node is perceived or when it receives activation from other nodes. This activation decays with time according to a power law to a base-level resting activation that also is strengthened or decays with experience. When new information is studied, two processes occur. First, the current and the resting level activation values of the corresponding preexisting concept nodes are increased. Second, if this is the first occurrence of the

study episode, a new event node is created and it gets associated with the corresponding concept nodes, as well as with the general and specific context nodes. If, however, the study event has occurred previously, the existing event node and its links with the concept and context nodes are strengthened instead.

Retrieval in SAC is based on the activation of the event and concept nodes and the process differs slightly between free recall, cued recall and recognition. During free recall, the general context node and the list node are activated and they spread activation to all episode nodes connected to them. During cued-recall or recognition, the concept node(s) for the cue(s) is also activated and it spreads activation to all episode nodes connected to it. Spreading activation is multiplied by the strength of each association, and divided by the sum total strength of associative links emanating from the sending node. This represents competition for retrieval. Finally, if an episode node's activation passes the retrieval threshold, an item is recalled (free and cued recall) or recollected (recognition). For recognition, if no episode node passes the threshold, the strength of the cue concept node is evaluated. If it passes its retrieval threshold, a familiarity-based response is made.

The majority of parameters in the model were imported from previous studies. Consistent with the fact that midazolam prevents the storage of novel associations in LTM, in the current simulation we manipulated the probability of encoding an episode node. During the first presentation of each word pair there is a certain probability that participants will fail to encode the event node due to inattention, fatigue or insufficient working memory (see Reder et al., 2007). In models of other studies, this value has been constant, but in the current implementation, we allowed it to vary between the saline and the midazolam conditions. The optimal value for the saline condition was estimated from the data ($p = 0.35$), while the encoding probability for the midazolam condition changed with time elapsed since the injection (see Table 1 for parameter estimates and descriptions, and Table 2 for full model specification). Immediately after the midazolam injection the encoding probability was 0, reflecting the inability to store new associations at maximum potency, and it gradually increased to half of the encoding probability in the saline condition in 31 minutes (drug halflife for memorial effects, Albrecht et al., 1999).

The overall model fit for all conditions is presented in Figure 2, and the fit for the specific subset of interest is overlaid on Figure 3. Importantly, the model was fit by predicting a single value for each participant – their overall cued-recall performance and by minimizing the RMSE between the predicted and the observed value. Given that the model had no information about the performance in each condition, we obtained a surprisingly good fit for the split by conditions (16 summary data points per participant; RMSE = 0.139, $R^2 = 0.8$). The model demonstrates that the beneficial effect of repetition under midazolam can be explained entirely by the strengthening of preexisting memory traces that were previously below the retrieval threshold.

One could question why practice pairs were not recalled better than control pairs in the saline condition, given that they should benefit both from strengthening a pre-existing trace as well as from creating novel associations for pairs that were previously unlearned, while control pairs benefit only from forming new associations. Indeed, while the overall fit of the model was quite

good, the model predicts that there should be a repetition advantage for the subset of analyzed practice pairs even in the saline condition (Figure 3). The behavioral data showed a small effect in that direction, which was not significant after correcting for multiple comparisons (p = .09). One possibility is that this is due to a selection bias – the practice pairs selected for analysis were those that showed no evidence of learning in three previous tests (~32%), thus they were generally hard to learn. Note that the control pairs we analyzed were those that had not been recalled on only 1 previous test (~76%) so they were probably not as difficult to learn. The greater difficulty of the selected practice pairs might have offset the *relative* repetition benefits under saline (as seen from Fig 2, practice pairs do benefit more under saline than under midazolam). Another possibility is that there were only few observations per cell, and the resulting noise might have obscured the effect in the saline condition. Despite this, the key prediction, namely the comparison between control and practice pairs under midazolam, was quite robust.

In summary, the current study provides evidence that one mechanism through which repetition benefits memory is the strengthening existing memory traces. Despite this result, we do not wish to argue that no additional information beyond strength is stored in memory with each repetition of an event. Based on JOR, JOF, LLE and LSE results reviewed in the introduction, and the results presented here, it is reasonable to conclude that repeated experiences affect memory through a multitude of mechanisms that include both strengthening of previously encoded traces that match in content, as well as storing novel traces to represent the unique features of the repeated experience. What part of that information is accessed likely depends on the nature of the task being performed. While accurate judgments of recency and frequency might require accessing and comparing information across multiple memory traces, recognition and recall can depend on the strength of any one of those traces.

Table 1 SAC model parameters

Par	Description	Value
	Imported parameters	
A_{boost}	Value added to current activation when an item is perceived	40
p_{decay}	Exponential decay constant for current activation	0.8
d_{node}	Power-law decay constant for base-level activation	0.175
c_{node}	Power-law growth constant for base-level activation	25
d_{link}	Power-law decay constant for link strength	0.12
c_{link}	Power-law growth constant for link strength	25
b_{freq}	Exponent for Kucera and Francis word frequency norms for estimating pre-existing base-level activation	0.4
l_{freq}	Exponent for Kucera and Francis word frequency norms for estimating preexisting link fan	0.7
	Estimated parameters	
$P_{baseline}$	Baseline probability of encoding an event node for a new word pair	0.35
$\sigma_{episode}$	Standard deviation of the episode node activation	1
$T_{episode}$	Retrieval threshold for episode node activation	2.9 ± 1.6*

* Parameter was fit individually for each participant (Mean ± SD)

Table 2 SAC model equation

Equation	Description
$B_0 = KF^{b_{freq}}$	Preexisting base-level activation; a function of Kurcera & Francis word frequency
$B = B_0 + c_{node} \sum t_i^{-d_{node}}$	Current base-level activation is a function of preexisting base-level activation and time since each presentation of a stimulus. t_i is the time since the i-th presentation
$S_{cue,episode} = c_{link} \sum t_i^{-d_{link}}$	Current strength of the link from the cue to the episode node is a function of time since each presentation of the stimulus. t_i is the time since the i-th presentation
$A_{cue} = B + A_{boost}$	Current activation of the cue is a function of base-level activation and a perceptual boost
$A_{input} = A_{cue} \dfrac{S_{cue,episode}}{\sum S_{cue}}$	The input to an episode node due to spreading activation from the cue is a function of the cue activation level, the strength between the cue and the episode node, and the fan of the cue
$A_{episode} = \ln(B + A_{input})$	Current activation of the episode node is the natural logarithm of the sum of the base-level activation and the received spreading activation
$P_{encoding} = P_{baseline} * (1 - C \times 2^{-\frac{t_{injection}}{t_{hl}}})$	The probability of encoding the episode node is a function of the baseline probability, whether a the drug was saline (C=0) or midazolam (C=1), the time since the injection and the half-life of the drug.
$P_{retrieval} = N(A_{episode} \mid \sigma_{episode}, \tau_{episode})$	The probability of retrieval of the episode node is the area to the left of the activation value under a standard normal distribution with the threshold as the mean.

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