

Modeling human sequence learning under incidental conditions.

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ABSTRACT

This research explored the role that associative learning may play in human sequence learning. Two-choice serial reaction time tasks were performed under incidental conditions using two different sequences. In both cases an experimental group was trained on four sub-sequences: i.e. LLL, LRL, RLR and RRR for Group ‘Same’ and LLR, LRR, RLL and RRL for Group ‘Different’, with left and right counterbalanced across participants. To control for sequential effects, sequence learning was assayed by comparing their performance to a control group, which had been trained on a pseudo-random ordering, during a test-phase in which both experimental and control groups experienced the same sub-sequences. Participants in both groups showed sequence learning, but the group trained on ‘Different’ learned more, and more rapidly. This result is the opposite to that predicted by the Augmented SRN used by Jones and McLaren (2009), but can be modelled using a re-parameterised version of this network that also includes a more realistic representation of the stimulus array, suggesting that the latter may be a better model of human sequence learning under incidental conditions.

1 Understanding human sequence learning under incidental conditions, whether it involves
2 learning a sequence of events or a sequence of actions, is key to explaining much of human
3 and infra-human behavior. In order to learn sequences, people and animals need to cope with
4 information embedded in a temporal context, adding an extra dimension to the more static
5 problems typically studied in research on associative learning, and bringing them closer to
6 those that occur in real situations outside the lab. This extra complexity also constrains the
7 modeling of human sequence learning, where it is often addressed by the addition of
8 recursion to otherwise static models, for example the SRN (Simple Recurrent Network,
9 Elman, 1990) and the Augmented SRN (Cleeremans & McClelland, 1991). The question that
10 this paper addresses is whether or not these models provide adequate accounts of sequence
11 learning under incidental conditions.

12 In the experiment reported here we focus on a very simple task in which sequence
13 learning is known to occur, even though it is not explicitly required, and is hence often cited
14 as a situation in which "implicit" learning occurs. This is the variant of the two-choice serial
15 reaction time (SRT) task recently developed by Jones and McLaren (2009). In this task
16 participants observe two circle outlines on a screen and are given two response keys, one for
17 each circle. On each trial one of the circles "fills in" and the participants press the
18 corresponding key as quickly and accurately as possible. Following this, the circle outlines
19 reappear for 500 msec before the next trial starts. Trials come rapidly one after the other, and
20 the experience is of a fast-paced task that emphasises speed and accuracy in reacting to the
21 stimuli and requires little else.

22 In fact, for the experimental groups in this task there is a probabilistic rule governing
23 the sequence of locations in which the circle appears, knowledge of which could enable
24 participants to prepare for the stimulus and so increase the speed and accuracy of their
25 responding. The roles of the two stimulus locations are counter-balanced across participants

26 and so henceforth will be referred to as X and Y, rather than right and left. In our previous
27 work (Jones & McLaren, 2009), we were able to show that the Augmented SRN (Cleeremans
28 & McClelland, 1991) could successfully model incidental learning of a sequence that
29 comprised sub-sequences XXX, YYX, XYY, YXY, which follow the rule "if the first two
30 locations are the same then the third is an X, if they are different then it's a Y".

31 In the current experiment we vary the sub-sequences to see if the Augmented SRN
32 can still model the results. Thus, one group in this experiment has XXX, YYY, XYX and
33 YXY as their sub-sequences, which follow the rule that the "third element is the same as the
34 first". By concatenating these sub-sequences (e.g. XXXYYYXYX... etc) we can produce a
35 sequential structure that has the property that two-thirds of the time a trial is the same as the
36 trial before last. The other group is trained on the complementary set: XXY, YYX, XYY and
37 YXX, where the rule is that the "third element is different to the first" so that after
38 concatenation, two-thirds of the time the current trial is different from the trial before last. In
39 our experiments learning is measured relative to pseudo-random control groups. The controls
40 experience a mixture of all eight sub-sequences so that the first trial has no predictive value
41 for the third. Our interest, then, is in comparison of the differences between experimental and
42 control groups for those participants trained on sequences in which the first trial is different
43 to the third (Group Different) to those trained on first same as third (Group Same). The factor
44 of Group, denoting the type of sub-sequences used during training, will be a dummy variable
45 for the controls; as all of these participants receive the pseudo-random mixture of all eight
46 sub-sequences throughout.

47 We are focussing on this comparison because a simple extrapolation from the
48 empirical results of Jones and McLaren (2009) leads one to predict that Group Different
49 should have an advantage. This is because the sub-sequences XXX and YYY can be expected
50 to be very difficult to learn based on these earlier findings, and both these sub-sequences fall

51 in Group Same. Intriguingly, when we ran the Augmented SRN on this new experiment with
52 the same parameters as those used to model Jones and McLaren (2009), the pattern we
53 obtained was actually the reverse, with Group Same sub-sequences learnt better than Group
54 Different sub-sequences. Thus evidence-based intuition and the model seem to be in conflict,
55 and an empirical test was needed to resolve the issue. We will return to a discussion of the
56 modeling once we have reported the results of our experimental work.

57 **Method**

58 *Participants*

59 The study was conducted on 128 participants, randomly divided into four groups (two
60 experimental and two control). There were 32 participants in each of the two experimental
61 conditions, and the same number in the two control conditions (both control conditions were
62 actually treated identically and participants were randomly assigned as the control for one of
63 the two experimental conditions). The participants were all students at the University of
64 Exeter, aged from 18 to 35 years old. Additionally, each of the participants was rewarded for
65 their contribution with £10 at the end of their second session.

66 *Materials*

67 The two-choice SRT task was run on an Apple Mac computer, with the basic display
68 being one of two white outline circles on a black background. The circles were 1.9cm in
69 diameter and each was positioned 2.2cm right/left of the middle of the screen, which was
70 approximately 0.5m from the participant. The stimulus was a white filled circle 1.9cm in
71 diameter that replaced either the right or left outline circle during the trials. The participants
72 were instructed to press the “x” key on a QWERTY keyboard if the target stimulus appeared
73 on the left, and the “.” key if the stimulus appeared on the right. These keys were chosen to
74 be spatially compatible with the two stimulus locations.

75 *Design*

76 The experiment consisted of a two-choice SRT task that was conducted over two sessions,
77 each lasting about an hour. The first session was usually undertaken in the morning, with the
78 second session typically commencing after a 3 to 4 hour break on the same day. Both
79 sessions consisted of 20 blocks of 120 trials, with the last five blocks of Session 2 acting as
80 the test phase. All other blocks acted as training. The blocks for each of the experimental
81 conditions were constructed by concatenating equal numbers of the relevant sub-sequences,
82 as already described. Thus, during the training phase of the SRT task, experimental
83 participants in Group Different were presented with sequences made up of sub-sequences
84 where the location of the third trial was opposite to the location of the first trial (e.g. XXY).
85 The rule was different for participants in the experimental condition of Group Same, as in
86 training they were presented with sequences made up of sub-sequences where the third trial
87 location was the same as the first trial location (e.g. YXX). During training, participants in
88 the control conditions experienced pseudo-random blocks, which were created by
89 concatenating equal numbers of the eight possible triplets in a random order (see Jones &
90 McLaren, 2009, for further details). Note that, for all the conditions and groups, when the
91 sub-sequences or triplets were concatenated they formed continuous strings of trials, and
92 previous evidence suggests that participants do not learn about the special status of the third
93 trials, but rather learn the contingencies on a trial-by-trial basis (Jones & McLaren, 2009).
94 When training blocks are considered trial-by-trial, trials consistent with the experimental
95 groups' sub-sequences occur two-thirds of the time, with the remaining third of trials being
96 inconsistent (e.g. in the experimental condition of Group Same, XX is followed by X twice as
97 often as it is followed by Y).

98 For all conditions, the last five blocks of Session 2 acted as the test-phase and
99 consisted of pseudo-random blocks only. By comparing experimental and control

100 performance on what are effectively the same types of sequence, possible confounds due to
101 sequential effects are controlled for (Jones & McLaren, 2009).

102 *Procedure*

103 As in Jones and McLaren (2009), the participants were instructed to respond as
104 quickly as possible whilst avoiding errors. No mention was made of any sequential structure
105 embedded in the task. On each trial, the stimulus remained on the screen until the participant
106 had responded or was timed-out for not having pressed a key within 4.25s of the stimulus'
107 appearance. RT was measured from the stimulus' appearance on screen until the computer
108 detected a key press, and a 500ms Response-Stimulus Interval (RSI) was used. If participants
109 pressed an incorrect key or were timed-out then the trial terminated and the computer issued a
110 short 'beep' sound. Similarly, if they anticipated a stimulus, that is responded less than
111 100ms after its onset, then the trial was aborted and a beep sounded. Following each block,
112 participants experienced a 30 second break during which they were shown their average
113 reaction time (in milliseconds) and their accuracy (as an error percentage) for the last block.
114 They were also informed whether these scores were better or worse than those from the
115 previous block.

116 **Results**

117 For the test-phase data, difference scores were computed by subtracting the mean RTs and
118 proportion of errors for trials that were consistent with each sub-sequence from the respective
119 scores for trials that were inconsistent. To illustrate, consider the Group Same, Experimental
120 Condition sub-sequence XXX. Any X trial that is preceded by XX (which we will label as an
121 XXX trial) is consistent with the sub-sequence XXX, while any Y trial that is preceded by
122 XX (i.e. XXY) is inconsistent with this sub-sequence. Therefore, the RT difference scores
123 for sub-sequence XXX were calculated by subtracting the mean RT for XXX trials from the
124 mean RT for XXY trials, and a similar subtraction gave us an equivalent difference for errors.

125 This was done for all experimental sub-sequences, for each group and condition. In the
126 Control Conditions consistent/inconsistent was a dummy variable that was determined by the
127 sub-sequences in the paired Experimental Condition.

128 Because the Experimental Conditions experienced different trial orders to their
129 respective Control Conditions in training, any difference between the conditions here could
130 be due to sequential effects (i.e. performance differences on different trial orders) instead of
131 sequence learning (cf. Soetens, Boer & Huetting, 1985). To minimise this confound, the
132 method of calculating inconsistent minus consistent training difference scores described by
133 Jones and McLaren (2009) was employed. Sequential effects of up to order $n-3$ were
134 controlled for by equal-weight averaging of the two versions of each sub-sequence that exist
135 when the $n-3$ trial is also considered (e.g. the score for XXX is the equal weighted average of
136 the scores for YXXX and XXXX). Insufficient data meant sequential effects of $n-4$ and
137 greater could not be controlled for in this way, but an inspection of our data suggests that $n-4$
138 sequential effects are in the order of 1ms (for a more detailed discussion, see Jones &
139 McLaren, 2009, p. 543). This type of analysis sometimes leads to decreased degrees of
140 freedom because some participants do not have sufficient data to contribute to all the
141 analyses. To minimise this, data from pairs of blocks were collapsed; i.e. in the analyses, data
142 from Blocks 1 and 2 were treated as being from one block.

143 Our expectation is that, for both training and test, sequence learning should increase
144 the difference scores of the Experimental Conditions compared to their respective Controls,
145 because it should increase RT and errors on inconsistent trials and decrease RT and errors on
146 consistent trials. Figure 1 shows these scores during training and test. We examine the
147 training data first.

148 Considering the Control Conditions, the pattern here can be attributed to sequential
149 effects. The Group Same Control Condition's scores are positive for both errors and RTs,

150 because the Group Same sub-sequences are trial-orders that participants find it relatively easy
151 to respond to, but note that the Group Same experimental scores are higher still, suggesting
152 that training on the Group Same sub-sequences has led to sequence learning. The Group
153 Different Control Condition scores are approximately the mirror image of those for the Group
154 Same Control Condition, with any (small) deviation from this being attributable to random
155 variation. Thus, their Inconsistent-Consistent scores are negative, because their sub-
156 sequences are those that participants do not find easy to perform. Once again the
157 Experimental Condition's scores are higher than their controls (much higher in the case of
158 errors) suggesting that training on the Group Different sub-sequences has resulted in
159 sequence learning.

160 We can assess the main effect of Condition (Experimental vs. Control) for Group
161 Same in RTs, $F(1, 48) = 68.96, p < .001$; and errors, $F(1, 57) = 7.97, p < .008$; and similarly
162 for Group Different in RTs, $F(1, 48) = 72.93, p < .001$; and errors, $F(1, 54) = 44.59, p < .001$.
163 In all cases there is good evidence of superior performance in the Experimental conditions,
164 and the difference between Experimental and Control Conditions increases over blocks in
165 both groups, further supporting the conclusion that the participants have learned at least some
166 of the statistical structure of these sequences during the course of training. The interaction
167 between Condition and Block that supports this assertion is significant in both RTs, $F(16,$
168 $768) = 5.31, p < .001$; and errors, $F(16, 864) = 3.82, p < .001$ in Group Different. The same
169 Condition by Block interaction is also significant in Group Same for RTs, $F(16, 768) = 5.93,$
170 $p < .001$; and errors, $F(16, 912) = 1.76, p < .04$. Hence, both groups exhibit reliable learning
171 of the sequences during our experiment, but the difference between Experimental and Control
172 Conditions over Blocks also differs for Group Different and Group Same, as the Group by
173 Condition by Block interaction is significant in the RTs: $F(16, 1536) = 1.98, p = .012$, (but
174 not in the errors: $F(16, 1776) = .812, p = .67$). Inspection of the graphs in Figure 1 suggests

199 for all groups and conditions. The effect is not large, but it is entirely reliable and not
200 compromised by any issues of speed vs. accuracy.

201 We are also able to offer some reassurance that our results were indeed obtained
202 under incidental conditions. By the end of the study, no participants were able to tell us what
203 the sub-sequences in the experiment were. This fits well with the claims made by Jones and
204 McLaren (2009) for this paradigm under the same conditions, and reassures us that our
205 instructions and procedures placed our participants in this experiment on a similar footing to
206 those in the earlier study. Given this, we can now enquire whether the model that fit the data
207 in Jones and McLaren (2009), the Augmented SRN (Cleeremans & McClelland, 1991), is
208 also able to fit these results.

209 We attempted to model the experiment using the Augmented SRN with exactly the
210 parameters given in Jones and McLaren (2009). In this model (shown in Figure 2, please
211 disregard the two input units at the bottom labelled "Next trial" for this purpose), the two
212 possible stimulus locations were each assigned an input unit (units labelled "Current trial")
213 and were activated as appropriate on each trial, following the same sequential structure,
214 number of trials, blocks and sessions as used for participants. These fed forward to a set of
215 hidden units, which in turn activated two output units (labelled "Prediction of next trial"),
216 with the activity of units in both layers determined by the logistic activation function
217 (Rumelhart, Hinton & Williams, 1986). At the end of each trial, the activations of the hidden
218 units were copied via one-to-one feedback connections to a set of "context units" on the input
219 layer (labelled as "Copy of last trial's hidden units"). This recurrence is the essence of
220 Elman's (1990) SRN architecture. The output units corresponded to the two possible stimulus
221 locations, and their activation represented the model's prediction of the identity of the next
222 trial.

223 The Augmented SRN has a similar architecture to the SRN, but differs in its feed-
224 forward connections. Where the SRN has modifiable network connections driven by a single
225 learning parameter, the Augmented SRN has two components to these connections, fast and
226 slow (not shown separately in the figure), which have higher and lower learning rates,
227 respectively. The fast components also decay by half their value at each time step, a feature
228 adopted by Cleeremans and McClelland (1991) to help account for the robust short-term
229 priming effect observed in their data. In addition, the Augmented SRN includes a set of
230 response units, to capture the effect that responding to the previous trial has on the current
231 trial, whereas the SRN lacks this component. Cleeremans and McClelland (1991),
232 Cleeremans (1993) and Jimenez, Mendez, and Cleeremans (1996) have shown that this model
233 can capture the detailed pattern of SRT data.

234 Using the stimulus location on the following trial as a training target, the weights
235 (both fast and slow in the Augmented SRN) determining the strength of each connection
236 between units were modified according to the back propagation algorithm (Rumelhart,
237 Hinton & Williams, 1986). As in the simulations conducted by Jones and McLaren (2009),
238 the networks used had 20 hidden units and a slow learning rate parameter of 0.4, with the fast
239 weights having a learning rate 1.33 times larger. Thirty-two networks were run in each of the
240 four cells of the experimental design. The results are shown in the top panels of Figure 3. If
241 we compare the simulations to the empirical results, then first impressions are that there is
242 some correspondence, especially given that we have not fit the model to the data by varying
243 parameters. The Augmented SRN does quite well in predicting the basic pattern during
244 training, in that Experimental and Control groups are appropriately placed with respect to one
245 another, and at least some of the trends observed in our data are also present in the
246 simulations.

247 But the crucial point is that there are areas of significant disagreement between our
248 data and the model predictions. Most important are those in the test data (top-right panel of
249 Figure 3). Both groups demonstrate a significant main effect of Condition: for Group
250 Different $F(1, 62) = 290.65, p < .001$, and for Group Same $F(1, 62) = 1383.87, p < .001$, but
251 the significant interaction between Condition and Group, $F(1, 124) = 11.61, p < .002$,
252 confirms reliably greater sequence learning in Group Same for the Augmented SRN, which is
253 the contrary pattern of results to that found in our empirical study. Thus, we have to reject the
254 Augmented SRN, or at least this version of it, as an adequate model for our data.
255 Furthermore, with this architecture, we have, to date, been unable to find a set of parameters
256 for the Augmented SRN that will allow it to correctly predict the ordering of Groups
257 Different and Same on test despite an extensive search over the parameter space for the
258 model, suggesting that, as it stands, it cannot model our data. In any case, we can conclude
259 that the version of the model that was successful in modeling the data in Jones and McLaren
260 (2009) is demonstrably falsified by our results.

261 This outcome was surprising, as the Augmented SRN is our benchmark model of
262 sequence learning, and coped very well with the pattern of results that we obtained in Jones
263 and McLaren (2009). Our initial response was to revert to the version of the SRN adopted in
264 Spiegel and McLaren (2006). This proved capable of simulating more learning in Group
265 Different than Group Same, but only at the cost of losing the ability to adequately simulate
266 the overall pattern for our dataset on test, and it does not provide as good a fit to the Jones
267 and McLaren (2009) data. After considering various other modifications of the network, we
268 finally realised that our simulation of this task using the Augmented SRN was unrealistic in
269 the following way. Recall that our architecture is feed-forward (and recurrent) with two input
270 units set according to which of the left or right locations were designated as responses on the
271 trial just past, and context units whose activation is set by the hidden unit activations on the

272 previous trial as well. What we had failed to include in our model was anything that
273 represented the stimulus - the filled circle – that always occurs just before a response is made.
274 This was because this stimulus completely specifies the response, and it would have seemed
275 odd to include something so directly predictive when we were interested in the ability of the
276 network to learn the sequential contingencies in play, not learn that when the left circle filled
277 it was to produce a left response! But, our participants would have been exposed to just such
278 a contingency, and so would have had the opportunity to learn about it. In some sense, this
279 captures the idea of some automatization occurring in the course of experience that takes over
280 from the instruction to press the corresponding key when one of the circles fills. Hence, we
281 included these inputs in our new network (the input units labelled "Next trial" in Figure 2),
282 but, because the circle only fills just before the response, we gave it a relatively low
283 weighting in our model¹. With this addition, we were able to successfully re-parameterise the
284 Augmented SRN to produce our pattern of results in this experiment and still generate the
285 pattern of results found by Jones and McLaren (2009). A typical set of simulation results for
286 the current experiment is shown in the bottom panels of Figure 3. These simulations were run
287 using the two additional input units, and activation of the unit corresponding to the response
288 required on the to-be-predicted trial was set to 0.1. Input activations corresponding to the
289 response units that had been activated on the previous trial were set to 0.75, and the context
290 unit activations were set to 1.3 times the hidden unit activations from the previous trial. These
291 parameters modulate the relative weightings of the contributions to learning from the
292 different inputs and the context units, and were deliberately chosen to allow us to simulate
293 our data. The learning rate parameters for the fast and slow weights were set to 0.5 and 0.2
294 respectively, and other parameters were the same as those used in Jones and McLaren (2009).

¹ The low weighting was intended to reflect the fact that the timing was sub-optimal for an associative network learning to predict the next response required – but this is a completely separate issue to the cue's predictiveness which, of course, was 100%.

295 The training data shown on the left of the bottom panel of Figure 3 again have the
296 different conditions / groups in their appropriate relative positions. The control groups are
297 once again approximate mirror images, and now produce a somewhat more stable pattern of
298 performance over time. Learning proceeds at approximately the same rate in the two
299 experimental groups (it's slightly faster overall for Group Different). The real data of interest
300 are those at test, however, and here the pattern corresponds closely to that in our empirical
301 data. There is a main effect of Group, $F(1, 124) = 1682, p < .001$, a main effect of Condition,
302 $F(1, 124) = 717, p < .001$, and importantly, an interaction between Group and Condition, $F(1,$
303 $124) = 4.08, p < .05$, such that the difference between Experimental and Control Conditions
304 for Group Different is significantly greater than that for Group Same. In other words, Group
305 Different sequences are better learned than Group Same, though the effect is relatively small
306 (roughly 10%) compared to the main effects. This is very much the pattern, and the power,
307 that we observe in the empirical data we report here. We can also confirm that this model
308 captures the pattern of sub-sequence learning observed in Jones and McLaren (2009), and
309 also predicts that it is the difference in performance between XXX and the other sequences
310 used in 2009, XYY, YXY and YYX that will be the easiest to detect. It would appear, then,
311 that this revised model is a candidate to be our new benchmark for modeling sequence
312 learning with this task under incidental conditions.

313 Why does this revised model succeed where the standard Augmented SRN failed?
314 The new version of the model differs from the old version in both including a more accurate
315 representation of the stimulus conditions in the experiment (an unambiguously good thing),
316 and in possessing more free parameters as a consequence of this modification. Is its success
317 simply a consequence of greater flexibility in fitting the data contingent on this increase in
318 free parameters? We believe this is not the right explanation, because when we simulated the
319 Jones and McLaren (2009) data with the new model we did not vary the parameters at all,

320 implying our success (the fit was actually better than in the Jones and McLaren paper) is
321 unlikely to represent "overfitting" of the data. Instead, we believe that the inclusion of the
322 units corresponding to the circle stimulus on the current trial is the critical feature making the
323 difference, and if we take this out of the simulation leaving everything else unchanged then
324 the model reverts to predicting that Group Same should perform better on test than Group
325 Different. The change made is in some sense minor, but is also important. It does not
326 represent a change in the algorithms used in the Augmented SRN, or even in its basic
327 architecture, but it is a departure from conventional simulation practice as far as the SRT task
328 is concerned. As far as we are aware, no one else modeling sequence learning involving this
329 type of task includes the current stimulus as an input in the model – but clearly it matters.
330 Why has it typically been left out when simulating sequence learning in the SRT task? We
331 think the reason is fairly straightforward – researchers were (are) interested in sequence
332 learning, and putting in this input contributes nothing to learning about the contingencies
333 between sequences of events in this task, it's just allows S-R learning. This type of
334 information cannot actually assist in learning sequential structure, and would be expected to
335 be the same in both experimental and control groups, and hence controlled for when assaying
336 sequence learning. But, whilst this S-R learning cannot produce a difference between
337 experimental and control groups in its own right, we now realize it can modulate our ability
338 to learn about sequential structure, and so influence the size of that difference. It does this, we
339 believe, via cue competition, which itself varies as a function of the local temporal sequence
340 of events experienced. To see this, consider as an example the sequence LLL (three left
341 responses required in a row). On the first two trials of this sequence the Augmented SRN will
342 learn (transiently) that a left on trial n-1 predicts a left on trial n, and also the association
343 between the left stimulus and the required left response will have been incremented. The first
344 effect makes learning of the "LL is followed by L" structure difficult, because it partially

345 blocks it (this is the explanation of why LLL is learned poorly under implicit conditions
346 given in Jones and McLaren, 2009). But the second effect, incrementing the S-R learning,
347 also contributes to blocking learning of the "LL is followed by L" contingency. Learning
348 LLR is, relatively speaking, easier, because the R is surprising in terms of the "L predicts L"
349 transient learning, and, in this case, the R stimulus to R response association will not have
350 just received two increments. Thus, the increments to the S-R associations are more of a
351 problem for some subsequences (which turn out to be those in Group Same) than others, and
352 so contribute to Group Same learning the sequential structure more slowly. For this reason,
353 we cannot simply disregard this S-R information any more on the basis that it will be the
354 same for both Experimental and Control groups. Clearly we should not disregard this aspect
355 of the task in any case, if we are to hold to the view that these models are automatic in their
356 operation and learn about all elements of the perceived stimulus array. If we are to believe
357 that this is a real psychological model of associative learning, then, because the circles in the
358 experiment flash signalling which response to make, there must be something in the model
359 that represents this, and the model will inevitably learn about this 100% reliable contingency.
360 But now we can see that if we do neglect this aspect of the stimulus conditions, then our
361 simulations do not match the empirical data, which is, in some sense, a rather encouraging
362 outcome for this modelling approach.

363 Are there any discrepancies between our model's simulation and the data? One
364 obvious discrepancy arises when comparing the training data from the model and our
365 empirical results. The change from session one to session two is not captured by the model –
366 but this is hardly surprising as we have no way of representing it in the model at present.
367 Perhaps the worst aspect of the model as it stands is that it has Group Same learning faster
368 than Group Different in the middle section of the graph, whereas the empirical data show the
369 reverse, but even here it is difficult to know if this is a reliable difference, and the analysis is

370 compromised both by the effect of a change of session and by a lack of power. We
371 acknowledge that it is also possible to criticise the current model for being rather slow to
372 learn. We speculate that a modification of back propagation, APECS (McLaren, 1993, 1994;
373 Le Pelley & McLaren, 2001; McLaren, 2011) instantiated in a recurrent architecture (Jones,
374 Le Pelley & McLaren, 2002) might be the way forward here in terms of improving learning,
375 and incorporating aspects of memory that might permit the session effect to be captured. But,
376 for now, on the basis of the data available in the literature on sequence learning in humans
377 and contained in this paper, the revised Augmented SRN is our benchmark model of
378 sequence learning.

379

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435 mile with the simulation work reported in this paper.

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438 **Figure legends**

439

440 Figure 1: This shows reaction time difference scores (top half) and proportion of errors
441 differences scores (bottom half), during training (left panels) and on test (right panels).
442 Blocks are given in block pairs (i.e. 2 means the average of blocks 1 and 2), and there was a
443 break between blocks 20 and 21 (two different sessions at least 2 hrs apart). Only 14 blocks
444 (7 block pairs) are shown in the second session as the last five blocks were used as the test
445 phase.

446

447 Figure 2. This shows the model architecture for the Augmented SRN used in Jones and
448 McLaren (2009, Figure 7), with the addition of two extra input units (corresponding to the
449 two response locations) labelled "Next trial". See text for a description of the model and its
450 operation.

451

452 Figure 3: This shows mean-square error difference scores during training and test for the
453 Augmented SRN (top) and the revised Augmented SRN (bottom). We did not attempt to
454 simulate the delay between blocks 20 and 21. Otherwise it is laid out exactly as Figure 1.

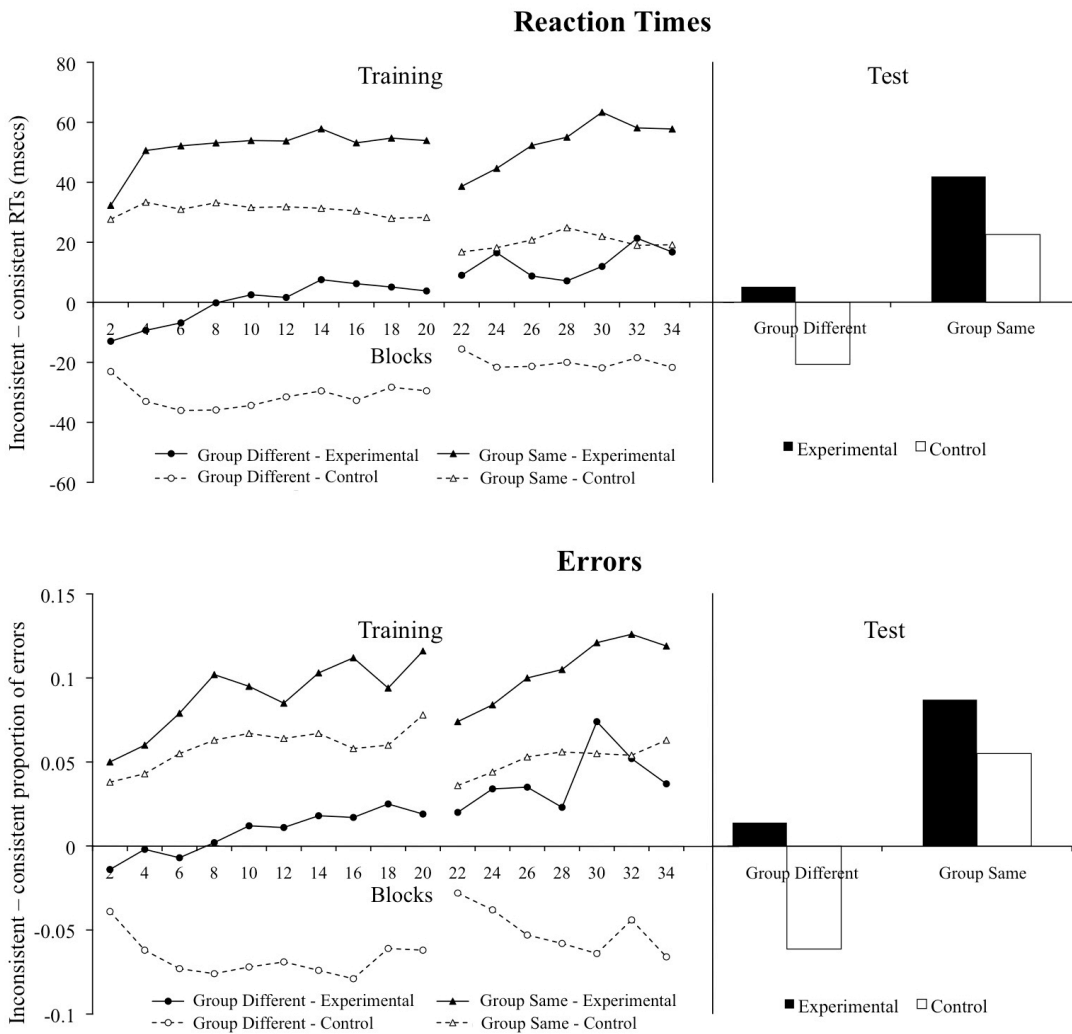
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458 FIGURE 1

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463 FIGURE 2

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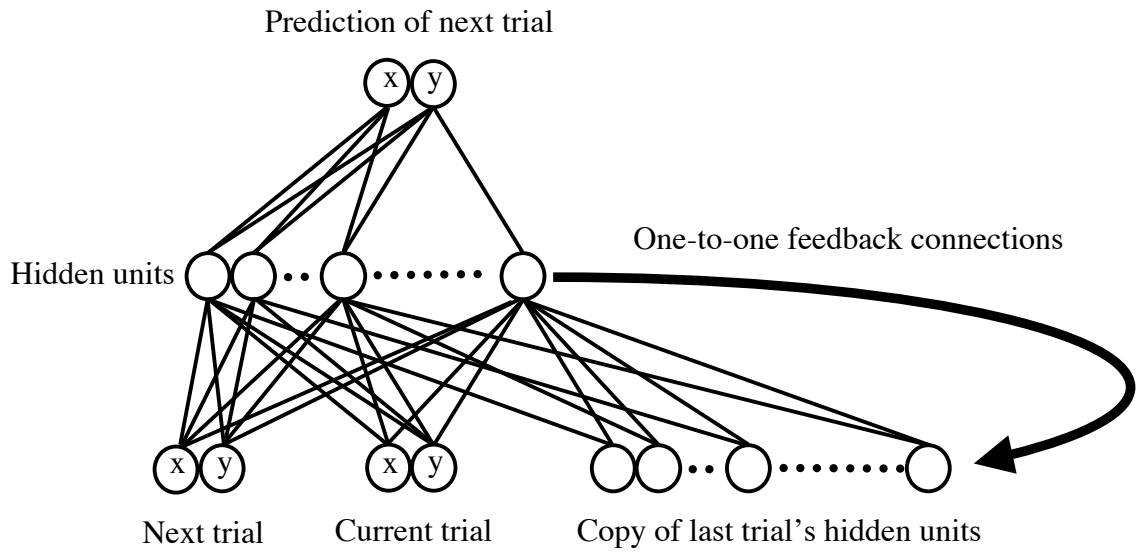
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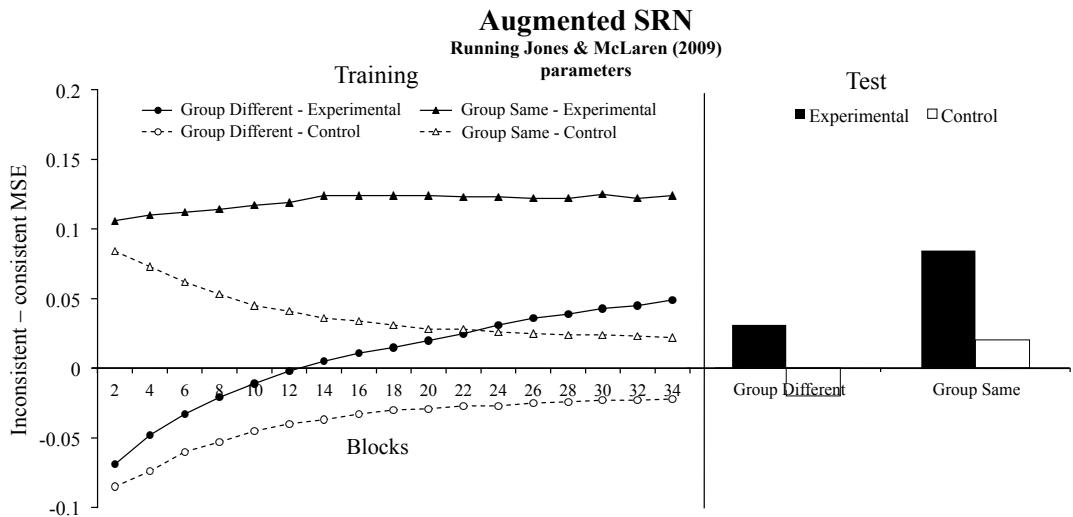
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480 FIGURE 3

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