Can US sensitization account for the electrodermal variant of the Perruchet effect?

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Abstract

During experiments employing Perruchet's (1985) paradigm there are runs of reinforced (CS-US) trials and non-reinforced (CS-noUS) trials. Conditioned responding (CR) is measured, for example, using blink responses (Perruchet, 1985), reaction times (Perruchet, Cleeremans, & Destrebecqz, 2006), or changes in skin conductance (SCR; McAndrew, Jones, McLaren, & McLaren, 2012), as well as an online measure of expectancy for the unconditioned stimulus (US). A double dissociation between CR and conscious expectancy of the US is typically found, whereby expectancy of the US decreases while the CR increases across runs of successively reinforced trials. A gambler’s fallacy explanation can be offered for the expectancy data, whereas an associative explanation can be used to explain variations in the CR (consistent with the dual processing theory of McLaren, Green, & Mackintosh, 1994). However, skeptics of this effect have proposed nonassociative explanations of the CR data seen in these experiments. They note that every CS-US pairing is confounded by the presence of the US. Therefore, it is possible that US sensitization, the phenomenon whereby repeated US presentations lead to stronger unconditioned responding to the US, could produce the increasing CR pattern with successive reinforcements (Weidemann, Tangen, Lovibond, & Mitchell, 2009). Two experiments are presented investigating whether US sensitization can explain the recently published electrodermal version of the Perruchet effect.

Keywords: Perruchet effect; US sensitization; Dual processing systems

Introduction

The Perruchet effect (Perruchet, 1985, Perruchet, Cleeremans, & Destrebecqz, 2006) is often cited as one of the most convincing pieces of evidence of a dissociation between explicit, conscious, propositional processes and implicit, automatic, associative processes (e.g. Mitchell, De Houwer, & Lovibond, 2009). McAndrew, Jones, McLaren and McLaren (2012) ran an electrodermal variation of the classic Perruchet experiment in which participants saw a visual conditioned stimulus (CS), a brown cylinder, which was partially reinforced by an electric shock (the unconditioned stimulus, US). The participants made online expectancy ratings on every trial as to whether they thought the US was going to occur. Changes in their autonomic skin conductance response (SCR) were also measured as an index of conditioned responding (CR).

We found that the SCR increased over successive reinforcements, while expectancy of the US decreased across the same sequence of trials. This mirrored the original findings of Perruchet and colleagues in both the eyelink (Perruchet, 1985) and reaction time (RT; Perruchet, Cleeremans, & Destrebecqz, 2006) variants of this paradigm. The gambler’s fallacy heuristic (Burns & Corpus, 2004), an explicit, propositional phenomenon, explained the expectancy data, implying that as participants experienced successive runs of reinforced (CS-US) trials, they were less likely to rate the subsequent trial as being paired with a US. Conversely, when participants experienced runs of successively non-reinforced (CS-noUS) trials they rated it as increasingly likely they would experience a US. However, this explanation did not apply to the SCR data, but an associative account did (e.g. McLaren, Forrest and McLaren, 2012). SCR increased over successive reinforcements, meaning that the CR was strongest when the participants had experienced a run of CS-US trials. In associative terms, during this type of Pavlovian conditioning, the link between the representation of the US and the representation of the US was strengthened by the successively reinforced trials, producing a larger CR. However, after a run of CS-noUS trials, the link between the representations of the CS and the US was weakened by extinction, causing smaller changes in SCR and therefore a weaker CR. Hence, these results are consistent with a dual processing systems account of learning, with an explicit propositional system generating the expectancy data and an associative system the changes in SCR.

The Perruchet effect is one of the most compelling examples of dual processing systems due to the simultaneous measurement of CR and expectancy. Previous research demonstrating dissociations between these two variables has often involved subliminal presentations of the CS (e.g. Balderston, & Helmstetter, 2010), but this research is often criticized about whether the presented stimuli are truly subliminal (e.g. Mitchell, De Houwer, & Lovibond, 2009). Alternatively, researchers have attempted to use post-conditioning questionnaires to assess contingency awareness, however it has been argued this type of measure could be subject to interference or forgetting influencing the reliability and validity of the awareness measure (Lovibond, & Shanks, 2002). The Perruchet paradigm however, overcomes these problems and is a more convincing demonstration of a double dissociation.

However, the dual processing system account of the Perruchet effect depends critically on the assumption that the linear trend in CRs is the result of associative learning. Alternately the pattern of CRs could be accounted for by US sensitization; this effect refers to the increase in unconditioned responding (UR) seen when there is repeated exposure to a US (Weidemann, Tangen, Lovibond, & Mitchell, 2009). In the Perruchet experiments, every CS-US pairing unavoidably involves presentation of the US. Therefore, it may not be the pairing of the two stimuli (CS and US) strengthening or weakening the associative link between the stimuli that is causing fluctuations in the SCR.
Instead, it could simply be exposure to the US driving this effect. If this were true, this would undermine the Perruchet effect as evidence for dual processing systems.

Research investigating US sensitization as an explanation of CR in the blink and RT variants of the Perruchet effect has produced mixed results; with US sensitization failing to account for data from the blink paradigm (e.g. Weidemann et al., 2009), but Mitchell et al. (2010) finding US sensitization a plausible explanation of the RT data despite Barrett and Livesey (2010) disagreeing. Given the inconsistency on this point, we felt that further investigation was important to determine whether US sensitization could account for the variations in the CR observed in the Perruchet effect. If it were found that US sensitization could adequately account for these results, a single, explicit, nonassociative processing explanation of the results would be sufficient.

In particular, it was important to try and determine whether US sensitization could explain the results reported in our 2012 paper. A lot of past research within the electrodermal domain finds a strong positive correlation between CR and conscious contingency knowledge, for example, if participants fail to develop CS-US contingency knowledge they often fail to show any CR (Lovibond & Shanks, 2002). The implication is that to see a CR using electrodermal procedures, participants must explicitly expect the shock to happen when they are presented with a CS. This view is in stark contrast to our earlier findings. We hypothesized that we were able to dissociate the CR and expectancy of the US because of the nature of the Perruchet task. In our experiment there was a partial reinforcement schedule, half the trials were followed by a shock and half were not, and the participants were made explicitly aware of this from the beginning of the experiment. The participants were therefore put into a state of uncertainty from the start, as they were unable to accurately predict when the shocks were going to happen. Consequently, given that the participants were unable to use their rational, inferential processes to determine when the shocks were going to happen, this provides a context in which some reliance on alternative processing systems, which could be implicit or associative, might be expected. There is some evidence in the electrodermal domain to support this hypothesis. One example is Knight, Nguyen, and Bandettini (2003), who presented participants with tone stimuli, one continually reinforced by white noise (CS+) and another never paired with white noise (CS-). They varied US predictability by presenting the CSs above and below the perceptual threshold and found that even in the absence of any conscious ability to discriminate between the stimuli, there was still evidence of higher CRs to the CS+ than the CS-. Additionally, evidence of an implicit/explicit learning distinction can be found within the neuroimaging literature. Different brain structures appear to be involved in different aspects of learning to the extent that one can differentiate brain regions involved in conscious and unconscious learning (e.g. Tabbert, Stark, Kirsch, & Vaitl, 2006).

Our aim here is to establish whether associative processes govern the CR in our experiments, by checking whether US sensitization can account for our findings. If we can rule out this explanation of our results, then we can add our experiment to the others that show that SCR and conscious expectancy can dissociate.

**Experiment 1**

One of the simplest ways to investigate whether US sensitization governs CR in our Perruchet experiment was to simply remove the CSs. In this way participants would only experience noCS-US trials and noCS-noUS trials. If the same increasing patterns in SCR were found as in the original experiment, this would imply that the result was not dependent on CS-US pairings, as there are no CSs presented in the experiment. Under these circumstances we could conclude that US sensitization would be driving responding. However, if SCR fails to increase over successive US presentations, this would tend to suggest that a US sensitization account could not explain the electrodermal variant of the Perruchet effect.

**Method**

**Participants**

24 University of Exeter students participated in this experiment, 16 women and 8 men; ages ranging from 18-35 (average, 21 years). All were paid £10.

**Stimuli**

The US was a 500ms electrical impulse administered with a PowerLab 26T generator using stainless steel electrodes attached to the left proximal and medial phalanges of the index finger. Participants set their own shock level between 5 and 20mA where they deemed the shock to be “definitely uncomfortable but not painful”.

Throughout the experiment there was a black cross (5 x 5cm onscreen) in the centre of a white screen. The cross was used to fixate participants’ attention.

Skin conductance was measured using LabChart software via MLT116F GSR electrodes attached to the medial phalanges on the left third and fourth fingers. Online explicit expectancy of the US was recorded using a Contour Shuttle Xpress device. Roughly every five seconds participants were required to make an expectancy rating about the extent they thought the shock would happen at that moment in time. The device had 5 buttons and fit nicely into participants’ hand whereby 1 button corresponded to 1 finger. The different expectancy values were: 1 “There will definitely not be a shock”, 2 “There might not be a shock”, 3 “Not sure either way”, 4 “There may be a shock”, and 5 “There will definitely be a shock”. A continuously available key explained which buttons represented which expectancy ratings.

**Design**

There were two repeated-measures factors in this experiment. The first was run length, i.e. the number of trials of the same type in a row; there were six different run
lengths: -3, -2, -1, +1, +2, and +3. The run length measure is taken on the trial after the run itself. For example, a +1 run length SCR is taken on the trial after a participant has previously experienced a US trial that itself was preceded by a noUS trial. A +2 measurement is taken on the trial following two consecutive US trials. Whereas, a -1 run length measurement is taken if the participant has just experienced exactly one noUS trial, and a -2 measurement is taken when a participant experiences two noUS trials in a row. A switch between a positive and negative run length measurement occurs when the trial type just experienced switches from a US to noUS trial and vice versa. The other factor used in the design and analysis is the presence or absence of the US on the trials in the run, i.e. shock (+) or no shock (-).

The trial sequences used in this experiment were matched to the sequences used in the original McAndrew, Jones, McLaren and McLaren (2012) experiment, using the same trial structure and run distributions, see Table 1. In the McAndrew et al. experiment, on shock (+) trials, a 500ms US was administered after 4500ms of the CS being on screen, whereas on no shock (-) trials no US occurred. SCR recordings were taken on every trial, during the five seconds prior to CS onset (PreCS), five seconds while the CS was on screen and five seconds after the CS (PostCS). The inter-trial interval (ITI) was randomly varied between 30 and 40 seconds on each trial in order to stop participants timing the trial interval (ITI) was randomly varied between 30 and 5 seconds before this as a “PreCS” measure and 5 seconds after this as a “CS” period. Long ITIs were required to allow the SCR to stabilize to enable direct comparisons to be made.

Two-way repeated measures analyses of variance (ANOVA) were carried out separately on the SCR and expectancy data. With regards to SCR, there was no significant linear trend over level, $F(2,46) = 0.26, MSE = 0.004, p = .774$, see Fig. 1, a Bayesian analysis (Dienes, 2011) confirmed that we have strong evidence for the null hypothesis, reducing US sensitization as an explanation of our effect, Bayes factor = 0.32. There was a significant effect of US presence, with a higher mean SCR after US absent trials (-0.02) than US present trials (-0.04, see Fig. 2), $F(1,23) = 5.43, MSE = 0.003, p = .029$. The interaction between level and US presence was also not significant ($p > .05$).

There was a significantly increasing linear trend over level in the expectancy data, $F(2,46) = 5.04, MSE = 0.257, p = .044$, see Fig. 1. There was also a significant effect of US presence with a higher mean expectancy rating for US absent trials (3.68) than US present trials (3.17, see Fig. 3), $F(1,23) = 18.41, MSE = 0.496, p < .001$. In addition, there was a significant linear interaction between level and US presence, $F(1,23) = 14.77, MSE = 0.213, p = .001$, reflecting the fact that measures taken after US present (+) trials increase as a function of level whereas those taken after US absent (-) trials slightly decrease.

**Results**

The SCR data was recorded in micro-Siemens in LabChart and exported to Excel. For each trial the mean SCR was taken for the hypothetical “PreCS” and “CS” periods. The data was standardised using a log transformation to reduce the variability between participants. The change in SCR prior to US onset (as a consequence of preceding runs), was calculated using the formula “CS-PreCS”. Mean CR for each run length was then calculated for each participant and across participants. For the expectancy data, the rating made closest to the hypothetical CS period was used as the participants’ expectancy of the US on that trial. A mean expectancy rating for each run length was calculated for each participant and then across participants. Additionally, as in the 2012 experiment, the SCR and expectancy data were collapsed to form levels 1, 2 and 3. Level 1 averages run lengths +1 and -3, level 2 run length +2 and -2, level 3 run lengths +3 and -1. This was done to treat the data in a similar fashion to the 2012 experiment to enable direct comparisons to be made.

# Table 1. Trial types by frequency of occurrence.

<table>
<thead>
<tr>
<th>Variable</th>
<th>noUS (-)</th>
<th>US (+)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Run length</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Frequency</td>
<td>2</td>
<td>4</td>
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**Procedure**

The participants were told they would receive shocks randomly throughout the experiment without any warning they were going to occur. The participants were asked to rate their expectancy that the shock would occur at that moment in time roughly every 5 seconds throughout the duration of the experiment. Expectancy ratings were made using the Shuttle Xpress device, on the scale 1 (definitely no shock) to 5 (definitely shock). Otherwise they were asked to remain still to avoid motion artifacts in the SCR.

![Figure 1. Graph depicting changes in SCR and expectancy as functions of level.](image-url)


**Discussion**

This experiment aimed to investigate the extent to which US sensitization is a plausible explanation of the SCR result observed in our original 2012 experiment. In that earlier experiment participants experienced exactly the same sequences of shocks as used here (NB. a different sequence for each participant), but the shocks followed a CS (a picture of a brown cylinder). This CS also occurred on no-shock trials, so that it had a 50% rate of reinforcement. McAndrew et al. (2012) found that autonomic SCR increased significantly with level, whilst explicit expectancy decreased significantly with level.

In the current experiment participants experienced runs of USs and noUSs in the absence of this CS. Hence, there was no associative structure to drive changes in SCR, and now SCR is essentially flat across level. This indicates that US sensitization is not occurring in this experiment, as successive shocks are not leading to an increase in SCR as would be expected if this were happening. This suggests that US sensitization is not responsible for the SCR pattern seen in the 2012 experiment, making the case that associative processes are responsible somewhat stronger.

Supporting this analysis, a significant effect of US presence was found in the SCR data, with higher SCRs observed on measurements taken after noUS (-) trials. Therefore, there were bigger changes in SCR just after non-reinforced runs (when shocks were not occurring; see Fig. 2). We conjecture that the SCR may be subject to habituation rather than sensitization using our procedures, because exposure to shocks appears to be causing smaller rather than larger SCR fluctuations. Alternatively, it could be that any learning to the temporal cues is being expressed more in the PreCS period than the CS period, explaining the negative difference scores.

![Changes in SCR](image)

Figure 2. Graph depicting SCR as a function of run length.

Regarding expectancy, the data is more complicated. A significantly increasing effect of level was found as well as a significant US presence effect such that expectancy of shock was higher after US absent (+) trials. These two findings at first seem paradoxical, with the latter suggesting participants gave higher expectancy ratings if there had not been any shocks, while the former implies the opposite (see Fig. 3). The increase in expectancy with level is entirely driven by the US present (+) trials, and could simply reflect use of another heuristic, the "hot hand" effect (Burns & Corpus, 2004). Here we speculate that participants are simply tracking runs of shocks, and once they have had two in a row decide that the run is likely to continue. Regarding the US presence effect, participants gave higher ratings of shock after US absent trials where no USs occurred. This implies participants expected shocks more when there had not been one recently. We speculate that because participants knew that in this experiment, the only thing that would happen was that intermittent shocks would occur, as time elapsed ratings for a shock occurring would tend to increase as they knew that eventually a shock had to happen. We recorded the expectancy ratings participants made over each trial in 5 second bins (we had to exclude one bin as this varied from 5 to 15 seconds due to recording issues caused by a variable ITI). Supporting our speculation, expectancy significantly increases as time elapses between trials, \( F(1,23) = 75.41, MSE = 11.99, p < .001 \), i.e. once a participant is shocked ratings of another shock occurring are lower just after this, but as time elapses the rating increases. In some sense this is a temporal equivalent of the gambler's fallacy heuristic, but this time it is an entirely rational reflection of the sequences experienced in this experiment.

By combining these two effects we can explain the pattern of results shown in Fig. 3. The gradual increase in expectancy with time since the last shock sets the overall trend, and tracking of runs of USs explains the increasing trend for positive runs superimposed on this overall effect.

This pattern of results conflicts with our 2012 expectancy finding that expectancy of shock decreased as a function of level. Comparing both experiments, we have two very distinct patterns of responding, which suggests we have fundamentally changed the paradigm and the demand characteristics from our original experiment, leading participants to approach the task differently. Given that both SCR and expectancy show an overall decline across run length in this experiment we could even claim that our data are consistent with the conscious expectancy-driven account of SCR often found in the literature on electrodermal conditioning. What this pattern of effects cannot do, however, is explain the quite different pattern found by McAndrew et al. (2012).
We hypothesise that the crucial factor causing the difference between the results of this experiment and the 2012 experiment is the absence of the CS. In this experiment participants only experience one stimulus (the US) as opposed to two interacting stimuli (the CS and the US). But can we be sure that sensitization is not a factor in our experiment? We see two possible issues that need to be investigated. The first stems from the possibility that any change in SCR consequent on experience of shock is being expressed in the PreCS period because of timing issues. If sensitization were occurring, but manifesting in the PreCS window, then this would have the effect of driving our SCR measure down. In the McAndrew et al. (2012) experiment the CS could be used to eliminate this timing issue, and so the sensitisation would now manifest in the CS period and drive the measure up. Weidemann et al. (2009) have also proposed that the expression of US sensitization is dependent on a weakly conditioned discrete cue being present. Given this, we cannot establish that US sensitization is not driving the SCR in our 2012 experiment as matters stand. This is addressed in Experiment 2.

**Experiment 2**

This experiment uses a discrete cue to provide the correct context for the expression of US sensitization (Weidemann et al., 2009). Therefore, there are CS-US, CS-noUS, noCS-US and noUS-noUS trials in this experiment. The US sequences used were mapped to those in the previous experiments except that we added strategically placed CSs, one per run length per block. These CSs were placed on these specific runs to avoid the build up of associative strength, and alternated in terms of being reinforced or not. SCR was measured on these trials, and, due to the absence of any associative structure during the preceding run, if an increase in SCR across run length (and level) is found, US sensitization would explain this. However, if we fail to find an increasing pattern an alternative explanation for our 2012 results must be sought, perhaps an associative one.

**Method**

**Participants**

24 people participated in this experiment, all University of Exeter undergraduate students, 15 women and 9 men, ages ranging from 18 to 24 years old (average, 19 years). All participants were paid £10.

**Stimuli**

The same stimuli were used in this experiment as in the previous one. However, on the trials where a CS was presented, a brown cylinder (19 x 13cm onscreen) appeared for 5 seconds (the same as used in McAndrew et al., 2012). Participants were asked to make explicit expectancy ratings just as they were in the first experiment, every 5 seconds.

**Design**

The sequences were the same as those used in Experiment 1, however, a CS was added to 6 trials per block, one on each of the -3, -2, -1, +1, +2 and +3 runs. There is only one +3 and -3 run per block so these always had a CS. A CS was then randomly allocated to a +2, -2, +1, and -1 run. Three additional trials were inserted at the start of each block, CS-US, CS-noUS, CS-US, in order to create the weakly conditioned discrete cue. Thus, overall there were 52 trials.

**Procedure**

Participants were told that sometimes they would see a brown cylinder come on the screen. Half the time it would be followed by a shock and half the time not, but sometimes they would receive a shock when the cylinder was not there. Other procedures were as in the previous experiment.

**Results**

The SCR data was treated in much the same way as in Experiment 1, with regards to data collection and log transformation. A measure of the CR was taken for each trial on which the CS was present using the formula, CS-PreCS. A mean SCR measure was then recorded for each run length and averaged across participants. With regards to the expectancy data, the expectancy rating made during the actual CS period was taken as participants' expectancy of the US on that trial. Again, a mean rating for each run length was calculated for each participant and then across participants. Both data sets were collapsed to form the variable level (see Fig. 4).

![Experiment 2 Results](image)

Figure 4. Graph depicting changes in SCR and expectancy as functions of level.

Two-way repeated measures ANOVAs were run on the SCR and expectancy data separately. In the SCR data, there was no significant linear trend over level, $F(1,23) = 1.82$, $MSE = .023$, $p = .190$, Bayes factor = 0.31, so we have strong evidence for the null hypothesis leading us to reject US sensitization as an account for this result. Nor was there a significant difference between the US present and US absent trials or any interaction ($p > .05$). With regards to the expectancy data, there was a significantly increasing linear effect across level, $F(1,23) = 5.52$, $MSE = 2.19$, $p = .028$. Additionally, there was a significant effect of US presence, with a higher mean expectancy rating for US absent (3.69) compared to US present (2.90) trials, $F(1,23) = 39.35$, $MSE = 22.56$, $p < .001$, see Fig 5. However there was no interaction between these two variables ($p > .05$).
Discussion
Weidemann et al. (2009) proposed that in order to see the effects of US sensitization, the experimental context had to incorporate a discrete CS. Therefore, Experiment 2 aimed to do this to keep the context of the experiment similar to that of the original 2012 experiment. The CSs were strategically placed on one of each US run length, to measure UR without the build up of associative strength. In some sense our manipulation has been successful, as now the SCR changes recorded are all positive, as was the case in our 2012 experiment.

Analysis of the SCR data shows we have found another case where SCR is flat across run length and level. There is no sign of an increasing trend, which would be expected if presentation of the US is sensitizing participants. This null result strengthens the case against the nonassociative US sensitisation account as an explanation of the SCR result seen in the original Perruchet experiment. In the absence of any evidence to the contrary, we propose that associative processes are driving performance in the original 2012 paradigm, consistent with a dual processing system account of learning (McLaren, Green, & Mackintosh, 1994).

Further Results

Figure 5. Graph depicting changes in SCR and expectancy as functions of run length.

With regards to the expectancy data, despite our changes to the paradigm, we have obtained the same pattern as we found in Experiment 1. There is a significant linear increase in expectancy across level, yet an overall decrease in expectancy from US absent to US present trials. The explanation we proposed for Experiment 1 can also account for this result. Despite CSs being present in this experiment, participants are still instructed that shocks will happen intermittently throughout the experiment regardless of the presence/absence of the CS. Therefore, as time elapses participants give higher ratings for shock, which then decrease once a shock has occurred. Once again we speculate that they track runs of USs, and this effect is superimposed on the overall trend due to elapsed time.

Conclusion
In two experiments we investigated whether nonassociative US sensitization could explain the original result found in McAndrew et al.’s (2012) experiment. In both cases there was no evidence for sensitization to the US, and the pattern of results was different to that obtained in the Perruchet paradigm. We conclude that a dual processing system explanation (e.g. McLaren et al., 1994), appealing to explicit, propositional processes to explain the expectancy data as opposed to associative, autonomic accounts of the SCR data, is still the most convincing account of McAndrew et al.’s (2012) results.

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REFERENCES