Facilitation of voluntary goal-directed action by reward cue

TITLE

Using a human fear paradigm, Lovibond et al (2013) attempted to show competition between an instrumental avoidance response and a Pavlovian safety signal for association with omission of shock. Pavlovian and instrumental conditioning are two forms of associative learning. Pavlovian conditioning involves humans learning that initially neutral conditioned stimuli (CSs), such as a tone or colour, predicts an outcome (US), such as electric shock, or in the case of safety signals, safety, such as an omission of shock. Instrumental learning refers to learning associations between voluntary responses (such as a button press, or an ‘ avoidance’ response) and outcomes or ‘ reinforcers’, such as shock or an omission of shock. In their first, overshadowing, experiment, expectancy data but not skin conductance levels (SCLs) suggested mutual overshadowing, as when the avoidance response (button press, *) and safety signal (C) were both presented with stimulus A, expectancy of shock was significantly lower than when A was only presented with the avoidance response or safety signal. In the second, blocking, experiment, no matter whether the avoidance response or C was pre-trained, the pre-trained element yielded the lowest expectancies of shock (i. e. greater safety learning), while safety learning of the alternate element was suppressed. Lovibond et al (2013) conclude that the expectancy data, as well as the non-significant SCL data, in the blocking and overshadowing paradigms exhibit evidence that competition occurred between the instrumental avoidance response and Pavlovian safety signal, and therefore a common learning mechanism underlies both forms of

associative learning. In this paper, Lovibond et al's (2013) experiments, and their conclusions, shall be critiqued.

Strengths

Lovibond et al (2013) exhibited considerable strength in the planning of their experiments. In both experiments, they used a variation of a previously used paradigm, such that their experiments already had relatively sound internal consistency and construct validity. They had the foresight to acknowledge the possibility that participants would learn a response-stimulus-outcome relationship rather than viewing the avoidance response and safety stimulus C as separate predictors. That is, they saw a potential weakness in their experimental design in that C could become a mediator of the causal efficacy of the avoidance response, rather than a competing cause. As such, in both experiments, they deliberately adjusted their design in order to prevent this by adding BC- trials and varying the time interval between the avoidance response and safety signal, to weaken the response-stimulus C association. They additionally asked participants to rate the degree of association between them, as well as with shock, so that they would know if response-stimulus-outcome learning had nonetheless occurred. Lovibond et al (2013) used previous research in order to resolve potential issues that could arise before running their experiment. For example, they doubled the number of B- trials in the pre-training phase because a prior study of theirs showed that predictors of no shock are more slowly learnt than predictors of shock, and they needed to ensure differential conditioning to stimuli A and B had occurred. Furthermore, aware that C being novel could be more anxiety-provoking and hence confound results by resulting in more conservative

expectancy ratings and a higher SCL, Lovibond et al (2013) ensured that the first trial of the compound phase was always a BC- trial to reduce the novelty of C before it was paired with stimulus A. They acknowledged , in experiment 1, the possibility of participants having never experienced a trial with just the instrumental response or just the safety signal before the test phase, and thus participants may have been more conservative in their judgments, and account for this through directly evaluating competition via a blocking paradigm in experiment 2 where one group pre-trained Pavlovian (AC- trials) and the other pretrained (A* (+)) to ensure wasn't just conservative ratings etcetera

Lovibond et al (2013) also exhibited strength in their rigorously controlled experimental design. The use of headphones constantly emitting white noise (except when the tone stimulus was presented), ensured safety signal-shock learning was not confounded by external, extraneous sounds. The 180 degree rotary dial presented a more accurate measure of expectancy than a typical Likert 1-10 confidence scale. Lovibond et al (2013) used inter-trial intervals to ensure adequate time between trials to prevent confusion, to ensure shock was paired with the correct stimulus (A or B), and to allow SCL to return to baseline levels. Furthermore, they used Bonferroni correction to control for the extra possibility of type I error from using two measurements (expectancy and SCL data).

In terms of theoretical strengths, Lovibond et al (2013) attempted to explain unexpected results; and provide alternate explanations for expectancy data. In experiment 1, they excuse the lack of difference in expectancy to shock between A+ and B- trials in the pre-training phase, by explaining that across

the remainder of the experiment, there was a significant difference in expectancies between the two (that is, differential conditioning occurred, it simply took longer than they expected). In experiment 1, they also provided an explanation for SCL unexpectedly increasing in the compound phase from trial 1 to trial 2, explaining that only 37% of participants made an instrumental response on the first trial, so that most participants received a shock then (so SCL would have been higher for trial 2 as they would be more anxious about being shocked), and from trial 2 onwards SCL declined appropriately. In experiment 1, they provided an alternate explanation for the expectancy data, by claiming that it may have just been the novelty of A*- and AC- (that is, the novelty of testing the avoidance response and safety signal individually) that may have lead to the more conservative expectancy ratings when they were presented individually compared to when in conjunction. That is, they highlighted that it may not have been mutual overshadowing or competition that lead to lowered shock expectancies when in conjunction compared to when elements were presented individually, but rather an effect of novelty. This retained a sense of objectivity that is often forgotten in psychological reports which are determined to present their findings as definitive conclusions. Furthermore, while they do not bring this argument up, it is clear that this was not the case based on similar expectancy data from the blocking paradigm in Experiment 2, where either A* or AC- were pre-trained (that is, they were not novel in the test phase), and similar results emerged. They conclude by mentioning that the evidence of a single learning mechanism found in the paper is preliminary, not definitive, which is a strength as it highlights the need for repetition and an

accumulation of more data to prove without a doubt that there is a single learning mechanism – Lovibond et al (2013) do not make any assumptions.

This is furthered by their outline of limitations in their own experiment – by attempting an objective evaluation of their own experiment, a practice which is sometimes forgotten by psychologists who wish to convince their readers of their findings. As they highlight, the strongest evidence for competition was a cross-experiment comparison. They attempt to dismiss this limitation by saying that the same participant pool was used, with the same equipment, experimenter and same time frame, and that the common trials (A+ and B-) gave highly congruent data, suggesting that the test phases could be directly compared across experiments. Nonetheless, they acknowledge that a within-subjects design would be better. They highlight the limitation that only the expectancy measure yielded significant effects, but attempt to excuse this by explaining that autonomic conditioning results are often insignificant due to large individual differences which inflate the error term and reduce power.

Weaknesses

Unfortunately, Lovibond et al (2013)’s design had some flaws. Although they added BC- trials and varied time intervals between the avoidance response and presentation of safety signal C to ensure the avoidance response and stimulus C were independent, competing causes of shock, the post-experiment questionnaires where participants rated the degree of relationship between the two revealed that they were aware of a relationship between them. This means that the results (the lowered expectancies to

shock when the avoidance response and safety signal were presented together, than when presented individually), which Lovibond et al (2013) saw as evidence for competition between an avoidance response and safety signal (and thus evidence for a single learning mechanism) may have simply occurred as the safety signal C, as a mediator of causal efficacy of the avoidance response, would have resulted in lower expectancy of shock when combined with the avoidance response, than when they were separate (no competition necessary), whether in the blocking or overshadowing paradigm. Lovibond et al (2013) failed to discuss this, brushing it off as an intrinsic problem when there are voluntary responses. Continuing, while not the most ethical option, conditioning may have been more robust (in particular, SCL results may have been significant) if the level of shock selected for participants was manageably painful instead of just uncomfortable. This is because more variability in SCL would have emerged as participants would have been more anxious. The highly constructed laboratory setting, where they deliberately presented twice as many B- trials, and made as many adjustments as possible to find significant results, begs the question as to how often competition between avoidance responses and safety signals occurs in real life, and whether the single mechanism of learning proposed by Lovibond et al (2013) really exists or is just a fabrication of the laboratory procedures used. Furthermore, humans are quite intelligent: by giving them instructions telling them that pressing a button or hearing a tone ‘ may or may not’ effect an outcome, it would be much easier for them to gain an accurate perception of expectancy of shock, particularly if they were undergraduate psychology students, which they probably were, and this may have confounded the results by lowering the expectancies in significant

amounts accordingly – that is, rather than genuine competition, participants may have just believed that there were connections from the instructions given, that there was less chance of shock when a button press or tone, and in conjunction, there was the least chance.

Continuing, Lovibond et al (2013) claim, in their first experiment, that they had 53 participants, and in their second experiment, 89 participants, but after exclusions, the sample sizes of these experiments were 30 and 57 respectively. While they still had significant expectancy data, Lovibond et al (2013) should have specified more accurately the number of participants in each experiment. Furthermore, if they had had a larger sample size, they may have found significant SCL results due to greater power.

Lovibond et al (2013), make faulty conclusions regarding SCL data. They conclude that the SCL data pattern mirrors that of the expectancy data across both experiments. However, as the SCL results were not significant, it is inappropriate to conclude this, as there is a higher probability that any mirrored ‘ pattern’ could be the result of chance alone. Statistically speaking, if the SCL data was not significant, than no real differences between the instrumental response and safety signal tested individually versus together have been found. Furthermore, Lovibond et al (2013) brush off the lack of findings in SCL data by claiming that the SCL measure is unreliable. However, it must be asked then, why Lovibond et al (2013) used such a measure in the first place if it is so ‘ unreliable’. They claim that SCL have greater individual variability and greater sensitivity to extraneous factors and that is why there were no significant results, but in real life, those extraneous factors are bound to interfere, and if there were non-significant

results with such factors, one must ask how applicable a single learning mechanism approach is. Granted, it could be argued that Lovibond et al (2013) is a highly theoretical paper by nature, interested in modeling conditioned learning (by claiming a single underlying mechanism defines conditioned learning structure), rather than application. However, one must ask how relevant or important a model could be if it does not have any external validity.

Lovibond et al (2013), furthermore, make assumptions in their conclusions. They fail to explain why it follows that because there seems to be a common associative mechanism that the critical association in instrumental learning is an R-O association in order to explain competition with a Pavlovian S-O association. They do not attempt to explain why, in their cross-experiment comparison, expectancy measure responding in the blocked condition was significantly higher than in the overshadowing condition. Continuing, they assume that if there is a single-learning mechanism, it must be propositional by nature. This is problematic, because while the common thought among single-learning mechanism theorists is that the mechanism is propositional, Lovibond et al (2013) do not explain how their experiment exhibits a propositional mechanism. Even if they have provided evidence for a single-learning mechanism, they have not provided evidence regarding the nature of this single-learning mechanism. Propositional accounts claim that associative learning depends on effortful, attention-demanding reasoning processes. However, one must ask which part of this experiment showed that learning was an effortful process. Continuing, propositional models are faulty. Propositional accounts of learning fail to align with animal and

developmental psychology. Non-human animals exhibit associative learning, although they do not have the language to deploy propositions to infer relations about events. “ If p, then q” (or contingency) propositions, are not understood until children are 6years old. However, despite lacking the language abilities and contingency propositions to infer relations about events, backward blocking and other evidence of associative learning has been shown in children as young as 8 months. AsXclaims, there is not enough evidence to justify structured mental representations existing when associative learning occurs (i. e. a propositional model), over a broad, non-propositional associative link between representations.

In their introduction, Lovibond et al (2013) are pedantic with their definitions in their introduction when explaining how Pavlovian and instrumental learning could be separate mechanisms. They differentiate between performance and learning claiming that Pavlovian performance is involuntary while instrumental responses are voluntary, but that does not mean they are not learnt the same way. However, if they are to be differentiated, as Lovibond et al (2013) do, whether in their experiment they are actually measuring an underlying mechanism or performance in the test phase, as generated expectancies could simply be another measure of performance – their anxiety levels (CR) conditioned to the safety signal or avoidance response. Continuing, they claim that the notation E1 and E2, where E1 could be a stimulus (Pavlovian) or action (Instrumental conditioning), and where E2 is the outcome, reinforces the notion that a single learning mechanism may underlie both types of associative learning. However, this is simply induced notation. Equally, one could use the notation S-S for

Pavlovian learning (the CS-US link, hence S-S), and R-O for instrumental learning (the response-outcome relationship), to portray them as separate learning mechanisms, and to support a dual-process model. Thus, Lovibond et al's (2013) proposal of a single learning mechanism is largely based on unfounded claims.

Furthermore, in their introduction, while Lovibond et al (2013) attempt to provide evidence for a single-learning mechanism, evidence can also be provided for a dual-process model. For example, a single learning mechanism assumes awareness is required for conditioning. However, Baeyens et al 1990 found flavour-flavour learning occurred in absence of any contingency awareness. Continuing, in Perruchet's task where a tone was either paired with an air-puff or was presented alone, when the tone and airpuff had recently been paired together, expectancy of an air puff on the next trial was reduced, the probability of an eyeblink CR occurring was heightened. Furthermore, neurological data suggests different brain regions are involved in different learning processes, for example, the amygdala plays a large role in fear conditioning. Therefore, it is possible that instrumental and pavlovian are equally run by different parts of the brain. Lovibond et al (2013) did not actually provide evidence against such a model. For example, they could have argued against the dual-process model by claiming that the dissociation between the eyeblink CR and expectancy when CS-US pairings have recently been presented in the Perruchet task, which some learning theorists use to support the dual-process model, that the eyeblink CR results from sensitisation from recent US presentation (a recent air puff). Alternately, they could counter-argue that while the amygdala has a large

part in fear learning, it could simply be a subcomponent of a broader, singular system of learning. It would have been a more convincing argument that the experiments were necessary and that a single learning mechanism were possible if they had had more depth in the lead up to their hypotheses.

Conclusion

Lovibond et al (2013) claim from their experiments that a single learning mechanism underlies Pavlovian and instrumental conditioning. However, despite their attempts to remain objective and their rigorous planning and control of their experiment, they fail to address vital problems to their experiment (such as the possibility of the safety signal being a mediator for the efficacy of the avoidance response), assume, without sufficient evidence, that if a single learning mechanism underlies both types of associative learning, it must be propositional in nature (a faulty assumption), speak of SCL data as if it were significant when it was not, and in the lead-up to their hypotheses regarding a single learning mehcanism, fail to dismiss the possibility of a dual-process model.

Reference

Lovibond, P. F. and Colagiuri, B., 2013. Facilitation of voluntary goal-directed action by reward cues. *Psychological Science* , *24* (10), pp. 2030-2037.