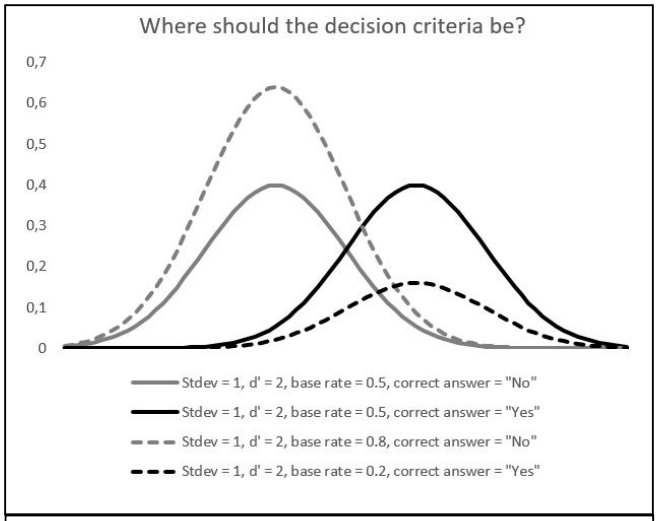
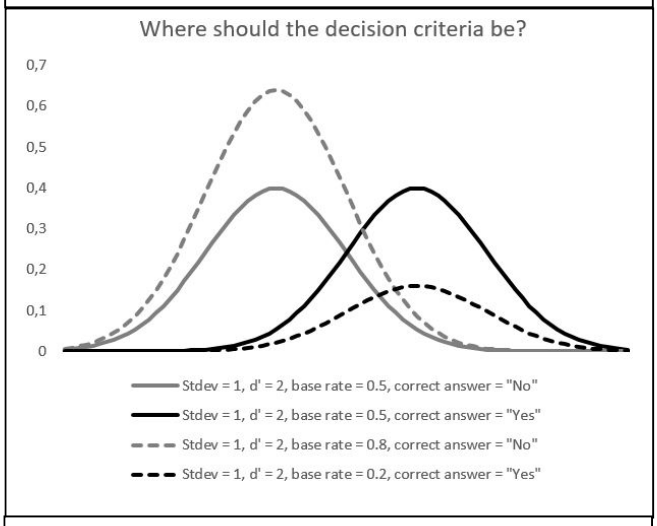
PSYPRO4412

Langsvarsspørsmål (Velg to av tre spørsmål, hvert av disse to spørsmålene teller 40% av endelig karakter)

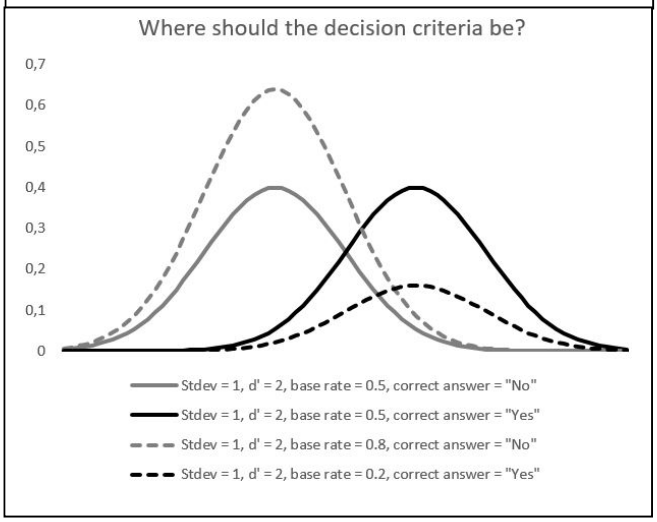
1. Anta at normalfordelingene vist i første figuren representerer høyde for kvinner (venstre, grå linje) og for menn (høyre, svart linje). Du får betalt 1kr hver gang du svarer korrekt på spørsmålet "Er denne personen en mann?", gitt at den eneste informasjonen du har om vedkommende er høyde. De hele linjene viser tilfeller der kvinner og menn står for lik andel av den totale populasjonen (grunnfrekvensen = 0.5 hver). De stripete linjene viser tilfeller der det er fire kvinner for hver mann (grunnfrekvensen = 0.8 for kvinner og 0.2 for menn). Forklar med utgangspunkt i figuren hvor beslutningskriterium bør være? Ingen kalkulering er nødvendig for besvarelsen.



Bruk andre figuren for å forklare hvor beslutningskriterium bør være når du igjen har fire kvinner for hver mann, men nå sammenligner du tilfeller med samme standardavvik som i forrige figur (stripete linjer, d' = 2) og de tilfeller der du har mindre gode mål på høyde, med større standardavvik, og derfor også dårligere evne til å skille disse (hele linjer, d' = 1,33)? Hva forteller dette deg om forholdet mellom d' og den optimale lokaliseringen av beslutningskriteriumet når det optimale beslutningskriteriumet utviser systematiske skjevhet?



Til slutt, anta at du har gode mål for begge sammenligninger, men en av dine sammenligninger er for en populasjon av relativt høye kvinner og relativt lave menn (hele linjer, d' = 1.33) som vist i tredje figur.



Sammenlign de optimale beslutningskriteria for den lette distinksjonen (d' = 2) og for den vanskelige (d'= 1.33).

Hvor ville det optimale beslutningskriteriet være hvis grunnfrekvensene var like, men du fikk betalt 4kr for hver kvinne du identifiserte korrekt, og fremdeles kun 1kr for hver mann? Kan du se noe relevans til McKay sin argument knyttet til den kombinerte underskudds og motivasjons forklaringen for vrangforestillinger? Hvordan kan forståelse av optimale beslutningskriterier ellers brukes i noen annen applikasjon til psykologi?

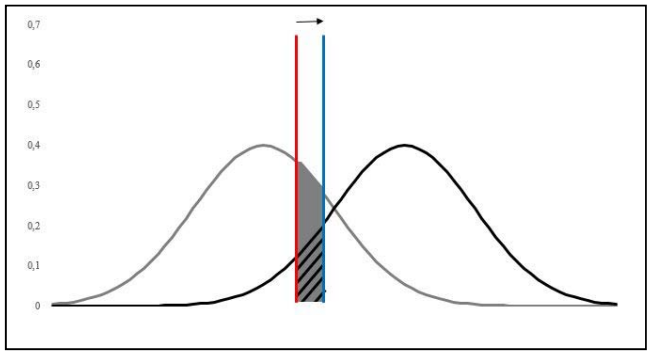
2. Gi et eksempel for hver av underskuddsmodellene (deficit model) for vrangforestillinger, ett for motivasjonsmodellen og ett eksempel for kombinasjonsmodellen. McKay et al. argumenterer for at enkelte vrangforestillinger kun kan forklares gjennom en kombinasjon av underskudd og motivasjonsmodellen. Kan du bedømme hvorvidt dette er alltid tilfellet eller hvorvidt noen vrangforestillinger kan forklares utelukkende ved bruk av underskudds eller motivasjonsmodellen?

3. Hvor pålitelig er minnet, og hvilke mekanismer kjenner du til som kan produsere falske minner?

Short questions (Choose one of two, each answer counts as 20% of the final grade)

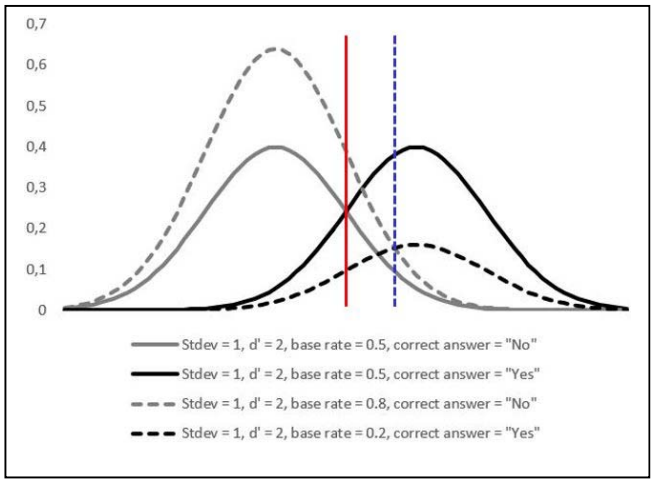
1. Hva er fremovermodellering (forward modelling), og hva kan skje hvis det går galt?
2. Hva er prediksjonsfeilens rolle i rekonsolidering?

Sensorveiledning oppg 1: Lynn and Barrett provide the equations to answer this question, but here I outline a purely graphical method which requires no calculation. Only two ideas are needed to work out the answer: first, that the area under each curve can be proportional to the base rate, the payoffs, or both; and second, that when the criterion is moved, then the change in false alarms (false positives) and correct rejections (true negatives) is proportional to the height of the left curve at the location of the decision threshold, while the change in misses (false negatives) and hits (true positives) is proportional to the height of the right curve at the decision threshold.



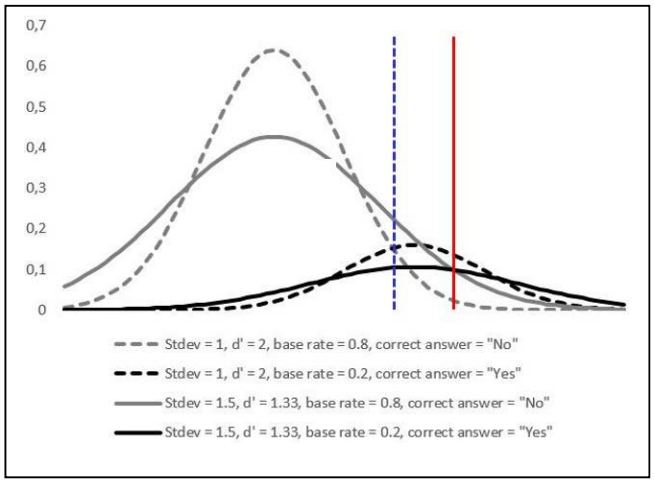
Taking the continuous lines in the first figure of the question, base rates are equal, and the areas under the two curves are equal. So if the decision criterion is moved from the location marked by the red line on the left to the blue line further right, then the gain in the number of women correctly identified in the example problem (true negatives to the question "is this a man?") is proportional to the grey area. The loss in the number of men correctly identified (true positives) is proportional to the cross-hatched area, and is smaller in this example. This movement in the decision threshold should increase the total payoff. If the movement of the decision threshold is reduced to infinitesimally small, the point where the gains in one kind of correct decision equal the losses in the other kind of correct decision is where the two curves cross. Beyond this point, further movement to the right would result in more losses of true positives than gains of true negatives. Therefore the optimal location for the decision threshold is where the two curves cross. When base rates and standard deviations are equal, that is exactly in the middle between the peaks of the two curves.

If payoffs remain symmetrical, and the areas under the curves represent base rates, the same argument still says that the optimal location for the decision threshold is where the two curves cross. However, that is no longer exactly in the middle between the peaks of the two curves, represented here by the red, continuous line. The optimal decision threshold is biased towards saying someone is female, represented here by the blue, stippled line.



If base rates are equal, but the areas under the curve present payoffs, the same argument applies. So if instead of having four times as many women as men in the poplation we have equal numbers, but the payoff for correctly identifying a woman is four times the payoff for correctly identifying a man, that can be represented by making the curve for women four times as high as that for men, and the optimal location for the decision threshold is in exactly the same location.

The next part of the question concerns the effects of different d' when base rates (or payoffs) are unequal and the optimal decision threshold is therefore biased. The example graph given shows that when d' is smaller because standard deviations are larger, the point where the curves cross is further out to the side (red, continuous line), and so the optimal decision threshold becomes more biased.



The same happens when d' becomes smaller because the peaks of the curves are closer together. So if the optimal decision threshold is biased, then the more difficult it is to distinguish the two populations, the more biased the optimal decision threshold will be.

That suggests an application to McKay et al.'s idea that many delusions are caused by a combination of motivation and deficit. One of the things that can create a very biased decision threshold is asymmetric payoffs. Therefore someone who believes that the cost of missing a threat is very high (a quite reasonable assessment for a member of a persecuted minority, or someone with an experience of abuse) should have a decision threshold biased to see threats. That assessment of costs can be seen as a motivational factor. If, in addition, this person also has a poor ability to distinguish people who are threats from those who are not, meaning that d' is low, then bias should be more extreme, possibly to the point where it is considered delusional. The reduced discrimination, meaning low d', would be the deficit. It is not clear whether this line of reasoning can be applied to all delusions.

Sensorveiledning oppg 2: An example of a purely motivational account of a delusion is the proposal that the Fregoli delusion, mistaking a stranger for a familiar person, such as the patient's dead father, is the result only of a strong wish that the father were still alive. Signal detection theory can be used to illustrate that a change in the subjective evaluations of the consequences of true and false positives and negatives also changes the where the optimal decision criterion is. Extreme evaluations lead to extreme biases, which may be dysfunctional enough to be called delusions. Higher incidence of persecutory delusions in members of disadvantaged groups is a plausible candidate for that kind of motivational factor, seeing that these people are more likely to be discriminated against, and missing a plot against them may have more severe consequences.

However, McKay et al. first propose that that there may also be cognitive deficits. For example, if people's voices seem to be fainter than they used to be, it could be that one's hearing is failing, or that people are whispering because they are conspiring. Someone may wish to believe that their faculties are not failing. That could be a motivational factor. If the patient needs to turn up the volume on media, if bird song seems fainter, and rainfall, and all sorts of

other sounds that can be expected to have remained as loud as they used to be, then believing in the conspiracy is implausible, and evidence for a cognitive deficit as well.

Second, McKay et al. propose that the existence of a second factor can be inferred if there are people who have a deficit that is associated with a delusion, but they lack that delusion. If the people who suffer from the delusion share both the first and a second factor, and the second factor alone is not enough to produce the delusion, then both factors are needed. (This translates to an interaction between first and second factor.)

Two factor accounts of the Fregoli and the Capgras delusion can differ in what kinds of factors they depend on. For the Fregoli delusion a first deficit in face processing leads to exaggerated feelings of familiarity and affect, and in the Capgras delusion a lack of familiarity and affect. The second factor in both is a deficit in belief evaluation, allowing bizarre beliefs to gain credence. These would be examples of two cognitive deficits.

Frith's explanation of delusions of control depends on one clearly cognitive deficit, the overestimation of prediction error, and a second factor, the overattribution of agency that could be argued to be either a cognitive deficit or a motivational factor. Deciding that would need more information on attribution of agency.

McKay et al. say that persecutory delusions are associated with two factors, namely high overt and low covert self esteem, and high need for closure (some facets only). They describe both as motivational factors.

In summary, McKay et al. list two factor accounts of several delusions. Adding in McKay's account of persecutory delusions following from deafness, and one interpretation of Frith's account of delusions of control, the proposals include accounts proposing two cognitive impairments, one cognitive and one motivational factor, or two cognitive factors.

Although McKay et al. only discuss theories with up to two factors, there is no guarantee that this is universal. There may well be delusions that are caused by a single factor, and others that only occur when more than two factors coincide.

Sensorveiledning oppg 3: Geraerts et al. divided memories of childhood sexual abuse into continuously remembered (there was never a time when the person reporting abuse had forgotten the abuse) and discontinuous, and they divided the discontinuous memories into those remembered within or outside of therapy. Geraerts et al. do not mention whether the therapy in question was specifically recovered memory therapy or a broader range of therapies. In this study, *none* of the memories recovered in therapy was corroborated by independent evidence, while memories recovered out of therapy were as likely to corroborated as continuous memories (those that had never been forgotten). The best predictor of the existence of corroborating evidence was being surprised by the memory. Memories that were corroborated also tended to be recovered suddenly, rather than gradually.

Shaw and Porter interviewed subjects three times, asking them both about an emotionally salient event that (according to the subjects' parents) they really had experienced, and about a

fictitious crime. Shaw and Porter were able to persuade 70% of subjects that they had committed a crime serious enough that police had gotten involved. Their interviews used tactics that had elicited false confessions in previous research, such as presenting false evidence, social pressure, building rapport, suggestive retrieval techniques including guided imagery, leaving pauses for the subjects to fill, and implying that experimenters knew further details.

The finding that corroborated discontinuous memories were recovered more suddenly and were more surprising than uncorroborated (and therefore more likely false) memories suggests that false memories are built up slowly. There are three candidate mechanisms within the pensum. One is reconsolidation. Hupbach et al demonstrated that episodic memory can be altered through reconsolidation. They showed people a list of objects on a Monday. On Wednesday, the reconsolidation group was asked to remember what they had done on Monday. Then they were shown a second set of objects. One control group saw the same second set of objects, but without being reminded of the previous set. Those two groups returned on Friday, joining the second control group. The experimental group encoded more Wednesday objects along with the Monday objects of which they had been reminded than the control group without the reminder. The Monday & Friday only group provided a baseline of spontaneous intrusions without exposure to the Wednesday objects. The intrusions were asymmetric. The experimental group did not include Monday items in their recall of the Wednesday items. The increased intrusions were of Wednesday items seen at a time when the Monday items were re-encoded after the reminder. Reconsolidation this provides a mechanism by which memories may altered, rather than merely new memories being established that may compete with the old ones. That matters in the context of false memories. Depending on the circumstances in which a false memory is established, the original information may be overwritten.

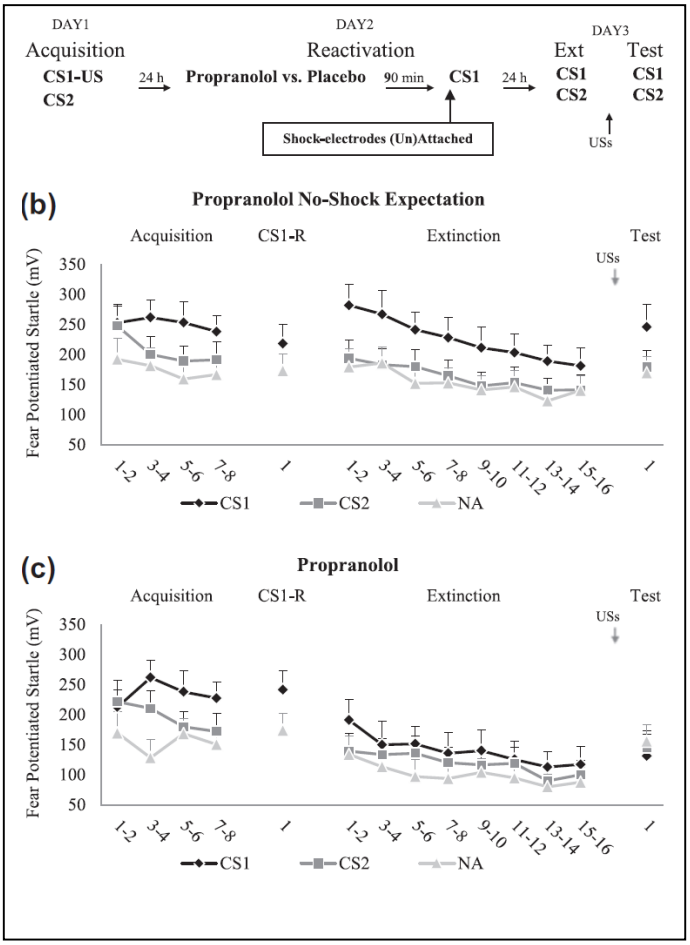
An alternative method of altering an existing memory is to change the probabilistic relationship between a conditioned and an unconditioned stimulus gradually. The theory behind this approach is that a sudden change in the relationship between conditioned and unconditioned stimulus acts like a context change and triggers new learning, represented in a separate, parallel association. Gradual change in the relationship targets the original association. It is unclear whether this would generalise from conditioning to episodic or semantic memory.

A sudden change in the CS-US relationship is more likely to generate new, parallel associations. In the case of conditioning, there would be no explicit information regarding the source of the information, and it would be impossible to tell directly which is the original memory.

The pensum in this course does not contain any studies that attempt to establish how much each of these mechanisms may contribute to any particular false memory. All that matters here is that students understand the distinct mechanisms, that two of them modify an original memory, while one establishes a new memory that is stored in parallel with the old.

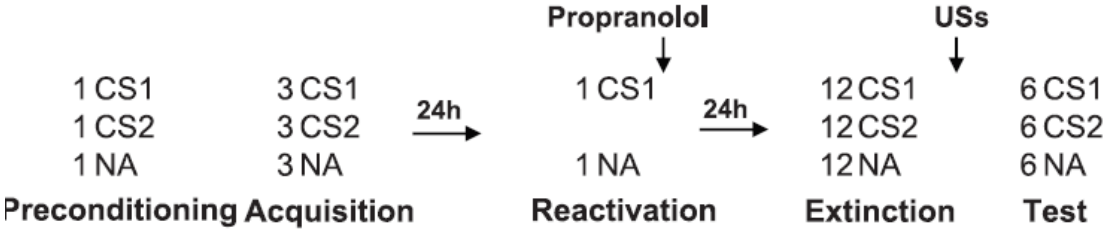
Sensorveiledning oppg 1: Forward modelling generates a prediction of the consequences of body movements, predicting both the trajectory of effectors (forward dynamic model) and the sensory consequences of actions (forward output model). The function of the forward output model is to separate sensory input caused by events in the environment from sensory input caused by one's own actions. The method is to compare an efference copy (copy of motor commands) is sensory feedback, usually by subtraction of predicted sensory information from perceived information. What remains is then perceived as caused by external events. If the forward modelling goes wrong, one's own movements may feel as if not under one's own control. That wrong perception classifies as a hallucination. In Frith's theory, that hallucination is one necessary condition for delusions of control. It is not a sufficient condition. The other necessary condition is a low threshold for attribution of agency. Students are not required to discuss that second condition in depth.

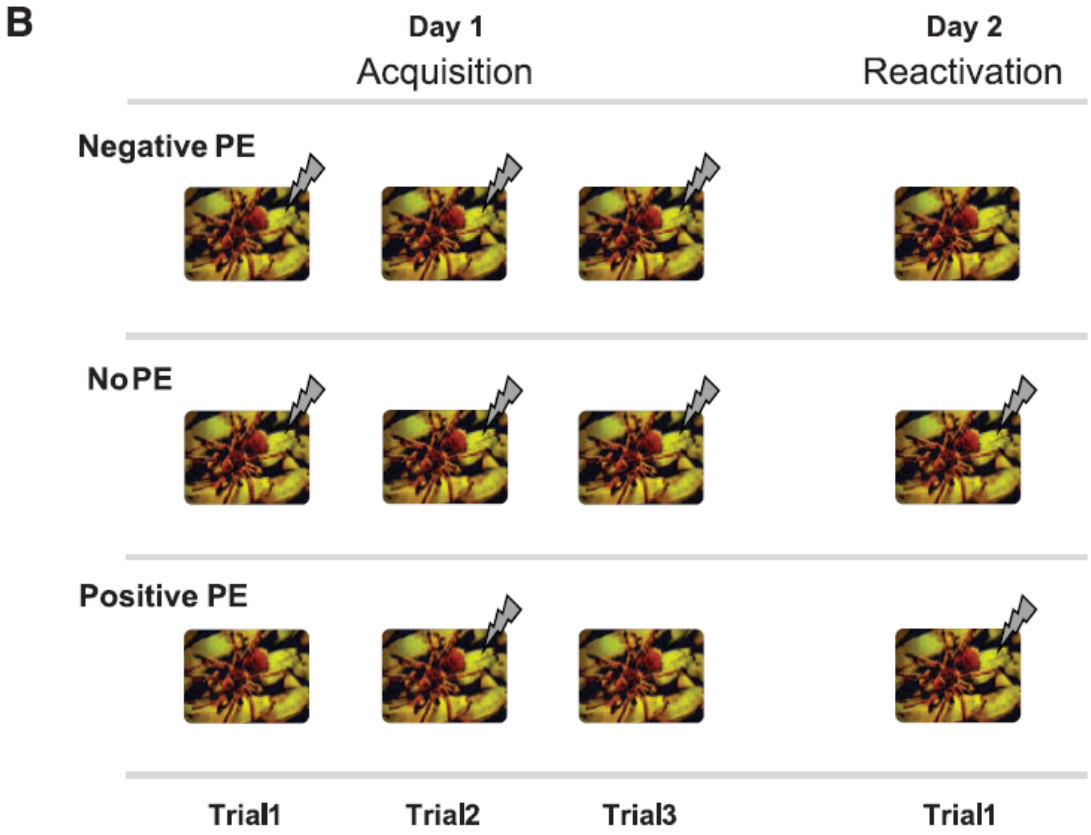
Sensorveiledning oppg 2: Reconsolidation occurs when being reminded of some information in memory makes that information easier to change during a reconsolidation time window. It occurs in multiple different memory systems. In associative learning, there is evidence that a reminder alone is not enough to trigger reconsolidation, that the association only becomes labile if the conditioned stimulus is not followed by its usual consequence during the reminder. Agren's review provides a verbal description of Sevenster et al.'s (2012) experimental design. In fear conditioning, administration of propranolol combined with a reminder was previously found to selectively reduce the emotional response to a CS. In Sevenster et al.'s study, this was true only for participants who were connected to the electrodes that had previously delivered electric shock. People who were not connected, and who had no reason to expect any shock, and who therefore experienced no prediction error when shock was absent during the reminder, behaved like the placebo group. Their fear potentiated start did not decrease from the end of acquisition to the beginning of extinction. More importantly, the group without shock expectation and thus no prediction error showed reinstatement after being shocked again, which indicates that the original association still



existed, and had only been masked by an inhibitory association created during extinction training.

A second study by Sevenster et al. (2013) is not described in Agren's review, but students may remember it from the lecture. Initial fear conditioning involved three presentations of a spider picture, with either one or three shocks in those three trials. A no prediction error group received a shock during the reminder trial, which was expected given the 100% reinforcement during acquisition. Negative prediction error came from the omission of an expected shock, positive prediction error came from a shock when past reinforcement history (spider picture followed by shock only one time out of three) indicated that no shock was more likely.





Although explicit expectation of shock depended on its past frequency, fear potentiated startle, the measure of emotional response, depended only on whether there was prediction error, not whether it was positive or negative. In the presence of propranolol, either prediction error led to an immediate decrease of fear potentiated startle after the reminder trial at the beginning of extinction, and after extinction training no reinstatement after being exposed to shock.

There is good evidence for prediction error being necessary for a reminder to make an association labile. It is not clear what prediction error even means in episodic memory. There, a reminder of a previously learned list can make the memory of that list vulnerable to intrusion errors from a newly learned list, consistent with the idea that the remembered list is being re-encoded while the new list is being learned, and so new list items are added to the memory of the old list. But what would be the prediction error in this situation? So the relevance of prediction error has been demonstrated when reconsolidating associative learning, so far specifically fear conditioning, but it is unclear whether prediction error is relevant to reconsolidation in all memory systems.