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Understanding individual variations in human associative learning

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Abstract
Associative learning has offered vital insights into psychopathology. However, illness exists on a continuum, and identifying disturbances in associative learning processes related to psychopathology demonstrates a general adaptability in human associative learning. A few studies have looked mainly at individual variations in human associative learning. Yet, while much work has focused on accounting for adaptability in learning caused by external factors, there has been little consideration of how to model the impact of dispositional factors. This review examines the spectrum of individual differences investigated in human associative learning, as well as attempts to understand and model this adaptability. To completely comprehend human associative learning, additional research must focus on the sources of diversity in human learning.

Keywords: Association learning, individual variations, depression, psychopathology, perceptual processing

Introduction
Individual variations in the human population have been studied to improve understanding of anything from academic achievement to crime and delinquency, money and poverty, and health (Lubinski, 2000) [40]. Individual variations in human learning have helped us understand the mechanisms behind psychopathology, primarily because learning identifies a process and, thus, a method by which individuals may differ. Because psychopathology traits vary across the population, our study of the relationship between psychopathology and disruptions in association learning processes may reveal significant information on the nature and breadth of diversity in human associative learning. While proof that people do not all learn in the same way has been used to assist us in comprehending aspects of psychopathology, this investigation of adaptability in human learning must be integrated into our overall understanding of learning strategies so that models can account for the factors that cause variance in learning. Investigating individual variations in all areas of associative learning would be an overly broad scope for this review. This research examines diversity in learning about stimulus pairings in order to focus on individual variations. Specifically, this paper gives a variety of instances indicating individual variations in learning selectivity and proclivity to learn about certain elements or combinations, as well as how theories of associative learning might account for this variation.

Associative learning theorists explore the acquisition and utilisation of connections between stimuli representations to interpret behaviour. Much of this research focuses on which factors affect learning and how they do so. The fundamental framework of error prediction learning, illustrated in Equation (1), gives us an idea of numerous elements that could influence learning. Rescorla and Wagner described this equation in 1972. $\Delta Vn = \alpha n \times \beta \times (\lambda - \sum V)$ (1)

This equation illustrates the change in associative intensity of a stimulus (ΔVn) as a function of prediction error, which is the difference between the expected and actual outcomes of a particular stimulus. The prediction error is calculated as the difference between the asymptote of learning (λ), the total associative intensity that the unconditioned stimulus (US)

may support, and the current associative intensity of all stimuli on the trial. The prediction error is compounded by the salience or strength of the stimulus (α) and the US (β).

Research has explored the impact of varying stimulus strength and salience (α) on learning (Logan, 1954; Perkins, 1953; Redhead & Pearce, 1995) [39, 70, 74]. There has also been considerable discussion of how focus shifts between different stimuli can impact learning (Pearce & Hall, 1980; Mackintosh, 1975; McLaren *et al*., 2010; de Wit & Dickinson, 2009; Harris & Livesey, 2010; Le Pelley & McLaren, 2004; Lubow, 2010) [67, 46, 53, 14, 24, 36, 42], as well as how previous experiences may influence the acquisition of new stimulus representations and their associations (Seligman, 1972; Kamin, 1968; Lubow *et al*., 1976) [80, 28, 44]. This review examines whether these elements are constant across the population or whether their impact on learning varies by individual. Because much of the research on individual variations in human associative learning is related to psychopathology, this review draws mainly on examples from clinically focused studies. The research presented here shows significant individual variability in key components of associative learning. The article ended with a brief discussion of how theories of associative learning can account for observed individual variations.

Stimulus Salience and Selective Prediction Error

Individual variations in perceptions of what is essential may influence association formation. The efficacy of associative learning increases with stimulus salience (Kamin & Schaub, 1963; Kamin & Brimer, 1963) [31, 30]. For example, if two stimuli of varying salience coexist, the more salient stimulus should develop stronger stimulus-outcome linkages (Mackintosh, 1971; Kamin, 1969) [45, 29]. Additionally, the potency of associative learning has been linked to that of the unconditioned stimulus (US; Pavlov, 1927) [63]. For example, conditioned response to shock in rabbits was found to be proportional to the shock's severity in the US (Smith, 1968) [82]. To summarise, with a somewhat simple example, a child playing with a toy could discover that pressing a lever on the object activates a light. The apparent intensity or salience of the light (the outcome of the behaviour) affects the associative intensity that can be maintained. The apparent kinaesthetic experience of touching the lever (the strength or salience of the stimulus) will also impact the learning degree. Variation in what people perceive as salient should have a significant impact on association acquisition, and it could result in disparities in associative learning in anxiety and depression.

Depression has been linked to a preference for negative information (Mogg *et al*., 1995; Gotlib *et al*., 2004; Bradley *et al*., 1997; Matthews *et al*., 1995; Rusting, 1998, 1999; Phillips *et al*., 2010; Chan *et al*., 2007) [56, 20, 9, 50, 77, 78, 71, 12]. This should affect the associations learnt. Salient stimuli will be used to learn to the detriment of less salient stimuli (Mackintosh, 1971) [45]. As a result, if those suffering from or at risk of developing depression find negative information more prominent, they are more likely to make associations with negative stimuli rather than good or neutral stimuli.

When learning happens, the amount of learning that may be maintained is determined by the strength of the consequence or unconditioned stimulus (Rescorla & Wagner, 1972) [76]. As in the case of the child playing with a toy, the link between pressing the leaver and the outcome of the light going on may be determined by both the brightness of the light and the child's interest in the lights. If the child shows little interest in lights, we may conclude that the lights perceived salience is restricted for that child. In this instance, the light's ability to facilitate learning should be minimised. Applying this logic to those suffering from depression, we can see how a propensity to find adverse information more salient may raise the perceived prominence of unfavourable outcomes. This should enhance adverse outcomes, allowing for a more substantial accumulation of associative strength. This could lead to those suffering from depression developing strong links between stimuli and unfavourable outcomes, increasing subsequent negative expectations. As a result, the inclination to prioritise negative information may reinforce the expectation of unfavourable outcomes.

Similar discrepancies in stimulus perception may characterise anxiety-related fear conditioning. Enhanced fear conditioning may play an essential role in anxiety disorders (Mineka & Zinbarg, 2006; Craske *et al*., 2006) [54, 13]. Variations in the perceived strength of a frightened stimulus may explain variations in how easily fear associations are learnt or maintained (Otto *et al*., 2007) [62]. For example, participants' judgements of the averseness of a US have been found to correlate strongly with their ability to learn to distinguish between a stimulus (CS) coupled with the aversive US and a CS not matched with the US (Joos *et al*., 2013) [27].

However, the prominence of a stimulus does not remain constant. Stimulus salience can vary with experience (Pearce & Hall, 1980; Pearce & Mackintosh, 2010; Le Pelley & McLaren, 2004; Mackintosh, 1975; Le Pelley *et al*., 2010) [67, 68, 36, 46, 37]. Learning may be easier with stimuli that have strong predictors of an outcome, whereas stimuli that are poor forecasters lose their capacity to capture attention (Mackintosh, 1975) [46]. Research investigating associative learning mechanisms, which may be underlying schizophrenia symptoms, has found examples of individual variations in stimulus salience changes across time.

Usually, frequent exposure to a stimulus uncorrelated with an outcome reduces later ability to learn about that stimulus (Lubow *et al*., 1976; Lubow & Moore, 1959; Lubow, 2010) [44, 43, 42]. This phenomenon has been referred to as latent inhibition. One explanation for this change is that repeated contact with the stimulus reduces its salience, particularly its attentional associability, so the level of attention given to the stimulus decreases compared to other stimuli (Le Pelley, 2004; Mackintosh, 1975) [35, 46]. Attentional associability determines which stimuli are accessible to learning and which are not (Le Pelley, 2004; Mackintosh, 1975) [35, 46]. Hence, a decrease in attentional associability should impair learning.

This process of latent inhibition is interrupted in schizophrenia, and it is linked to negative symptoms precisely (Vaitl & Lipp, 1997; Lubow *et al*., 1976; Lubow, 1989, 2010; Grey *et al*., 1995; Baruch *et al*., 1988; Gal *et al*., 2009; Rascle *et al*., 2001) [88, 44, 41, 42, 22, 7, 19, 72]. In contrast, animal models of positive schizophrenia symptoms have shown persistent latent inhibition or compelling latent inhibitory mechanisms (Weiner, 2003) [90]. Compared to the abundance of research on interrupted latent inhibition in human participants, there has been little investigation into the impact of persistent latent inhibition in the community. Further research would help determine whether associative learning mechanisms are relevant to understanding positive

symptoms of schizophrenia. However, the disruption of latent inhibition linked to negative schizophrenia symptoms shows that negative symptoms are related to a deficiency in selective attention (Weiner *et al*., 1981, 1984; Solomon *et al*., 1981) [91, 92, 83] or selective prediction error (Hasselgrove & Evans, 2010) [26].

Haselgrove & Evans (2010) [26] employed the blocking effect to investigate the link between selective prediction error and schizophrenia. Blocking is assumed to be based on selective prediction error. Kamin (1968, 1969) [28, 29] discovered that past training with one stimulus interacts with the acquisition of associative intensity with a second stimulus when delivered concurrently with the first stimulus. For example, suppose a stimulus is paired with an outcome (A+) before matching two stimuli with the same result (AXE+). In that case, the associative intensity acquired by the second stimulus (X) is lower than that of the control. According to Haselgrove & Evans (2010) [26], this impact is explained by selective prediction error. The Rescorla and Wagner model of learning, outlined above in Equation 1, employs a summed error term and anticipates that the change in the associative intensity of a stimulus is proportional to the difference between the asymptote of learning supported by the result and the associative strength of all stimuli offer on a trial. For example, A has previously predicted the result in the AXE compound trial. Therefore, the prediction error is minimal, prohibiting learning with X. A failure to display blocking may indicate that prediction error is non-selective, i.e., on the AXE compound trial, the associative intensity acquired by A is not considered when learning with X, and so learning with X may take place (Haselgrove & Evans, 2010) [26].

Blocking is disturbed in schizophrenia, which is linked to the disease's negative and depressive symptoms (Moran *et al*., 2008; Bender *et al*., 2001) [58, 8]. This impact has been reproduced in a non-clinical population; individuals with high levels of introverted anhedonia, a negative symptom of schizotypy, exhibit impaired blocking (Haselgrove & Evans, 2010) [26]. The observation of this impact with the dimension of schizotypy shows that individuals in the general population vary significantly in the selectivity of their learning.

Attending To the Cues or the Context

In an associative learning model, participants are typically given the chance to learn how a stimulus predicts the result. Specificity is a key component of this learning. Learning that particular stimulus, rather than the context in which it is presented or any other given stimuli, predicts the result of interest. To return to the original example of a child playing with a toy, pressing the lever activates a light. Playing with the toy allows the child to experience the possibility of leaver pressing and the appearance of the light. Experience with this contingency should help you learn that pressing the leaver, instead of any other indication in the environment, turns on the light.

A lack of specificity in learning could explain the link between anxiety and high levels of conditioned fear (Baas *et al*., 2008; Baas, 2013) [3, 2]. For example, suppose an unpleasant stimulus (US) is delivered in a specific context. In that case, it is possible that the context will become associated with the US, and so the context may begin to elicit a fear response. If the aversive US is always and only shown immediately following a given cue, the cue can be utilised to predict the aversive US. Learning the precise relationship between the cue and the US should lessen the link between the context and the aversive US, as the context is a less accurate predictor of the US than the cue. Failure to learn this precise association may result in ongoing overall anxiety about the context. Studies have found a link between learning a specific association between a danger cue and the unpleasant US and decreasing overall dread of the situation in which the cue and the aversive US are given. Baas (2013) [2] found that those who failed to understand the relationship between a specific threat cue and the unpleasant US assessed the situation in which that stimulus was given as frightening Participants who learnt the specific CS-US association experienced lower fear ratings for the scenario (Baas, 2013) [2]. However, this study found no link between trait anxiety and failure to learn the specific connection, while it is plausible that failure to learn the particular association is related to anxiety traits such as attentional control (Baas, 2013; Derryberry & Reed, 2002) [2, 16].

Individual variations in the specificity of learning about cues in a situation can be observed in contingency learning in humans. Learning contingencies enables people to judge how well events and actions predict future outcomes, enabling experience to guide behaviour (Baker *et al*., 2001) [6]. While positive contingencies, in which the probability of an event occurring rises with the presence of a stimulus, are common, we also encounter zero contingencies, in which the outcome is equally likely to happen in the presence or absence of a stimulus. The accuracy in detecting zero contingencies is relatively low, particularly when people are asked to examine whether their actions produce an outcome (Baker *et al*., 2010; Alloy & Abramson, 1979) [5, 1]. Alloy & Abramson (1979) [1] asked participants to press a light switch and assess how much control they had over a light turning on and off. There was no contingency connection between pressing the light switch and the light turning on; the light was equally likely to turn on during trials when the light switch was not pressed as during trials when it was pressed. Alloy & Abramson (1979) [1] discovered that sad individuals correctly perceived that they had no control over the light. Non-depressed individuals wrongly assumed they had control over the light. This phenomenon was dubbed depressive realism (Alloy & Abramson, 1979) [1]. Recent studies on this effect imply it is less susceptible to context information (Msetfi *et al*., 2005) [59]. Msetfi *et al*. (2005) [59] altered two factors when rerunning the original Alloy and Abramson experiment: result density and inter-trial interval (ITI). This experimental design divides the chance to press a light switch into trials based on the occurrence or non-occurrence of the light. The ITI, or the amount of time between trials, can be modified. Event density, or the fraction of trials in which an event happens, can likewise be adjusted while keeping a zero contingency. For instance, in a low outcome density situation, the light may illuminate 25% of the trials in which the light switch is pressed and 25% of the trials in which it is not pressed. In a high outcome density situation, the light may turn on in 75% of trials when the light switch is pressed and 75% when it is not.

Msetfi *et al*. (2005) [59] found that the original Depressive Realism impact occurred only when the ITI was long, and the resulting density was high. Non-depressed respondents did not overestimate their influence over the light during shorter ITIs or when the outcome density was lower.

Interestingly, in a long ITI design, respondents are exposed to the context despite the outcome; that is, they have a more significant experience with no-action (those who participated cannot press the light switch during the ITI) and no outcome (the light never turns on during the ITI). Getting more exposed to the no-action, no-outcome contingency raises the contingency between action and outcome. As a result, given these conditions, non-depressed individuals were correct in assuming they had some influence over the outcome. The inability of depressed participants to enhance their judgements of control shows that they were unresponsive to the no-action, no-outcome information offered during the ITI (Baker *et al*., 2010; Msetfi *et al*., 2005) [5, 59].

Learning About Constituent Elements or Configurations

While linear learning is the acquisition and application of associations between single stimuli and outcomes, non-linear learning is the learning of compound stimuli in separate configurations associated with different outcomes than those associated with the compound's constituent stimuli. The Rescorla &Wagner (1972) [76] model of elemental learning posits that each stimulus is processed separately, resulting in its own associative relationship to the outcome. When learning about and reacting to compound stimuli, this fundamental method assumes that each component stimulus forms its own associative relationship with the result. As a result, the model suggests that the associative intensity of a compound stimulus (Vab) is the algebraic sum of the associative strengths of the stimuli delivered (Vab = Va + Vb). While elemental theory actually accounts for situations in which the outcome of the co-occurrence of stimuli is higher than that of the separate constituent stimuli, non-linear bias tasks necessitate learning the opposite relationship: where the outcome of the co-occurrence of stimuli is lower than, or opposite to, that of the separate constituent stimuli. Non-linear bias, such as negative patterning, can be solved successfully by humans and animals (Shanks & Darby, 1998; Deisig *et al*., 2001; Redhead & Pearce, 1995; Myers *et al*., 2001; Grand & Honey, 2008; Harris *et al*., 2008; Pearce & George, 2002;) [81, 15, 74, 60, 21, 25, 66]. This cannot be explained using the standard Rescorla & Wagner (1972) [76] elemental model. Non-linear bias learning, on the other hand, can be explained using configural theory (Pearce, 1987) [64]. Configural theory (Pearce, 1987) [64] holds that linkages emerge between outcomes and unitary or configural representations of the stimuli present in a given trial. As a result, the configuration contained in a compound trial (AB) should associate with an outcome irrespective of the associations built between the constituent stimuli and outcomes. Though these two types of models make opposing predictions about how the link between constituent stimuli and configurations should be taught, both models have substantial support, indicating significant diversity in non-linear learning. Melchers *et al*. (2008) [55].

It has been proposed that the perceptual qualities of stimuli impact whether learning occurs with individual constituent stimuli (elemental) or configurations (Kehoe *et al*., 1994; Myers *et al*., 2001; Rescorla & Coldwell, 1995; Lachnit, 1988) [32, 60, 75, 34]. Others suggest that these are two distinct forms of learning, each mediated by a different neural substrate (Sutherland & Rudy, 1989; Fanselow, 1999) [84, 18].

Several studies have examined whether people differ in their proclivity to learn about constituent elements or arrangements. The negative patterned discrimination (A+, B+, AB−) is an adequate test of configural learning, as it requires participants to learn that the compound stimulus has a different outcome than each constituent stimulus. Shanks and Darby (1998) [81] proposed that humans' ability to learn non-linear discriminations, like negative patterning, may be contingent on rule use. Shanks & Darby (1998) [81] found that the capacity to learn negative patterning discrimination was linked to later usage of rule-based generalisation rather than feature-based generalisation. Rule-based generalisation relies on the abstraction and generalisation of a rule. Surface similarity across different stimuli and molecules is required for feature-based generalisation. As a result, it is hypothesised that rule-based generalisation is more complex. It may be a deeper grasp of the discrimination (Shanks & Darby, 1998) [81] or increased working memory capacity (Wills *et al*., 2011) [93].

In the Shanks & Darby (1998) [81] experiment, respondents were trained on negative patterning discrimination (A+, B+, AB−) intermixed with trials in which separate stimuli were paired with the outcome (I+, J+) before being prompted to predict the result following the co-occurrence of the separately trained stimuli (IJ?). Some of the respondents predicted the event to occur after the IJ compound, demonstrating feature-based generalisation. Others showed the use of a negative patterning rule, which predicted no effect after the IJ compound. Rule-based generalisation was linked to significant initial discrimination learning (Shanks & Darby, 1998) [81]. Wills *et al*. (2011) [93] discovered that people who completed a concurrent activity while learning the same initial discrimination were prone to exhibit feature-based generalisation (Wills, 2011) [93]. As a result, larger working memory capacity may correlate with more substantial non-linear discrimination learning and rule-based generalisation. Baker (2013) [4] found that success in Raven's Progressive Matrices (Raven, 2000) [73] was associated with the capacity to learn about negative patterning discrimination. Ravens Matrices are intended to measure reasoning abilities. Therefore, these findings may support the hypothesis that rule use enhances non-linear discrimination learning, like negative patterning.

Negative patterning, on the other hand, necessitates learning about a configuration (i.e., the coexistence of stimuli) in addition to learning about the constituent stimuli. We can thus assume that a proclivity to perceive or interpret groupings of stimuli as a unitary configuration rather than just a cluster of co-occurring inputs may influence performance. Similar requirements for tasks have been investigated in other fields of psychology. For example, face recognition is assumed to rely on configural processing (Tanaka &Farah, 1993; Diamond & Carey, 1986; Maurer *et al*., 2002; Leder & Bruce, 2000) [85, 17, 51, 38]. Strong face recognition has been linked to a general advantage in global processing (Perfect, 2003; Macrae & Lewis, 2002) [69, 47], which tends to process global information first or with a greater priority than the particular elements that make up the global stimuli (Navon, 1977). [61]

Individual variations in their inclination to demonstrate a global or local processing advantage (Navon, 1977) [61], and it may be that this variation relates to, or impacts, the ability to learn about combinations of inputs and so learn a non-linear discriminate. Byrom & Murphy (under review) found

that individuals with a global processing advantage were more likely to discriminate BC from ABC in an improved negative patterning task (A+, BC+, ABC−), which is similar to the discrimination task developed by Shanks and Darby.

Modeling Individual Difference in Human Associative Learning

Using associative learning to investigate clinical events has helped us better understand the mechanisms behind cognitive aspects of psychopathology. Given that psychopathology is widely considered to occur on a spectrum, the clinical cases offered here help to demonstrate significant individual variations in associative learning processes. For example, whereas schizophrenia is a primary mental health disease with a prevalence of approximately 0.4% (McGrath *et al*., 2008; Saha *et al*., 2005) [52, 79], schizotypy, a dimension reflecting schizophrenia features, varies across the population (Mason & Claridge, 2006; Mason *et al*., 2005) [48, 49]. Schizotypy, like schizophrenia, is linked to disruptions in latent inhibition and blocking (Haselgrove & Evans, 2010; Moran *et al*., 2003) [26, 57], and poor conditional task performance (Haddon *et al*., 2011) [23] and visual context processing (Uhlhaas & Silverstein, 2005; Uhlhaas *et al*., 2004) [86, 87].

Models of learning might be required to account for this adaptability. If associative learning mechanisms differ throughout the population, focusing on the average performance of a sample while creating learning models may result in models that do not accurately capture the population's performance. Simple learning models have seen numerous revisions over time. While these improvements help the models to capture a greater variety of experimental results, many different parameters change during learning. Thus, it may not be appropriate to look for a single alteration to account for all variability in learning. One measure is unlikely to capture all components contributing to individual variability in human associative learning.

Individual variations in many of the above characteristics can be reflected by changing the parameters in the Rescorla & Wagner (1972) [76] learning model, as given in Equation (1). Modifying α or β can account for individual variations in the perceived salience of the CS or US. Varying λ accommodates individual variations in learning rates. Furthermore, Haselgrove & Evans (2010) [26] found that it may be possible to account for individual variations in learning selectivity by altering the extent to which a separable (Bush & Mosteller, 1951) [10] rather than a summed (Rescorla & Wagner, 1972) [76] error term is used. The variation and integration of summed and separable error terms, as well as their relationship to attention processes, have been extensively studied elsewhere (Pearce & Mackintosh, 2010; Lepelley, 2004) [68, 35].

On the other hand, individual variations in the capacity to solve negative patterning to discrimination cannot be explained by changing existing parameters in this model. At least three ways have been proposed to offer adaptability between elemental and configural learning models: the sampling capacity parameter, the discriminability parameter and the replacement parameter. Each is discussed below.

The Replaced Elements Model (Wagner, 2003; Brandon *et al*., 2000) [89, 11] views stimuli as having several features or elements. The model focuses on the characteristics of all stimuli and how they interact with elements distinctive to each stimulus. In a compound representation, context-independent elements are thought to be activated anytime the stimulus is delivered. In contrast, context-dependent elements can be stimulated or inhibited based on the combinations of stimuli shown (Brandon *et al*. 2000) [11]. For example, if stimulus A is delivered alone, representations of items A1 and A2 may be engaged. When stimulus A is provided along with stimulus B, the element A2 can be replaced by a new element, A3. The paradigm requires that a compound have no more remarkable ability to evoke associative power than any of its individual constituents. As a result, adding and inhibiting elements causes a qualitative change in represented elements instead of a quantitative change.

The replacement parameter r allows for variation in the proportion of context-dependent elements replaced when stimuli are given in compound form (Wagner, 2003) [89]. When r is zero, no replacement occurs, and so a considerable generalisation of associative power between stimuli and compounds is expected. When r is 1, elements are significantly replaced; hence, the generalisation predicted between compounds and constituent stimuli is diminished. With maximal element replacement, the compound representation must be unique from the representations of the individual stimuli.

The discriminability parameter proposed by Kinder & Lachnit (2003) [33] adds flexibility to a configurable learning model (Pearce, 1987) [64], enabling the perceived similarity between stimuli and compounds to be adjusted. This also influences how much generalisation of associative power is projected. The change implies that as it gets more challenging to determine constituent stimuli within compounds, the discriminability parameter will fall, lowering the prediction of perceived similarity between compounds and constituent stimuli (Kinder & Lachnit, 2003) [33].

Although the replacement and discriminability parameters were designed to account for the influence of external factors like stimulus modality (Kehoe *et al*., 1994) [32], the sampling capacity parameter was created to account for individual variations found in human associative learning. Sampling capacity is defined as the number of stimulus features that are capable of being sampled within a single trial. To learn about and react to the co-occurrence of stimuli as a distinct combination, Byrom & Murphy (under review) propose that aspects of each co-occurring stimulus be collected concurrently to represent a configuration in any given sample. Variations in sampling capacity must result in variations in the amount to which co-occurring stimulus features can be sampled, as well as variations in the ability to encode and learn about the various combinations of stimuli needed to acquire non-linear discrimination. Byrom & Murphy (under review) propose that the influence of changing sampling capacity can be modelled by integrating a parameter, f, into a variant of Pearce's configural model of associative learning. This value reflects the chance of encoding a configuration computed using sampling capacity. The likelihood of sampling a configuration with a set number of features for a particular sample size grows as sampling capacity rises.

According to Pearce's (1987, 1994) [64, 65] configural model of learning, the configurations of stimuli presented determine associative power. Individuals with limited sampling capacity can learn about the individual stimuli rather than the combinations. To account for this

adaptability, Byrom & Murphy (under review) propose revising Pearce's (1987, 1994) [64, 65] configural model of learning so that input can activate two sets of nodes: distinct stimuli (A, B, and C) and presented combinations (A, BC, and ABC). Both sets of nodes can create associations in response to an unconditioned stimulus, and generalisation can occur across all nodes. This can be accomplished by changing Pearce's (1987, 1994) [64, 65] configural model of associative learning so that variations in the excitatory intensity of the individual stimuli and presented configurations are controlled by the parameter f, representing sampling capacity. At a large sampling capacity, the excitatory power of presented configurations varies between learning trials. At low sampling capacity, the excitatory intensity of the individual stimuli varies between learning trials. Because Pearce's (1987, 1994) [64, 65] configural model heavily relies on the influence of generalisation, any modifications to this model must consider generalisation, which, like changes in excitatory strength, is controlled by the parameter f. As a result, at a high sampling capacity, associative strength to separate stimuli and between presented configurations will be high, whereas, at a low sampling capacity, associative strength to separate stimuli and between presented configurations will be low, but generalisation from separate stimuli to presented configurations will be high.

The ability to specify parameters a priori determines how much they can be utilised to predict learning and behaviour in unexpected contexts. Each of these alterations encounters difficulties in determining parameters in advance. The replacement parameter is determined by the fraction of elements replaced when a stimulus is delivered in compound form. The discriminability parameter is based on the ability to distinguish between stimuli. Calculating any of these values for a specific stimulus set is achievable. Still, several factors are likely to interact to affect "element replacement" and stimulus discriminability, restricting the extent to which these parameters may be stated in advance. Individual variations in tendencies to exhibit local or global processing can be used to calculate sampling capacity. This requires relevant data, which includes participant performance on a task like the Navon task (Navon, 1977) [61].

Conclusion

Individual variations in human associative learning appear to impact learning significantly. This adaptability must be expressed in terms of specific parameters to comprehend and fully describe human associative learning. Though adding new parameters for improving the adaptability of learning models has limitations, investigating the level at which variation among particular parameters can be used to understand certain individual variations in human associative learning should improve understanding of the associative learning mechanism.

References

1. Alloy LB, Abramson LY. Judgment of contingency in depressed and nondepressed students: sadder but wiser. Journal of Experimental Psychology: General. 1979;108(4):441-485.
2. Baas JM. Individual differences in predicting aversive events and modulating contextual anxiety in a context and cue conditioning paradigm. Biological Psychology. 2013;92:17-25.
3. Baas JM, van Ooijen L, Goudriaan A, Kenemans JL. Failure to condition to a cue is associated with sustained contextual fear. Acta Psychologica. 2008;127:581-592.
4. Baker AG. Individual differences in associative learning. Cardiff: Paper presented at the Associative Learning Symposium; 2013.
5. Baker AG, Msetfi RM, Hanley N, Murphy RA. Depressive realism? Sadly not wiser. In: Haselgrove M, Hogarth L, editors. Clinical applications of learning theory. Hove: Psychology Press; 2010. p. 153-177.
6. Baker AG, Murphy RA, Vallée-Tourangeau F, Mehta R. Contingency learning and causal reasoning. In: Mowrer RR, Klein SB, editors. Handbook of contemporary learning theories. Mahwah (NJ): Lawrence Erlbaum Associates; 2001. p. 255-306.
7. Baruch I, Hemsley DR, Gray JA. Differential performance of acute and chronic schizophrenics in a latent inhibition task. Journal of Nervous and Mental Disease. 1988;176:598-606.
8. Bender S, Müller B, Oades RD, Sartory G. Conditioned blocking and schizophrenia: a replication and study of the role of symptoms, age, onset-age of psychosis and illness-duration. Schizophrenia Research. 2001;49:157-170.
9. Bradley BP, Mogg K, Lee SC. Attentional biases for negative information in induced and naturally occurring dysphoria. Behaviour Research and Therapy. 1997;35:911-927.
10. Bush RR, Mosteller F. A model for stimulus generalization and discrimination. Psychological Review. 1951;58:413-423.
11. Brandon SE, Vogel EH, Wagner AR. A componential view of configural cues in generalization and discrimination in Pavlovian conditioning. Behavioural Processes. 2000;51:67-72.
12. Chan SWY, Goodwin GM, Harmer CJ. Highly neurotic never-depressed students have negative biases in information processing. Psychological Medicine. 2007;37:1281-1291.
13. Craske MG, Hermans D, Vansteenwegen D. *Fear and learning: from basic processes to clinical implications*. Washington (DC): American Psychological Association; 2006. p. 1-312.
14. de Wit S, Dickinson A. Associative theories of goal-directed behaviour: a case for animal-human translational models. Psychological Research. 2009;73:463-476.
15. Deisig N, Lachnit H, Giurfa M, Hellstern F. Configural olfactory learning in honeybees: negative and positive patterning discrimination. Learning and Memory. 2001;8:70-78.
16. Derryberry D, Reed MA. Anxiety-related attentional biases and their regulation by attentional control. Journal of Abnormal Psychology. 2002;111:225-236.
17. Diamond R, Carey S. Why faces are and are not special: an effect of expertise. Journal of Experimental Psychology: General. 1986;115:107-117.
18. Fanselow MS. Learning theory and neuropsychology: configuring their disparate elements in the hippocampus. Journal of Experimental Psychology: Animal Behavior Processes. 1999;25:275-283.
19. Gal G, Barnea Y, Biran L, Mendlovic S, Gedi T, Halavy M, *et al*. Enhancement of latent inhibition in patients with chronic schizophrenia. Behavioural Brain Research. 2009;197:1-8.

20. Gotlib IH, Krasnoperova E, Yue DN, Joormann J. Attentional biases for negative interpersonal stimuli in clinical depression. Journal of Abnormal Psychology. 2004;113:127-135.
21. Grand C, Honey RC. Solving XOR. Journal of Experimental Psychology: Animal Behavior Processes. 2008;34:486-493.
22. Gray NS, Pilowsky LS, Gray JA, Kerwin RW. Latent inhibition in drug-naive schizophrenics: relationship to duration of illness and dopamine D2 binding using SPET. Schizophrenia Research. 1995;17:95-107.
23. Haddon JE, George DN, Grayson L, McGowan C, Honey RC, Killcross S. Impaired conditional task performance in a high schizotypy population: relation to cognitive deficits. Quarterly Journal of Experimental Psychology. 2011;64:1-9.
24. Harris JA, Livesey EJ. An attention-modulated associative network. Learning and Behavior. 2010;38:1-26.
25. Harris JA, Livesey EJ, Gharaei S, Westbrook RF. Negative patterning is easier than a biconditional discrimination. Journal of Experimental Psychology: Animal Behavior Processes. 2008;34:494-500.
26. Haselgrove M, Evans LH. Variations in selective and nonselective prediction error with the negative dimension of schizotypy. Quarterly Journal of Experimental Psychology. 2010;63:1127-1149.
27. Joos E, Vansteenwegen D, Hermans D. Worry as a predictor of fear acquisition in a non-clinical sample. Behaviour Modification. 2013;36:723-739.
28. Kamin LJ. Attention-like processes in classical conditioning. In: Jones MR, editor. Miami symposium on the prediction of behaviour: aversive stimulation. Miami: University of Miami Press; 1968. p. 9-33.
29. Kamin LJ. Predictability, surprise, attention and conditioning. In: Campbell BA, Church RM, editors. Punishment and aversive behaviour. New York: Appleton-Century-Crofts; 1969. p. 279-296.
30. Kamin LJ, Brimer CJ. The effects of intensity of conditioned and unconditioned stimuli on a conditioned emotional response. Canadian Journal of Psychology. 1963;17:194-200.
31. Kamin LJ, Schaub RE. Effects of conditioned stimulus intensity on the conditioned emotional response. Journal of Comparative and Physiological Psychology. 1963;56:502-507.
32. Kehoe EJ, Horne AJ, Horne PS, Macrae M. Summation and configuration between and within sensory modalities in classical conditioning of the rabbit. Animal Learning and Behavior. 1994;22:19-26.
33. Kinder A, Lachnit H. Similarity and discrimination in human Pavlovian conditioning. Psychophysiology. 2003;40:226-234.
34. Lachnit H. Convergent validation of information-processing constructs with pavlovian methodology. Journal of Experimental Psychology: Human Perception and Performance. 1988;14:143-152.
35. Le Pelley ME. The role of associative history in models of associative learning: a selective review and a hybrid model. Quarterly Journal of Experimental Psychology B. 2004;57:193-243.
36. Le Pelley ME, McLaren IPL. Associative history affects the associative change undergone by both presented and absent cues in human causal learning. Journal of Experimental Psychology: Animal Behavior Processes. 2004;30:67-73.
37. Le Pelley ME, Turnbull MN, Reimers SJ, Knipe RL. Learned predictiveness effects following single-cue training in humans. Learning and Behavior. 2010;38:126-144.
38. Leder H, Bruce V. When inverted faces are recognized: the role of configural information in face recognition. Quarterly Journal of Experimental Psychology A. 2000;53:513-536.
39. Logan FA. A note on stimulus intensity dynamism. Psychological Review. 1954;61:77-80.
40. Lubinski D. Scientific and social significance of assessing individual differences: “sinking shafts at a few critical points”. Annual Review of Psychology. 2000;51:405-444.
41. Lubow RE. Latent inhibition and conditioned attention theory. New York: Cambridge University Press; 1989.
42. Lubow RE. Latent inhibition. In: Corsini Encyclopedia of Psychology. John Wiley & Sons, Inc; 2010.
43. Lubow RE, Moore AU. Latent inhibition: the effect of nonreinforced pre-exposure to the conditional stimulus. Journal of Comparative and Physiological Psychology. 1959;52:415-419.
44. Lubow RE, Schnur P, Rifkin B. Latent inhibition and conditioned attention theory. Journal of Experimental Psychology: Animal Behavior Processes. 1976;2:163-174.
45. Mackintosh NJ. Analysis of overshadowing and blocking. Quarterly Journal of Experimental Psychology. 1971;23:118-125.
46. Mackintosh NJ. Theory of attention variations in associability of stimuli with reinforcement. Psychological Review. 1975;82:276-298.
47. Macrae CN, Lewis HL. Do I know you? Processing orientation and face recognition. Psychological Science. 2002;13:194-196.
48. Mason O, Claridge G. The Oxford-Liverpool Inventory of Feelings and Experiences (OLIFE): further description and extended norms. Schizophrenia Research. 2006;82:203-211.
49. Mason O, Linney Y, Claridge G. Short scales for measuring schizotypy. Schizophrenia Research. 2005;78:293-296.
50. Matthews G, Pitcaithly D, Mann RLE. Mood, neuroticism, and the encoding of affective words. Cognitive Therapy and Research. 1995;19:563-587.
51. Maurer D, Le Grand R, Mondloch CJ. The many faces of configural processing. Trends in Cognitive Sciences. 2002;6:255-260.
52. McGrath J, Saha S, Chant D, Welham J. Schizophrenia: a concise overview of incidence, prevalence, and mortality. Epidemiologic Reviews. 2008;30:67-76.
53. McLaren IPL, Wills AJ, Graham S. Attention and perceptual learning. In: Mitchell C, Le Pelley MEL, editors. Attention and associative learning: from brain to behaviour. Oxford: Oxford University Press; 2010. p. 133-158.
54. Mineka S, Zinbarg R. A contemporary learning theory perspective on the etiology of anxiety disorders it’s not what you thought it was. American Psychologist. 2006;61:10-26.
55. Melchers KG, Shanks DR, Lachnit H. Stimulus coding in human associative learning: flexible representations of parts and wholes. Behavioural Processes. 2008;77:413-427.
56. Mogg K, Bradley BP, Williams R. Attentional bias in anxiety and depression the role of awareness. British Journal of Clinical Psychology. 1995;34:17-36.
57. Moran PM, Al-Uzri MM, Watson J, Reveley MA. Reduced Kamin blocking in non-paranoid schizophrenia: associations with schizotypy. Journal of Psychiatric Research. 2003;37:155-163.

58. Moran PM, Owen L, Crookes AE, Al-Uzri MM, Reveley MA. Abnormal prediction error is associated with negative and depressive symptoms in schizophrenia. Progress in Neuro-Psychopharmacology and Biological Psychiatry. 2008;32:116-123.
59. Msetfi RM, Murphy RA, Simpson J, Kornbrot DE. Depressive realism and outcome density bias in contingency judgments: the effect of the context and intertrial interval. Journal of Experimental Psychology: General. 2005;134:10-22.
60. Myers KM, Vogel EH, Shin J, Wagner AR. A comparison of the Rescorla-Wagner and Pearce models in a negative patterning and a summation problem. Animal Learning and Behavior. 2001;29:36-45.
61. Navon D. Forest before trees precedence of global features in visual perception. Cognitive Psychology. 1977;9:353-383.
62. Otto MW, Leyro TM, Christian K, Deveney CM, Reese H, Pollack MH, *et al*. Prediction of "fear" acquisition in healthy control participants in a de novo fear-conditioning paradigm. Behaviour Modification. 2007;31:32-51.
63. Pavlov IP. Conditioned reflexes. Oxford: Oxford University Press; 1927.
64. Pearce JM. A model for stimulus-generalization in pavlovian conditioning. Psychological Review. 1987;94:61-73.
65. Pearce JM. Similarity. Psychological Review. 1994;101:587-607.
66. Pearce JM, George DN. The effects of using stimuli from three different dimensions on autoshaping with a complex negative patterning discrimination. Quarterly Journal of Experimental Psychology B. 2002;55:349-364.
67. Pearce JM, Hall G. A model for pavlovian learning variations in the effectiveness of conditioned but not of unconditioned stimuli. Psychological Review. 1980;87:532-552.
68. Pearce JM, Mackintosh NJ. Two theories of attention: a review and possible integration. In: Mitchell C, Le Pelley MEL, editors. Attention and associative learning: from brain to behaviour. Oxford: Oxford University Press; 2010. p. 11-40.
69. Perfect TJ. Local processing bias impairs lineup performance. Psychological Reports. 2003;93:393-394.
70. Perkins CC. The relation between conditioned stimulus intensity and response strength. Journal of Experimental Psychology. 1953;46:225-231.
71. Phillips WJ, Hine DW, Thorsteinsson EB. Implicit cognition and depression: a meta-analysis. Clinical Psychology Review. 2010;30:691-709.
72. Rascle C, Mazas O, Vaiva G, Tournant M, Raybois O, Goudemand M, *et al*. Clinical features of latent inhibition in schizophrenia. Schizophrenia Research. 2001;51:149-161.
73. Raven J. The Raven's progressive matrices: change and stability over culture and time. Cognitive Psychology. 2000;41:1-48.
74. Redhead ES, Pearce JM. Stimulus salience and negative patterning. Quarterly Journal of Experimental Psychology B. 1995;48:67-83.
75. Rescorla RA, Coldwell SE. Summation in autoshaping. Animal Learning and Behavior. 1995;23:314-326.
76. Rescorla RA, Wagner AR, editors. A theory of Pavlovian conditioning: variations in the effectiveness of reinforcement and nonreinforcement. New York: Appleton-Century-Crofts; 1972.
77. Rusting CL. Personality, mood, and cognitive processing of emotional information: three conceptual frameworks. Psychological Bulletin. 1998;124:165-196.
78. Rusting CL. Interactive effects of personality and mood on emotion-congruent memory and judgment. Journal of Personality and Social Psychology. 1999;77:1073-1086.
79. Saha S, Chant D, Welham J, McGrath J. A systematic review of the prevalence of schizophrenia. PLoS Medicine. 2005;2:e141.
80. Seligman ME. Learned helplessness. Annual Review of Medicine. 1972;23:407-412.
81. Shanks DR, Darby RJ. Feature- and rule-based generalization in human associative learning. Journal of Experimental Psychology: Animal Behavior Processes. 1998;24:405-415.
82. Smith MC. CS-US interval and US intensity in classical conditioning of the rabbit's nictitating membrane response. Journal of Comparative Physiology. 1968;66:679-687.
83. Solomon PR, Crider A, Winkelman JW, Turi A, Kamer RM, Kaplan LJ. Disrupted latent inhibition in the rat with chronic amphetamine or haloperidol-induced supersensitivity relationship to schizophrenic attention disorder. Biological Psychiatry. 1981;16:519-537.
84. Sutherland RJ, Rudy JW. Configural association theory the role of the hippocampal formation in learning, memory, and amnesia. Psychobiology. 1989;17:129-144.
85. Tanaka JW, Farah MJ. Parts and wholes in face recognition. Quarterly Journal of Experimental Psychology A. 1993;46:225-245.
86. Uhlhaas PJ, Silverstein SM. Perceptual organization in schizophrenia spectrum disorders: empirical research and theoretical implications. Psychological Bulletin. 2005;131:618-632.
87. Uhlhaas PJ, Silverstein SM, Phillips WA, Lovell PG. Evidence for impaired visual context processing in schizotypy with thought disorder. Schizophrenia Research. 2004;68:249-260.
88. Vaitl D, Lipp OV. Latent inhibition and autonomic responses: a psychophysiological approach. Behavioural Brain Research. 1997;88:85-93.
89. Wagner AR. Context-sensitive elemental theory. Quarterly Journal of Experimental Psychology B. 2003;56:7-29.
90. Weiner I. The 'two-headed' latent inhibition model of schizophrenia: modeling positive and negative symptoms and their treatment. Psychopharmacology. 2003;169:257-297.
91. Weiner I, Lubow RE, Feldon J. Chronic amphetamine and latent inhibition. Behavioural Brain Research. 1981;2:285-286.
92. Weiner I, Lubow RE, Feldon J. Abolition of the expression but not the acquisition of latent inhibition by chronic amphetamine in rats. Psychopharmacology. 1984;83:194-199.
93. Wills AJ, Graham S, Koh ZS, McLaren IPL, Rolland MD. Effects of concurrent load on feature- and rule-based generalization in human contingency learning. Journal of Experimental Psychology: Animal Behavior Processes. 2011;37:308-316.