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ELEMENTAL AND CONFIGURAL ASSOCIATIVE PROCESSES IN JUDGEMENTS OF THE CONTINGENCY BETWEEN COMPOUND PREDICTORS AND AN OUTCOME

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For my wife Antonietta, and my sons

Emilio Saverio and Samuele Domenico, with love. I gratefully

acknowledge the intellectual guidance and support of my

supervisor and mentor Pierre Mercier.

ABSTRACT

In 4 experiments, participants judged the contingency between compound predictors and an outcome, as well as the contingency between the compounds' constituent elements and the outcome, in different contingency and similarity conditions. The Rescorla-Wagner (1972) and Pearce (1987) models of associative learning describe different processes through which a compound predictor becomes associated with an outcome, and how responses to a compound are mediated by the association between its constituent elements and the outcome. According to the Rescorla-Wagner model, when a compound is paired with an outcome an association will develop between each element of the compound and the outcome, and responding to the compound will reflect the associative strength accrued to each element in an additive fashion. According to the Pearce model, a compound is associated with the outcome in its entirety (i.e., as a configural cue), and responding to the compound is related to both the associative strength of the configural cue and the associative strength generalised to the compound from other predictors as a function of similarity. Across experiments and conditions, compound predictors were assessed independently of the normative relation between their constituent elements and an outcome. Manipulations of the similarity among predictors, measured as the proportion of elements they share, did not impact judgements of compound predictors. Findings are consistent with the notion that compound predictors are functionally independent of their constituent elements, and possible modifications of the Rescorla-Wagner and Pearce models to account for these findings are discussed. A configural associative model that assumes no generalisation of associative strength among predictors appears to provide the best fit to the empirical findings.

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INTRODUCTION

With the sound of approaching thunder, a farmer's fear for a meagre harvest is allayed by the anticipation of irrigated crops. The farmer's response is consequent to an assessment of the naturally occurring covariation of thunder and rain. One putative statistical measure of the relation between pairs of binary events is the delta-p (Δp) coefficient, measured as the difference between the two independent conditional probabilities P(outcome|predictor) and P(outcome|~predictor) (Allan, 1980; Jenkins & Ward, 1965). The contingency between thunder and rain is calculated as the difference between the probability of rain in the presence of thunder and the probability of rain in the absence of thunder. In common everyday circumstances, however, an outcome could result from many possible causes either individually or in compound (Downing, Sternberg, & Ross, 1985). Judgements of the likelihood of rain may be more accurate, for example, if based on the co-presence of thunder and a decrease in atmospheric temperature. The ability to induce these relations between events, as well as between behaviours and their outcomes, is essential for all aspects of behavioural functioning and adaptation to the environment (Einhorn & Hogarth, 1986; Richardson, 1992; Saffran, Aslin, & Newport, 1996; Shaklee & Elek, 1988; Shaklee & Paszek, 1985; Tarabulsy, Tessier, & Kappas, 1996; White, 1988; Young, 1995), and a failure in this ability is associated with various forms of psychopathology (Alloy & Abramson, 1979; Alloy & Tabachnik, 1984; de Jong, Merckelbach, & Arntz, 1995; Ohr & Fagen, 1994; Pauli, Montoya, & Martz, 1996; Tomarken, Sutton, & Mineka, 1995). Accordingly, the process by which humans arrive at judgements of these relations is of considerable theoretical and empirical interest (Allan, 1993; Cheng, 1993; Shanks, 1993a; Wasserman & Miller, 1997).

Different psychological processes are proposed for the assessment of event contingency (Allan, 1993; Cheng, 1993; Kelly, 1967; Shanks, 1993a, 1993b; Waldmann & Holyoak, 1992). Many of the most recent reports focus on the applicability of associative theories derived from animal learning (Allan, 1993, López, Shanks, Almaraz, & Fernández, 1998; Shanks, 1993a; Siegel & Allan, 1996; Wasserman & Berglan, 1998). This conceptual approach is supported by findings that contingency judgements reflect processes similar to those presumed to underlie Pavlovian and Thorndikian conditioning (Allan, 1993; Alloy & Tabachnik, 1984; Dickinson & Shanks, 1985; Shanks, 1993a, 1993b; Shanks & Dickinson, 1987; Wasserman & Miller, 1997; Young, 1995). These reports focus primarily on judgements of the contingency between single predictors and an outcome, with or without the presence of other predictors of the same outcome (e.g., Baker, Mercier, Vallée-Tourangeau, Frank, & Pam, 1993; Chapman & Robbins, 1990; Price & Yates, 1993). Little is known of the associative processes underlying judgements of the contingency between compound predictors and an outcome. There are two general classes of associative theory relevant to the consideration of compound predictor processing: elemental and configural. The two classes describe different processes through which a compound predictor becomes associated with an outcome, and how responses to a compound are mediated by the association between its constituent elements and the outcome (Kehoe & Graham, 1988).

The traditional elemental account of multiple cue processing, as espoused by most contemporary Pavlovian theories of animal learning (e.g., Mackintosh, 1975; Pearce & Hall, 1980; Rescorla & Wagner, 1972) presumes that, when multiple predictors are paired with an outcome, the opportunity is present for each predictor to become associated with the outcome. This assumption is difficult to reconcile with findings from animal research that are more

consistent with the notion that a compound predictor becomes associated with an outcome in its entirety (Darby & Pearce, 1995; Pearce, Aydin, & Redhead, 1997; Pearce & Redhead, 1995; Pearce & Wilson, 1990a, 1990b, 1991, 1992). Pearce (1987, 1994; Pearce & Wilson, 1990b) developed a configural model to account for findings from research with non-human animals that appear incompatible with an elemental view of associative processing (Pearce & Redhead, 1993; Wilson & Pearce, 1992). This configural model posits that compound stimuli are associated with an outcome in their entirety (i.e., as configural cues), and that responding to a compound is related to both the associative strength of the configural cue and the associative strength generalised to the compound from other cues as a function of similarity (Pearce, 1987, 1994; Pearce & Wilson, 1990b).

Associative Models: Theoretical Background

A judgement of the contingency between a predictor and an outcome is an elemental causal attribution. These attributions are a function of both data driven and theory driven processes (Alloy & Tabachnik, 1984; Young, 1995). Associative models characterise data driven processing through which events, or behaviours and their outcome, become connected by virtue of their pairing in an individual's past experiences (Baker & Mercier, 1989; Wasserman & Miller, 1997; Young, 1995). When a predictor is paired with an outcome, an association is formed between them in a manner analogous to how a conditioned stimulus (CS) and an unconditioned stimulus (US), or how an operant and a reinforcer, become associated (Dickinson & Shanks, 1985; Young, 1995). The associative strength (V) of a predictor is analogous to its predictive or signal value (Williams, Sagness, & McPhee, 1994). The acquisition or formation of associations between a predictor and an outcome is accomplished through a process that relies

only on event contiguity (Rescorla & Wagner, 1972; Shanks & Dickinson, 1987). Sensitivity to the contingency between a predictor and an outcome is a property that emerges from this contiguity-sensitive process, and is related to a competition for associative strength among different predictors of the same outcome (Rescorla & Wagner, 1972; Young, 1995; see the formal description of the Rescorla-Wagner model below for a more complete characterisation of the relation between associative strength and contingency).

The psychological processes proposed for the assessment of event contingency have been cast at both the individual and group levels. Some researchers have suggested that individual differences in contingency judgements reflect the use of different judgement rules (Shaklee & Mims, 1982). Associative processes, however, have been more commonly used to describe contingency judgements at the level of groups (e.g., Baker et al., 1993). Although associative models can be formulated to characterise individual variations through differences in model parameter values, no systematic process has yet been described for changing parametric values at the individual level. Associative models are primarily models of information acquisition as opposed to behavioural expression (Baker & Mercier, 1989; Wasserman & Miller, 1997), and various theories have been advanced to elucidate the processes through which associative strength is expressed behaviourally (e.g., Frey & Sears, 1978; Miller & Matzel, 1988). In general, contingency judgements are assumed to have a monotonic relation with associative strength (Gluck & Bower, 1988; Miller, Barnet, & Grahame, 1995; Rescorla & Wagner, 1972; Shanks, 1993a). While associative theories do not provide an incontrovertible framework for understanding either animal learning (Baker & Mercier, 1989; Miller et al., 1995; Wasserman & Miller, 1997) or human contingency judgements (Allan, 1993; Catena, Maldonado, & Cándido,

1998; Shanks, 1993a; Siegel & Allan, 1996; Van Hamme & Wasserman, 1994; Wasserman & Berglan, 1998;), they have powerful heuristic value for generating research at least in part because of their clear ordinal predictions (Wasserman & Miller, 1997; Young, 1995).

Associative models are often contrasted with statistical models of contingency judgements (Allan, 1993; Cheng, 1993; López et al., 1998; Shanks, 1993b; Young, 1995). Researchers advocating statistical accounts view judgements of contingency as reflecting processes analogous to those employed by statisticians to evaluate the mathematical relatedness of events (Busemeyer, 1991; Cheng & Holyoak, 1995; Einhorn & Hogarth, 1986; Kelly, 1967). For example, to assess the mathematical relation between several predictors and an outcome people may instantiate a process roughly analogous to multiple linear regression (Koh & Meyer, 1991). Alternatively, people may evaluate the relative frequencies or conditional probabilities of co-occurring events in their environment akin to the calculation of the Δp coefficient or similar statistical rule (Allan & Jenkins, 1980; Allan & Jenkins, 1983; Chatlosh, Neunaber, & Wasserman, 1985; Cheng & Novick, 1990a, 1990b, 1992; Melz, Cheng, Holyoak, & Waldmann, 1993; Wasserman, Chatlosh, & Neunaber, 1983). Contingency judgement by this latter analysis is presumed to be based on a cognitive representation of the conditional probability of the outcome both in the presence and in the absence of a predictor (Busemeyer, 1991; Einhorn & Hogarth, 1986). While contemporary statistical models can effectively describe much of the empirical data in contingency judgements (Cheng, 1993; Cheng & Holyoak, 1995), associative models appear to provide a parsimonious description of a broader range of empirical phenomena (Allan, 1993; Shanks, 1993a, 1993b; López et al., 1998).

One of the central and enduring questions in associative theory concerns what is learned

when a predictor composed of several elements is followed by an outcome (Miller et al., 1995; Spence, 1952; Wasserman & Miller, 1997). Elemental and configural associative models relate the change of associative strength between a predictor and an outcome with experience, and describe interaction effects among predictors all associated with the same outcome. However, the two models describe different processes through which a compound predictor becomes associated with an outcome, and how associative strength is generalised between a compound predictor and the individual elements that compose it. Most contemporary models of Pavlovian associative learning may be characterised as elemental in nature (e.g., Frey & Sears, 1978; Mackintosh, 1975; Pearce & Hall, 1980; Rescorla & Wagner, 1972; Wagner, 1981). These theories share the common assumption that conditioning to a compound composed of two or more elements provides the opportunity for the formation of associations between each element of the compound and the outcome (Wasserman & Miller, 1997). Judgements of the compound predictor will reflect the individual associative strength acquired by each element of the compound in a linear or additive fashion (Rescorla & Wagner, 1972; Rescorla, Grau, & Durlach, 1985). Configural associative models (e.g., Kruschke, 1992; Pearce, 1987, 1994), on the other hand, share the assumption that when a compound of several predictors is paired with an outcome, the only association that will develop is that between the compound as a whole (i.e., as a configural cue) and the outcome. Any change in the pattern of predictors presented will result in the formation of an association between a new configural predictor and the outcome. Judgements to the compound will reflect the sum of the associative strength of the configural cue and the associative strength generalised to the compound from other predictors as a function of similarity (Pearce, 1987, 1994). According to the elemental approach, if a compound predictor

composed of two separable elements is followed by an outcome (AB+), the associative strength of the compound may be an additive function of the individual predictive values of the elements ($V_{AB} = V_A + V_B$) (e.g., Frey & Sears, 1978; Mackintosh, 1975; Pearce & Hall, 1980; Rescorla & Wagner, 1972). Alternatively, the associative strength of the compound predictor may be predominantly a function of the predictive value of a unique configural representation of the compound ($V_{AB} = V_{\text{"AB"}}$, where "AB" represents the configural cue) (Pearce, 1987, 1994).[1] As the Rescorla-Wagner model is the predominant associative characterisation of human contingency judgements (Siegal & Allan, 1996), it will be contrasted with the most current configural model of animal discrimination learning as proposed by Pearce (1987, 1994).

Rescorla-Wagner Model

Investigators assessing the associative processes underlying contingency judgements generally rely on the Rescorla-Wagner model (1972; Wagner & Rescorla, 1972) as an exemplar of the associative view (Shanks, 1993a; Siegel & Allan, 1996). The Rescorla-Wagner model, as most contemporary elemental associative models (e.g., MacKintosh, 1975; Pearce & Hall, 1980), was designed to account for data that the associative strength of a predictor is mitigated by the associative strength of other predictors of the same outcome (Shanks, 1993a; Wasserman & Miller, 1997). This is accomplished by assuming that the associative strength accruing to a CS on a reinforced trial is limited by the difference between the maximum associative strength supported by a US and the associative strength already gained by other predictors present on that trial. Through this competitive process, the difference between the maximum associative strength

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[1] Configural predictors are indicated with double quotation marks.

supported by a US and the associative strength already accounted for by predictors decreases trial-by-trial to yield the classic learning curve (Shanks, 1993a). One characterisation of this process is that the US becomes less able to confer associative strength to a predictor as learning progresses. The nature of this process contrasts with that presumed by some other contemporary elemental associative models (e.g., Frey & Sears, 1978; MacKintosh, 1975; Pearce & Hall, 1980), which posit that the effectiveness of the US does not vary but rather that the CS varies in associability with experience (Mercier, 1996; Miller et al., 1995).

Formally, The Rescorla-Wagner model assumes that the strength of association between a predictor and an outcome will change as

$$\Delta V_A^{n+1} = \alpha_A \beta_1 (\lambda_1 - V_T^n), \quad (1)$$

where

$$V_T^n = V_A^n + ... + V_X^n, \quad (2)$$

and

$$V_A^{n+1} = V_A^n + \Delta V_A^{n+1}. \quad (3)$$

Equation 1 describes the change in associative strength (V) of a predictor A as a result of a pairing with outcome$_1$ on trial n + 1 (ΔV_A^{n+1}), where α_A is the associability of predictor A, β_1 is the associability of outcome$_1$, λ_1 is the maximum associative strength supported by the outcome$_1$, and V_T^n is the sum of the associative strength already gained by all predictors present on that trial including A, and is given by Equation 2. In Equation 3 V_A^{n+1}, the associative strength of predictor A after trial n + 1, is the sum of the associative strength of predictor A immediately before trial n

+ 1 (V_A^n) and the change in associative strength of predictor A (ΔV_A^{n+1}) as determined by Equation 1. The learning rate parameters α and β can have values between 0 and 1, whereas λ takes on a value of 1 in the presence of the outcome and 0 on trials when the outcome does not occur. Associative models do not specify the precise values for these parameters. The Rescorla-Wagner model assumes that the associability parameters α and β do not change as a function of experience. The parenthetic term $\lambda_1 - V_T^n$ represents the maximum gain possible in a predictor's associative strength on a given trial. As V_T^n is the sum of the associative strength already gained by all predictors present on a trial, the associative strength gained by one predictor will directly affect the associative strength gained by a second predictor. For example, on a trial when both stimuli A and B are presented in compound $V_T^n = V_A^n + V_B^n$, and the value of the parenthetic term $\lambda_1 - V_T^n$ will be relatively smaller than on a trial when either A or B are presented alone. Consequently, each of stimuli A and B will acquire less associative strength when they are presented in compound than when they are presented individually.

To account for findings that conditioning appears to depend on both the probability of an outcome in the presence of a predictor [P(US|CS)] and the probability of an outcome in the absence of that predictor [P(US|~CS)] (Rescorla, 1968), Rescorla and Wagner (1972; Wagner & Rescorla, 1972) made the assumption that conditioning always occurs against a background of stimuli. This contextual cue is present on every trial of a conditioning experiment and acquires associative strength in a fashion analogous to other predictors. Consequently, an individual predictor never occurs in isolation, but rather is always presented in compound with the contextual cue. By assuming the existence of a contextual cue, the model accounts for

contingency learning through the simple contiguity between a CS and a US (Rescorla & Wagner, 1972; Siegel & Allan, 1996). For example, when a US is present without a CS, the contextual cue gains in associative strength as suggested by Equation 1. When a CS and the US are present, the associative values of both the CS and the contextual cue will contribute to V_T^n. Gains in the associative strength of the CS will be hindered if the contextual cue acquires sufficient associative strength. Through the competition for associative strength between the contextual cue and the CS, the associative strength of the CS will appear to reflect the contingency between the CS and the US (Rescorla & Wagner, 1972; Young, 1995). Through this contiguity mechanism, then, a decrease in the contingency between a CS and a US reflects an increase in the associative strength of the contextual cue and a concomitant decrease in the associative strength of the CS (Rescorla & Wagner, 1972; Shanks, 1993a; Young, 1995).

To illustrate the application of the Rescorla-Wagner model in the context of discrimination learning, a computer simulation of an A+, AB- discrimination was conducted. There were a total of forty trials in this simulation, twenty A+ trials and twenty AB- trials. A contextual cue (X) was assumed to be present throughout. Further, α was set at .3 for predictors A and B, while α for the X was .1. The salience of the outcome (β) was .3 both when it was present and when it was absent. As participants are frequently asked to make judgements of contingency on a scale that ranges from +100 to –100 (e.g., Baker et al., 1993; Baker, Berbrier, & Vallée-Tourangeau, 1989; Chapman & Robbins, 1990; Shanks, 1987), λ was set at 100 with the outcome present and 0 with the outcome absent so that model theoretical values and empirical judgements are comparable without a scale transformation. The result of the simulation represents mean predicted associative strengths of twenty-four different randomisations of forty

trials, after every four trials. With the contextual cue, the discrimination could be conceptualised as XA+, XAB-, and each individual predictor (X, A, and B) acquires associative strength when it is presented as indicated by Equation 1. The top two panels of Figure 1 depict the predicted mean associative strengths for the compounds and the separate elements after every four trials derived from the Rescorla-Wagner model. The CR to a compound is assumed to reflect the sum of the associative strength of its constituent elements so that: XA = $V_X + V_A$, XB = $V_X + V_B$, and XAB = $V_X + V_A + V_B$. Mean XAB initially rises as mean V_X and mean V_A undergo rapid increases in the first few trials. As mean V_B becomes increasingly negative, mean XAB declines towards zero. Appendix A contains a sample of the trial-by-trial changes in associative strength derived from an application of Equations 1 through 3, for one run of forty randomised trials of X, A, and B.

Pearce Model

Pearce's (1987, 1994) configural associative model presumes that compound predictors may be assessed independently of their constituent elements, and proposes a process through which associative strength is generalised among predictors as a function of their similarity (Pearce, 1987, 1994). According to Pearce (1987), a pattern of stimulation is represented in a limited capacity perceptual buffer. The perceptual buffer represents the overall pattern of stimulation that an organism is currently being exposed to. The perceptual representation present in the buffer will enter into an association in its entirety (i.e., as a configural representation) with the US, and represent the effective CS. Any change in the perceptual buffer will lead to a new CS-US association. The strength of the CR to any representation in the perceptual buffer is determined by its own pairing history with the outcome and by its similarity with other representations previously presented in the perceptual buffer. The Pearce model adopts the same

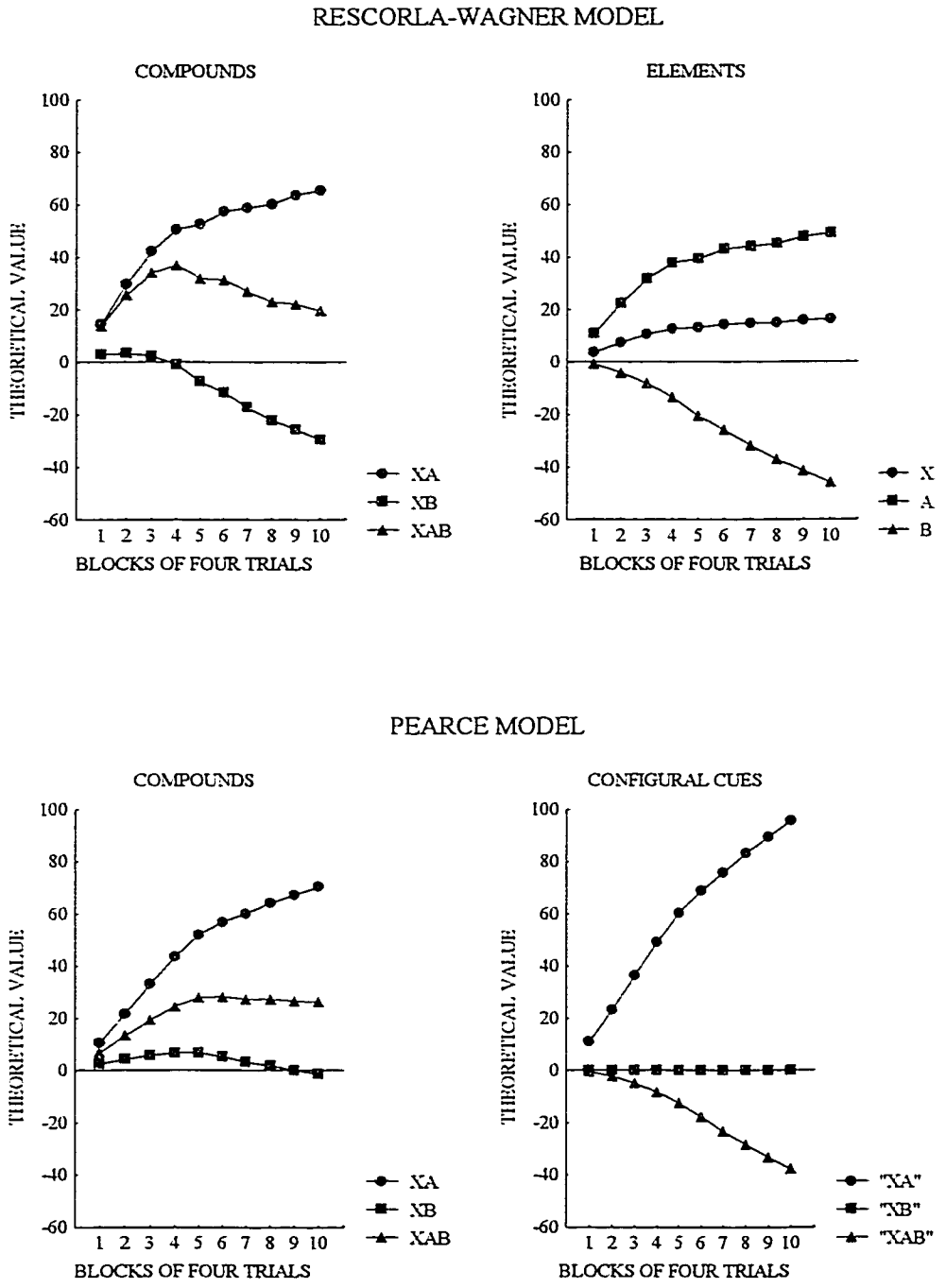

FIGURE 1. Theoretical values derived from the Rescorla-Wagner and Pearce models for an A+, AB- discrimination.

competitive learning rule as in Equation 1.

Formally, the Pearce model assumes that the strength of association between a predictor and an outcome will change as

$$\Delta V_A^{n+1} = \alpha_A \beta_1 (\lambda - V_{A+G}^n), \quad (4)$$

where,

$$V_{A+G}^n = V_A^n + V_G^n, \quad (5)$$

and

$$V_A^{n+1} = V_A^n + \Delta V_A^{n+1}. \quad (6)$$

Equation 4 describes the change in associative strength (V) of a predictor A as a result of a pairing with outcome$_1$ on trial n + 1 (ΔV_A^{n+1}), where α_A is the associability of predictor A, β_1 is the associability of outcome$_1$, λ_1 is the maximum associative strength supported by the outcome$_1$, and V_{A+G}^n is the sum of, a) the associative strength already gained by predictor A immediately before trial n + 1 (V_A^n), and b) the associative strength that generalises to A from other predictors previously paired with the outcome (V_G^n). In Equation 6, V_A^{n+1}, the associative strength of predictor A after trial n + 1, is the sum of the associative strength of predictor A immediately before trial n + 1 (V_A^n) and the change in associative strength of predictor A (ΔV_A^{n+1}) determined by Equation 4. The associative strength that generalises to predictor A (V_G^n) is given by

$$V_G^n = \Sigma S_{A,i} \times V_i^n, \quad (7)$$

and reflects the sum of the products of: a) the associative strengths of all other predictors of the same outcome (V_i, where i = 1 to number of predictors), and b) their respective similarities to

predictor A ($S_{A,i}$). Pearce (1987) originally proposed that the similarity between the current contents of the perceptual buffer and a previously established configural representation is a function of the perceived intensity of the shared elements and the total perceptual intensity of the two representations. For purposes of this report, we will assume that all the CS (i.e., predictors) are of equal intensity. Consequently, the generalisation between CS representations will be proportional to the number of elements they share (Pearce, 1994), and is given by

$$S_{A,i} = [n_C(1/\sqrt{n_A} \times 1/\sqrt{n_i})]^2, \quad (8)$$

where n_C is the number of elements in common between configural representations A and i, n_A the number of elements in representation A, and n_i the number of elements in representation i. Equation 8 is formally equivalent to Pearce's (1987) original conceptualisation of generalisation as a function of perceived intensity (Pearce, 1994).

As with the Rescorla-Wagner model, the learning rate parameters α and β can have values between 0 and 1, whereas λ takes on a value of 1 in the presence of the outcome and 0 on trials when the outcome does not occur. The Pearce model also assumes that the associability parameters α and β do not change as a function of experience. The parenthetic term $\lambda_1 - V^n_{A+G}$ represents the maximum gain possible in a predictor's associative strength on a given trial. Unlike the Rescorla-Wagner model, the associative strength that may accrue to predictor A will be constrained by its associative strength immediately before trial n + 1 and the associative strength generalised to it from other predictors as a function of their similarity. Thus, if other predictors of the same outcome do not share any elements with A, changes in V_A will be constrained only by its own associative strength immediately before trial n + 1. By this model, if

both predictors A and B are presented in compound and reinforced on the first trial of conditioning, the only association that will develop is that between configural predictor "AB" and the outcome. Since this is the first trial of conditioning, and there are no other predictors of the same outcome, $V^{n}_{AB+G} = V^{n}_{-AB-}$. In order to account for certain associative phenomena in animal conditioning, Pearce (1994) assumes the presence of a contextual cue (X). The effect of the contextual cue is to increase the similarity among predictors as it becomes an element common to all predictors. However, X only acquires associative strength independently on trials when no other predictors are present. That is, on a trial where predictor A is presented with the outcome, an association will develop between the configural predictor "AX" and the outcome, and the associative strength of X will remain unaffected.

To illustrate the application of the Pearce model in the context of discrimination learning, a computer simulation of an A+, AB- discrimination, identical to the one presented for the Rescorla-Wagner model above, was conducted. Again, the simulation included a total of forty trials, twenty A+ trials and twenty AB- trials. The presence of a contextual cue (X) was assumed for this simulation. With the contextual cue, the discrimination could be conceptualised as XA+, XAB-, and each configural predictor (i.e., "XA", and "XAB") acquires associative strength when it is presented as indicated in Equation 4. As the Pearce model presumes that only one associative strength will change during every conditioning trial (i.e., between a configural cue and the outcome), two configural predictors are present in this simulation, "XA" and "XAB". The CR to XA will reflect V_{-XA-} and the associative strength generalised from "XAB" as a function of their similarity given by Equation 8. The CR to XAB will reflect V_{-XAB-} and the associative strength generalised to it from "XA". The salience parameter α was set at .3 for both

predictors, and .1 for the X. The salience of the outcome (β) was .3 both when it was present and when it was absent, and λ was set at 100 with the outcome present and 0 with the outcome absent. The result of the simulation represents mean predicted associative strengths of twenty-four different randomisations of forty trials, after every four trials. As stated above, the CR to a compound is assumed to reflect the sum of the associative strength acquired by its configural cue and that generalised to it as a function of similarity so that: XA = $V_{\text{-XA-}} + V_G$, and XAB = $V_{\text{-XAB-}} + V_G$. The bottom two panels of Figure 1 depict the predicted mean associative strengths for the compounds and the separate configural cues derived from the Pearce model. Note that although the left panels of Figure 1 are qualitatively similar, the net associative strength accruing to the compounds reflects different components. For example, the net associative strength accruing to compound XB in Figure 1 reflects the associative strength generalised from configural cues "XA" and "XAB" in the simulation of the Pearce model, and the sum of the associative strength of elements X, and B in the simulation of the Rescorla-Wagner model. Indeed, configural cue "XB" has no associative strength according to the Pearce model (i.e., bottom right panel of Figure 1) as B was never presented on its own. According to Pearce's model, compound XB eventually becomes a conditioned inhibitor based on a progressively increasing generalisation of negative associative strength from configural cue "XAB". This contrasts with the Rescorla-Wagner model simulation, where compound XB (i.e., top left panel of Figure 1) becomes a conditioned inhibitor primarily as a result of the negative associative strength acquired by element B individually (i.e., top right panel of Figure 1).

Differences between the Rescorla-Wagner and Pearce models could be demonstrated by considering the conditioning that will take place after a reinforced presentation of the compound

AB. According to the Rescorla-Wagner model, an AB+ trial will provide the opportunity for the acquisition of associations to each element of the compound. The associative strength that will accrue to A will be limited both by its own associative strength immediately prior to this trial and by that already gained by B. The associative strength that will accrue to B will be similarly constrained. Finally, the CR to compound AB will reflect the associative strength of both A and B in an additive fashion. In contrast, the Pearce (1987) model predicts that AB will enter into an association with the outcome as a whole (i.e., as a configural cue) leaving the associative strength of cues A and B unaffected. The associative strength accruing to the configural cue "AB" will be limited by its own associative strength immediately prior to this trial and by the associative strength generalised to it from all other predictors previously paired with the outcome as a function of similarity. The CR to compound AB will reflect the associative strength of the configural cue "AB", as well as the associative strength of all other cues in proportion to their similarity. Thus, whereas the Rescorla-Wagner model presumes that the generalisation of associative strength between a compound and its elements is complete (Rescorla, 1997), Pearce's (1987, 1994) model predicts that generalisation is somewhat less than complete and related to the similarity among the predictors. Also, in the Rescorla-Wagner model, only stimuli that are present on a given trial can influence the change in associative strength, whereas in Pearce's model generalisation occurs from stimuli not currently present.[2]

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[2] For the purpose of deriving theoretical values from the Rescorla-Wagner and Pearce models, the presence of a contextual cue will always be assumed unless otherwise stated. Consequently, the contextual cue (X) will not be included in subsequent notation that characterises model simulations, or that describes experimental manipulations. For example, although an A+, AB- discrimination is accurately characterised as XA+, XAB- according to both

Associative Processes in Contingency Judgements

Parallels between animal conditioning and human contingency judgements have prompted researchers to investigate the applicability of associative models to higher level judgements (Allan, 1993; Baker & Mercier, 1989; Dickinson & Shanks, 1985; López et al., 1998; Mercier, 1996; Mercier & Parr, 1996; Shanks, 1993a, 1993b; Shanks & Dickinson, 1987; Waldmann & Holyoak, 1992; Wasserman, Elek, Chatlosh, & Baker, 1993; Young, 1995). The research strategy is to assess the extent to which contingency judgements are subject to the same processing constraints delineated in animal associative learning (Young, 1995). Of particular significance in assessing the applicability of associative models to human contingency judgements are two phenomena that have come to be prototypical of animal conditioning processes: cue interaction effects, and acquisition functions. Among the cue interaction effects replicated in the context of contingency judgements are: blocking, relative validity, conditioned inhibition, and the signal effect. Cue competition refers to the differential ability of a CS to elicit responding when it is conditioned along with other CS (Wasserman & Miller, 1997). Acquisition functions refer to the characteristic rate of acquisition of behavioural control by a CS, and is presumed to reflect trial-by-trial increments in the association between representations of the CS and the US as described by Equation 1.

Chapman and Robbins (1990, Experiment 1) assessed whether contingency judgements are subject to the blocking phenomenon well established in the animal learning literature (see

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models, notation will not reflect the presence of X unless this is necessary in order to distinguish between the theoretical values of the two models.

also, Chapman, 1991, Experiment 1; Dickinson, Shanks, & Evenden, 1984; Jones, Gray, & Hemsley, 1990; Shanks, 1985a). In a blocking preparation, initial pairings of a CS (A) and a US are followed by compound presentation of A and a new CS (B) once again paired with the US. The typical result of this preparation is that the prior experience with A "blocks" or prevents learning about B (Kamin, 1968). By Equation 1, initial pairings of A and the US will result in the value $\lambda_1 - V_T^n$ approaching zero. Consequently, little associative strength will be available for B to acquire in the second phase of the procedure. Chapman and Robbins (1990) asked participants to provide judgements of contingency between the rise in prices of four fictitious corporation stocks P, N, B, and C (predictors) and the rise in a fictitious stock market (outcome). In the first phase of the experiment, a rise in the price of stock P was always accompanied by a rise in the market (P+) whereas a rise in the price of stock N was never accompanied by a rise in the market (N-). Trials were also included in which neither the stocks nor the market rose. In the second phase of the experiment, stocks P and N were compounded with new stocks B and C respectively. Specifically, a rise in the price of stock P was accompanied by a rise in the price of stock B, which was followed by a rise in the market (PB+). Further, a rise in the price of stock N was accompanied by a rise in the price of stock C, which was followed by a rise in the stock market (NC+). As in the first phase, trials were included in which neither of the compounds nor the market rose. Although the contingency between the outcome and either of the new stocks was equal in the second phase, judgements of the contingency between B and the outcome were consistently lower than judgements of the contingency between C and the outcome. Thus, prior experience with the P+ trials blocked subjects' judgements of the relation between stock B and the market in the second phase.

A conditioning phenomenon closely related to blocking is the relative validity effect. This phenomenon is variously referred to as overshadowing or discounting in the contingency judgement literature (e.g., Price & Yates, 1993; Vallée-Tourangeau, Baker, & Mercier, 1994; Wasserman & Berglan, 1998). Whereas in overshadowing the contingency between different predictors and an outcome (e.g., one strong and one moderate contingency) are established simultaneously, in blocking a strong contingency between one predictor and an outcome is established before introducing a second predictor. Wagner (1969; Wagner, Logan, Haberlandt, & Price, 1968) reported that the ability of a CS to elicit a CR was relative to the ability of other CS to elicit the same CR. Suppose an outcome is strongly contingent on predictor A and moderately contingent on predictor B in one experimental group (group 1), and weakly contingent on predictor A and again moderately contingent on predictor B in another experimental group (group 2). The ability of the moderate predictor B to elicit a CR is generally observed to be higher in the presence of the weak predictor (group 2) than in the presence of the strong predictor (group 1), although the contingency between the predictor B and the outcome is the same in both experimental groups (Wagner, 1969; Wagner et al., 1968). The presence of the strong predictor in group 1 is said to overshadow the moderate predictor's ability to elicit a CR. In group 1, predictor A will gain associative strength to the detriment of predictor B as it is more frequently paired with the US. In group 2, the US is only weakly contingent on the presence of predictor A and consequently predictor B will acquire a larger proportion of the total associative strength supported by the US in group 2 relative to group 1.

In a report by Baker et al. (1993, Experiment 1), participants judged the effectiveness of camouflage in preventing the destruction of a tank (i.e., the outcome) when it passed through a

mine field. The camouflage could help the tanks successfully avoid the colour sensitive mines in the minefield. On some trials, a plane appeared along with the tank. Participants were told that the plane could help the tank successfully traverse the minefield by providing it information about the mines. Participants were presented with two contingencies between camouflage and the outcome (i.e., avoidance of destruction), and two contingencies between the plane and the outcome in a 2 X 2 factorial design. The contingency between the camouflage and the tank avoiding destruction was either .5 or 0. The contingency between the plane and the tank avoiding destruction was either 1 or 0. In condition .5/0, the presence of camouflage was associated with a moderate reduction in the probability of the tank avoiding destruction relative to the absence of camouflage, while the plane had no effect on the outcome. In condition .5/1, the contingency between camouflage and the outcome remained unchanged while the plane only appeared on trials when the tanks avoided destruction. Consequently, the plane was a perfect predictor of a successful crossing of the minefield. Baker et al. reported that judgements of the camouflage were lower in the presence of the strong plane contingency relative to judgements in the presence of a zero plane contingency. These results are analogous to those reported by Wagner (1969; Wagner et al., 1968), and indicate overshadowing of one predictor when in the presence of a more valid predictor of the same outcome (see also, Gluck & Bower, 1988, Price & Yates, 1993, Vallée-Tourangeau et al., 1994, and Shanks, 1991a, 1991b, for other examples of overshadowing in contingency judgements).

Suppose animals are conditioned with only two trial types, A+ and AB-. Presentations of predictor A are always followed by the outcome, whereas presentations of predictor A in compound with a second predictor B are never followed by the outcome. The result of this

preparation is typically that predictor B acquires negative associative strength relative to another predictor that was presented singly with no outcome (Rescorla, 1969; Rescorla & Holland, 1977). Equation 1 accounts for this finding with the assumption that the value of λ is greater on reinforced trials than on un-reinforced trials. As predictor A acquires increasingly positive associative strength, the value of $\lambda_1 - V_T^n$ on un-reinforced trials becomes negative. Consequently, un-reinforced compound presentations of predictors A and B will result in B acquiring negative associative strength. Chapman & Robbins (1990, Experiment 2) demonstrated inhibitory judgements following a conditioned inhibition preparation analogous to the effects of a conditioned inhibitor in studies with non-humans.

The signal effect refers to the increase in the CR to a predictor (A) when a second predictor (the signal) is presented on trials when the outcome occurs without A, relative to a condition during which some outcomes occur without the signal (Durlach, 1983). The increased CR to A in the signal condition occurs despite the fact that the contingency between A and the outcome remains the same in both conditions. On trials when the signal occurs, associative strength is distributed between the signal and the contextual cue with the result that the contextual cue acquires less associative strength than it would otherwise acquire without the signal. Consequently, predictor A receives less competition from the contextual cue on reinforced trials and acquires greater associative strength than in an un-signalled condition (Allan, 1993; Shanks, 1989, 1993a). In a procedure similar to that used by Baker et al. (1993), Shanks (1986, Experiments 3 and 4) asked participants to judge the effectiveness of an artillery shell in destroying a tank that traversed a computer screen. The destruction of the tank could be attributed to either the artillery shell or to hitting a mine in a minefield. In a signalled condition, a

plane appeared every time the tank exploded in the absence of a hit by the artillery shell. As predicted by an associative model, judgements of the effectiveness of the artillery shell were greater in the signalled condition that in the un-signalled condition.

The rate of acquisition of behavioural control by a CS may be characterised as a negatively accelerated function of the subjects' experience with the contingency (Rescorla & Wagner, 1972; Wagner & Rescorla, 1972). Behavioural control by a CS typically begins near zero and increases toward an asymptote that reflects the actual CS-US contingency (Shanks, 1993a). This pattern of acquisition is presumed to reflect the trial-by-trial increments in associative strength to a CS as described by Equation 1. Initial CS-US pairings will result in relatively large increments in the associative strength of the CS as the value of $\lambda_1 - V_T^n$ is large. Successive CS-US pairings will result in progressively smaller increments in associative strength as the value of $\lambda_1 - V_T^n$ decreases. Shanks (1985b) monitored contingency judgements as they developed in a task similar to that used by Baker et al. (1993) described above. Participants were asked to judge the extent to which hitting a tank with a delayed-action artillery shell resulted in the tank's destruction relative to when the tank was not hit with the artillery, after every five trials. The tank could be destroyed either by the artillery shell or by a mine located in a minefield traversed by the tank. As predicted by an associative model, Shanks (1985b) found that judgements of the artillery shell's effectiveness followed growth functions with asymptotes similar to the contingency between the shell and the outcome despite the fact that the actual Δp's remained constant across trials. These growth functions were found for both positive and negative contingencies between the shell and the tank's destruction. Acquisition functions in contingency judgements have been found in several experiments (e.g., Baker et al., 1989,

Experiment 1; Chatlosh et al., 1985; Dickinson & Shanks, 1985; Shanks, 1987; but see Baker et al., 1989, Experiment 3, and Catena et al., 1998, who did not find changes in judgements across trials), and do not appear to be related to changes in confidence (Shanks, 1987, Experiment 2).

In addition to cue competition and acquisition functions, there are several other phenomena in human contingency judgements that presumably reflect associative processes (Allan, 1993; Shanks, 1993a; Wasserman & Miller, 1997). One, human judgements are sensitive to manipulation of both the contingency (Allan & Jenkins, 1980, 1983; Alloy & Abramson, 1979; Dickinson et al., 1984; Neunaber & Wasserman, 1986; Wasserman et al., 1993) and contiguity between a predictor and an outcome (Reed, 1992; Shanks, 1986, 1989; Shanks & Dickinson, 1987). Two, if the contingency between a predictor and an outcome is 0, judgements are often a function of the frequency of the outcome (e.g., Allan & Jenkins, 1983; Alloy & Abramson, 1979, Experiment 2; Baker et al., 1989, Experiments 1 and 3; Chatlosh et al., 1985, Experiment 1; Dickinson at al., 1984, Experiment 1; Shanks, 1985b, Experiments 1, 2, and 3, 1987, Experiment 1). All other things being equal, judgements are typically greater when the probability of the outcome is high relative to when the probability of the outcome is low (Shanks, 1993a; but see Neunaber & Wasserman, 1986, who failed to observe this bias). Three, when the outcome has a high probability of occurring, judgements are often seen to increase initially, reach a maximum, and subsequently decrease toward zero (Rescorla & Wagner, 1972; Shanks, 1985b, 1987). Four, according to associative models, the associative strength of a predictor is updated on a trial-by-trial basis, and is influenced by the associative strength already gained by other predictors of the same outcome. Consequently, the order of trial presentation can have a large influence on a predictor's asymptotic associative strength (López et al., 1998;

Chapman, 1991; but see Catena et al., 1998 for trial order effects inconsistent with an associative analysis). Five, Mercier and Parr (1996) assessed the effect of processing requirements on contingency judgements by manipulating the number of trials that participants were exposed to, inter-trial intervals, and stimulus durations. Consistent with Wagner's (1981) associative model, the accuracy of judgements was mostly related to the time participants had to process the experimental trials (Mercier & Parr, 1996). Although a broad range of empirical phenomena appear to reflect associative processes, it remains unclear which class of associative model (i.e., elemental or configural) most accurately characterises both non-human learning and contingency judgements.

Elemental and Configural Processes in Associative Learning

Evidence for both elemental and configural associative processes in animal conditioning is abundant (Kehoe & Graham, 1988). The cue interaction effects described above are thought to reflect the operation of elemental processing, and are presumed to provide support for an elemental conceptualisation of conditioning (Wasserman & Miller, 1997). For example, one predictor may acquire greater associative strength than a second as a consequence of either its prior history with the outcome (i.e., blocking), or its greater salience (i.e., overshadowing) relative to the second predictor. Similarly, a predictor acquires negative associative strength if paired with a second predictor with positive associative strength, and the compound is un-reinforced (i.e., conditioned inhibition). Each of these phenomena, thus, appear to rely on an elemental conceptualisation of associative learning.

Elemental associative processing has been most directly demonstrated in the context of stimulus compounding. In the stimulus compounding procedure, the response to a compound

stimulus is assessed after training with the individual components of the compound (Weiss, 1972). For example, after separate training with two stimuli followed by an outcome (i.e., A+, and B+), responding to a compound of the stimuli (i.e., AB) is compared with responding to the elements. In general, the result of the stimulus compounding procedure is summation; responding to the compound is higher than that to either of its constituent elements individually (Kehoe & Graham, 1988; Kehoe, A. J. Horne, P. J. Horne, & Macrae, 1994; Rescorla, 1997; Wasserman & Miller, 1997). Greater responding to the compound than to its elements does not appear to be attributable to other properties of the compound, such as greater intensity or complexity relative to its elements (Rescorla, 1997; Whitlow & Wagner, 1972). Analogously, if one stimulus is trained to elicit a CR, and a second is trained to inhibit the occurrence of a CR that would otherwise accrue (i.e., conditioned inhibitor), the CR to a compound of the two stimuli will be less than to stimulus A alone (Rescorla, 1969). Again, responding to a compound appears to reflect the sum of the associative strengths of its components, in this case one component has positive associative strength and the other has negative associative strength. These results are taken to mean that responding to the compound is some additive function of the predictive values of its elements (Kehoe, 1986; Kehoe et al., 1994; Kehoe & Graham, 1988; Wagner, 1971; Weiss, 1972).

Despite the evidence for summation in compound predictor processing and the ability of elemental associative models to predict cue interaction phenomena, data consistent with the notion that compounds may acquire predictive value independently of their constituents (i.e., as configural predictors) dates from early on in conditioning research (Spence, 1952; Wasserman & Miller, 1997). For example, Razran (1939a) reviewed research from Pavlov's laboratory

suggesting that a compound CS may elicit a CR independently of the training histories of its constituent elements. Some of this early research assessed the role of Gestalt grouping principles (i.e., similarity, continuity, and proximity) on configural conditioning (Razran, 1939b, 1939c). The most persuasive evidence for the configural nature of associative processing comes from research with the negative patterning and conditional discrimination paradigms (Rescorla et al., 1985).

In a classic experiment, Woodbury (1943) used an instrumental procedure to assess the ability of dogs to discriminate among a compound composed of two CS, and the CS individually. Two buzzers with different auditory characteristics were used as the CS (B_1, B_2). In one experimental condition, a negative patterning procedure was used whereby responses following the simultaneous sounding of the two buzzers were not reinforced (B_1B_2-) and responses following the sounding of either of the buzzers individually were reinforced (B_1+, B_2+). Woodbury (1943) found that responding to the compound was reliably less than responding to either of the elements. Elemental associative models predict that responding to the compound should be a function of the predictive values of the constituent elements, and less responding to the compound than to its element could not be accounted for by any additive rule. Consequently, data from studies of negative patterning are interpreted to imply that compound predictors are assessed as unique configural entities (Pearce & Redhead, 1993; Rescorla, 1972; Rescorla at al., 1985; Whitlow & Wagner, 1972). Analogous results have been obtained with human participants using a Pavlovian skin conductance conditioning paradigm (e.g., Kimmel & Lachnit, 1991; Kleinschmidt & Lachnit, 1993; Lachnit & Kimmel, 1993).

Saavedra (1975) employed a biconditional discrimination to assess the role of configural

cues in discrimination learning. In a Pavlovian eyelid conditioning experiment with rabbits, two different compound stimuli were consistently reinforced (T_1L_1+, T_2L_2+; T = tone, L = light), whereas two different compounds of the same stimuli were consistently un-reinforced (T_1L_2-, T_2L_1-). As each element is included in both reinforced and un-reinforced compounds an equal number of times, differential responding to the reinforced and un-reinforced compounds could not be attributed to the operation of an elemental associative process. Saavedra (1975) reported that rabbits effectively discriminated between the reinforced and un-reinforced compounds, suggesting that this discrimination was acquired by a configural associative process whereby the tone and light compounds were associated with the outcome in their entirety, independently of their constituent elements.

Researchers have attempted to reconcile elemental associative theories with evidence of configural processing while maintaining the summation principle central to elemental accounts (e.g., Whitlow & Wagner, 1972). For example, Wagner (1971) suggested that when predictors occur together (i.e., in compound), conditioning will result in the formation of an association between the compound as a whole and the outcome in addition to the associations between the elements of the compound and the outcome (Saavedra, 1975; Rescorla, 1972, 1973; Rescorla et al., 1985). For example, when the compound AB is paired with an outcome, elements A, B, and the configural cue "AB" will enter into an association with the outcome. The net associative strength accruing to the compound AB is the sum of the associative strengths of elements A, B, and "AB".[3] Wagner and Rescorla (1972) suggested that the influence of a configural cue on

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[3] Most contemporary models of associative learning also presume that associations will develop between the

Pavlovian conditioning is small relative to the influence of the individual elements that composed it. That is, the associative strength of a compound predictor is largely dependent on the sum of the associative strengths of its constituent elements (Rescorla, 1997).

If a unique configural cue is assumed, then the Rescorla-Wagner model correctly predicts the acquisition of both negative patterning and biconditional discriminations. In the case of negative patterning (e.g., Woodbury, 1943), the presence of configural cue "AB" with negative associative strength will lead to a net associative strength to compound AB of less than the sum of the associative strengths that accrue to both A and B individually. In the case of a biconditional discrimination (e.g., Saavedra, 1975), differential responding to the reinforced and un-reinforced compounds could be completely attributed to the associative strength that accrues to the configural cues (i.e., in the study described above by Saavedra (1975) the configural cues are: "T_1L_1", "T_2L_2", "T_1L_2", "T_2L_1"). Difficulties remain, however, with this conceptualisation of associative processing. Increasing the number of elements in a compound will result in a combinatorial explosion of configural cues with even a modest number of elements (Pearce, 1994). A compound composed of three elements (ABC) can potentially result in the formation of seven different associations (i.e., between each of A, B, C, "AB", "AC", "BC", and "ABC", and the outcome), while a compound composed of five elements could result in the formation of thirty-one different associations. Even if the presence of configural cues is assumed, an

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elements of a compound cue (Rescorla, 1980; Rescorla & Durlach, 1981), and it has been suggested that these within-compound associations may mediate retrospective revaluation phenomena (e.g., backward blocking, and recovery from overshadowing) in human causal judgements (Wasserman & Berglan, 1998). However, the effect of within-compound associations on judgements of contingency is not considered in this report.

elemental associative model that characterises compound predictor processing as reflecting the operation of summation of associative strength continues to make empirically unsupported predictions.

In the classic feature-negative discrimination paradigm animals are presented with two different trial types: A+, and AB-. The result of this preparation is that A elicits a strong CR whereas AB elicits little responding. B, on the other hand, should pass both summation and retardation tests for conditioned inhibition (Rescorla, 1969). Minimal responding to the compound, therefore, reflects the summation of positive associative strength from A and negative associative strength from B. If a third element is added, yielding an AC+, ABC- discrimination, the Rescorla-Wagner model predicts that this new discrimination will be acquired more quickly relative to one without the common element (Pearce, 1987, 1994). This is because excitatory conditioning on a trial with two CS present (i.e., AC+) is predicted to progress more rapidly than excitatory conditioning on a trial with only one CS (i.e. A+). Similarly, B will acquire negative associative strength faster on a trial when it is presented along with two CS (i.e., ABC-) than on a trial when it is presented along with only one other CS (i.e., AB-). That is, on an ABC- trial the value of V_T^n in Equation 2 will be greater, and hence from Equation 1 the negative associative strength acquired by B on this trial will be greater, than on an AC- trial. Note that assuming the existence of configural cues does not change this prediction. If we assume the existence of a configural cue, on an AC+ trial associations will develop between A, C, and "AC" and the outcome, and the net associative strength to the AC compound will reflect the three cues in an additive fashion. By Equation 2, the net associative strength to the AC compound on AC+ trials will nevertheless remain higher than the associative strength acquired

by A on A+ trials. The Rescorla-Wagner model thus makes the counterintuitive prediction that increasing the similarity between cues by adding common elements renders the cues easier to discriminate (Rescorla, 1972; Pearce, 1987, 1994).

Using an autoshaping procedure where pigeons were conditioned to discriminate among different randomly presented geometric figures on a television screen, Pearce and Redhead (1993, Experiment 1) assessed the effect of adding a common element to a feature negative discrimination. With one group of pigeons, pecking at a screen was reinforced in the presence of red rectangles (A+), and not reinforced in the presence of red and green rectangles (AB-). With a second group, pecking in the presence of red and white rectangles was reinforced (AC+), while pecking in the presence of red, green, and white rectangles was not reinforced (ABC-). Contrary to the predictions of the Rescorla-Wagner model, increasing the similarity between the cues by adding a common element retarded the rate at which the discrimination was acquired (Pearce & Redhead, 1993). The detrimental effect of increasing similarity among cues in a discrimination procedure occurs also in the negative patterning paradigm (Pearce & Redhead, 1993, Rescorla, 1972). These results could be readily explained by Pearce's (1987) configural associative model. First, regardless of the number of elements presented in compound on any given trial, conditioning would only provide the opportunity for the formation of one association between the compound as a whole and the outcome. From the standpoint of the net gain in associative strength on a given trial, AC- and ABC- trials are analogous. Second, the net associative strength accruing to a compound is the sum of the associative strength of the configural cue and the associative strength generalised to it from other cues as a function of their similarity. There is less generalisation of associative strength between A+ and AB-, than between AC+ and ABC- as

these latter two cues are more similar than the former. Consequently, Pearce's (1987) configural model makes the prediction that increasing the similarity among cues will retard rather than improve the acquisition of discrimination learning.

Despite the mounting data in support of a configural approach to non-human learning (e.g., Darby & Pearce, 1995; Pearce et al., 1997; Pearce & Redhead, 1995), neither the elemental nor configural approach appears to provide an incontrovertible framework for characterising associative learning (Aydin & Pearce, 1995; Kehoe & Graham, 1988). Redhead and Pearce (1998) reported results consistent with a configural associative model, but that also incorporates the notion of changes in the α of a CS with experience as proposed by some elemental associative models (e.g., Pearce & Hall, 1980). Finally, some recent theoretical attempts have focussed on unifying configural and elemental associative processes in one conceptual scheme (Kehoe & Graham, 1988).

Elemental and Configural Processes in Contingency Judgements

The available data provides little insight into elemental and configural associative processes in contingency judgements. In reports investigating competition among predictors in contingency judgement tasks, participants are typically presented with several predictors of the same outcome, sometimes individually and sometimes in compound with other predictors. Following the experimental treatments, participants are requested to judge the contingency between each of the predictors individually and the outcome. In a report by Baker et al. (1993) described above, participants were asked to judge the contingency between two separate predictors considered individually (camouflage and spotter plane) and an outcome (successful crossing of a mine field) in the context of a tank game. The crucial comparison in Experiment 1

was between two conditions: one where Δp for the camouflage was .5 and Δp for the spotter plane was 1, and the other where Δp for the camouflage remained at .5 but Δp for the spotter plane was 0. As described earlier, judgements of the camouflage were reduced in the condition with a high plane contingency relative to the condition with a low plane contingency, despite the fact that the contingency between the camouflage and the outcome remained constant in the two conditions. Although not considered by the authors, Δp for the compound composed of both predictors varied across experimental conditions. For example, in the condition when Δp for the camouflage was .5 and Δp for the plane was 1, there were fifteen trial during which both the camouflage and the plane occurred in compound with the outcome, giving a Δp for the camouflage/plane compound of .8. In the remaining experimental conditions, Δp for the compound was either 0 or .33. As in other reports of contingency judgements, participants were not requested to judge the effect of the joint presence of the two predictors on the outcome (e.g., Dickinson et al., 1984; Dickinson & Shanks, 1985; Shanks, 1985a, 1986). Similarly, many reports of predictor interactions in contingency judgement request participants to judge the contingency between several symptoms and a disease. Again, participants are typically requested to judge the contingency between the presence of each symptom individually and the outcome, and not the contingency between configurations of symptoms and the disease (e.g., Gluck & Bower, 1988; Price & Yates, 1993).

Some reports, although not explicitly contrasting elemental and configural models, present data consistent with configural processing in contingency judgements (e.g., Vallée-Tourangeau, Murphy, Drew, & Baker, 1998). For example, Williams (1995) assessed the inhibitory properties of a predictor in the context of a fictitious stock market game similar to the

one used by Chapman & Robbins (1990) described above. Participants were asked to assess the extent to which an increase in the value of certain fictitious stocks (predictive cues, P) affected a change in the value of a stock market (outcome). The findings that are crucial for the present discussion relate to differences between the double/element and double/compound groups of the report by Williams (1995). In the double/element group, participants received trials with P_1+, P_2+, and N- in the first phase of the experiment, and trials with P_1+, P_2+, and P_1N- in the second phase. Note that un-reinforced pairings of N with the positive predictor P_1 in the second phase should endow N with inhibitory properties. In the double/compound group, participants received trials with P_1+, P_2+, and P_2N- in the first phase of the experiment, and trials with P_1+, P_2+, and P_1N- in the second phase. In the double/compound group N is paired with a positive predictor both in the first phase of the experiment (P_2N-) and the second phase (P_1N-). The Rescorla-Wagner model assumes that N will acquire inhibitory strength only during un-reinforced pairings with a positive predictor. As N is paired with a positive predictor in both phases of the experiment in group double/compound, and in only the second phase in group double/element, the inhibitory strength accruing to N should be greater in the double/compound group. Contrary to this prediction, Williams (1995) reported that the inhibitory strength of N, assessed through a transfer of inhibition paradigm, was greater in the double/element group than in the double/compound group. This finding appears to be consistent with Pearce's (1987) configural associative model. According to Pearce's model, the associative strength of N will reflect the inhibitory strength of configural cues "N" and "P_1N" in the double/element group, and configural cues "P_2N" and "P_1N" in the double/compound group. As the similarity between N and the configural cues is higher in the double/element group than in the double/compound group (i.e., N

is more similar to "N" in the double/element group than it is to "P_2N" in the double/compound group), N will reflect more inhibitory strength in the double/element group. This explanation of the results is post hoc, however, as the report was not designed to contrast elemental and configural associative processes.

In two recent reports, elemental and configural associative processes were directly assessed in the context of contingency judgements. Williams et al. (1994) reported that contingency judgements may be based on either configural or elemental strategies depending on the mental set adopted by the participants. For example, blocking in contingency judgements was only observed among participants given pre-treatment problems encouraging elemental processing. When given pre-treatment problems not encouraging elemental processing, selectional effects were not observed, presumably because participants were employing a configural strategy. This conclusion is based on the assumptions that configural strategies are inimical to selectional effects in contingency judgements, and that the failure to observe selectional effects is evidence that a configural strategy was used by participants. In fact, the configural model of associative processing proposed by Pearce (1987, 1994) can readily account for cue interaction phenomena such as reported by Williams et al. (1994). In a blocking preparation, initial pairings of predictor A and the outcome (A+) are followed in a second phase by compound presentation of A and a new predictor B once again paired with the outcome (AB+). The typical result of this preparation is that the prior experience with A+ "blocks" or prevents learning about B in the second phase (Chapman & Robbins, 1990). According to a configural model, the compound of A and B in the second phase will become associated to the outcome in its entirety (i.e., as a configural cue). However, accrual of associative strength to

"AB" will be limited by the associative strength already acquired by "A" in proportion to their similarity. Consequently, "AB" will only acquire a small amount of associative strength in this second phase. Judgements of B, then, will reflect a small quantity of associative strength generalised to it from "AB" as a function of their similarity. That is, B will reflect a much smaller amount of associative strength than A, and judgements of B will be blocked by the prior experience with A. In the report by Williams et al. (1994), the associative strategy employed by the participants was induced from judgements of the individual stocks. As elemental and configural model propose different processes through which compound predictors are assessed, a more direct strategy to assess the relative merits of the models is to request judgements of compounds. This was one of the strategies used in the study described below by Shanks, Darby, and Charles (1998).

Shanks et al. (1998) reported a series of experiments designed specifically to contrast the predictions of the Rescorla-Wagner and Pearce models. Shanks et al. (Experiment 2) assessed the susceptibility to interference of human discrimination learning in a two stage experimental design. Participants assessed if eating certain foods (predictors) was related to getting a food allergy (outcome). In the first phase of the experiment, participants received trials during which a compound of food A and food B always signal allergy 1 ($AB+_1$), and a compound of food C and food D never signalled an allergy (CD-). In the second phase of the experiment, participants received trials during which food A never signalled an allergy (A-), food B never signalled an allergy (B-), food C always signalled allergy 2 ($C+_2$), and food D always signalled allergy 2 ($D+_2$). In the final test phase of the experiment, participants were asked to predict whether food compounds AB and CD signalled an allergy. According to the Pearce model, after the first phase

of the experiment, assuming no common contextual cue, the associative strength of compound AB should be 1 and that of compound CD should be 0. Phase two training should leave the associative strength of these two compounds unaffected. At the end of phase two, the associative strength of A and B should each be at -.5, while the associative strength of C and D should each be 1. Judgements of the AB compound will reflect the associative strength of configural cue "AB" (1), and half the associative strength of each of A (.5 x -.5 = -.25) and B (.5 x -.5 = -.25), giving a total associative strength to AB of .5. Judgements of CD will reflect the associative strength of "CD" (0), as well as half associative strength of each of C (.5 x 1 = .5) and D (.5 x 1 = .5), giving a total associative strength to CD of 1. The Pearce model, therefore, predicts that compound AB will be judged to be less related to the occurrence of an allergy than compound CD. That is, the effect of the second phase of training will be to reverse the relative predictive status of compounds AB and CD in the first phase. The results were opposite to those predicted by the Pearce model. The compound AB was judged to be more predictive of allergy than the compound CD, reflecting their relative predictive status after the first phase of the experiment. Based on this finding, and on similar findings from the other experiments in the report, Shanks et al. concluded that compound predictors appear to be functionally independent of their constituent elements. The authors suggested that if generalisation among predictors is assumed to be much less than that envisioned in the Pearce model much of their findings could be accommodated. If generalisation among predictors is set to zero, judgements to compounds AB and CD will only reflect their respective associative strength at the end of the first phase of the experiment (i.e., consistent with the findings).

Rationale and Objective

Despite the widespread influence of the Rescorla-Wagner model (Siegel & Allan, 1996), support for it as an account of either associative learning or contingency judgements is by no means unanimous (Shanks, 1993a; Young, 1995), and various other associative models have support among researchers (e.g., Mackintosh, 1975; Pearce & Hall, 1980; Pearce, 1987; Wagner, 1981). The inability of the Rescorla-Wagner model to fully account for associative phenomena (Miller et al., 1995; Siegel & Allan, 1996) does not represent the failing of an associationist theoretical approach but rather reflects the inadequacies of one instantiation of general associative principles. Consequently, contrasting between different variants of associative models in the context of contingency judgements appears to be justified (López et al., 1998; Mercier, 1996; Shanks et al., 1998).

The objective of this report is to examine the operation of elemental and configural associative processes in judgements of the contingency between compound predictors and an outcome. This will be accomplished by contrasting the predictions of Pearce's configural model with those of the Rescorla-Wagner model. Pearce's model has only seen extensive application in the context of Pavlovian conditioning paradigms with non-human participants (e.g., Pearce & Redhead, 1993; Rescorla, 1997), and has just begun to be assessed in the context of contingency judgements (e.g., Shanks et al., 1998). Although Shanks et al. (1998) have suggested that compounds are functionally independent from their constituent elements, the extent of this independence can only be assessed in a paradigm that systematically manipulates the predictive value of the compound independently of the predictive value of its constituent elements. In the four experiments of this report, judgements of a compound are assessed after systematic

manipulations of the contingency between its constituent elements and an outcome. Further, the effect of manipulating the contingency between a compound and an outcome on judgements of its elements is also assessed. The Pearce model describes a process of generalisation through which the associative strength of one predictor will affect responses to another predictor. Early indications are that contingency judgements are not subject to the generalisation of predictive value among cues (Shanks et al., 1998). Consequently, the effect of systematic manipulations of the similarity among predictive cues on contingency judgements will also be assessed. The contingency and similarity manipulations will be implemented in the context of discrimination paradigms from the animal learning literature that have generated findings difficult to reconcile with elemental theories of Pavlovian learning (e.g., Pearce & Redhead, 1993; Wilson & Pearce, 1992). Specifically, feature-negative and negative-patterning paradigms are modified to enable contingency manipulations of the relevant predictors. As outcomes typically result from many possible causes occurring in compound (Downing et al., 1985), assessing how judgements of compound predictors are arrived at promises to provide a more realistic assessment of contingency judgements in the course of everyday experiencing. Accordingly, participants in this report are requested to judge the predictive value of two- and three-element cues.

EXPERIMENT 1

This experiment establishes a fictitious medical context in which participants judged the relation between taking different medications (predictors) for a given disease and the occurrence of a facial rash (outcome). Each predictor was represented with a different colour. Participants were asked to make judgements of the contingency between each predictor and the outcome after every four experimental trials. Judgements of the contingency between a two-element compound predictor and an outcome are assessed as a function of: a) the contingency between one of its constituent elements and the outcome, and b) the similarity between its elements.

In the standard feature-negative discrimination paradigm, animals are presented with two different trial types: A+, and AB-. The outcome occurs every time A is presented alone but never when it is presented in compound with B. This paradigm was modified in this experiment to include trials with each of the compound's elements presented alone, as well as trials during which no predictors were presented. The contingency (Δp) between a predictor and the outcome (O) was calculated as the difference between the two independent conditional probabilities, P(O|predictor) and P(O|~predictor) (Allan, 1980; Jenkins & Ward, 1965). Conditions varied with regard to the contingency between one or more elements of a two-element compound predictor and the outcome, as well as the similarity between the elements of the compound predictor. The colour of the predictors was the primary attribute used to calculate the normative contingencies, and upon which similarity was manipulated. In a low similarity condition, participants were presented with predictors A and B, two physically separate stimuli of distinct colour, and an AB compound, half the colour of A and half the colour of B. In a high similarity condition, the AB compound was identical but when the A and B elements were presented outside the AB

compound, they were embedded in two other compounds, AC and BC, sharing the common colour C.

In the low similarity condition, there were A, B and AB trials, as well as trials during which no predictors were presented. Participants were asked to make judgements about each predictor under two contingency conditions. The contingency between predictor A and the outcome was either .6 [P(O|A) = .8, P(O|~A) = .2] or .3 [P(O|A) = .65, P(O|~A) = .35], while the contingency between predictor B and the outcome, and between predictor AB and the outcome remained constant at 0 [P(O|B) = .5, P(O|~B) = .5] and -.34 [P(O|AB) = .2, P(O|~AB) = .54] respectively. In the high similarity condition, participants were presented with AC, BC, and AB trials, as well as trials during which no predictors were presented, and asked to make judgements about each predictor under two contingency conditions. When the contingency for predictor A was .6 [P(O|A) = .8, P(O|~A) = .2], Δp for AC was .8 [P(O|AC = 1, P(O|~AC) = .2], and Δp for C was .91 [P(O|C) = 1, P(O|~C) = .09]. When the contingency for predictor A was .3 [P(O|A) = .65, P(O|~A) = .35], Δp for AC was .48 [P(O|AC = .8, P(O|~AC) = .32], and Δp for C was .6 [P(O|C) = .83, P(O|~C) = .23]. The contingency between predictor BC and the outcome remained constant at .54 [P(O|BC) = 1, P(O|~BC) = .46]. The contingency for the predictors B, and AB, was the same as in the low similarity condition; 0 [P(O|B) = .5, P(O|~B) = .5] and -.34 [P(O|AB) = .2, P(O|~AB) = .54] respectively. Although the high similarity condition involved seven different predictors (A, B, C, AB, AC, BC, and AB), predictors A, B, and C were always presented within two-element compounds and never alone. The similarity between predictor's AC and BC was presumed to be higher than the similarity between predictors A and B due to the presence of the common element C. The four conditions are referred to as: low/.6, low/3, high/.6,

Table 1. Predictor contingencies (Δp) by condition in the four experiments in this report.

		Predictors (Δp)						
Experiment	Condition	A	B	C	AB	AC	BC	ABC
1	low/.6	.6	0		-.34			
	low/.3	.3	0		-.34			
	high/.6	.6	0	.91	-.34	.8	.54	
	high/.3	.3	0	.6	-.34	.48	.54	
2	.6/0	.6	0		-.34			
	.3/0	.3	0		-.34			
	.6/-.5	.6	-.5		-.34			
	.3/-.5	.3	-.5		-.34			
3	.6/.54/-.35	.54	0	-.35		.6		-.34
	.3/.54/-.35	.54	0	-.35		.3		-.34
	.6/-.35/.54	-.35	0	.54		.6		-.34
	.3/-.35/.54	-.35	0	.54		.3		-.34
4	low/.6	.6	0		-.34			
	low/.3	.3	0		-.34			
	high/.6		0			.6		-.34
	high/.3		0			.3		-.34

and high/.3; the word indicates the similarity and the number indicates the Δp for predictor A. Table 1 summarises the Δp's for the predictors in each of the four experiments in this report.

Table 2 lists the frequency and probability of occurrence of each trial type in the two similarity and two contingency conditions. The probability (p) associated with each trial type is the ratio of the number of trials of a given type to the total number of trials presented for that condition (i.e., forty). For example, the condition with low similarity and Δp for A set at .6 contained fifteen trials when predictor A was presented alone with the outcome (p = .38), three trials when predictor B was presented alone with the outcome (p = .08), and one trial during which predictors A and B were presented together and with the outcome (p = .03). Seventeen of

Table 2. Frequency (f) and probability (p) of occurrence of each event in Experiment 1.

	Δp_A			
	.6		.3	
Event	f	p	f	p
Low Similarity				
A+	15	.38	12	.3
B+	3	.08	3	.08
AB+	1	.03	1	.03
~+	1	.03	4	.1
A-	0	0	3	.08
B-	0	0	0	0
AB-	4	.1	4	.1
~-	16	.4	13	.33
High Similarity				
A+	0	0	0	0
B+	0	0	0	0
C+	0	0	0	0
AC+	15	.38	12	.3
BC+	3	.08	3	.08
AB+	1	.03	1	.03
~+	1	.03	4	.1
A-	0	0	0	0
B-	0	0	0	0
C-	0	0	0	0
AC-	0	0	3	.08
BC-	0	0	0	0
AB-	4	.1	4	.1
~-	16	.4	13	.33

the forty trials presented to participants did not include any predictor, of which one contained the outcome ($p = .03$) and sixteen did not ($p = .4$).

Table 3 lists trial frequency by outcome combinations, as well as the associated conditional probability and contingency, for each predictor in the four experimental conditions. For example, in the low similarity condition with Δp for predictor A at .6 (low / .6), there are

Table 3. Trial frequency (f), conditional probability (c/p), and contingency (Δp) associated with each predictor by condition for Experiment 1.

	Condition (similarity / Δp_A)											
	low / .6			low / .3			high / .6			high / .3		
	f	c/p	Δp	f	c/p	Δp	f	c/p	Δp	f	c/p	Δp
Predictor A												
A+	16			13			16			13		
A-	4	.8		7	.65		4	.8		7	.65	
~A+	4			7			4			7		
~A-	16	.2	.6	13	.35	.3	16	.2	.6	13	.35	.3
Predictor B												
B+	4			4			4			4		
B-	4	.5		4	.5		4	.5		4	.5	
~B+	16			16			16			16		
~B-	16	.5	0	16	.5	0	16	.5	0	16	.5	0
Predictor C												
C+							18			15		
C-							0	1		3	.83	
~C+							2			5		
~C-							20	.09	.91	17	.23	.6
Predictor AC												
AC+							15			12		
AC-							0	4		3	.8	
~AC+							5			8		
~AC-							20	.2	.8	17	.32	.48
Predictor BC												
BC+							3			3		
BC-							0	1		0	1	
~BC+							17			17		
~BC-							20	.46	.54	20	.46	.54
Predictor AB												
AB+	1			1			1			1		
AB-	4	.2		4	.2		4	.2		4	.2	
~AB+	19			19			19			19		
~AB-	16	.54	-.34	16	.54	-.34	16	.54	-.34	16	.54	-.34

twenty trials on which predictor A is presented, sixteen are paired with the outcome (A+) and four are not (A-), for a conditional probability of an outcome given the presence of predictor A of .8. There are twenty trials during which predictor A does not occur, four contain an outcome (~A+) and sixteen do not (~A-), for a conditional probability of an outcome given the absence of predictor A of .2, and a Δp for predictor A of .6 (.8 - .2). Treatment of compound predictors is analogous to that of single predictors. For example, in every experimental condition there are five trials during which predictors A and B occur in compound, once paired with the outcome and four times without for a conditional probability of the outcome given predictor AB of .2. There are thirty-five trials during which predictors A and B are not presented in compound (they may occur in isolation or not at all), nineteen of these trials include the outcome and sixteen do not for a conditional probability of an outcome given the absence of predictor AB of .54, and a Δp for predictor AB of -.34 (.2 - .54). In Table 3, frequencies are tabulated according to the universal set of events, that is for all occurrences or non-occurrences of a predictor independently of the presence of other predictors. For instance, a trial during which both the predictors A and B are present is tabulated as one occurrence each of three separate predictors (i.e., A, B, and AB). Tabulation of frequencies for predictors A and B individually in the low/.6 condition include trials during which both predictors are presented in compound, such that one of the sixteen A+ trials include predictor B, while all four of the A- trials include predictor B. Consequently, the nominal contingency for each predictor is calculated independently of the presence of other predictors. This method of calculating the contingency of single and compound predictors is consistent with the method employed in other reports (e.g., Baker et al., 1993).

Model predictions for the low and high similarity condition were derived from computer

simulations based on Equations 1 and 4. For purposes of these simulations, the learning rate parameter (α) was set at .3 for each predictor, while α for the context (X) was .1. The salience of the outcome (β) was .3 both when it was present and when it was absent. The values of these parameters are consistent with those employed in other reports assessing the predictions of associative models (e.g., Redhead & Pearce, 1995). The result of each simulation represents average predicted judgements of twenty-four different randomisations of forty trials.

The theoretical values derived from the Rescorla-Wagner model are depicted in Figure 2. The corresponding theoretical values of the Pearce model are depicted in Figure 3. If the value of a compound predictor is primarily a function of the sum of the associative strengths accrued to its elements as suggested by the Rescorla-Wagner model, then the value of compound AB in the low similarity condition will remain consistently higher than the value of either A or B. That is, AB will reflect $V_A + V_B$, and changes in the Δp for predictor A will be directly reflected in the value of both A and AB. Alternatively, the value of compound AB may be more consistent with the operation of the configural associative process described by Pearce (1987). By this account, AB will reflect the associative strength accrued to three different configural cues. That is, AB will reflect the associative strength of configural cue "AB", as well as the associative strength generalised to the compound from two other predictors, "A" and "B", as a function of similarity. Note that the contextual cue also generates a configural cue (i.e., "X") because there are several trials in each condition during which no predictors are presented. On these trials, the pattern of background stimulation becomes the effective CS. The presence of the contextual cue is omitted from the notation as it does not impact significantly on the theoretical values. Consequently, the value of AB remains consistently lower than the value of A and higher than the value of B. This

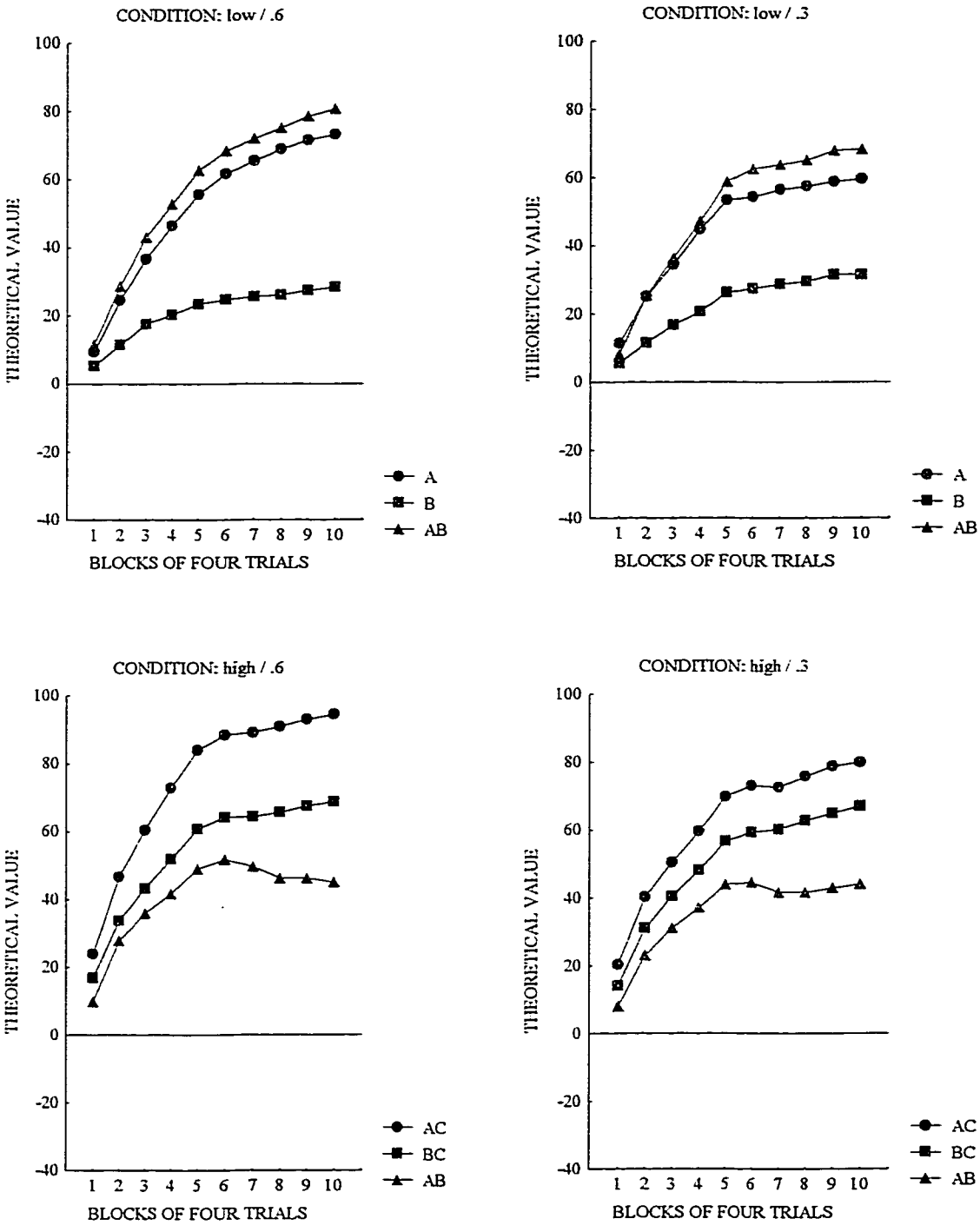

Figure 2. Theoretical values derived from the Rescorla-Wagner model in the four conditions of Experiment 1.

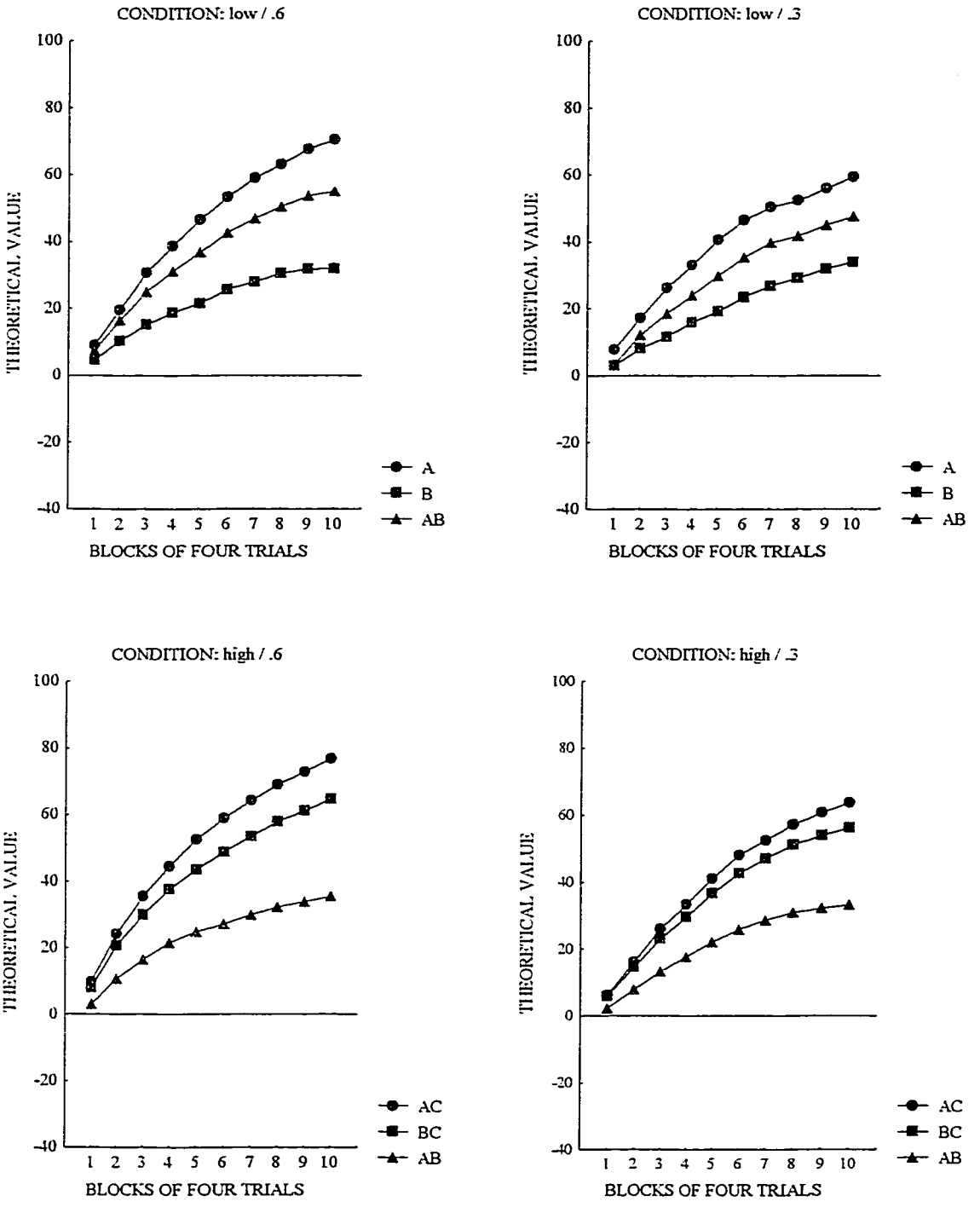

Figure 3. Theoretical values derived from the Pearce model in the four conditions of Experiment 1.

is because AB reflects the negative associative strength accrued to configural cue "AB" , and the positive associative strength accrued to "A" and "B". According to both models, the value of B will be moderate and positive, and the value of the compound AB will remain positive despite its negative contingency with the outcome.

In the high similarity condition, the models generate qualitatively similar theoretical values. According to both models, the value of A and B should be lower than the value of AC and BC respectively. According to the Rescorla-Wagner model, this is because the two element compounds should reflect the associative strength of each element in an additive fashion. Consequently, $A = V_A$, whereas $AC = V_A + V_C$; and $B = V_B$, whereas $BC = V_B + V_C$. According to the Pearce model, the value of the compounds will reflect primarily the associative strength gained by their configural representations. Δp for A and B in the low similarity condition are lower than Δp for AC and BC in the high similarity condition respectively. Specifically, when Δp for A is .6, Δp for AC is .8, whereas when Δp for A is .3, Δp for AC is .48. Similarly, while Δp for B remains constant at 0, Δp for BC remains constant at .54. Consequently, the Pearce model makes qualitatively similar predictions as the Rescorla-Wagner model regarding the value of AC and BC relative to the value of A and B respectively.

Both models anticipate that increasing the similarity between A and B will reduce the value of the AB compound. The Rescorla-Wagner model presumes that the compound AB will reflect the associative strength of A and B in an additive fashion. Increasing the similarity between A and B by adding the element C will decrease the associative strength that accrues to A and B individually, and consequently decreases the net associative strength to compound AB. This stems from the fact that the associative strength that will accrue to A and B will be limited

on trials where C is present. For example, $\lambda - V_T^n$ (Equation 1) will be smaller on an AC+ trial (where $V_T^n = V_A + V_C$) than on an A+ trial (where $V_T^n = V_A$). The Pearce model arrives at a similar conclusion through a different process. Judgements of compound AB will reflect the associative strength generalised from "A" and "B" in the low similarity condition, and from "AC" and "BC" in the high similarity condition. As the similarity between "AC" and "BC", and the compound AB is lower than the similarity between "A", "B", and AB (Equation 8), AB will benefit from less generalisation of positive associative strength in the high similarity condition than in the low similarity condition. Consequently, the value of AB will be lower when the similarity between A and B is high than when the similarity between A and B is low. Note, however, that the Pearce model anticipates a much smaller change in the value of AB across similarity conditions than the Rescorla-Wagner model. The decrease in the value of AB predicted by the Rescorla-Wagner model is about twice as large as that predicted by the Pearce model.

Method

Participants

Twenty-four undergraduate students (11 female, 13 male, mean age = 22 years) were recruited at the University of Ottawa to serve as participants for this experiment. After 24 participants were tested, one person was chosen randomly and awarded a $50 prize.

Apparatus

IBM compatible microcomputers, located in individual testing rooms, served to administer the tasks and collect data for this experiment. Each computer was equipped with a keyboard, a mouse, and a 14 in VGA colour monitor. The computer program used for task

presentation and data collection was developed using Microsoft Visual Basic Professional 4.0.

Stimuli

All stimuli were presented sequentially, in a discrete trial procedure, on 10 cm by 15 cm graphical windows centred in the middle of the computer screen. Appendix B presents the graphical components used in this experiment. Within each experimental condition, a window represented the medical file of one patient who participated in the clinical trial for a given fictitious disease. The left-hand portion of the window displayed the medication treatment(s) administered to the patient, while the right hand portion displayed the treatment outcome (i.e., facial rash, or no facial rash). The medication treatments were represented with oval-shaped medication pills. In the low similarity condition, medication pills were red (predictor A), green (predictor B), and half red/half green (predictor AB). In the high similarity condition, medication pills were half red/half white (predictor AC), half green/half white (predictor BC), and half red/half green (predictor AB). Each medication pill measured approximately 9 mm vertically and 5 mm horizontally. The outcome was represented with a round yellow icon depicting a face, which measured approximately 12 mm in diameter. The presence of rash on the face was indicated with red spots. The display of each medical file remained visible on the computer screen for 3 sec, with a 1 sec inter-trial interval.

Procedure

The participants first read task instructions on the computer screen. They were told to imagine they had access to the files of patients who took part in a clinical trial to assess the effectiveness of certain medications for treating a disease. It was explained that the patients were all ill with the disease, that one possible symptom of the disease was facial rash, and that the

medications given to the patients could affect the likelihood with which they get facial rash. It was emphasised that their task was to judge the effect of the medications on the likelihood of the facial rash. It was explained that they would be asked to make these judgements in the context of six different clinical trials, each for a different fictitious disease. The first two clinical trials were practices during which they could request the clarification of task instructions.

Following the initial task instructions, participants saw the graphical components that represented patient files, possible medications (i.e., red pill, green pill, half red/half white pill, half green/half white pill, and half red/half green pill), and possible outcomes (i.e., facial rash, and no facial rash). It was explained that clinical trials could be of two types distinguished by the medications given to patients. Red pills, green pills, and half red/half green pills were administered to patients in one type of clinical trial, while half red/half white pills, half green/half white pills, and half red/half green pills were administered in the second type of clinical trial. They were told that a medication treatment could either increase, decrease, or leave the likelihood of facial rash unchanged relative to the absence of the treatment under consideration. It was emphasised that the relations between the various medications and the facial rash remained constant within a clinical trial, but varied between clinical trials.

Each clinical trial consisted of 40 individual trials (i.e., patient files), and participants judged the relations between each medication treatment and the facial rash after every 4 trials for a total of 10 judgements per medication. Judgements were made on a response screen that presented each possible treatment with its respective icon on a different line. Responses were recorded by manipulating a horizontal scroll bar placed to the right of each medication treatment with a mouse. Manipulations of the scroll bar were reflected with a number in a data box placed

between the treatment icons and the scroll bars. Possible responses ranged from -100 to + 100, in increments of 1. The location of the slider on the horizontal scroll bar was reset to 0 for each new judgement screen, so that participants did not have access to their previous responses. Since judgements were made after every 4 trials, it was emphasised that judgements should be based on all the files seen up to the current response screen for a given disease.

After the initial task instructions, participants completed two practice clinical trials. One practice clinical trial was a low similarity condition and the other was a high similarity condition. The practice always began with the low similarity condition. The practice clinical trials were identical to the experimental clinical trials except for the normative contingencies between the predictors and the outcome. In the low similarity practice, the contingency for A was .5 [P(O|A) = .75, P(O|~A) = .25], the contingency for B was -.5 [P(O|B) = .25, P(O|~B) = .75], and the contingency for the AB compound was 0 [P(O|AB) = .5, P(O|~AB) = .5]. In the high similarity practice, the contingency for AC was .6 [P(O|AC = .92, P(O|~AC) = .32], the contingency for BC was -.6 [P(O|C) = .08, P(O|~C) = .68], and the contingency for both AB and C was 0 [P(O|AB) = .5, P(O|~AB) = .5]. The contingency for predictors A and B was the same as in the low similarity practice. The name of the fictitious diseases considered in a given clinical trial (i.e., Laparosis, Oxyopathy, Hypermegia, Anoperosis, Dendropathy, or Osteosis) was displayed at the top of the computer screen throughout the clinical trial.

Within each experimental condition, the order of trial presentation was randomised with one constraint. At least one of the first four trials contained the presentation of the half red/half green predictor, thereby exposing participants both to red and green pill colours prior to the first judgement screen. This was to ensure that even the early judgements were based on a minimum

of empirical information. The order of presentation of experimental conditions was counterbalanced between participants, according to the 24 possible permutations of four objects. The assignment of pill colour to contingency was counterbalanced between conditions and between subjects. Between each condition participants saw a screen explaining that a new clinical trial was beginning, and were instructed to disregard all they had seen previously and start their evaluations afresh. The frequency of the outcome was kept constant across conditions (i.e., 20) as judgements are reported to vary with outcome density (Allan & Jenkins, 1983; Alloy & Abramson, 1979; Baker et al., 1989; Chatlosh et al., 1985; Dickinson et al., 1984; Shanks, 1985b, 1987, 1993b).

Results

A within-observation analysis of variance, with individual judgements as the dependent variable, was used to examine the variables of similarity (low, high), contingency (Δp for A = .6, and .3), predictor (A B, and AB in the low similarity condition, and AC, BC, and AB in the high similarity condition), and trial block (1 to 10).[4] A posteriori comparisons were protected with the Bonferroni procedure. A Type I error rate of .05 was used throughout.

Figure 4 depicts the empirical judgements for Experiment 1. Inspection of the judgements in the low similarity condition suggests that judgements of A are higher when Δp of A is .6 (top left panel) than when it is .3 (top right panel). Varying the Δp of A does not appear to have

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[4] It is not clear whether the data collected with the procedure used in this thesis yields an interval or ordinal measurement. Nevertheless, the data were analyzed using the ANOVA technique because continuity and monotonicity of the underlying psychological dimension could be assumed.

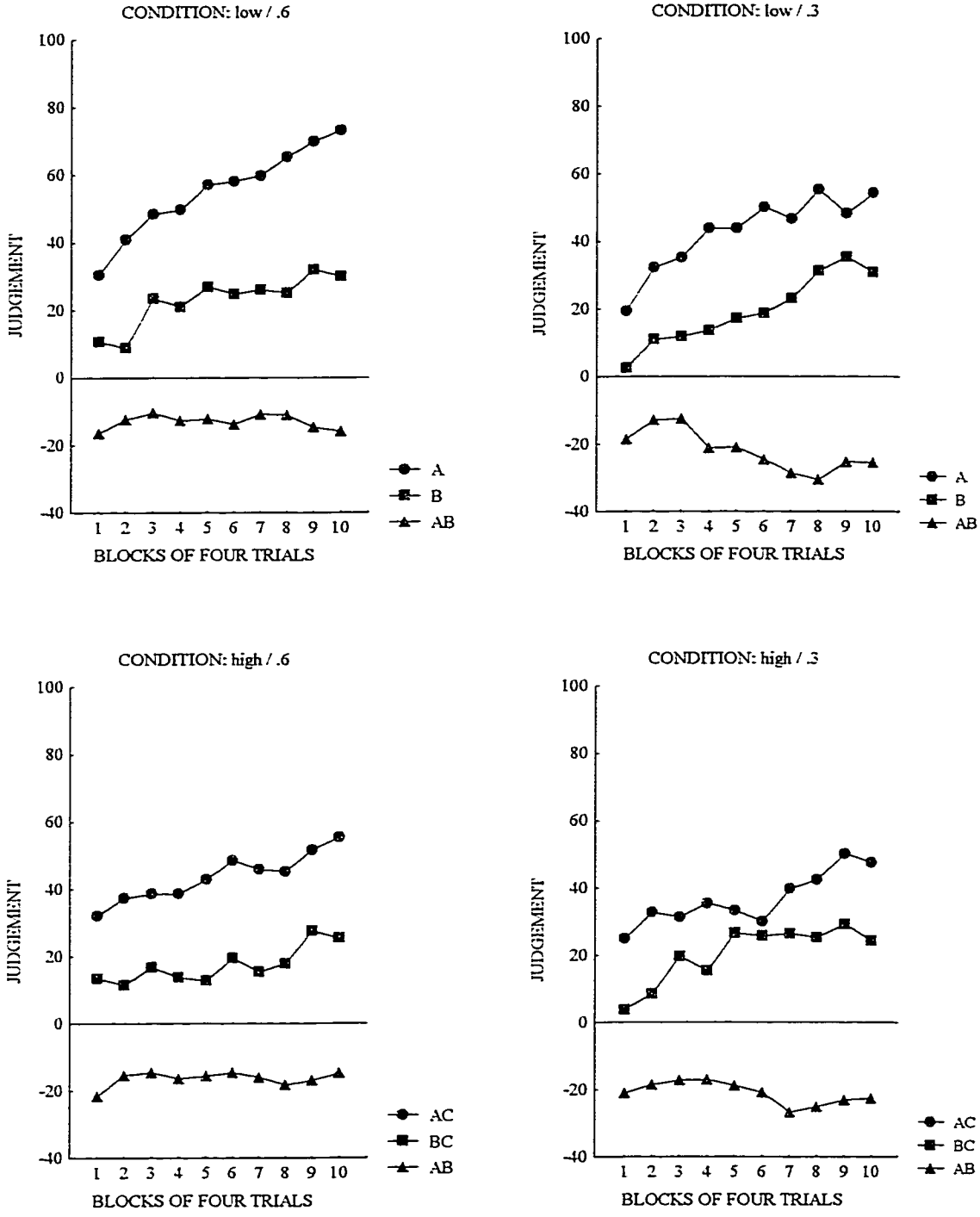

Figure 4. Empirical judgements in the four conditions of Experiment 1.

affected judgements of either B or AB. The same appears to be evident in the high similarity condition. That is, participants discriminated between the contingencies of AC, but judgements of BC and AB remained similar across levels of AC. Judgements of AB appear to remain both consistent and moderately negative across experimental conditions.

The results of the statistical analyses support these impressions. Judgements were reliably higher in the low similarity condition relative to the high similarity condition ($F(1,23) = 5.05$, $MSE = 2439.55$), judgements were higher when Δp for A was .6 than when it was .3 ($F(1,23) = 11.61$, $MSE = 1891.97$), judgements differed by predictor ($F(2,46) = 39.75$, $MSE = 24140.75$), and judgements increased with trial block ($F(9,207) = 19.59$, $MSE = 388.76$). The analysis also revealed reliable interactions between predictor and trial block ($F(18,414) = 6.97$, $MSE = 478.3$), and between contingency, predictor, and trial block ($F(18,414) = 1.91$, $MSE = 275.13$).

Protected a posteriori comparisons of contingencies and predictors within the three-way interaction revealed no reliable effects. Consequently, the reliable three-way interaction probably results from differences involving trial block. This interpretation is consistent with the reliable main effect of contingency and with the two-way interaction between predictor and trial block. As differences involving trial block are only relevant to the effect of similarity on judgements, and similarity did not interact with any variable, a posteriori comparisons within the three-way interaction involving the ten levels of trial block were not conducted. Analogously, a posteriori comparisons within the two-way interaction between predictor and trial block were not conducted.

Discussion

This experiment assessed judgements of the contingency between a two-element

compound predictor and an outcome as a function of: a) the contingency between one of its elements and the outcome, and b) the similarity between its elements. Varying the contingency between predictor A and the outcome had no effect on judgements of the AB compound, although participants reliably discriminated between the two levels of contingencies for A. Similarly, varying the similarity between predictors A and B by adding a common element had no effect on judgements of the AB compound. Judgements in the low similarity condition (pooled across contingency, predictor, and trial block) were higher than judgements in the high similarity condition. Participants judged the AB compound to be equivalent and consistently negative across all experimental conditions.

With regards to B, both models accurately predicted the non-normative assessment of this predictor. That is, although Δp for predictor B remained constant at 0, participants constantly rated B to be a moderately positive predictor. The verification of this highly counter-intuitive prediction attests to the already considerable theoretical value of the associative approach. At the same time, the findings that judgements of the compound predictor were independent of the contingency and similarity manipulations have considerable theoretical impact. These findings are incompatible with the notion that judgements of a compound reflect the associative strength of its elements in an additive fashion. The results are also incompatible with the notion that judgements of a compound reflect a portion of the associative strength generalised from other predictors as a function of similarity. Rather, it appears that judgements of a compound such as AB only reflect the associative strength accruing to it as a configural cue. According to both the Rescorla-Wagner and Pearce models, manipulating the contingency between predictor A and the outcome should affect judgements of the AB compound. This is related to the summation of

associative strength to a compound in the Rescorla-Wagner model, and to generalisation of associative strength among predictors in the Pearce model. Similarly, both models anticipate that the value of the AB compound would be lower in the high similarity condition than in the low similarity condition. According to the Rescorla-Wagner model this occurs because A and B acquire less associative strength when paired with the common element C than when presented individually. Consequently, the sum of the associative strength of A and B is lower in the high similarity condition than in the low similarity condition. The Pearce model anticipates a similar result based on the assumption that generalisation of positive associative strength to AB is lower in the high similarity condition than in the low similarity condition.

The notion that judgements of the contingency between a compound and an outcome are independent of the contingency between the compounds constituent elements and the outcome is tempered by two potential methodological difficulties in this experiment. One, the configural processing that was observed may have been a consequence of the method used to represent the AB compound. The AB compound was represented with a single pill sharing half of its colour with each of two single predictors (i.e., AB = half red/half green pill). Pill colour was the attribute used to calculate the normative contingencies, and upon which similarity was manipulated. As the AB compound was a visually distinct pill (i.e., distinct from A and B), participants may have considered AB to represent a different medication altogether. It is possible, then, that judgements of AB reflected the contingency between this distinct medication and the outcome, rather than the contingency between pill colour and the outcome. Two, this experiment included a manipulation of the contingency between only one of the compound's elements and the outcome. It remains possible that larger changes in the contingency of the

elements will be reflected in the judgements of the compound. This hypothesis is rendered highly unlikely by the fact that there was a reliable discrimination between the two values of A, thus confirming that the manipulation was indeed effective for the elemental stimulus. Nevertheless, an additional test with a manipulation of both elements would be further reassuring. Both of these issues are addressed in Experiment 2.

EXPERIMENT 2

This experiment replicates the low similarity condition of Experiment 1 with two changes. The AB compound is visually represented with two separate pills (i.e., one red and one green) rather than with one distinct pill as in Experiment 1. This should reduce the likelihood that participants will consider the AB compound as a distinct medication treatment. In fact, on any experimental trial, participants will only see two different pills. Any configural processing observed could only result if participants consider the simultaneous presentation of two different pills to be a unique configuration. In Experiment 1, changes in the contingency of one of the compound's elements did not affect judgements of the compound. In this experiment, the contingencies of both elements of a two-element compound were systematically manipulated. The sum of the normative contingencies between the compound's constituent elements and the outcome was varied systematically from .6 to -.2 among experimental conditions. In Experiment 1, this sum ranged only from .6 to .3. If judgements of a compound reflect the associative strengths accrued to its elements, this manipulation should be more powerful and result in observable differences in judgements of AB across conditions.

Participants were presented with A, B, and AB trials. The contingencies between the three predictors and the outcome were presented in a 2 X 2 factorial design. The four conditions each involved two contingencies between A and the outcome, and two contingencies between B and the outcome. The contingency between A and the outcome was either .6 [$P(O|A) = .8$, $P(O|\sim A) = .2$], or .3 [$P(O|A) = .65$, $P(O|\sim A) = .35$], as in Experiment 1. The contingency between B and the outcome was either 0 [$P(O|B) = .5$, $P(O|\sim B) = .5$], as in Experiment 1, or a new value of -.5 [$P(O|B) = .25$, $P(O|\sim B) = .75$]. As before, the contingency between the AB

Table 4. Frequency (f) and probability (p) of occurrence of each event in Experiment 2.

	Δp (Predictor A / Predictor B)							
	.6 / 0		.3 / 0		.6 / -.5		.3 / -.5	
Event	f	p	f	p	f	p	f	P
A+	15	.38	12	.3	15	.38	12	.3
B+	3	.08	3	.08	4	.1	4	.1
AB+	1	.03	1	.03	1	.03	1	.03
~+	1	.03	4	.1	0	0	3	.08
A-	0	0	3	.08	0	0	3	.08
B-	0	0	0	0	11	.28	11	.28
AB-	4	.1	4	.1	4	.1	4	.1
~-	16	.4	13	.33	5	.13	2	.05

compound and the outcome remained constant at -.34 across conditions [P(O|AB) = .2, P(O|~AB) = .54]. The frequency and probability of occurrence of each possible event (i.e., trial) in the four contingency conditions are listed in Table 4. Trial frequency by outcome combinations, as well as the associated conditional probability and contingency, for each predictor in the four conditions are listed in Table 5.

Simulations of the Rescorla-Wagner and Pearce models were conducted, with the same parameters used in Experiment 1, of the four conditions in this experiment. The results of this simulation are depicted in Figure 5 and 6 for each of the four conditions. As the .6/0 and .3/0 conditions are a replication of the low similarity condition of Experiment 1, values are similar to those depicted in the top two panels of Figure 2 for the Rescorla-Wagner model and Figure 3 for the Pearce model. As a consequence of summation, the Rescorla-Wagner model anticipates that the value of AB will be consistently higher than the value of A and B. According to the Pearce model, the value of AB will remain consistently lower than the value of A and higher than the

Table 5. Trial frequency (f), conditional probability (c/p), and contingency (Δp) associated with each predictor by condition for Experiment 2.

	Condition (Δp_A / Δp_B)											
	.6 / 0			.3 / 0			.6 / -.5			.3 /-.5		
	f	c/p	Δp	f	c/p	Δp	f	c/p	Δp	f	c/p	Δp
Predictor A												
A+	16			13			16			13		
A-	4	.8		7	.65		4	.8		7	.65	
~A+	4			7			4			7		
~A-	16	.2	.6	13	.35	.3	16	.2	.6	13	.35	.3
Predictor B												
B+	4			4			5			5		
B-	4	.5		4	.5		15	.25		15	.25	
~B+	16			16			15			15		
~B-	16	.5	0	16	.5	0	5	.75	-.5	5	.75	-.5
Predictor AB												
AB+	1			1			1			1		
AB-	4	.2		4	.2		4	.2		4	.2	
~AB+	19			19			19			19		
~AB-	16	.54	-.34	16	.54	-.34	16	.54	-.34	16	.54	-.34

value of B. Both models anticipate that the value of both A and AB will be lower when Δp for A is .3 than when Δp for A is .6. The two bottom panels of Figures 5 and 6 depict the theoretical values when the contingency for predictor B was -.5. According to both models, the lower contingency for predictor B will be reflected in lower values for both B and AB, and the value of B will remain moderately positive even when its Δp is -.5.

Method

Participants

Twenty-four undergraduate students (22 females, 2 males; mean age = 26 years) were recruited at the University of Ottawa to serve as participants for this experiment. After 24

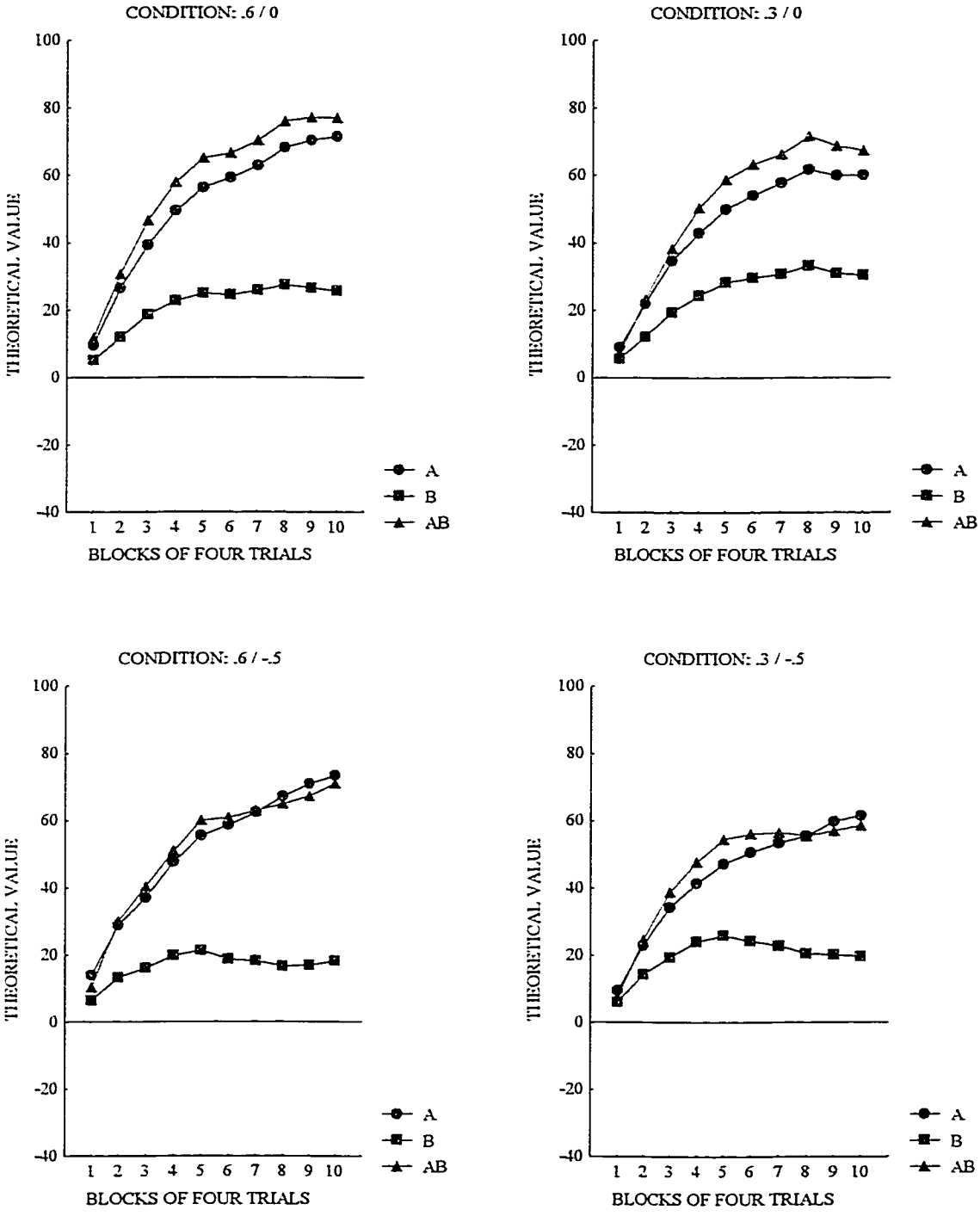

Figure 5. Theoretical values derived from the Rescorla-Wagner model in the four conditions of Experiment 2.

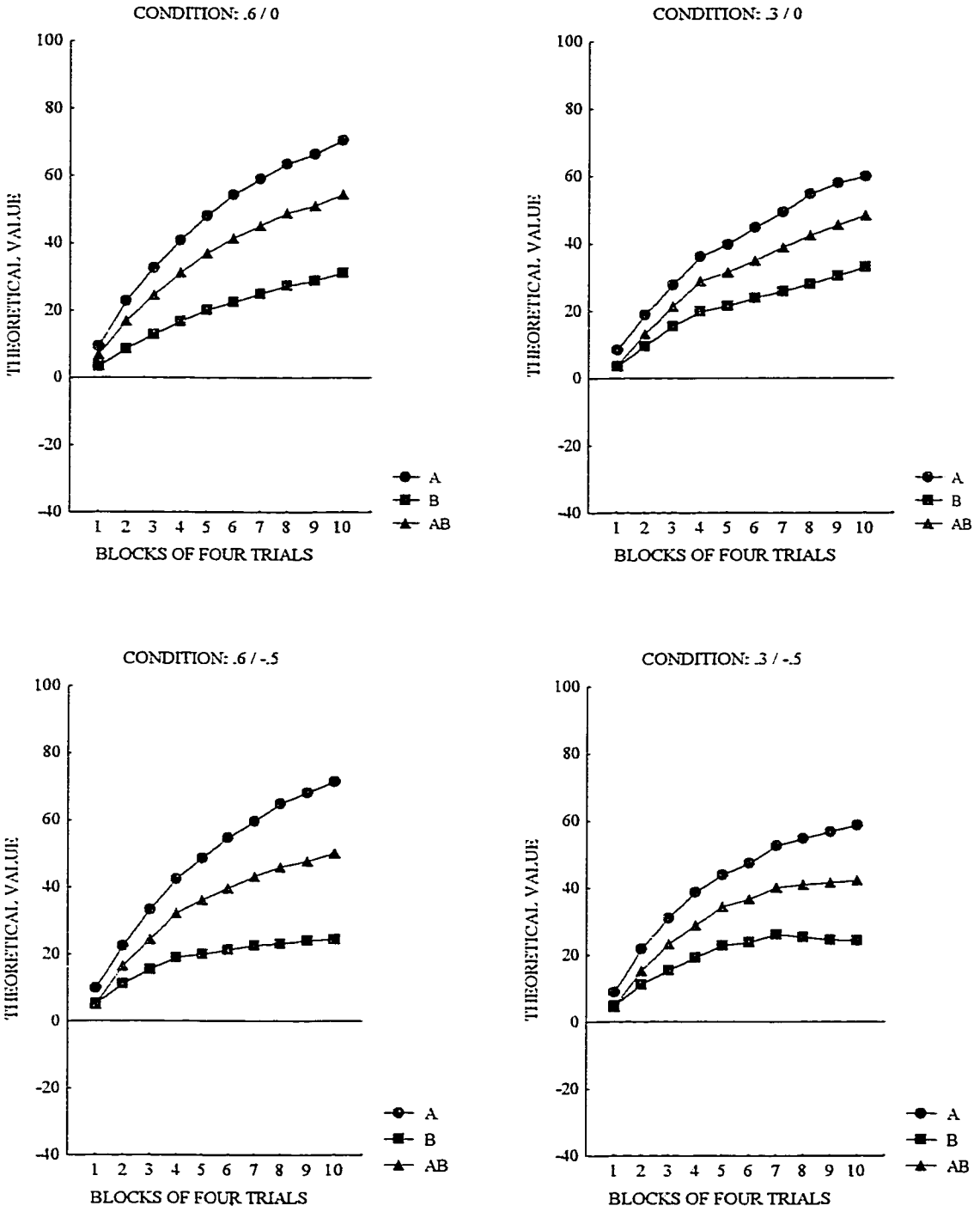

Figure 6. Theoretical values derived from the Pearce model in the four conditions of Experiment 2.

participants were tested, one person was chosen randomly and awarded a $50 prize.

Apparatus

The apparatus was the same as that employed in Experiment 1.

Stimuli

Stimuli were the same as those used in the low similarity condition of Experiment 1, with the exception that the AB treatment was represented with two different oval-shaped medication pills, one red and one green.

Procedure

The same procedure as in Experiment 1 was used. After the initial task instructions, participants completed one practice clinical trial. The practice was identical to the four experimental clinical trials except for the normative contingencies between the predictors and the outcome. In the practice trial, the contingency for A was .5 [$P(O|A) = .75$, $P(O|\sim A) = .25$], the contingency for B was -.5 [$P(O|B) = .25$, $P(O|\sim B) = .75$], and the contingency for the AB compound was 0 [$P(O|AB) = .5$, $P(O|\sim AB) = .5$]. As in Experiment 1, each clinical trial consisted of 40 individual trials (i.e., patient files), and participants judged the relations between each medication treatment and the facial rash after every 4 trials for a total of 10 judgements per medication. Also, the order of presentation of experimental conditions was counterbalanced between participants, according to the 24 possible permutations of four objects. The assignment of pill colour to contingency was counterbalanced between conditions and between subjects.

Results

A within-observation analysis of variance, with individual judgements as the dependent variable, was used to examine the variables of contingency of A ($\Delta p = .6$ or .3), contingency of B

(Δp = .5 or 0), predictor (A, B, and AB), and trial block (1 to 10). A posteriori comparisons were protected with the Bonferroni procedure. A Type I error rate of .05 was used.

Figure 7 depicts the empirical judgements in the four conditions of this experiment. From these figures, it appears that judgements of predictor A were higher when the Δp for A was .6 than when it was .3, and changes in the Δp for A did not affect judgements of either B or AB. This appears to be the case both when Δp for B is 0 (top two panels of Figure 7) and when Δp for B is -.5 (bottom two panels of Figure 7). Similarly, judgements of B appear to be lower when Δp for B is -.5 than when Δp for B is 0, and changing Δp for B appeared not to affect judgements of either A or AB. As in Experiment 1, judgements of AB appear to be unaffected by the contingency manipulations; remaining both relatively consistent and negative across experimental conditions.

The statistical analyses confirmed these impressions. Judgements were higher when the Δp for A was .6 than when it was .3 ($F(1, 23) = 7.80$, $MSE = 4600.88$), judgements were higher when the Δp for B was 0 than when it was -.5 ($F(1, 23) = 36.57$, $MSE = 3812.71$), judgements differed by predictor ($F(2, 46) = 33.58$, $MSE = 15704.37$),), and judgements increased with trial block ($F(9, 207) = 21.62$, $MSE = 294.54$). The analyses also revealed two-way interactions between Δp of A and predictor ($F(2, 46) = 4.93$, $MSE = 11777.39$), Δp of B and trial block ($F(9, 207) = 2.89$, $MSE = 356.86$), and predictor and trial block ($F(18, 414) = 5.41$, $MSE = 476.64$); and three-way interactions between Δp of A, predictor, and trial block ($F(18, 414) = 1.87$, $MSE = 316.6$), and between Δp of B, predictor, and trial block ($F(18, 414) = 2.79$, $MSE = 364.24$).

Protected a posteriori comparisons were used to examine the three-way interactions. In

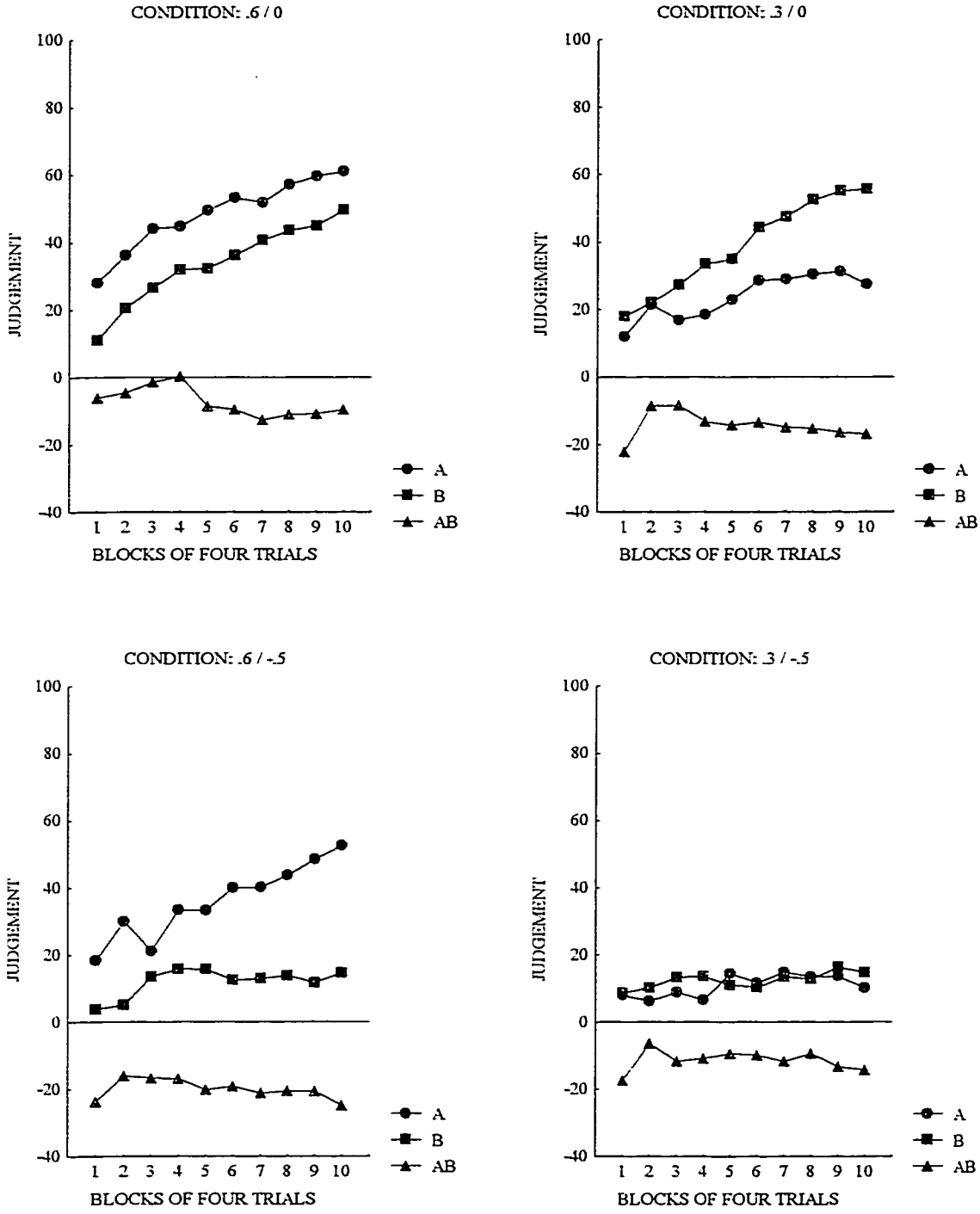

Figure 7. Empirical judgements in the four conditions of Experiment 2.

the three-way interaction between Δp of A, predictor, and trial block, participants discriminated between the two contingencies of A ($F(1, 23) = 14.08$, $MSE = 10633.2$), but varying the contingency between A and the outcome did not affect judgements of either B or AB. In the three-way interaction between Δp of B, predictor, and trial block, participants discriminated between the two contingencies of B ($F(1, 23) = 11.45$, $MSE = 12277.8$), but varying the contingency between B and the outcome did not affect judgements of either A or AB.

Discussion

This experiment assessed judgements of the contingency between a two-element compound predictor (AB) and an outcome as a function of the contingency between each of its elements (A, and B) and the outcome. Although participants reliably discriminated between the different contingencies for both A and B, judgements of AB were unaffected by the contingency manipulations. As in Experiment 1, judgements of AB were equivalent and consistently negative across experimental conditions. Judgements of the relation between the AB compound and the outcome remained constant across conditions, never approaching the sum of the normative contingencies between its constituent elements and the outcome, a sum that varied systematically from .6 to -.2 among conditions. The invariance of judgements of AB is inconsistent with the notion that judgements of a compound reflect the associative strength of its elements in an additive fashion. According to the Pearce model, the value of a compound reflects a portion of the associative strength generalised from other predictors. As judgements of the compound were completely unaffected by the contingency manipulations in both Experiments 1 and 2, these findings also appear to be inconsistent with a configural process that emphasises the generalisation of associative strength among predictors as a function of similarity.

The findings of this experiment replicate those of Experiment 1, and strengthen the conclusion that judgements of a compound predictor are arrived at through a configural process with little generalisation of associative strength. In Experiment 1, the AB compound was represented with a single pill sharing half of its colour with each of two single predictors (i.e., AB = half red/half green pill). In this context, it was possible to argue that judgements of AB reflected the contingency between a unique medication and the outcome, rather than the contingency between pill colour and the outcome. In Experiment 2, the AB compound was represented with two different pills, one red and one green. That participants considered the simultaneous presentation of two different pills as a unique configuration (i.e., distinct from either of the pills alone) argues in favour of configural processing.

According to the Pearce model, the generalisation of associative strength among predictors is a function of the number of elements they share (Equation 8). It is possible that the similarity among predictors in the first two experiments was not high enough to detect an effect of generalisation. For example, in Experiment 1 the similarity between predictors A and B was varied from .25 in the low similarity condition to .44 in the high similarity condition. This magnitude of change in similarity may not be sufficient to detect an effect of generalisation on judgements. If the number of elements shared by two predictors is increased, presumably the amount of generalisation between them will also increase. For example, in the procedure used for the first two experiments, participants were typically presented with at least two predictors sharing an element (e.g., A, and AB). The similarity between these predictors could be increased, and presumably the generalisation between them as well, by adding another common element (e.g., AC, ABC). The similarity between A and AB, assuming the existence of a common

contextual cue, given by Equation 8 is .67, whereas the similarity between AB and ABC is .75. By increasing the similarity between the predictors, then, the effect of manipulating the contingency between one predictor (e.g., AC) and the outcome may be more readily observed in judgements of the second (e.g., ABC). Finally, the first two experiments assessed judgements of a compound as a function of contingency manipulations of its constituent elements. If a compound predictor is truly functionally independent of its elements as suggested by Shanks et al. (1998), then judgements of its elements should also be unaffected by manipulating the compound's contingency. Experiment 3 explores both of these issue.

EXPERIMENT 3

The Pearce model specifies that constituent elements should affect judgements of the compound through a process of generalisation of associative strength, and that the generalisation of associative strength between a compound and its elements is less than complete and related to the number of shared elements (Equation 8). No evidence of the effect of generalisation on judgements was found in Experiments 1 and 2. From the point of view of Pearce's model however, the similarity among predictors in the first two experiments may not have been high enough to detect an effect of generalisation on judgements. In this experiment, the Δp of compound predictor AC is manipulated, and judgements of compound predictor ABC are assessed. The similarity between AC and ABC, assuming the existence of a common contextual cue, is as high as .75 (Equation 8). If the generalisation of associative strength among predictors is a function of the number of elements they share, the effect of manipulating the contingency between predictor AC and the outcome should be observed in judgements of predictor ABC. Also assessed in this experiment is the effect of manipulating the Δp of predictor AC on judgements of predictors A and C individually. Experiments 1 and 2 demonstrated that manipulations of the elements of a two-element compound did not affect judgements of the compound. Strictly speaking, if compound predictors are assessed uniquely and independently of their constituent elements, then manipulations of a compound should not be reflected in judgements of its constituent elements. However, as they stand, neither model is making this prediction because each has some generalisation from compound to element in addition to generalisation from elements to compound.

There are five different predictors in this experiment: A, B, C, AC, and ABC. The

contingencies between these predictors and the outcome were presented in a 2 X 2 factorial design, with AC and A the only predictors varying systematically across conditions (refer to Table 1 for a summary of the Δp for each predictor). The contingency between AC and the outcome was either .6 [P(O|AC) = .8, P(O|~AC) = .2], or .3 [P(O|AC) = .65, P(O|~AC) = .35]. Predictors A and C were each involved in two negative and two positive contingencies with the outcome. The contingency between A and the outcome was yoked with the contingency between C and the outcome as follows. When Δp for A was -.34 [P(O|A) = .46, P(O|~A) = .8] Δp for C was .55 [P(O|C) = .76, P(O|~C) = .21], when Δp for A was -.36 [P(O|A) = .42, P(O|~A) = .78] Δp for C was .52 [P(O|C) = .71, P(O|~C) = .19]. Similarly, when Δp for A was .55 [P(O|A) = .76, P(O|~A) = .21] Δp for C was -.34 [P(O|C) = .46, P(O|~C) = .8], when Δp for A was .52 [P(O|A) = .71, P(O|~A) = .19] Δp for C was -.36 [P(O|C) = .42, P(O|~C) = .78]. The two positive and two negative contingencies for A and C were considered equivalent for the purposes of statistical analysis and interpretation, thus yielding one level of A at .54 (with C at -.35), and another level of A at -.35 (with C at .54). The contingency between ABC and the outcome remained constant at -.34 [P(O|ABC) = .2, P(O|~ABC) = .54], and the contingency between B and the outcome remained constant at 0 [P(O|B) = .5, P(O|~B) = .5], in all conditions. The four conditions are referred to as: .6/.54/-.35, .3/.54/-.35, .6/-.35/.54, and .3/-.35/.54; the first number represents the Δp for predictor AC, the second number the Δp for predictor A, and the third number the Δp for predictor C. The frequency and probability of occurrence of each event (i.e., trial) in the four conditions are listed in Table 6. Trial frequency by outcome combinations, as well as the associated conditional probability and contingency, for each predictor in the four conditions are listed in Table 7.

Table 6. Frequency (f) and probability (p) of occurrence of each event in Experiment 3.

	Δp (Predictor AC / Predictor A / Predictor C)							
	.6/.54/-.35		.3/.54/-.35		.6/-.35/.54		.3/-.35/.54	
Event	f	p	f	p	f	p	f	p
A+	0	0	4	.1	0	0	0	0
B+	3	.15	3	.15	3	.15	3	.08
C+	0	0	0	0	0	0	4	.1
AC+	15	.38	12	.3	15	.38	12	.3
ABC+	1	.03	1	.03	1	.03	1	.03
~+	1	.03	0	0	1	.03	0	0
A-	1	.03	0	0	15	.38	11	.28
B-	0	0	0	0	0	0	0	0
C-	15	.38	11	.28	1	.03	0	0
AC-	0	0	3	.08	0	0	3	.08
ABC-	4	.4	4	.1	4	.1	4	.1
~-	0	0	2	.05	0	0	2	.05

Simulations of the Rescorla-Wagner and Pearce models were conducted, with the same parameters used in the previous experiments. Figures 8 and 9 depict the theoretical values for the Rescorla-Wagner and Pearce models respectively. The two models make different predictions regarding the ordinal relation between the values of predictors ABC and AC, reflecting their different conceptualisations of generalisation. According to the Rescorla-Wagner model, the value of ABC will be consistently higher than the value of AC in all conditions. This is because a compound is expected to reflect all of the associative strength of its constituent elements. That is, $ABC = V_A + V_B + V_C$, whereas $AC = V_A + V_C$, and B is presumed to gain some positive associative strength despite its Δp of 0. According to the Pearce model, the value of ABC will remain consistently below the value of AC in all conditions. This occurs for two reasons. One, the configural cue "ABC" is expected to acquire negative associative strength. Two, the value of

Table 7. Trial frequency (f), conditional probability (c/p), and contingency (Δp) associated with each predictor by condition for Experiment 3.

	Condition (Δp AC / Δp A / Δp C)											
	.6/.54/-.35			.3/.54/-.35			.6/-.35/.54			.3/-.35/.54		
	f	c/p	Δp	f	c/p	Δp	f	c/p	Δp	f	c/p	Δp
Predictor A												
A+	16			17			16			13		
A-	5	.76		7	.71		19	.46		18	.42	
~A+	4			3			4			7		
~A-	15	.21	.55	13	.19	.52	1	.8	-.34	2	.78	-.36
Predictor B												
B+	4			4			4			4		
B-	4	.5		4	.5		4	.5		4	.5	
~B+	16			16			16			16		
~B-	16	.5	0	16	.5	0	16	.5	0	16	.5	0
Predictor C												
C+	16			13			16			17		
C-	19	.46		18	.42		5	.76		7	.71	
~C+	4			7			4			3		
~C-	1	.8	-.34	2	.78	-.36	15	.21	.55	13	.19	.52
Predictor AC												
AC+	16			13			16			13		
AC-	4	.8		7	.65		4	.8		7	.65	
~AC+	4			7			4			7		
~AC-	16	.2	.6	13	.35	.3	16	.2	.6	13	.35	.3
Predictor ABC												
ABC+	1			1			1			1		
ABC-	4	.2		4	.2		4	.2		4	.2	
~ABC+	19			19			19			19		
~ABC-	16	.54	-.34	16	.54	-.34	16	.54	-.34	16	.54	-.34

ABC will reflect the negative associative strength of "ABC" to a greater extent than the value of AC, due to the ABC's higher similarity with "ABC".

According to both models, changing the Δp for AC from .6 to .3 will affect the value of A

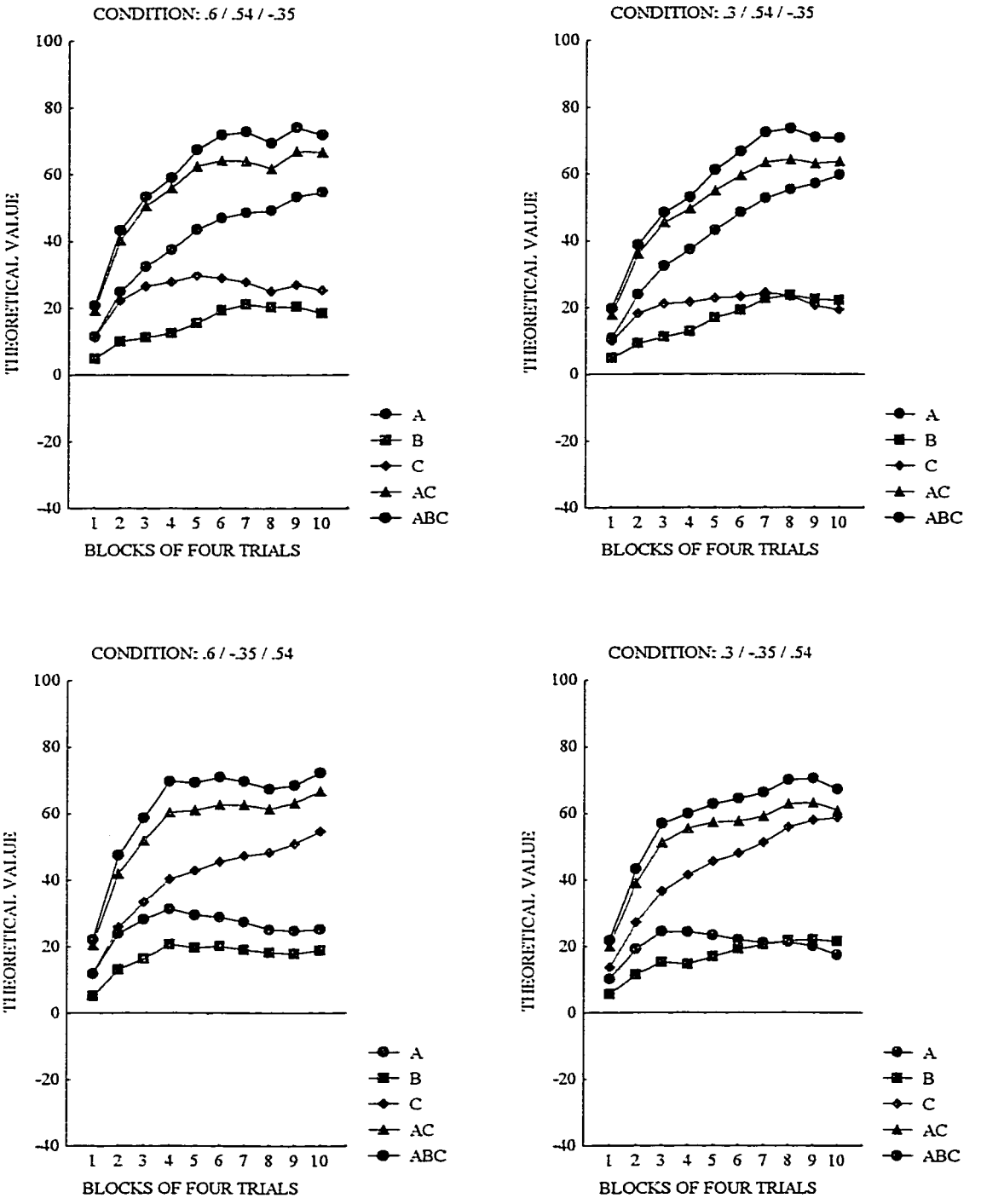

Figure 8. Theoretical values derived from the Rescorla-Wagner model in the four conditions of Experiment 3.

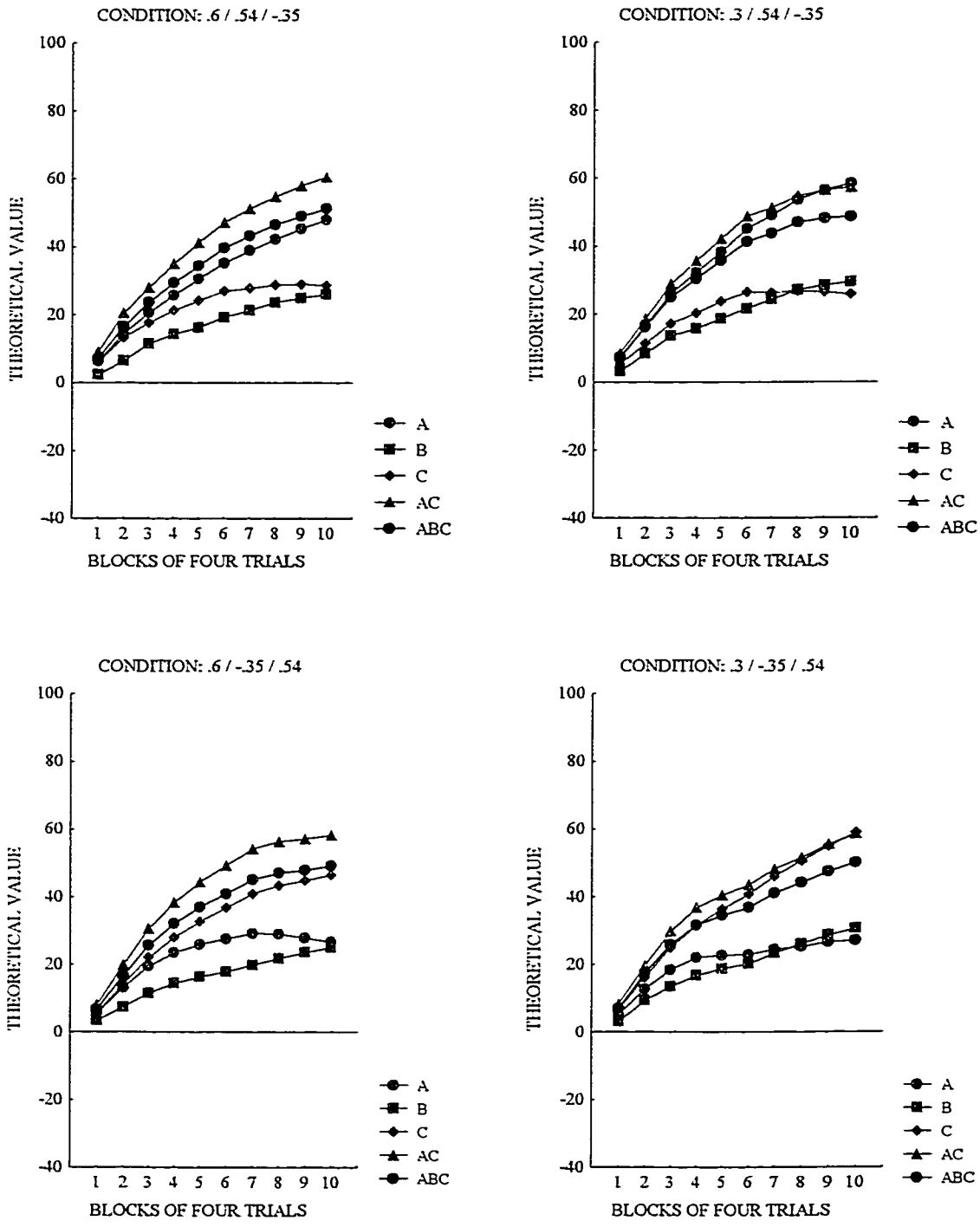

Figure 9. Theoretical values derived from the Pearce model in the four conditions of Experiment 3.

and B. The difference between the theoretical values of A and C is expected to be greater when the Δp for AC is .3 than when the Δp for AC is .6. To understand this, consider the .6/.54/-.35 and .3/.54/-.35 conditions (the theoretical values and presumed processes are the same in the conditions where the Δp for A and C are reversed [condition .6/-.35/.54, and .3/-.35/.54]). The value of A is higher when Δp for AC is .3 (condition .3/.54/-.35), relative to when Δp for AC is .6 (condition .6/.54/-.35). At the same time, the value of C is lower when Δp for AC is .3 (condition .3/.54/-.35), relative to when Δp for AC is .6 (condition .6/.54/-.35). According to the Rescorla-Wagner model, this results primarily from changes in the number and type of trials during which A and C are presented, at the different levels of Δp for AC. Predictor A occurs sixteen times with the outcome when the Δp for AC is .6 (fifteen times within the AC compound, and one time within the ABC compound), and seventeen times with the outcome when the Δp for AC is .3 (four times alone, twelve times within the AC compound, and one time within the ABC compound). The consequence of changing the Δp for AC from .6 to .3 is that A occurs proportionally more frequently alone. As A is assumed to gain more associative strength on trials when it is presented alone than on trials when it must compete for associative strength with B or C, the value of A will be higher when the Δp for AC is .3. Changing the Δp for AC has a different consequence for the number of trials during which C is presented. Predictor C occurs sixteen times with the outcome when the Δp for AC is .6 (fifteen times within the AC compound, and one time within the ABC compound), and thirteen times with the outcome when the Δp for AC is .3 (twelve times within the AC compound, and one time within the ABC compound). Predictor C occurs less frequently with the outcome when the Δp for AC is .3, and consequently

gains less positive associative strength, than when the Δp for AC is .6. The Pearce model anticipates a similar result by way of a different process. In the condition when the Δp for AC is .6, predictor A never occurs alone. Consequently, no associative strength accrues to configural cue "A". In the condition when the Δp for AC is .3, configural cue "A" gains positive associative strength as predictor A is presented alone with the outcome on four trials. Predictor A will benefit from the generalisation of positive associative strength from its configural cue when the Δp for AC is .3 but not when the Δp for AC is .6. Consequently, the value of A is higher in the .3/.54/-.35 condition than in the .6/.54/-.35 condition. Predictor C, however, never occurs alone with the outcome. Its value will be determined primarily by the positive associative strength generalised to it from "AC". As AC occurs less frequently when its Δp is .3 than when its Δp is .6, C will benefit from less generalisation of positive associative strength when Δp for AC is .3. Consequently, the value of C will be lower when the Δp for AC is .3 than when the Δp for AC is .6.

Both models anticipate that the ordinal relation of A and C will reflect their respective Δp. For example, in conditions .6/.54/-.35 and .3/.54/-.35, the value of A is higher than the value of C. Analogously, in conditions .6/-.35/.54 and .3/-.35/.54, the value of C is higher than the value of A. Finally, the models anticipate that the value of B will remain positive despite its Δp of 0. This is analogous to the model predictions in the first two experiments. Neither model anticipates negative values although the Δp for A is negative in conditions .6/-.35/.54 and .3/-.35/.54, the Δp for C is negative in conditions .6/.54/-.35 and .3/.54/-.35, and the Δp for ABC remains negative in all conditions.

Method

Participants

Twenty-four undergraduate students (21 females, 3 males; mean age = 24 years) were recruited at the University of Ottawa. After 24 participants were tested, one person was chosen randomly and awarded a $50 prize.

Apparatus

The apparatus was the same as that used in Experiment 1.

Stimuli

Stimuli were the same as those used in Experiment 2. A blue pill represented the new medication treatment (i.e., predictor C). Predictor AC was represented with two separate pills (i.e., one red pill, and one blue pill), and predictor ABC was represented with all three distinct coloured pills (i.e., one red pill, one green pill, and one blue pill).

Procedure

The procedure used in Experiment 1 was modified to accommodate five predictors on the response screen. During the instruction phase, participants were shown the icons for five possible treatments (red alone, green alone, blue alone, red and blue together, and red, green, and blue together). Trials did not include treatments for which participants were not asked to make judgements. For example, BC was not a possible treatment type. In the practice trial, the contingency for A was .5 [P(O|A) = .75, P(O|~A) = .25], the contingency for B was -.5 [P(O|B) = .25, P(O|~B) = .75], the contingency for C was .5 [P(O|C) = .75, P(O|~C) = .25], the contingency for the AC compound was .5 [P(O|AC) = .75, P(O|~AC) = .25], and the contingency for the ABC compound was 0 [P(O|ABC) = .5, P(O|~ABC) = .5]. Each clinical trial consisted of 40 individual

trials (i.e., patient files), and participants judged the relations between each medication treatment and the facial rash after every 4 trials for a total of 10 judgements per medication. Once again, the order of presentation of experimental conditions was counterbalanced between participants, according to the 24 possible permutations of four objects. The assignment of pill colour to contingency was counterbalanced between conditions and between subjects.

Results

A within-observation analysis of variance, with individual judgements as the dependent variable, was used to examine the variables of contingency of AC ($\Delta p = .6$ and .3), contingency of A ($\Delta p = -.35$ and .54), predictor (A, B, C, AC, ABC), and trial block (1 to 10). A posteriori comparisons were protected with the Bonferroni procedure. A Type I error rate of .05 was used throughout.

Figure 10 depicts the empirical judgements in the four conditions of this experiment. Judgements of AC appear to be higher when the Δp for AC is .6 than when the Δp for AC is .3, and judgements of A and C individually appear to be consistent with their Δp. Judgements of A are higher than judgements of C when the Δp for A is .54 (condition .6/.54/-.35, and .3/.54/-.35), and judgements of C are higher than judgements of A when the Δp for C is .54 (condition .6/-.35/.54, and .3/-.35/.54). Judgements of A and C are lower when the Δp for AC is .6 (A in condition .6/.54/-.35, and C in condition .6/-.35/.54), than when the Δp for AC is .3 (A in condition .3/.54/-.35, and C in condition .3/-.35/.54). Changing the Δp for AC appears not to have affected judgements of either B or ABC. Judgements of B remain moderately positive, and judgements of ABC remain moderately negative, in all conditions.

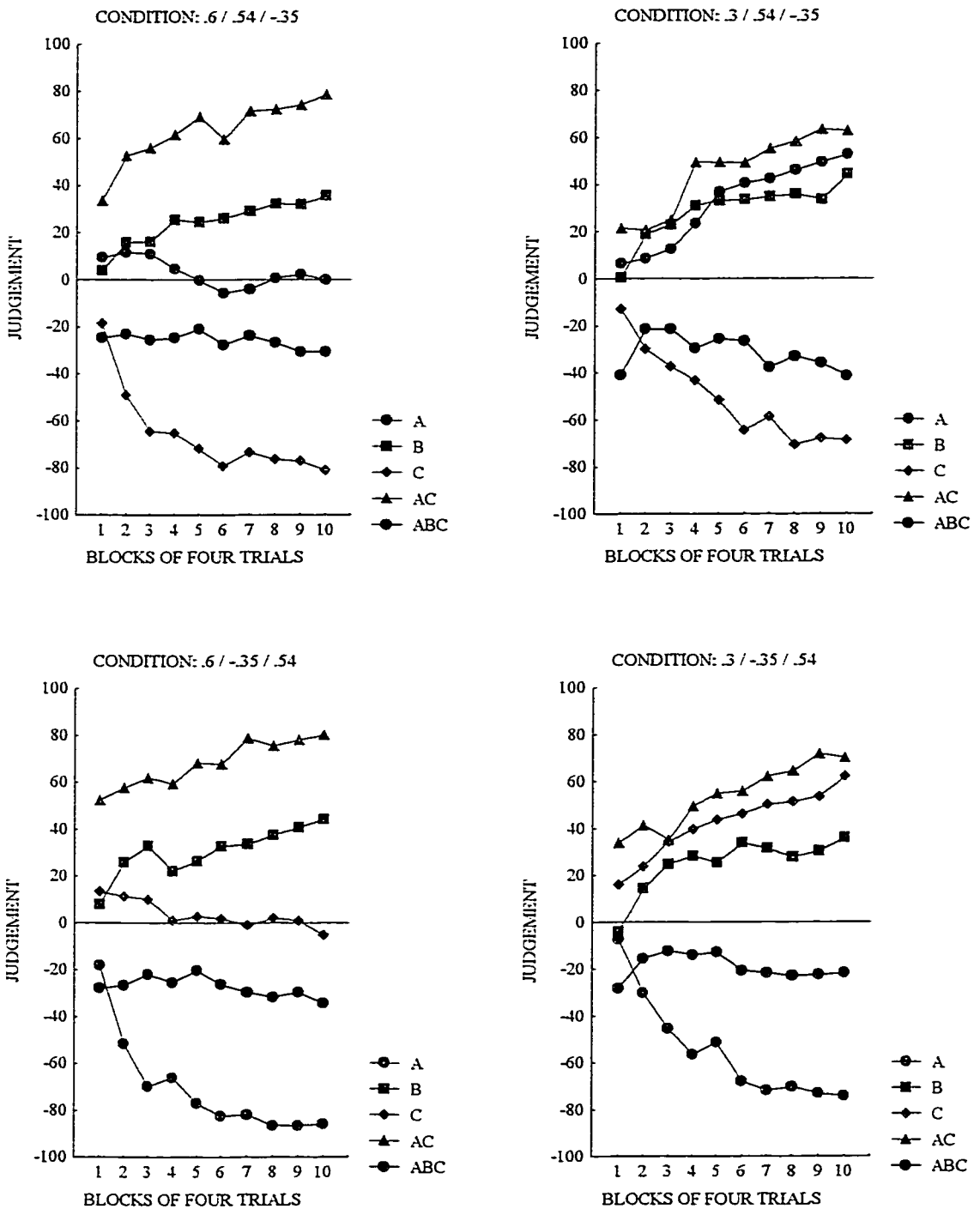

Figure 10. Empirical judgements in the four conditions of Experiment 3.

Statistical analyses confirmed these impressions. Judgements of AC were higher when the Δp for AC was .6 then when it was .3 ($F(1, 23) = 6.92$, $MSE = 8156.59$), and judgements differed by predictor ($F(4, 92) = 60.69$, $MSE = 21605.39$). The analyses also revealed interactions between the Δp for AC and trial block ($F(9, 207) = 6.56$, $MSE = 533.25$), the Δp for AC and predictor ($F(4, 92) = 10.75$, $MSE = 6861.58$), the Δp for A and predictor ($F(4, 92) = 75.95$, $MSE = 10280.18$), and between trial block and predictor ($F(36, 828) = 14.51$, $MSE = 673.92$). Three-way interactions were found between the Δp for AC, trial block, and predictor ($F(36, 828) = 1.44$, $MSE = 638.77$), and between the Δp for A, trial block, and predictor ($F(36, 828) = 11.65$, $MSE = 671.08$). Finally, the four-way interaction between the variables was reliable ($F(36, 828) = 2.81$, $MSE = 640.4$).

Planned comparisons with Bonferroni correction were used to examine the four-way interaction. Varying the contingency between AC and the outcome from .6 to .3 did not affect judgements of either B or ABC ($p > .8$). Similarly, changes in the contingency between A and the outcome from -.35 to .54 did not affect judgements of ABC ($p > .3$). Changes in Δp for AC affected judgements of A in conditions when A was involved in a positive contingency with the outcome ($F(1, 23) = 13.72$, $MSE = 8736.4$), such that judgements of A were lower when Δp for AC was .6 than when Δp for AC was .3. Similarly, changes in Δp for AC affected judgements of C in conditions when C was involved in a positive contingency with the outcome ($F(1, 23) = 22.72$, $MSE = 7556.6$), such that judgements of C were lower when Δp for AC was .6 than when Δp for AC was .3.

Discussion

This experiment assessed the effect of changes in the Δp for a two-pill compound, AC, on judgements of a three-pill compound, ABC, of which it was a subset. The effect of varying the Δp for AC on judgements of its constituent elements (i.e., A, and C) was also assessed. Both the Rescorla-Wagner and Pearce models predict that judgements of a compound will reflect the associative strength of its constituent elements, although to different degrees. The Rescorla-Wagner model presumes that judgements of a compound reflect all of the associative strength accrued to its constituent elements whereas, according to the Pearce model, judgements of a compound reflect a portion of the associative strength accrued to other predictors as determined by similarity. In the first two experiments of this report, the similarity among the predictors was relatively low. While the results of these experiments were incompatible with the notion of total generalisation of associative strength between a compound and its constituent elements as envisioned by the Rescorla-Wagner model, it remained conceivable that an effect of generalisation could be observable at a higher level of similarity. Although participants reliably discriminated between the two levels of Δp for AC, judgements of ABC remained both consistent and moderately negative in all conditions. Consequently, even in the context of a high degree of similarity, judgements of a compound appear unaffected by the relation between its constituent elements and the outcome.

As stated in the introduction to this experiment, both models predicted that the value of A would be higher when Δp for AC was .3 (condition .3/.54/-.35) than when Δp for AC was .6 (condition .6/.54/-.35). Consistent with this prediction, judgements of A and C (when each had a positive Δp) were higher when the Δp for AC was .3 than when the Δp for AC was .6. However,

the pattern of empirical judgements appears to reflect a process different from that presumed to be responsible for the predicted effects. The consequence of changing the Δp for AC from .6 to .3 is that A occurs proportionately more frequently alone. According to the Rescorla-Wagner model this results in A acquiring more associative strength when the Δp for AC is .3 than when Δp for AC is, as A is predicted to gain more associative strength on trials when it is presented alone than on trials when it must compete for associative strength with other predictors. According to the Pearce model, this effect occurs because the value of A will reflect the generalisation of positive associative strength from the configural cue "A" in condition .3/.54/-.35 but not in condition .6/.54/-.35, where A is never presented alone with the outcome and does not generate a configural cue. The model predictions, and the presumed processes, are the same when the Δp for A and C are reversed (condition .6/-.35/.54, and .3/-.35/.54). The pattern of empirical judgements suggests a different process. Judgements of A in condition .3/.54/-.35, and of C in condition .3/-.35/.54, remain consistently below judgements of AC and above judgements of B, consistent with the ordinal relation of their respective Δp. Judgements of A in condition .6/.54/-.35, and of C in condition .6/-.35/.54, are consistently below judgements of both AC and B, although the Δp for both A and C remained at .54. This result is more consistent with the notion that judgements of A and C were suppressed when the Δp for AC was .6, rather than the notion that judgements of A and C were increased when the Δp for AC was .3. It appears that the presence of a compound predictor with a moderate positive contingency with the outcome (i.e., AC, Δp = .6) suppressed judgements of a slightly weaker one-element predictor (i.e., both A and C, Δp = .54). Consequently, instead of being consistent with generalisation in either an elemental

or a configural process, this result is more suggestive of a form of overshadowing (Baker et al., 1993) of a one-element predictor by a configural compound predictor. This finding is consistent with the notion that compound predictors are assessed as unique configural cues. The implications of this finding for the nature of the associative process underlying contingency judgements will be considered in more detail in the general discussion.

Taken together, the results of the first three experiments strongly suggest that the notion of summation of associative strength to a compound is not a component of the process through which judgements of contingency are arrived at. This conclusion is strengthened by the results of the current experiment where changing the Δp for AC had no effect on judgements of ABC, and where judgements of AC never approached the sum of the judgements of A and C individually. Further, generalisation of associative strength appears not to contribute to judgements of compound predictors. This was found even though the similarity between AC and ABC was quite high. However, a proper contrast of the different conceptualisations of generalisation presumed by the Rescorla-Wagner and the Pearce models requires a within-experiment similarity manipulation for which the models make different predictions. An objective of Experiment 1 was to assess the effect of changing the similarity between the elements of the compound predictor on judgements of both the compound and the individual elements. Both associative models made qualitatively similar predictions regarding the effect of this similarity manipulation, although as a consequence of different underlying associative processes. Consider the effect of increasing the similarity between a one-element predictor, and the compound of which it is a subset. This could be accomplished by adding a common element. For example, a low similarity condition may include A+ and AB- trials, while a high similarity condition may include trials with AC+ and

ABC-. As a consequence of the assumption of complete generalisation of associative strength between a compound and its elements, the Rescorla-Wagner model predicts that judgements of AC and ABC will be higher than judgements of A and AB respectively. This stems from the fact that $V_A = V_A$, whereas $V_{AC} = V_A + V_C$, and $V_{AB} = V_A + V_B$, whereas $V_{ABC} = V_A + V_B + V_C$. According to the Pearce model, judgements should be approximately the same in both similarity conditions. This follows from the principle that A+ and AC+ trials are fundamentally equivalent. Only one associative connection will change on both types of trials; that between "A" and the outcome or that between "AC" and the outcome. Experiment 4 examines these predictions.

EXPERIMENT 4

This experiment was designed to contrast the different conceptualisations of generalisation presumed by the Rescorla-Wagner and Pearce models. Although Experiment 1 examined the effect on judgements of a similarity manipulation, the two models made qualitatively similar predictions in the two similarity conditions. This experiment implements a similarity manipulation for which the two models make different predictions. Participants were asked to make judgements of predictor-outcome contingency in the context of a feature-negative discrimination at two level of similarity. In the low similarity condition, participants judged the contingency for predictors A, B, and AB. In the high similarity condition, participants judged the contingency for predictors AC, B, and ABC. The presence of predictor C results in a higher similarity between AC and ABC, than between A and AB. The models differ regarding the effect of similarity on the values for the predictors.

In the low similarity condition, participants judged a compound predictor with two elements (AB), as well as its constituent elements (A and B), at two levels of Δp for A. The contingency between A and the outcome was either .6 [P(O|A) = .8, P(O|~A) = .2], or .3 [P(O|A) = .65, P(O|~A) = .35]. The contingency between AB and the outcome remained constant at -.34 [P(O|AB) = .2, P(O|~AB) = .54] across levels of Δp for A. In the high similarity condition, participants made judgements of a three element compound predictor (ABC), a two element compound predictor (AC), and a single predictor (B), at two levels of Δp for AC. The contingency for AC was either .6 [P(O|AC) = .8, P(O|~AC) = .2], or .3 [P(O|AC) = .65, P(O|~AC) = .35]. The contingency for ABC remained constant at -.34 [P(O|ABC) = .2, P(O|~ABC) = .54] across levels of Δp for AC. The contingency for B remained constant at 0

Table 8. Frequency (f) and probability (p) of occurrence of each event in Experiment 4.

	Δp A/AC			
	.6		.3	
Event	f	P	f	p
Low Similarity				
A+	15	.38	12	.3
B+	3	.08	3	.08
AB+	1	.03	1	.03
~+	1	.03	4	.1
A-	0	0	3	.08
B-	0	0	0	0
AB-	4	.1	4	.1
~-	16	.4	13	.33
High Similarity				
A+	0	0	0	0
B+	3	.08	3	.08
C+	0	0	0	0
AC+	15	.38	12	.3
ABC+	1	.03	1	.03
~+	1	.03	4	.1
A-	0	0	0	0
B-	0	0	0	0
C-	0	0	0	0
AC-	0	0	3	.08
ABC-	4	.1	4	.1
~-	16	.4	13	.33

[P(O|B) = .5, P(O|~B) = .5)] in all four experimental conditions (refer to Table 1 for a summary of the Δp for the predictors). The four conditions are referred to as: low/.6, low/.3, high/.6, and high/.3; the word low or high refers to the similarity condition, and the number represents the Δp for predictor A in the low similarity condition and the Δp for predictor AC in the high similarity condition. The frequency and probability of occurrence of each event (i.e., trial) in the four conditions are listed in Table 8. Trial frequency by outcome combinations, as well as the

Table 9. Trial frequency (f), conditional probability (c/p), and contingency (Δp) associated with each predictor by condition for Experiment 4.

	Condition (similarity / Δp A / AC)											
	low / .6			low / .3			high / .6			high / .3		
	f	c/p	Δp	f	c/p	Δp	f	c/p	Δp	f	c/p	Δp
Predictor A												
A+	16			13			16			13		
A-	4	.8		7	.65		4	.8		7	.65	
~A+	4			7			4			7		
~A-	16	.2	.6	13	.35	.3	16	.2	.6	13	.35	.3
Predictor B												
B+	4			4			4			4		
B-	4	.5		4	.5		4	.5		4	.5	
~B+	16			16			16			16		
~B-	16	.5	0	16	.5	0	16	.5	0	16	.5	0
Predictor AB												
AB+	1			1								
AB-	4	.2		4	.2							
~AB+	19			19								
~AB-	16	.54	-.34	16	.54	-.34						
Predictor AC												
AC+							16			13		
AC-							4	.8		7	.65	
~AC+							4			7		
~AC-							16	.2	.6	13	.35	.3
Predictor ABC												
ABC+							1			1		
ABC-							4	.2		4	.2	
~ABC+							19			19		
~ABC-							16	.54	-.34	16	.54	-.34

associated conditional probability and contingency, for each predictor in the four conditions are listed in Table 9. Simulations of the Rescorla-Wagner and Pearce models were conducted, with the same parameters used in the previous experiments. Figures 11 and 12 depict the theoretical

values for the Rescorla-Wagner and Pearce models respectively. As in the first three experiments, the Rescorla-Wagner model anticipates that the value of the compounds (both AB and ABC) will be consistently higher than the values of the other predictors in all four conditions. This stems from the assumption of complete generalisation of associative strength between a compound and its elements, whereby $V_A = V_A$, whereas $V_{AC} = V_A + V_C$, and $V_{AB} = V_A + V_B$, whereas $V_{ABC} = V_A + V_B + V_C$. The Pearce model, on the other hand, anticipates that the value of AB will be consistently below the value of A in the low similarity condition, and the value of ABC will remain consistently below the value of AC in the high similarity condition. This because both AC and ABC will reflect the generalisation of negative associative strength from their respective configural cues. According to both models, changing the Δp for A from .6 to .3 will result in lower values for both A and AB, and changing the Δp for AC from .6 to .3 will result in lower values for both AC and ABC. The two models anticipate different results across similarity conditions. According to the Rescorla-Wagner model, the net associative strength acquired on a trial is related to the number of individual elements present. A trial with AC will result in a greater net change in associative strength than a trial with A alone. Similarly, a trial with ABC will result in a greater change in associative strength than a trial with AB. Consequently, the rate at which associative strength is acquired will be greater for AC and ABC than for A and AB respectively. According to the Pearce model, the difference between the values of A and AB will be larger than the difference between the values of AC and ABC. That is, similarity will determine the extent to which the value of A and AB, and of AC and ABC, will diverge. This prediction rests on the assumption that configural cues "A" and "AC" will acquire positive associative strength, while configural cues "AB" and "ABC" will acquire negative

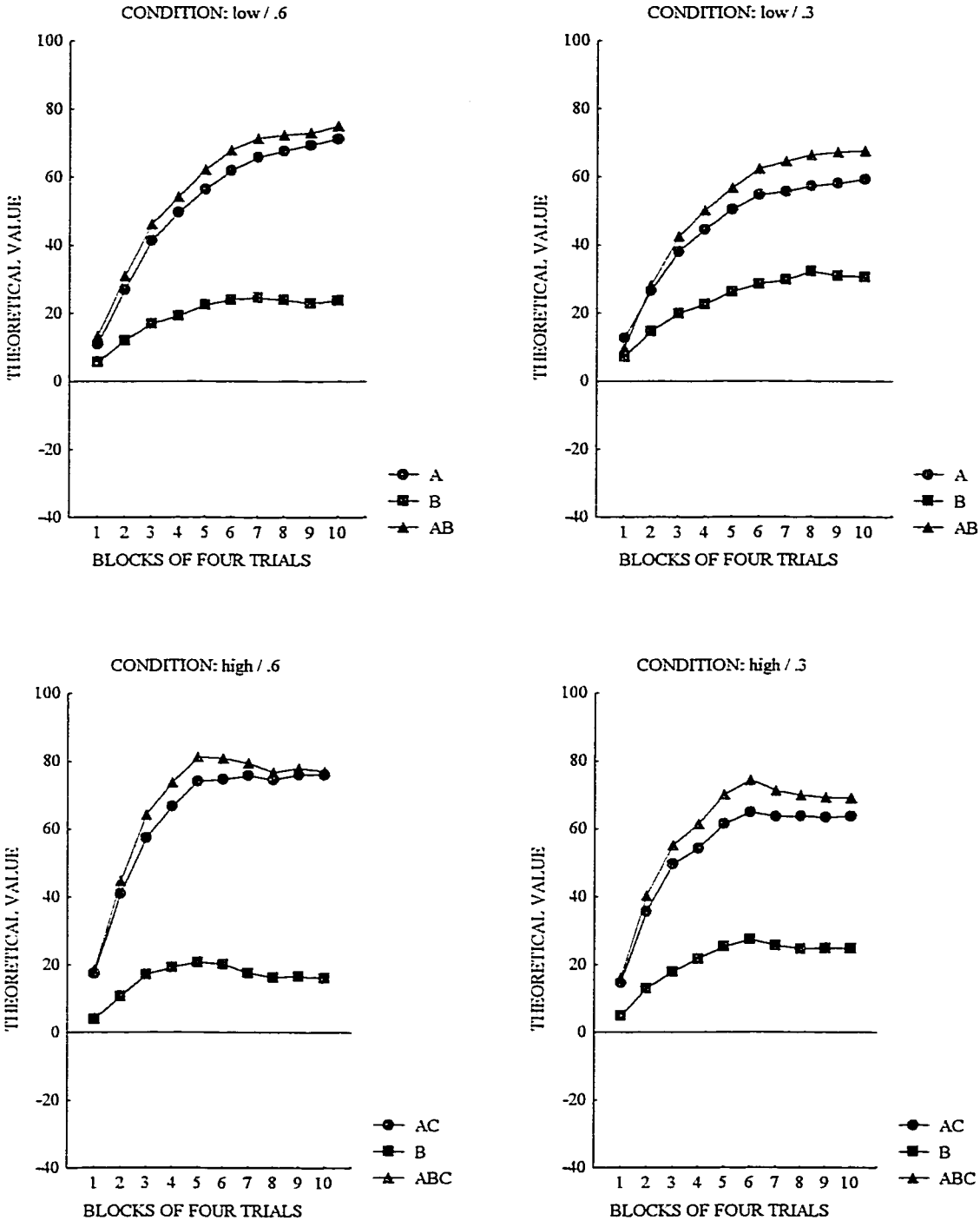

Figure 11. Theoretical values derived from the Rescorla-Wagner model in the four conditions of Experiment 4.

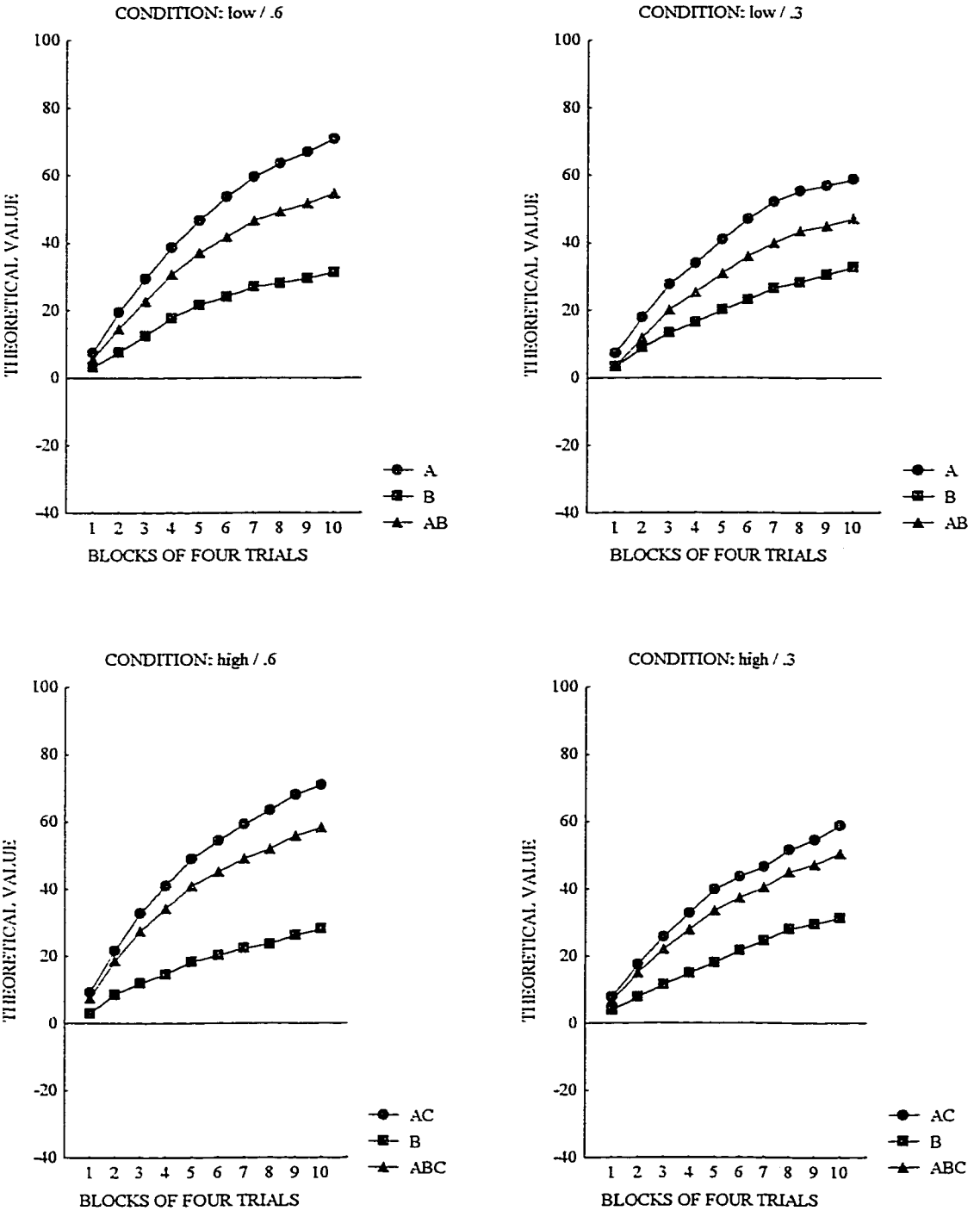

Figure 12. Theoretical values derived from the Pearce model in the four conditions of Experiment 4.

associative strength. As the similarity between AC and ABC is higher than the similarity between A and AB, AC will receive the generalisation of more negative associative strength than A. Consequently, the value of AC will be lower than the value of A. A similar analysis applies to the predicted value of AB and of ABC. As the similarity between ABC and AC is greater than the similarity between AB and A, ABC will receive the generalisation of more positive associative strength than AB. Consequently, the value of ABC will be higher than the value of AB. However, the net associative strength that accrues to A and AB should be equivalent to the net associative strength that accrues to AC and ABC. According to both models, the value of B will remain relatively consistent and moderately positive in the four conditions.

Method

Participants

Twenty-four undergraduate students (19 females, 5 males; mean age = 21 years) participated. After 24 participants were tested, one person was chosen randomly and awarded a $50 prize.

Apparatus and Stimuli

The apparatus and stimuli were the same as in Experiment 2.

Procedure

The procedure was generally the same as in Experiment 3. In the low similarity condition, treatments consisted of either a red pill, a green pill, or a red pill and a green pill presented together. In the high similarity condition, treatments consisted of either a red pill and a blue pill presented together, a red pill a green pill and a blue pill presented together, or a green pill presented alone. Each participant received one practice with the low similarity condition, and

one practice with the high similarity condition, always beginning with low similarity condition. In the low similarity practice trial, the contingency for A was .5 [P(O|A) = .75, P(O|~A) = .25], the contingency for B was -.5 [P(O|B) = .25, P(O|~B) = .75], and the contingency for the AB compound was 0 [P(O|AB) = .5, P(O|~AB) = .5]. In the high similarity practice trial, the contingency for the AC compound was .5 [P(O|AC) = .75, P(O|~AC) = .25], the contingency for B was -.5 [P(O|B) = .25, P(O|~B) = .75], and the contingency for the ABC compound was 0 [P(O|ABC) = .5, P(O|~ABC) = .5]. As in the previous experiments, each clinical trial consisted of 40 individual trials (i.e., patient files), and participants judged the relations between each medication treatment and the facial rash after every 4 trials for a total of 10 judgements per medication. The order of presentation of experimental conditions was counterbalanced between participants, according to the 24 possible permutations of four objects. The assignment of pill colour to contingency was counterbalanced between each level of the two similarity conditions and between subjects.

Results

A within-observation analysis of variance, with individual judgements as the dependant variable, was used to examine the variables of similarity (low, high), contingency for constituent (A or AC, $\Delta p = .6$ and .3), predictor (A/AC, B, and AB/ABC), and trial block (1 to 10). A posteriori comparisons were protected with the Bonferroni procedure. A Type I error rate of .05 was used throughout.

Figure 13 depicts the empirical judgements in the four conditions of this experiment. Judgements of A appear to be higher when the Δp for A was .6 than when the Δp for A was .3. Similarly, judgements of AC appear to be higher when the Δp for AC was .6 than when the Δp

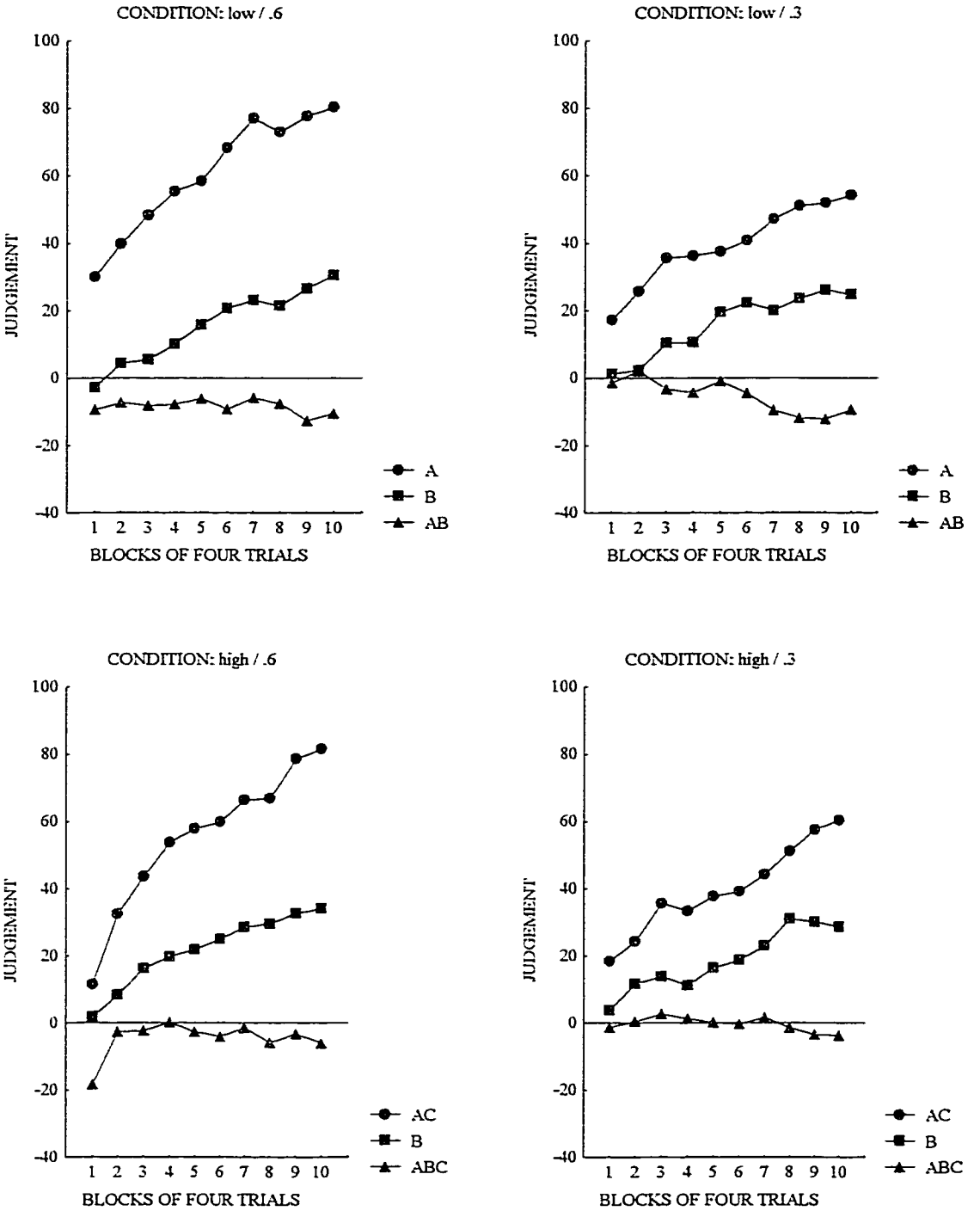

Figure 13. Empirical judgements in the four conditions of Experiment 4.

for AC was .3. Changing the Δp for A or AC does not appear to have affected judgements of AB or ABC respectively. Judgements of AB and ABC remained consistent across levels of Δp for A and AC respectively. Finally, judgements appear to be consistent across levels of similarity.

The statistical analyses confirm these impressions. Judgements differed both by predictor ($F(2, 46) = 35.37$, $MSE = 19717.89$), and trial block ($F(9, 207) = 33.97$, $MSE = 576.36$). The analyses also revealed two-way interactions between Δp for A / AC and predictor ($F(2, 46) = 23.68$, $MSE = 1308.68$), Δp for A / AC and trial block ($F(9, 207) = 4.59$, $MSE = 307.4$), and predictor and trial block ($F(18, 414) = 16.38$, $MSE = 430.25$).

A posteriori comparisons with Bonferroni correction revealed that participants discriminated between the levels of Δp for A / AC ($F(1, 23) = 28.1$, $MSE = 2780.43$), and that changing Δp for A / AC had no effect on judgements of any other predictor. Judgements of AC and ABC in the high similarity condition did not differ from judgements of A and AB in the low similarity condition ($p > .3$).

Discussion

In this experiment, judgements of the contingency between a compound and an outcome were assessed as a function of: a) the contingency between one of its elements and the outcome, and b) the similarity between the compound and one of its elements. Although participants reliable discriminated between the levels of contingency, judgement of compound AB in the low similarity condition, and of ABC in the high similarity conditions remained invariant. Overall, judgements remained equivalent across similarity conditions. The invariant judgements of the AB / ABC compounds are consistent with a configural view of stimuli. However, contrary to the predictions of Pearce's (1987, 1994) configural model, there was no effect involving similarity.

This implies that generalisation of associative strength among predictors is negligible. Thus the results are consistent with one assumption of the model, that stimuli are configural, but not with another assumption, that of similarity. Further, the results are inconsistent with the concept of generalisation from elements to compounds even when such generalisation may be based on a mechanism other than similarity such as in the Rescorla-Wagner model.

Rescorla-Wagner and Pearce models differ in their analysis of the nature of the associations that will accrue on a conditioning trial. According to the Rescorla-Wagner model, the presentation of a reinforced compound will provide the opportunity for each element of the compound to acquire associative strength. The Pearce model presumes that only one association will develop on any conditioning trial, that between the pattern of stimulation as a whole (i.e., a configural predictor) and the outcome. Experiment 4 assessed the effect of adding a common element to predictors A and AB, yielding predictors AC and ABC. According to the Rescorla-Wagner model, predictors AC and ABC should be rated higher than predictors A and AB respectively, as the net associative strength accrued on a trial is proportional to the number of predictors presented. Contrary to this prediction, judgements were equivalent for the two sets of predictors. This finding is more consistent with the notion that a conditioning trial results in the formation of only one association, between a configural representation of all the predictors presented on that trial and the outcome. Participants assessed A+ and AB- trials in a fashion equivalent to AC+, and ABC- trials respectively. These findings strengthen the conclusions of the first three experiments in this report. Compound predictors appear to be functionally independent of their constituent elements.

GENERAL DISCUSSION

Four experiments assessed the processing of compound predictors in contingency judgements. Participants judged the relation between compound predictors and an outcome, as well as the relation between their constituent elements and the outcome, under different contingency and similarity manipulations. These manipulations were implemented in a fictitious medical context in which taking different medications (predictors) for a given disease was related to the occurrence of a facial rash (outcome).

Two general conclusions may be drawn from the results of these experiments. The first conclusion is that a theory of contingency judgement must incorporate the notion of configural cues. Across experiments, judgements of a compound were made independently of the normative relation between its constituent elements and the outcome. Specifically, judgements of two- (Experiments 1, 2, 3, and 4) or three-element (Experiments 3 and 4) compounds remained unaffected by changes in the contingency between one or more of their constituent elements and the outcome. In fact, changes in the contingency of a compound left judgements of its constituent elements similarly unaffected (Experiment 3). These results are consistent with the notion that compound predictors are assessed as unique configural representations, and inconsistent with the notion that a process of summation of associative strength mediates the assessment of compounds. The second conclusion is that similarity does not represent a basis for the generalisation of associative strength among predictors. Changing the similarity among predictors did not have the theorised effects. In Experiment 1, judgements in a high similarity condition were lower than judgements in a low similarity condition, contrary to the predictions of both models. Further, manipulating the similarity between a compound and one of its

constituent elements did not affect judgements of the compounds in Experiment 4. The results of Experiment 4 are consistent with the notion that a conditioning trial provides the opportunity for the formation of only one associative strength; between a configural representation of the predictors present and the outcome. Some might consider these conclusions weak because they rely on the null hypothesis. It must be pointed out, however, that the invariance of the judgements of the compound predictors has been obtained each time in the context of other statistically reliable differences caused by the experimental manipulations. This indicates that the task is sensitive enough, and that the compounds are indeed invariant.

In the light of data from four experiments that is largely inconsistent with the predictions of either associative model, the extent to which modifications of the models could account for the findings remains an important theoretical objective. Computer simulations of the Rescorla-Wagner and Pearce models were conducted with various parametric and structural changes that nevertheless maintained the essential character of the models. For these simulations, a variety of values for α and β were used. Although an exhaustive parameter fitting processes was not undertaken, the values used for these simulations are consistent with those in other reports (e.g., Redhead & Pearce, 1995), and the pattern of theoretical values was qualitatively the same for all the parametric values attempted. A consideration of the fit between the theoretical values and the empirical judgements raises the issue of the accuracy of judgements. Although high levels of absolute accuracy have been reported (Chatlosh et al., 1985), it is generally accepted that absolute discrepancies between subjective judgements and normative contingencies are less informative than qualitative discrepancies (Shanks, 1993a). For example, judgements of zero contingencies are often rated to be moderately positive when a unidirectional scale is used

(Dickinson et al., 1984; Neunaber, & Wasserman, 1986; Shanks, 1993a). However, when a bi-directional scale is used, as in this report, non-contingent relations are more frequently rated as close to zero (Neunaber, & Wasserman, 1986). There is reason to believe that while the magnitude of judgements may reflect a measurement artefact when a unidirectional scale is used, at least the polarity of the contingency assessment on a bi-directional scale reflects distinct psychological meanings. Most would appreciate that playing golf in the middle of a thunderstorm increases the likelihood of being struck by lightening compared to staying home. Conversely, most sufferers of hypertension understand the importance of following the appropriate drug regimen in order to decrease the likelihood of having a cardiac arrest. Sensitivity to both ends of a bi-directional rating scale was evident in Experiment 3 where judgements systematically reflected the reversal of positive and negative contingencies across conditions. It is noteworthy, then, that judgements of B were moderately positive both when the Δp for B was 0 (Experiments 1 to 4), and when the Δp for B was -.5 (Experiment 2, conditions .6/-.5 and .3/-.5). With these considerations in mind, model theoretical values were subjected to two different evaluation criteria. One, model predictions were evaluated on the basis of whether they accurately reproduced the ordinal relations found in the empirical judgements. Two, predictions were evaluated on the basis of whether they reproduced the negative judgements found in the experiments. The effectiveness with which model predictions reproduced the empirical judgements was thus evaluated against both of these criteria.

Evaluation of the Rescorla-Wagner Model

The Rescorla-Wagner model does not adequately describe the pattern of empirical judgements in any of the four experiments. This inability stems primarily from the assumption

that judgements of a compound reflect the associative strength of its constituent elements. Across experiments, however, judgements of a compound did not reflect either the normative contingency or subjective assessment of its constituent elements. Related to this is the assumption that the net associative strength accruing to a compound will be directly proportional to the number of its elements. This led the model to predict that the value of AC and BC in Experiment 1 would be higher than the value of A and B respectively, and that the value of AC and ABC in Experiment 4 would accrue at a faster rate than the value of A and AB respectively. Both of these predictions were disconfirmed.

The Rescorla-Wagner model was modified in an attempt to address these inconsistencies without sacrificing the essential elemental character of the model. The elemental nature of the Rescorla-Wagner model is captured by the assumption that a conditioning trial provides the opportunity for the formation of an association between each element on that trial and the outcome. Judgements to a compound of several elements will then reflect the associative strength accrued to each element of the compound in an additive fashion. One modification may be to assume that conditioning to a compound will result in the formation of an association between the compound as a whole and the outcome in addition to the associations between the elements of the compound and the outcome (Saavedra, 1975; Rescorla, 1972, 1973; Rescorla et al., 1985; Whitlow & Wagner, 1972). When a compound AB is paired with an outcome, elements A, B, as well as the configural cue "AB" may enter into an association with the outcome. The net associative strength accruing to the compound AB would be the sum of the associative strengths of elements A, B, and of "AB" (Whitlow & Wagner, 1972). A difficulty with assuming configural cues within an elemental model, already alluded to in the general introduction, is that

of a combinatorial explosion of configural cues with even a modest number of elements (Pearce, 1994). A compound composed of three elements such as in experiment 4 (i.e., ABC) can potentially result in the formation of seven different associations (i.e., between each of A, B, C, "AB", "AC", "BC", and "ABC", and the outcome), while a compound of five elements could result in the formation of thirty-one different associations. Given inherent limitations in information processes capacity, such an associative process would quickly exhaust the available cognitive resources. Another strategy is to allow only the highest order configural cue. A trial with compound ABC would then result in the formation of four different associations; between each of A, B, C, and "ABC", and the outcome. A trial with compound AB would provide the opportunity for the formation of three associations: A, B, and "AB". Only the latter, or partial, configural implementation of the Rescorla-Wagner model was investigated further.

A simulation was conducted of the partial configural implementation of the Rescorla-Wagner model, with the same parameters used in the previous simulations. Even with configural cues, the Rescorla-Wagner model was unable to predict many of the ordinal relations found in the empirical judgements. For example, in Experiment 4 the value of ABC remains higher than the value for AC. This is because the value for ABC now reflects $V_A + V_B + V_C + V_{\text{"ABC"}}$, and all the elements including the configural cue "ABC" acquire positive associative strength. Figure 14 depicts the theoretical values of the modified Rescorla-Wagner model for Experiment 4. These values are qualitatively similar to the values of the unmodified model for Experiment 4 depicted in Figure 11. In fact, the addition of configural cues now creates theoretical values for ABC that are greater than the theoretical values for ABC in the original simulation.

The inability of even a configural implementation of the Rescorla-Wagner model to

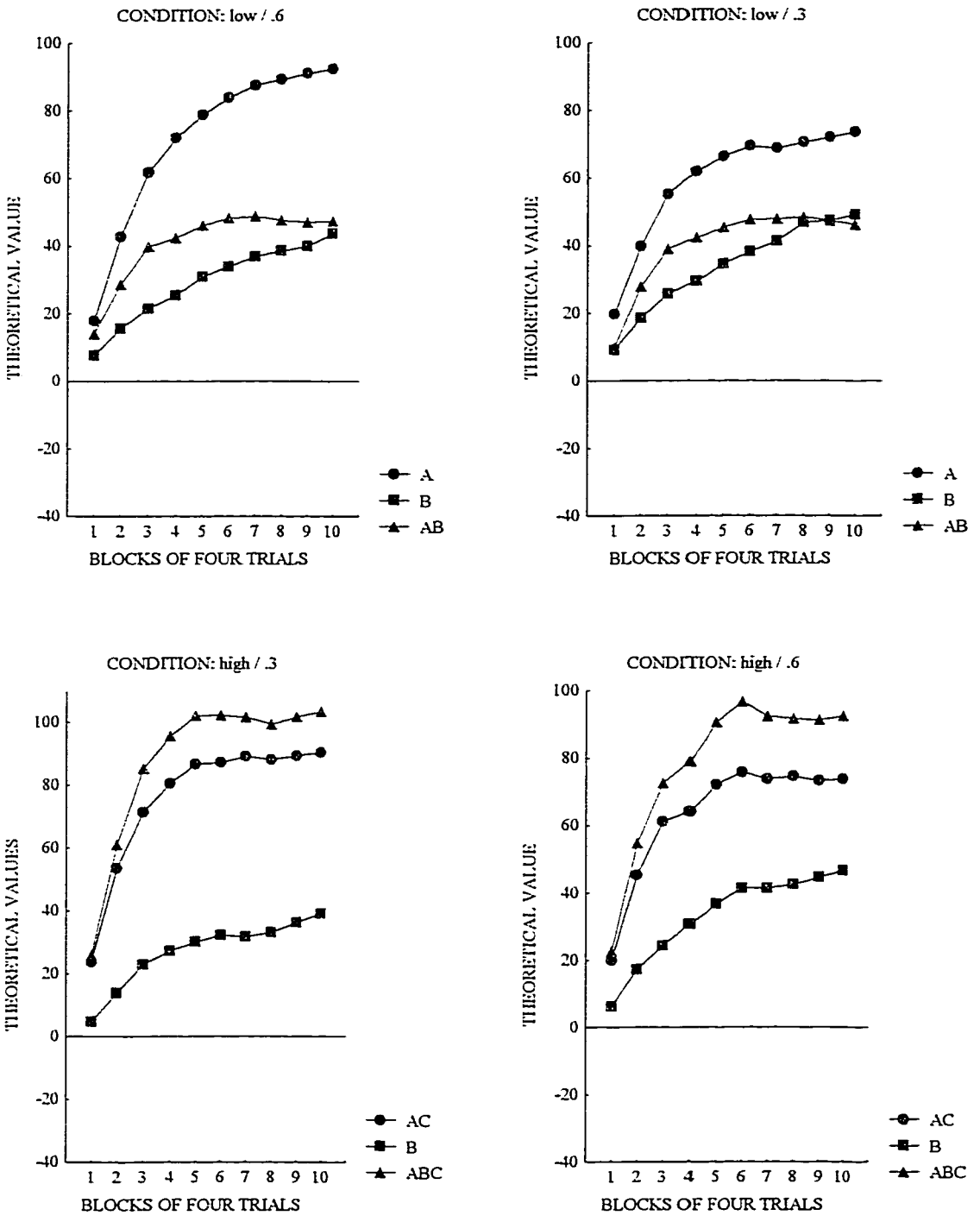

Figure 14. Theoretical values derived from the configural implementation of the Rescorla-Wagner model in the four conditions of Experiment 4.

capture the ordinal relation of the empirical judgements appears to stem from the fact that no predictors, including configural cues, acquire negative values. This is related to a fundamental asymmetry between the effects of reinforcement and non-reinforcement inherent in the model. The change in associative strength on a conditioning trial is dependent on the value $\lambda_1 - V_T^n$; where λ_1 is the maximum associative strength supported by the outcome$_1$, and V_T^n is the sum of the associative strength already gained by all predictors present on that trial. The value of λ is 1 in the presence of the outcome and 0 on trials when the outcome does not occur. As a consequence of this process, positive associative strength will accrue at a faster rate than negative associative strength. Positive increments in associative strength can occur on any trial when the outcome is present, but negative increments can occur only if two conditions are met, a) the outcome is not present, and b) the value V_T^n is greater than zero. For example, if A is presented without the outcome on the very first conditioning trial, it will not gain negative associative strength as $V_T^n = 0$, and the difference $\lambda_1 - V_T^n = 0$. Thus, a predictor will only gain negative associative strength when presented in compound along with other predictors whose net associative strength is greater than 0 (Rescorla, 1969; Rescorla & Holland, 1977). It remains possible that a more symmetrical treatment of reinforcement and non-reinforcement will generate theoretical values that are more consistent with the empirical judgements.

A second simulation of the partial configural implementation of the Rescorla-Wagner model was conducted. For this simulation λ was 1 in the presence of the outcome and -1 in the absence of the outcome. With both modifications, the Rescorla-Wagner model captures a larger portion of the relations found in the empirical judgements with several notable exceptions. The

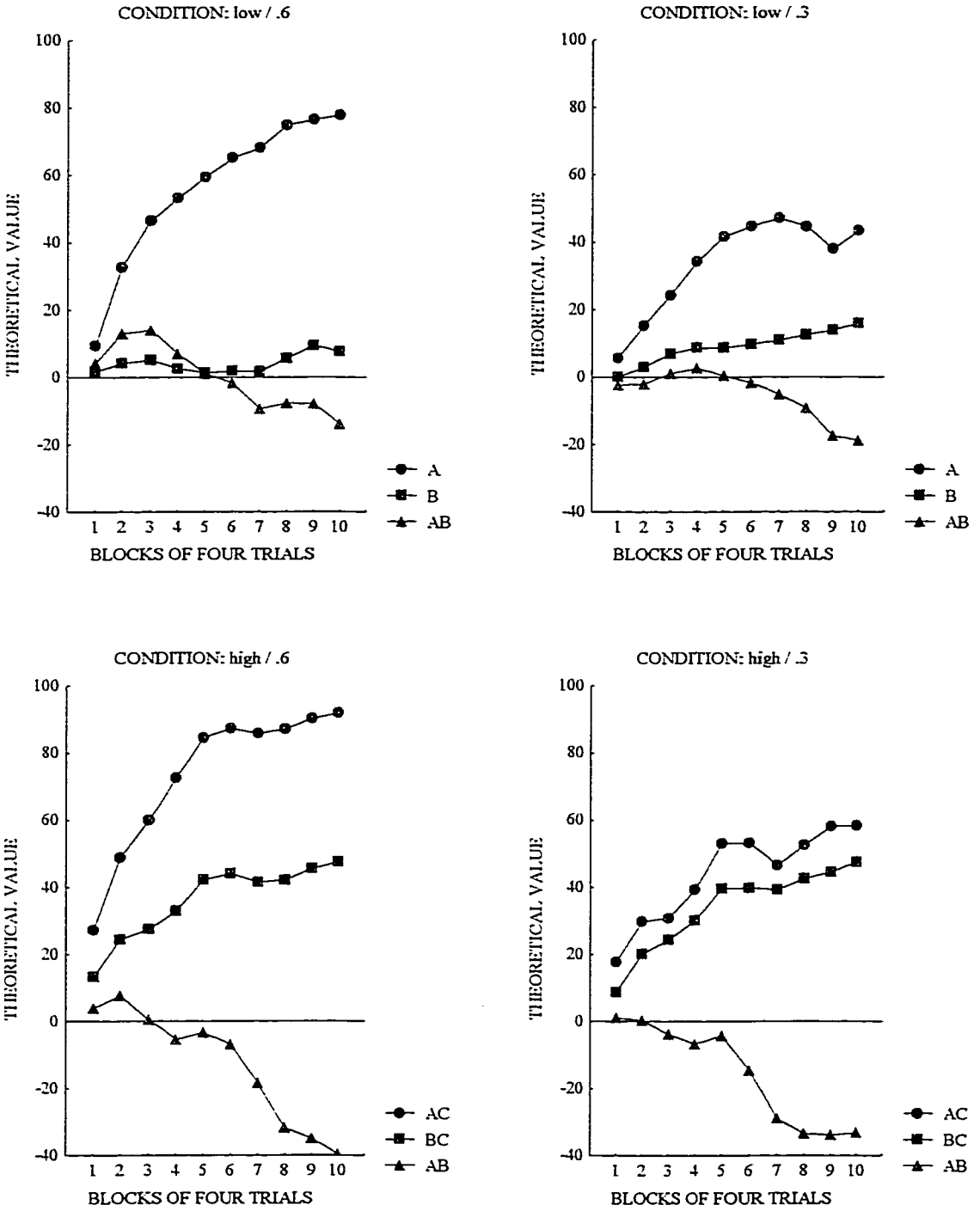

Figure 15. Theoretical values derived from the configural implementation of the Rescrola-Wagner model with lamda-no outcome set at -1, in the four conditions of Experiment 1.

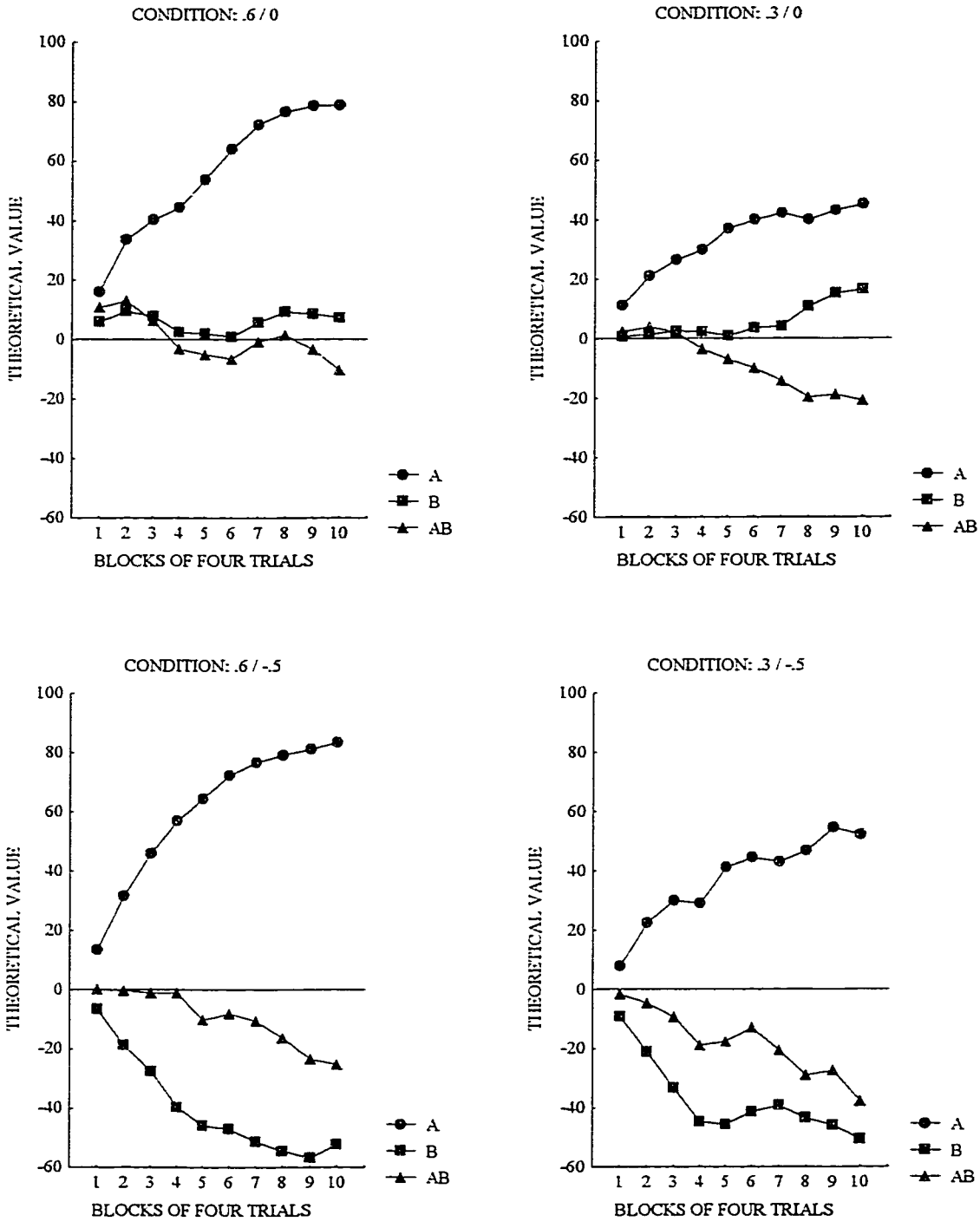

Figure 16. Theoretical values derived from the configural implementation of the Rescorla-Wagner model with lambda-no outcome set at -1, in the four conditions of Experiment 2.

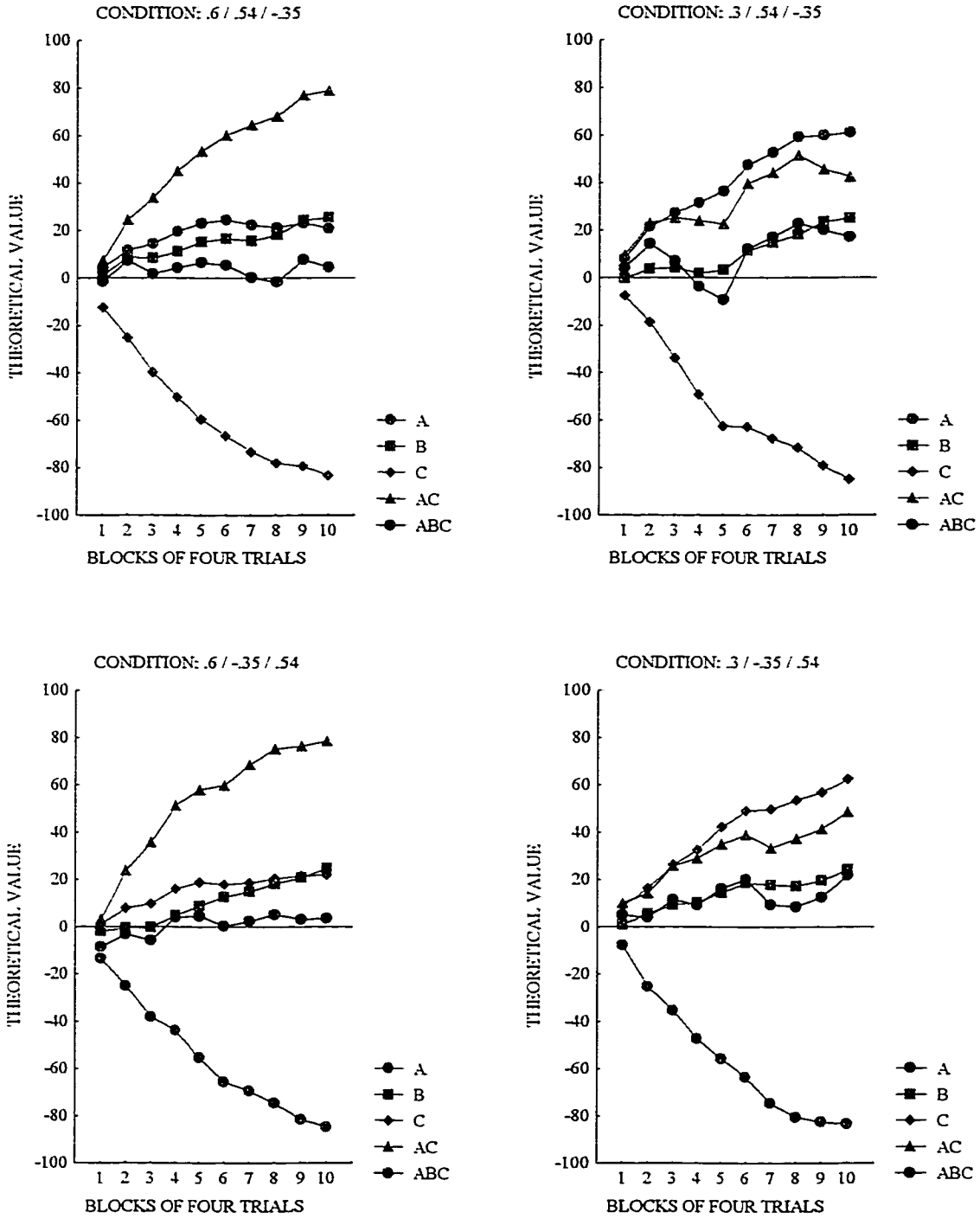

Figure 17. Theoretical values derived from the configural implementation of the Rescorla-Wagner model with lamda-no outcome set at -1, in the four conditions of Experiment 3.

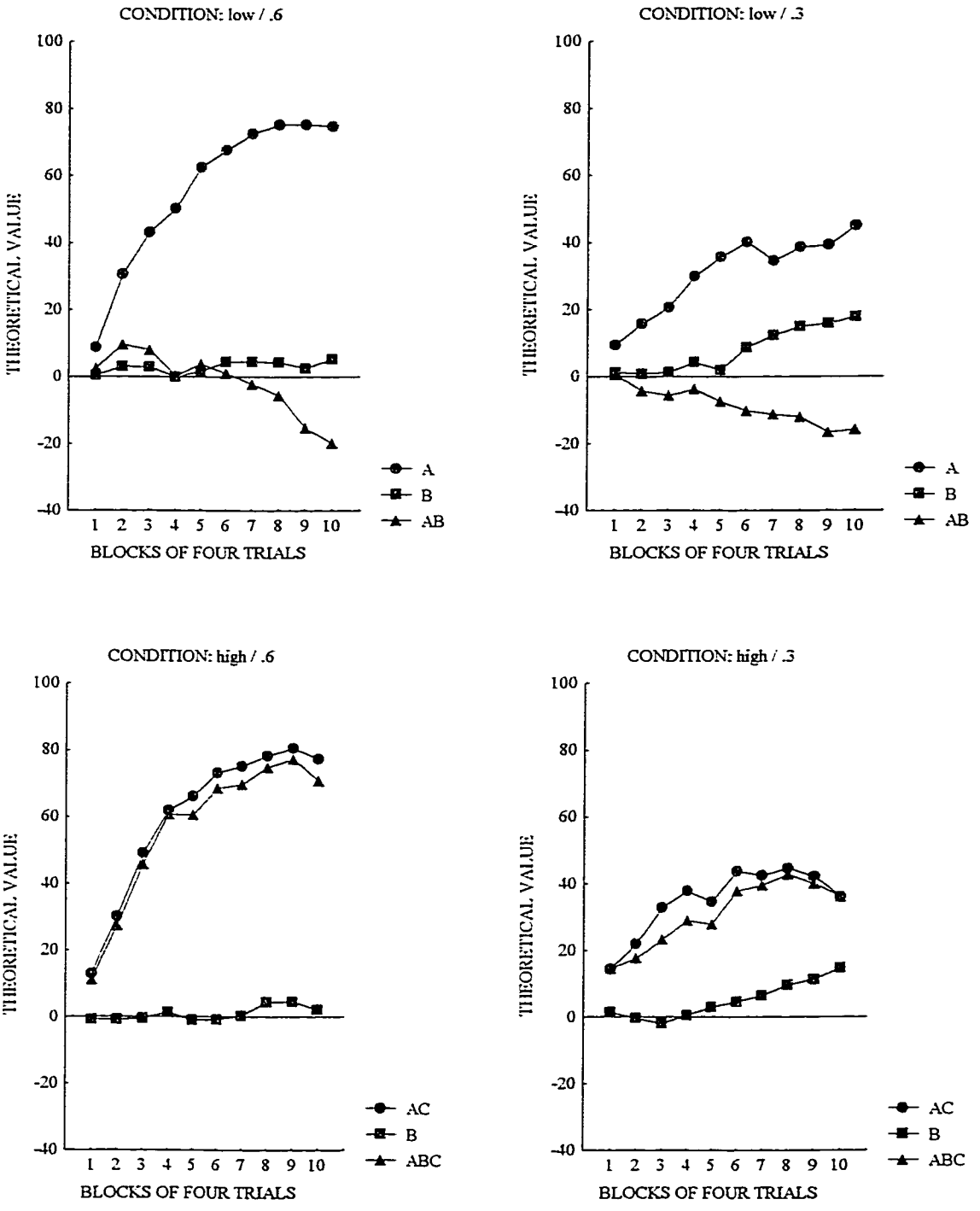

Figure 18. Theoretical values derived from the configural implementation of the Rescorla-Wagner model with lamda-no outcome set at -1, in the four conditions of Experiment 4.

theoretical values derived from the second revision of the Rescorla-Wagner model in the four experiments are depicted in Figures 15 through 18. The revised model accurately describes the ordinal relations found in Experiment 1, including the contingency effect and negative values for the AB compound. The value of the AB compound remains relatively unchanged across levels of contingency for A and AC, but changes across levels of similarity. Contrary to the empirical findings the values for A and B continue to be lower than the values for AC and BC respectively. With regard to Experiment 2, the model accurately describes the ordinal relations in the .6/0 and .3/0 conditions, but not in the .6/-.5 and .3/-.5 conditions. The theoretical value for B is lower than that for AB when the Δp for B is -.5. Empirical judgements of B, however, remained positive despite its negative contingency with the outcome. Contrary to the findings, the value of AB appears to differ slightly between the conditions when the Δp for B is 0 and when the Δp for B is -.5. In the conditions of Experiment 3, the ordinal relations among theoretical values are accurate except for a reversal in the ordinal relation between A and AC in the .3/.54/-.35 condition, and between C and AC in the .3/-.35/.54 condition. The model also accurately depicts the increase in A between conditions .6/.54/-.35 and .3/.54/-.35, as well as the increase in the value for C between conditions .6/-.35/.54 and .3/-.35/.54. However, the revised model does not accurately characterise the negative value for ABC. With Experiment 4, the revised model accurately depicts the ordinal relations only in the low similarity condition. In the high similarity condition, the theoretical value for ABC remains positive and above the value for B. Another simulation that also used $\lambda = -1$ but without configural cues, was not successful.

In sum, a revision of the Rescorla-Wagner model that incorporates both configural cues and a symmetrical characterisation of reinforcement and non-reinforcement does not accurately

reflect the empirical findings. In contrast to the empirical findings, the revised model continues to generate theoretical values for compounds that are sensitive to both contingency and similarity manipulations. Although other modifications of the model may result in theoretical values that more accurately reflect the empirical findings, these modification would probably change the essential character of the model. For example, simulations were conducted of the partial configural implementation of the model that eliminated the contribution of the individual elements by setting their α values to 0. This new model, that incorporated only configural cues, was relatively more accurate in reflecting the ordinal relations found in the empirical judgements. This model, however, bears very little theoretical resemblance to the original formulation. At this point, it is difficult to conceptualise of a modification to the Rescorla-Wagner model that would both maintain the essential elemental character of the model and accurately describe the empirical results.

Evaluation of the Pearce Model

Although the theoretical values derived from the Pearce model are relatively more accurate than those derived from the unmodified Rescorla-Wagner model, significant discrepancies between theoretical and empirical values remain. These discrepancies stem primarily from the notion of similarity proposed by the Pearce model. The essential configural nature of the Pearce model is that a conditioning trial provides the opportunity for the formation of only one association; that between a configural representation of all the elements presented on that trial and the outcome. Judgements to a compound will reflect the associative strength of the configural representation of the compound, as well as the associative strength generalised to the compound from other configural representation as a function of similarity. Across contingency

and similarity manipulations, however, judgements of a compound more closely reflected its normative contingency and remained unaffected by the presence of other predictors sharing elements with it. These findings are consistent with the notion that compounds are assessed as unique configural representations, and that judgements of a compound do not reflect the generalisation of associative strength based on similarity.

A simulation of the Pearce model was conducted with the same parameter values as in previous simulations. As the assumption of configural processing with limited generalisation of associative strength to a compound appears to be a viable description of the empirical data, it was assumed that judgements of a compound reflect only the associative strength of its configural cue. That is, the generalisation of associative strength to a compound as a function of similarity was assumed to be nil. Figures 19 through 22 depict the results of this simulation for the four conditions in each experiment. The revised model accurately captures all of the major empirical findings including: the effect on judgements of a predictor when its normative contingency is changed, positive judgements of B both when its Δp is 0 and when its Δp is -.5 (Figure 20), overshadowing of the judgements of a moderate positive predictor by a compound with a slightly higher contingency with the outcome (Figure 21, conditions .6/.54/-.35 and .6/-.35/.54), judgements of compounds that are unaffected by either contingency or similarity manipulations, and the consistently negative judgements of compound predictors with a negative normative contingency. With regard to the last point, terminal theoretical values of the AB compound (Experiments 1, 2, and 4) or of the ABC compound (Experiments 3 and 4) more closely approximate the negative empirical judgements with the added assumption that α for the configural cues is higher than .3. The theoretical values derived from this revised configural

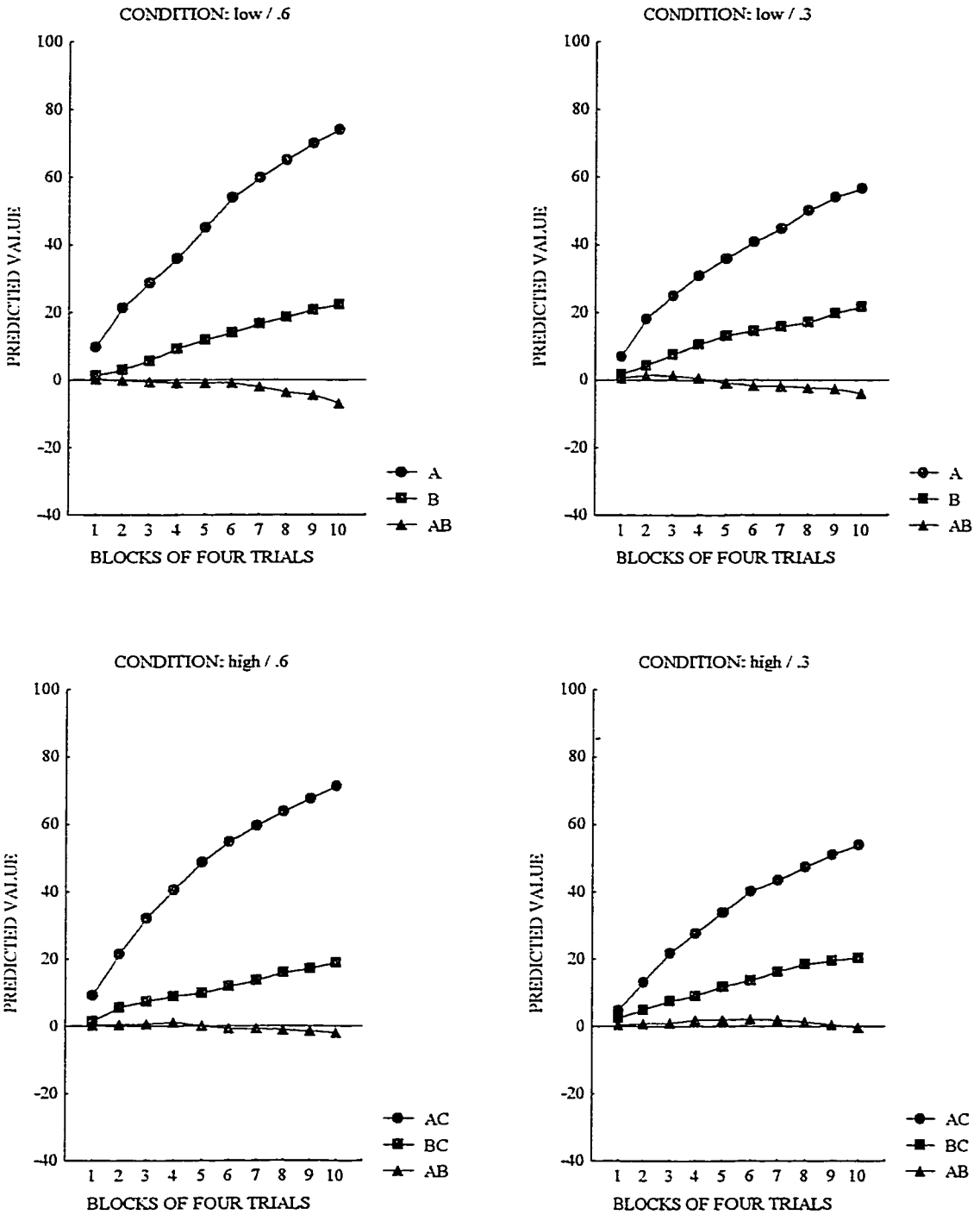

Figure 19. Theoretical values derived from the Pearce model with the assumption of no generalisation of associative strength among predictors, in the four conditions of Experiment 1.

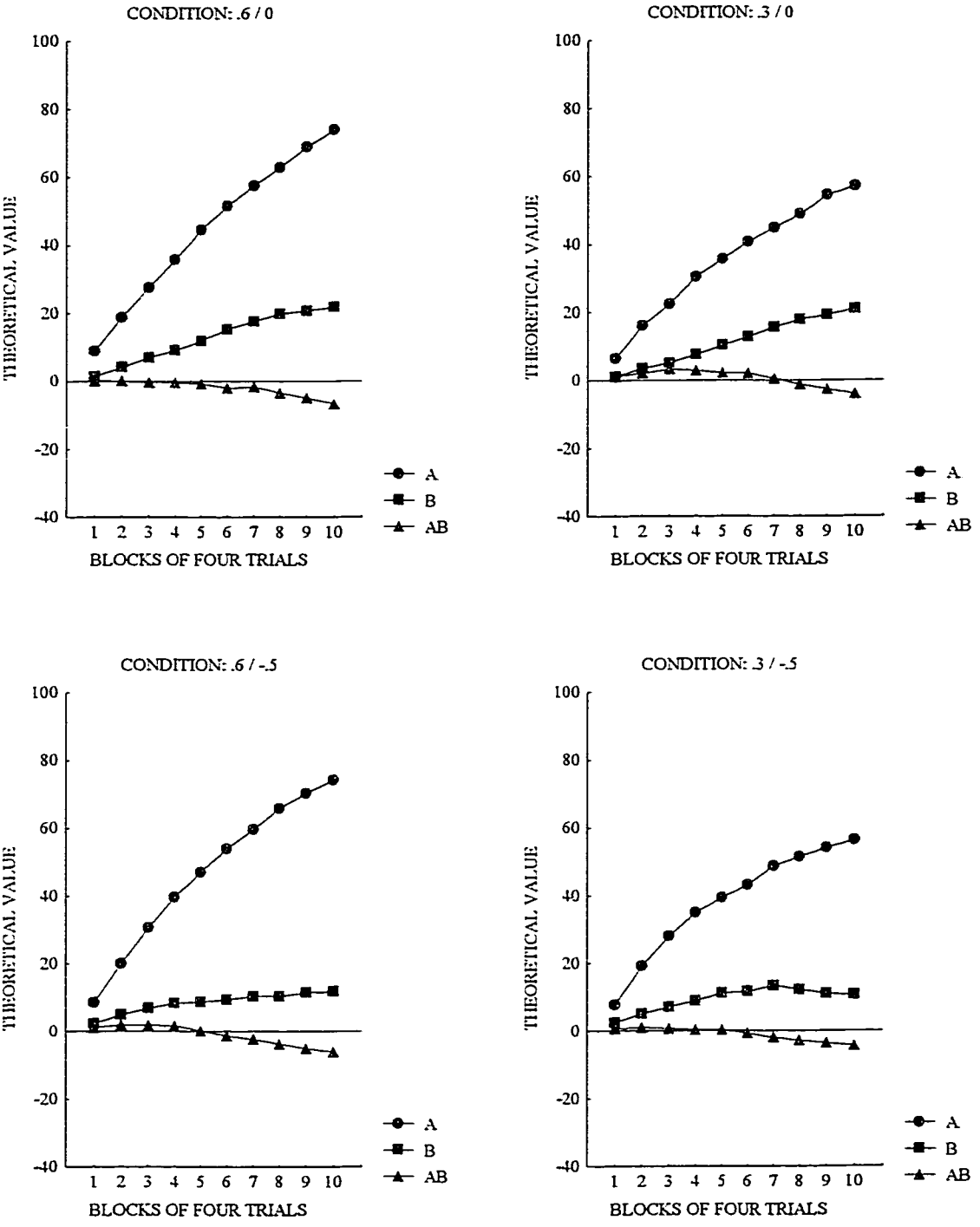

Figure 20. Theoretical values derived from the Pearce model with the assumption of no generalisation of associative strength among predictors, in the four conditions of Experiment 2.

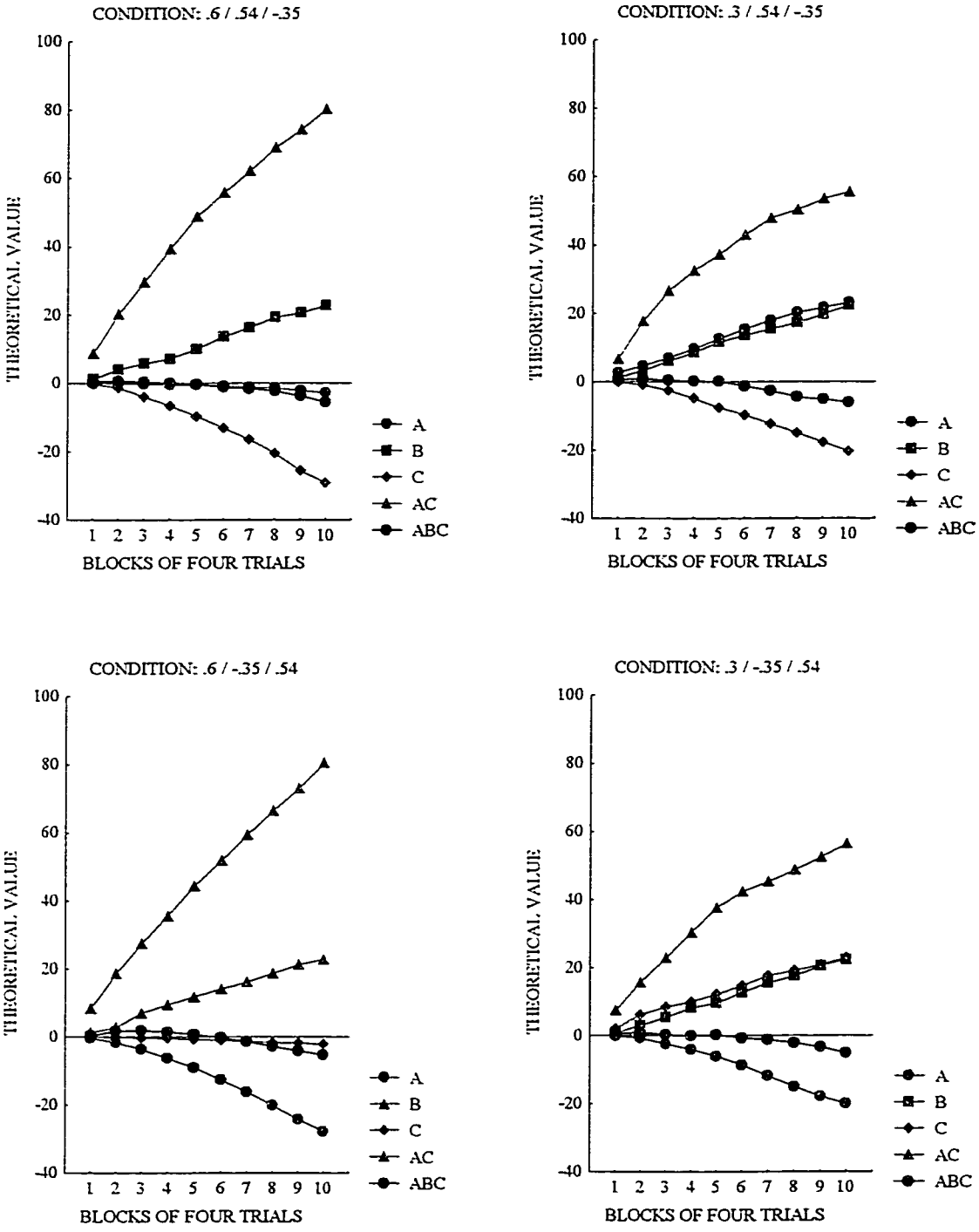

Figure 21. Theoretical values derived from the Pearce model with the assumption of no generalisation of associative strength among predictors, in the four conditions of Experiment 3.

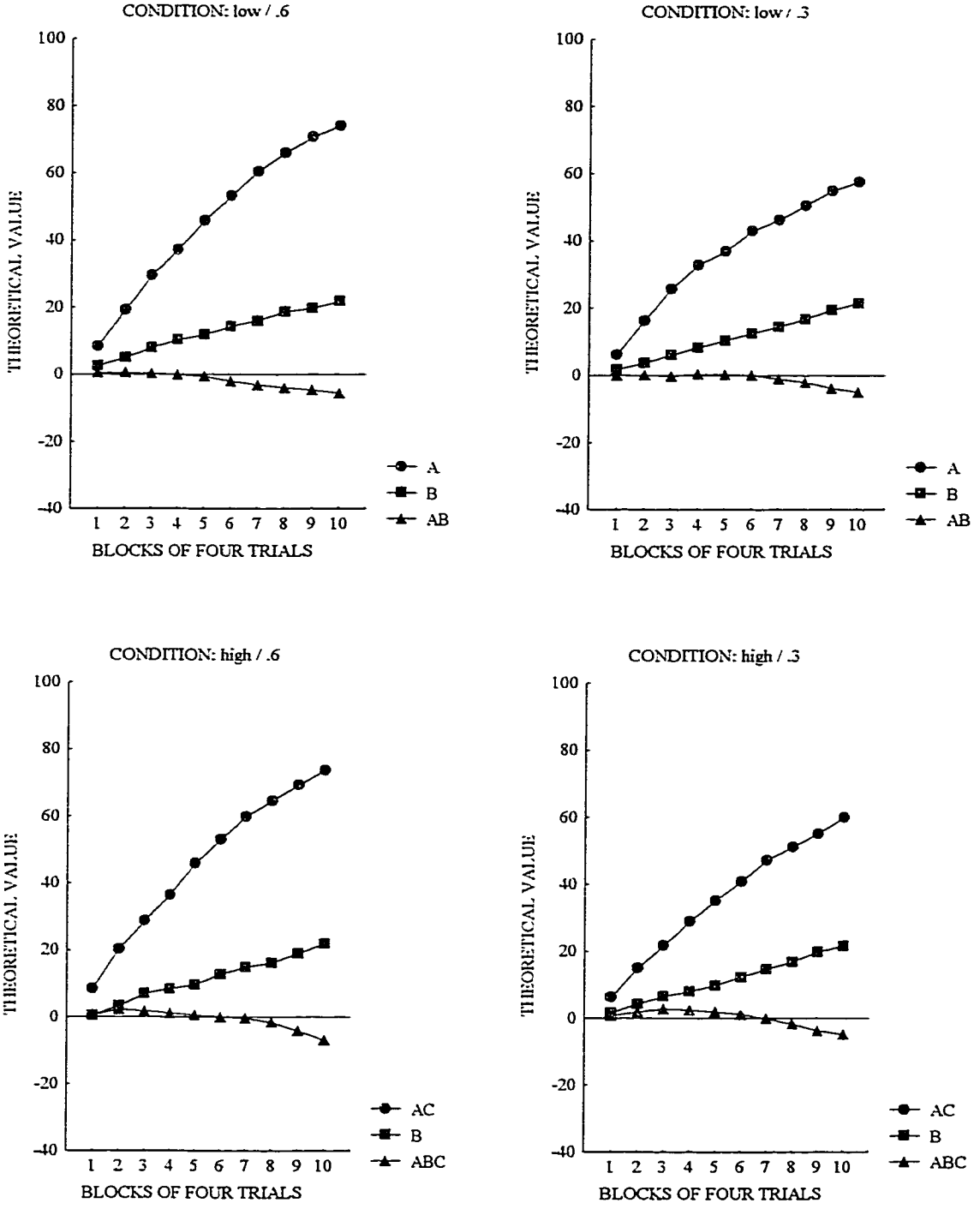

Figure 22. Theoretical values derived from the Pearce model with the assumption of no generalisation of associative strength among predictors, in the four conditions of Experiment 4.

model accurately reflect all the ordinal relations in the empirical judgements with two exceptions. In Experiment 2, participants judged A to be lower than B in condition .3/0, and participants judged A and B to be equivalent in condition .3/-.5, whereas the theoretical values for A remain higher than the values for B in the both conditions. Finally, the sum of the theoretical values in the low similarity condition of Experiment 1 is equivalent to the sum of the theoretical values in the high similarity condition. Empirical judgements, however, were lower in the high similarity condition.

The inability of the revised Pearce model to capture the main effect of similarity in Experiment 1 is a shortcoming that needs to be addressed. It is not clear at this point how this can be done within the confines of the conceptualisation offered by Pearce (1994). The finding itself, however, is not inimical to an associative interpretation per se. Another associative model proposed by Wagner (1981), and consistent with the 1972 formalisation, presumes that the number of stimulus elements that could be simultaneously conditioned is limited to two or three. Due to this capacity constraint, primarily related to a limitation in the short term memory buffer, the presentation of three or more stimulus elements simultaneously could result in an overall level of conditioning less than that to a presentation of fewer stimulus elements. The high similarity condition in Experiment 1 contained more separable stimulus elements than the low similarity condition. Consequently, less associative strength may have accrued in the high similarity condition.

Implications and Future Directions

The most crucial findings in this report are that a compound predictor is assessed independently of the relation, normative or judged, between its constituent elements and the

outcome, and that judgements appear to be unaffected by the similarity among predictors. Also of theoretical importance is the finding of a main effect of similarity in Experiment 1 that runs counter to the theoretical values derived from either model. Thus, a comprehensive associative model that would seem to work for contingency judgement would be one that: a) incorporates the notion of configural stimuli, b) minimises the notion of generalisation among predictors, and c) imposes limitations on the short-term memory buffer.

The pattern of empirical results in this report is consistent with the notion that compound predictors are assessed as unique configural representations. The assessment of a compound predictor was done independently of the predictive value, normative or subjective, of its constituent elements. Further, the assessment of individual predictors was done independently of the predictive value, normative or subjective, of a compound of which they were elements. It also appears that a configural representation can interact with other predictors of the same outcome in a fashion consistent with classic cue interaction effects in associative learning. It is generally recognised in both non-human associative learning and contingency judgements that the value of a predictor is relative to the value of other predictors of the same outcome (Gluck & Bower, 1988; Price & Yates, 1993; Vallée-Tourangeau et al., 1994; Wagner, 1969; Wagner et al., 1968; Wasserman & Berglan, 1998). In Experiment 3, judgements of a moderately positive single predictor were suppressed in the presence of a compound with a slightly higher contingency with the outcome. This finding seems to reflect a process similar to the relative cue validity, or overshadowing, phenomenon. However, this finding also suggests differences between the processing of compound and single predictors. In the standard overshadowing effect, a single predictor with a strong contingency with the outcome overshadows another single

predictor with a much weaker contingency with the outcome. For example, in the study reported by Baker et al. (1993), the overshadowing cue was a perfect predictor of the outcome ($\Delta p = 1$) whereas the overshadowed cue was only a moderate predictor ($\Delta p = .5$). In Experiment 3, however, the overshadowing cue was a moderate compound predictor ($\Delta p = .6$) and the overshadowed cue was a moderate single predictor ($\Delta p = .54$). Configural processing of compound predictors by non-humans does not appear to be related to the perceptual properties of the compound (i.e., its greater intensity or complexity) (Wasserman & Miller, 1997; Whitlow & Wagner, 1972). It remains possible that compound processing in humans is related to some other general characteristics of the configural representation in addition to its normative status. So, for example, predictor AC in Experiment 3 may have overshadowed judgements of predictor A (conditions .6/.54/-.35, and .3/.54/-.35) because of some general property of compound predictors that renders them psychologically more salient to humans relative to single predictors.

A modification of Pearce's (1987; 1994) configural model that assumes no generalisation of associative strength among predictors is able to describe the great majority of empirical findings. Note that according to the modified configural model presented above, similarity among predictors still impacts on the conditioning of a configural cue. That is, on a given conditioning trial the associative strength that accrues to a configural predictor is limited by the sum of two separate components: a) its own associative strength immediately before the current trial, and b) the associative strength of all other configural predictors previously paired with the outcome as a function of similarity. Similarity among predictors, then, continues to impact on the associative strength that may accrue to any one configural representation although not on the generalisation of associative strength among predictors.

It is generally recognised that generalisation is a fundamental aspect of associative learning (Pearce, 1987). Williams et al. (1994, page 707) alluded to the fact that a configural associative process with no generalisation would be handicapped in the face of novel circumstances. As predictors in everyday experiencing often occur in compound, a cognitive process that does not allow for generalisation would be unable to draw inferences about an individual predictor that never occurred alone. Consequently, a further assessment of the effect of generalisation on judgements of contingency would be to investigate judgements about predictors that do not occur alone. In the four experiments presented in this report, all the predictors that participants were required to assess occurred individually on at least one trial. For example, each of the five predictors in Experiment 3 (A, B, C, AC, and ABC) was presented at least once. Future experiments may assess judgements of predictors that participants are never exposed to. If the effect of generalisation is negligible, then such judgements should be close to zero as participants would have no basis for inference.

On two separate occasions in this report the issue of limits in processing capacity has emerged. Relatively little research has addressed the issue of capacity constraints on contingency judgement (Mercier & Parr, 1996). As explained earlier, a full configural implementation of the Rescorla-Wagner model would result in compound ABC generating seven different cues (i.e., A, B, C, "AB", "AC", "BC", and "ABC"). It seems reasonable to conclude that such an implementation of configural processing would quickly exhaust the available processing resources of an organism, with any number of cues approaching what is commonly experienced. Accordingly, the simulations were conducted with only the highest order configural cue present on a trial. Similarly, in Experiment 1 the number of separable stimulus elements was related to

overall judgements. Judgements were lower in the condition with more separable elements than in the condition with less separable elements. As suggested, this result is consistent with the associative model proposed by Wagner (1981) that imposes a limit on the number of stimulus elements that can be represented in the short term memory buffer simultaneously, and consequently on the total associative strength that can accrue on a conditioning trial. Research with non-humans suggests that compounds of multiple predictors require more time to "fuse" into configural representations than single predictors. For example, Kehoe & Schreurs (1986) reported that the discrimination between a compound and its constituent elements is directly related to the duration of the CS. A longer stimulus duration presumably increase the speed at which a configural representation develops. Given that configural representations may require a longer time to consolidate, and the detrimental effect of decreasing processing time on the accuracy of contingency judgements (Mercier & Parr, 1996), investigating the central processing constraints on configural processing may prove fruitful.

In sum, the empirical findings from four experiments provide a preliminary analysis of compound predictor processing in contingency judgements. Compound predictors are assessed in a fashion more consistent with configural associative processes than with elemental processes. Consistent with the results reported by Shanks et al. (1998), a compound predictor appears to be functionally independent of its constituent elements. These findings lend support to the notion of contrasting among different types of associative models in contingency judgement (López et al., 1998; Mercier, 1996). Further investigations in the processing of configural predictors promises to provide a more authentic understanding of the multicausal nature of common everyday experiences.

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APPENDIX A

Tr	X	A	B	O
1	1	1	1	0
2	1	1	1	0
3	1	1	0	1
4	1	1	1	0
5	1	1	0	1
6	1	1	1	0
7	1	1	1	0
8	1	1	1	0
9	1	1	0	1
10	1	1	0	1
11	1	1	1	0
12	1	1	0	1
13	1	1	1	0
14	1	1	1	0
15	1	1	1	0
16	1	1	0	1
17	1	1	0	1
18	1	1	0	1
19	1	1	0	1
20	1	1	1	0
21	1	1	0	1
22	1	1	1	0
23	1	1	0	1
24	1	1	0	1
25	1	1	1	0
26	1	1	1	0
27	1	1	0	1
28	1	1	0	1
29	1	1	0	1
30	1	1	1	0
31	1	1	0	1
32	1	1	0	1
33	1	1	0	1
34	1	1	0	1
35	1	1	1	0
36	1	1	0	1
37	1	1	1	0
38	1	1	1	0
39	1	1	1	0
40	1	1	1	0

dX	dA	dB
0	0	0
0	0	0
3	9	0
0	-1	-1
3	8	0
-1	-2	-2
0	-1	-1
0	-1	-1
3	8	0
2	7	0
-1	-3	-3
2	6	0
-1	-3	-3
-1	-2	-2
-1	-2	-2
2	6	0
2	6	0
2	5	0
1	4	0
-1	-4	-4
1	4	0
-1	-4	-4
1	4	0
1	4	0
-1	-4	-4
-1	-3	-3
1	4	0
1	4	0
1	3	0
-1	-4	-4
1	3	0
1	3	0
1	2	0
1	2	0
-1	-4	-4
1	2	0
-1	-4	-4
-1	-3	-3
-1	-2	-2
-1	-2	-2

vX	vA	vB	vT
0	0	0	0
0	0	0	0
3	9	0	0
3	8	-1	12
5	16	-1	11
5	14	-3	20
4	13	-4	16
4	12	-5	13
6	19	-5	15
9	26	-5	26
8	23	-8	29
10	29	-8	31
9	27	-11	31
8	24	-13	25
8	23	-15	19
10	29	-15	30
11	34	-15	39
13	39	-15	46
15	44	-15	52
13	40	-19	43
15	44	-19	53
13	40	-22	40
15	45	-22	54
16	48	-22	59
15	44	-26	42
14	41	-29	33
15	45	-29	55
16	49	-29	61
17	52	-29	65
16	48	-33	40
17	52	-33	65
18	54	-33	69
19	57	-33	73
20	59	-33	76
18	55	-37	46
19	57	-37	73
18	54	-40	40
17	51	-43	31
16	49	-45	25
16	47	-47	20

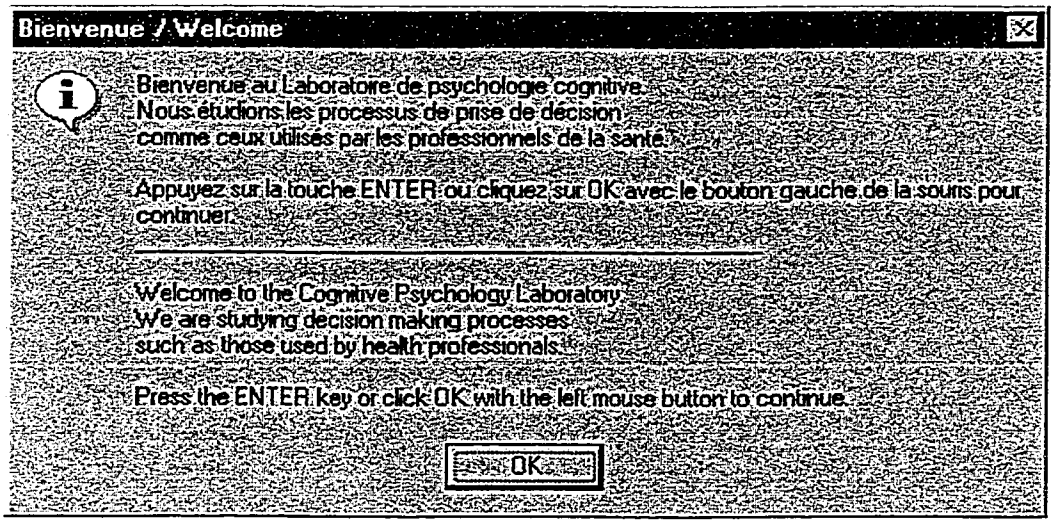
crXA	crXB	crXAB
0	0	0
0	0	0
12	3	12
11	2	09
21	4	20
19	2	16
17	0	13
15	-2	10
26	1	20
35	3	29
31	0	23
39	2	31
36	-2	25
33	-5	19
30	-7	15
39	-5	24
46	-3	31
52	-2	38
58	0	43
53	-6	34
59	-4	40
54	-9	31
59	-8	37
64	-6	42
59	-11	33
55	-15	26
61	-14	32
65	-13	36
70	-12	40
65	-17	32
69	-16	36
73	-15	40
76	-14	43
79	-13	46
73	-19	36
76	-18	40
72	-23	31
68	-26	25
65	-29	20
63	-32	15

The table on the preceding page is a sample of the trial-by-trial changes in associative strength derived from the Rescorla-Wagner model for an A+, AB- discrimination, for one run of forty randomised trials. The salience parameter α was set at .3 for predictors A and B, while α for the contextual cue X was .1. The salience of the outcome (β) was .3 both when it was present and when it was absent, while λ was 100 in the presence of the outcome and 0 in the absence of the outcome.

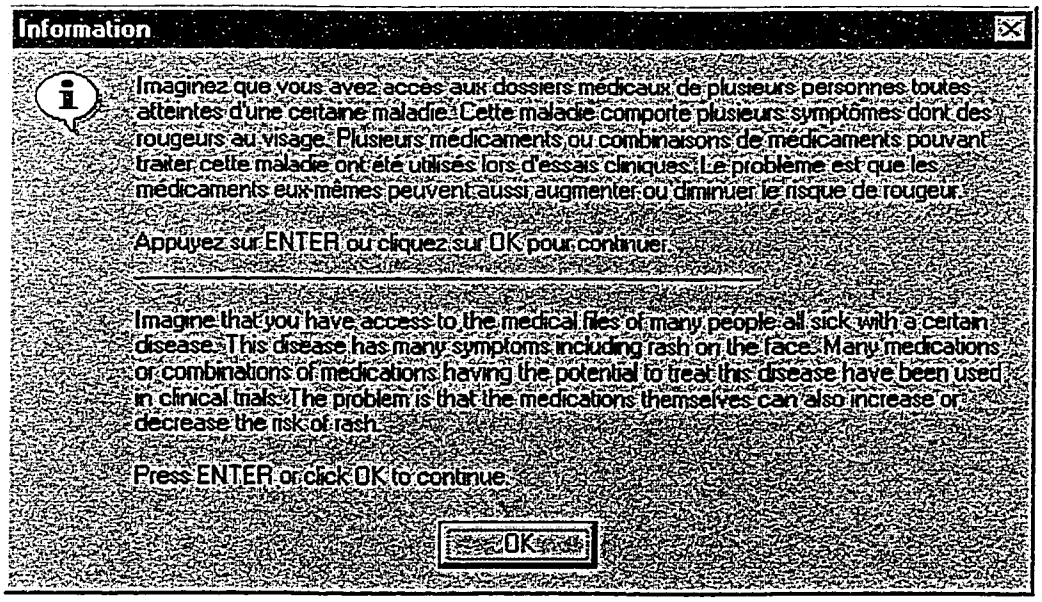
The columns labelled X, A, B, and O code for the presence or absence of the respective cue. For example, on trial 1 the contextual cue, A, and B were presented, and were not followed by the outcome. Columns dX, dA, and dB represent the change in associative strength to the respective cues following conditioning on that trial. These values are determined on a trial-by-trial basis through an application of Equation 1. For example, on trial 10 the associative strength of X is incremented by 2, while the associative strength of A is incremented by 7. The larger increment to A reflects its higher salience relative to X. The cumulative change in associative strength after every trial is given by Equation 3, and is displayed for each predictor in columns vX, vA, and vB. Column vT (representing the value V_T^n in Equations 1 and 2) cumulates the total associative strength after every trial (Equation 2), and is used in Equation 1 to determine the change of associative strength to each predictor present on that trial. The last three columns display the net associative strength that accrues to the compounds XA, XB, and XAB after every trial. For Example, as the Rescorla-Wagner model presumes that $V_{XAB} = V_X + V_A + V_B$, the net associative strength accrued to compound XAB after 10 trials is 29.

APPENDIX B

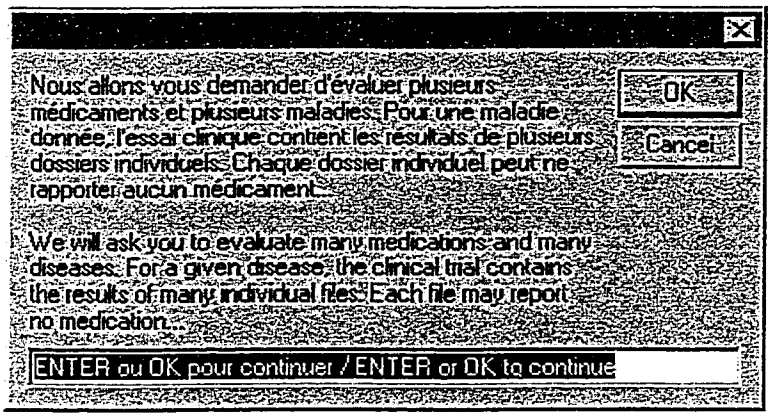
Screen 1:



Screen 2:

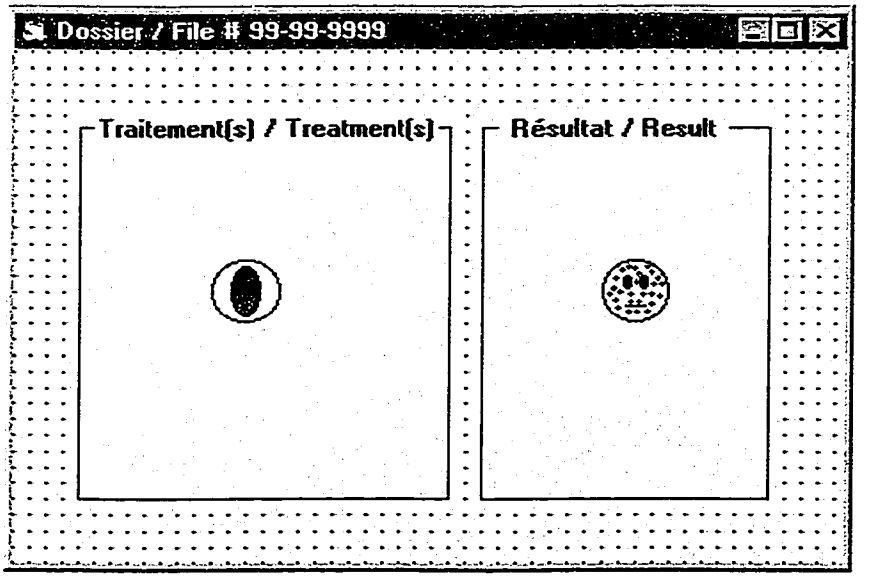


Screen 3:

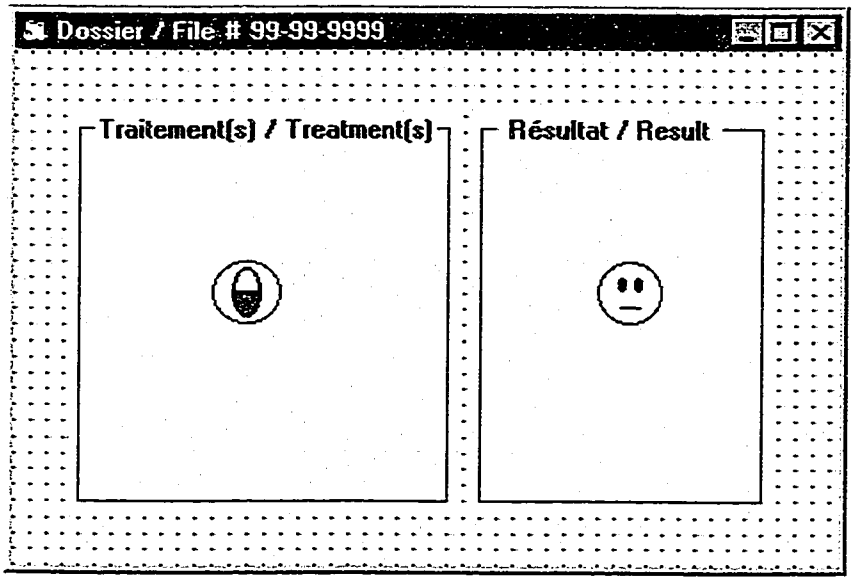


Following the first three screens, participants saw the graphical components that represented patient files, possible medications and possible outcomes. Examples of only two different trial types, of the high similarity condition, are depicted below. Screen 4 depicts the file of a patient who received the half red/half green pill, and who developed a facial rash. Screen 5 depicts the file of a patient who received the half green/half white pill, and who did not develop a facial rash.

Screen 4:

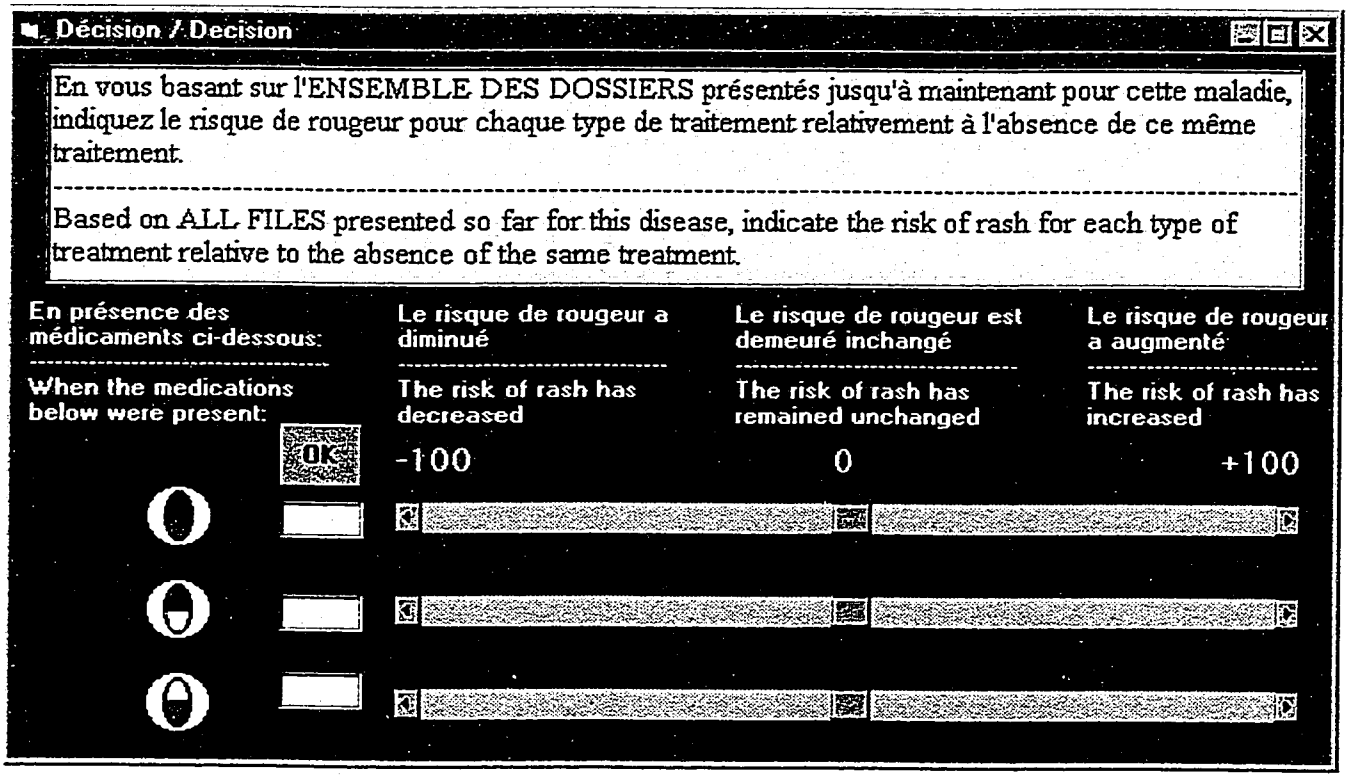


Screen 5:



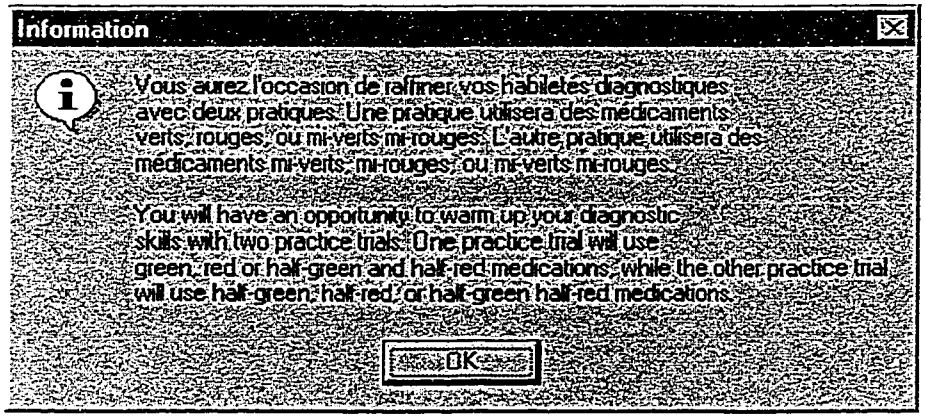
Screen 6 depicts the response screen that participants saw after every four trials in the high similarity condition of Experiment 1.

Screen 6:



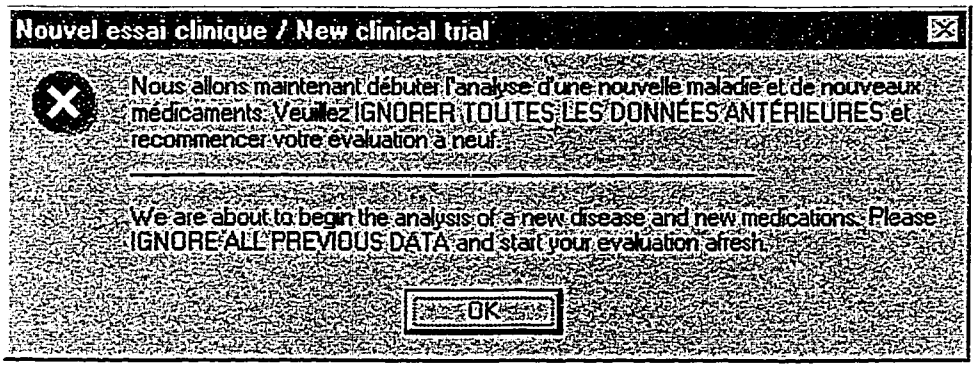
Before the practice trials, Screen 7 was presented to the participants.

Screen 7:



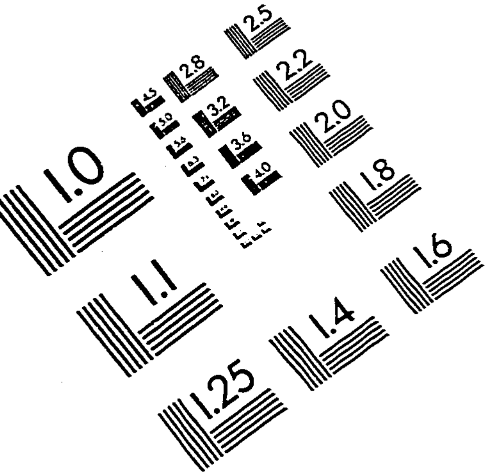
Screen 8 was presented before the first experimental condition, and between each successive experimental condition.

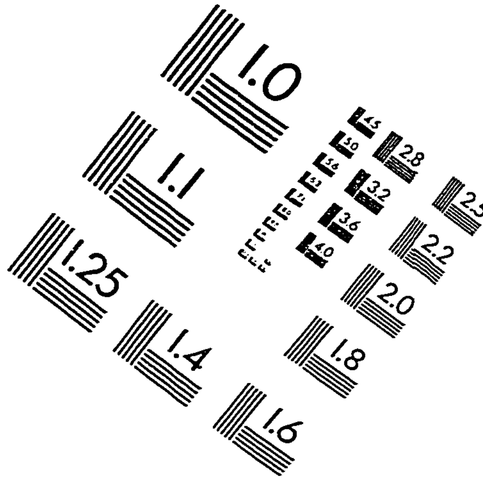
Screen 8:

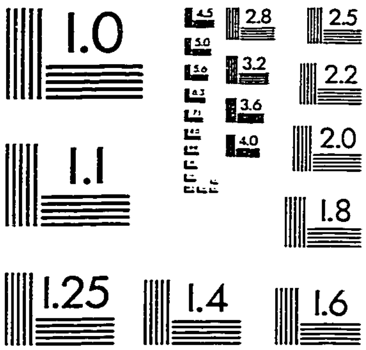


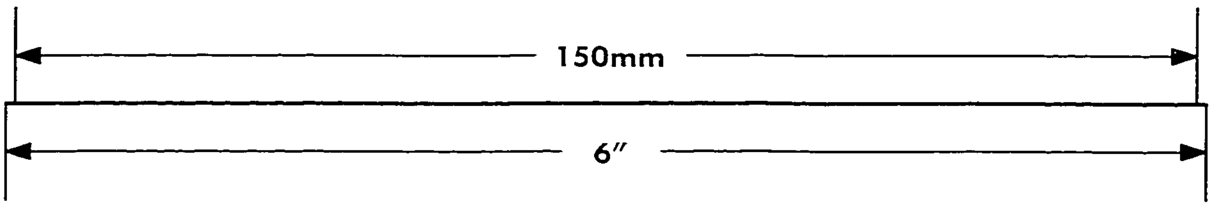
The instructions and graphical components were modified for each experiment to reflect the different number and types of possible treatments, as well as the different number of practice clinical trials.

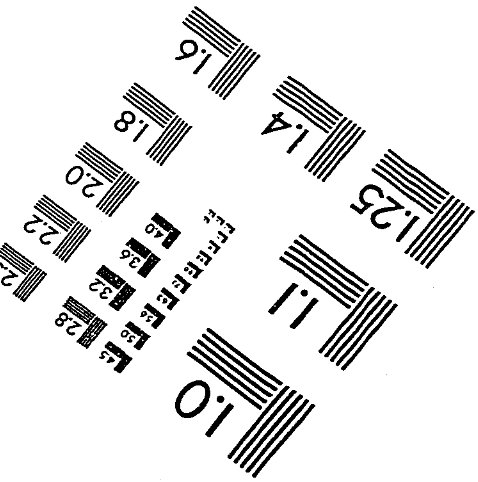
IMAGE EVALUATION TEST TARGET (QA-3)











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