Learning about predictive contingent relationships allows a window into the underlying causal structure of the environment. The alternative might be to work out the true physical mechanism of any caused event. For example, that flowers on fruit trees precede the arrival of fruit can be understood on the basis of a complete understanding of the ontogenesis of fruit, but even this knowledge is based on a chain of predictive contingencies. The epistemological nature of causal knowledge has been questioned for centuries (see also chap. 2, this volume). The idea that causal knowledge might be founded on an understanding of correlation is attributed to Hume (1739/1960). He suggested that detecting a positive contingency between flowers and fruit is enough information to infer a causal connection or, minimally, to allow us to act as if there was an understanding of the causal relation. Unlike working out the complete physical model, learning a contingency simply requires remembering the likelihood of fruit following flowers and comparing this memory with one of the likelihood of fruit in the absence of flowers.

A great deal of research first with laboratory animals (Rescorla, 1968) and then with humans (for reviews, see Allan, 1993; Baker, Murphy, & Vallée-Tourangeau, 1996; Shanks, 1993) has been devoted to showing sensitivity to contingent relationships. Psychological theories have been applied to the data to try and explain the processes that allow this learning to take place. We would argue that the most successful to date have been those based on the formation of associations (Baker, Murphy, Vallée-Tourangeau, & Mehta, 2001).

Doubts have been raised as to the adequacy of these accounts (e.g., Buhener & May, 2003; Cheng, 1997; De Houwer, chap. 3, this volume;
Melz, Cheng, Holyoak, & Waldmann, 1993). Not surprisingly, some of the evidence that has been recruited to undermine the associative account is from experiments showing that judgments sometimes deviate from the predictions of associative models. We discuss how this problem may be a general issue related to the fact that the associative models that have been recruited are sometimes used in a manner that omits important temporal parameters that may influence judgments. This problem is also relevant for theories that use normative statistical measures of contingency. In both cases the models assume that causes and events are binary and that some of the temporal variables that can be used to relate events are irrelevant.

We first discuss experimental evidence examining how people respond to contingent relationships and illustrate how associative models were crucial in distinguishing between contingency learning and learning based on simpler heuristics. We then discuss some experimental evidence that illustrates how varying some of the temporal variables in contingency tasks can influence learning. These results suggest that we need to modify our models. But generally they also suggest that both contingency-based and associative accounts still have much to offer (e.g., Allan, 2003).

ASSOCIATIVE THEORY AND CONTINGENCY

Associative theories generally have a single common mechanism to account for learning. They rely on the close pairing of two events in space and time. This experience leads to an internal connection between events. The strength of the connection is determined in part by how often the events are paired, how often either one occurs alone, and how often both are absent. Research into human learning of event–outcome contingency relationships has attempted to relate the underlying learning processes to associative models originally developed to account for animal learning phenomenon (e.g., Baker, Berbrier, & Vallée-Tourangeau, 1989; Dickinson, Shanks, & Evenden, 1984; Shanks, 1987). For example, Pavlovian learning has been carefully analyzed from an associative framework. Learning that a particular conditioned stimulus (CS) occurs immediately prior to an unconditioned stimulus (US) is helped if the CS and US occur together with greater regularity than they occur apart. One interpretation of these events is that an association between the CS and US is formed. The strength of this association provides an accurate representation of the predictive relatedness of the two events. Some human learning involves conditioning with the same sorts of biologically significant events and neutral sensory stimuli that have been shown with animals, but human learning also involves linguistically based stimuli such as words. With the full range of sensory input, associative models have been quite successful. In the causal learning literature, where differences have been found between judgments of relatedness
and the experimenter's programmed relationship, some researchers have proposed that, in addition to associative learning, causal schemas influence how judgments are made (e.g., Cheng, 1997) or that inherent biases or predispositions influence stimulus processing (Alloy & Abramson, 1979; Wasserman, Elek, Chatlosh, & Baker, 1993).

**SIGNAL-OUTCOME PAIRINGS VERSUS CONTINGENCY**

Early theories of learning relied on the pairing between two events to determine the strength of the association between them. For instance, Hull's (1943) theory for Pavlovian conditioning assumed that conditioned behavior resulted from pairing an outcome with a signal. A neutral CS (e.g., a 10-second light presentation) could be paired with a US (e.g., food). Conditioning was successful if experimental animals acquired a conditioned response (e.g., food searching) when the CS was presented. Associative accounts of this learning relied on the number of spatially and temporally contiguous pairings between the two events to explain the strength of the association and thus the level of responding. If the CS was presented without the US, behavior would diminish, presumably reflecting the weakened association between the two events. In terms of heuristics conditioning was thought to be determined by the likelihood of the US in the presence of the CS, P(US|CS). This was shown to be inadequate by experiments demonstrating that it was the CS's overall contingency that mattered.

Experiments by Rescorla (e.g., 1968) were the first to unambiguously demonstrate that the likelihood of the outcome in the absence of the CS [P(US|¬CS)] also contributed to the strength of conditioned responding. In turn this result led to the development of an explanation of contingency sensitivity using associative principles. To accommodate this result, Rescorla and Wagner (1972) suggested that learning about a single CS actually involves forming two associations: (a) an association between the CS and the US (as before), but also, (b) an association involving the context, in which the CS occurred, and the US. Moreover, these two associations are involved in a competition with one another for association with the US. This idea allows associative models to be sensitive to the overall contingency between the CS and the US. If the likelihood of the US is greater in the presence of the CS [i.e., P(US|CS) > P(US|¬CS)], then the CS–US association will be stronger than the context–US association. Whereas, if the likelihood of the US in the absence of the CS is greater, then the context–US association will be stronger (for a more detailed review of associative accounts of contingency learning, see Baker et al., 2001).

However, it is important to remember throughout this somewhat superficial discussion that some of the temporal characteristics related to the CS and US are lost in this analysis. For example, CSs, contexts, and USs are temporally extended and importantly the temporal characteristics of these events and their contiguity influence the
course of learning. For example, whether the CS and the US are presented simultaneously or sequentially influences the strength of learning. Even if, as is usual, the CS and the US are presented sequentially, the degree to which they overlap also influences the extent of learning. As a general rule, the closer the two events are presented in time the stronger the learning. But there are important qualifications to be made as a function of the organism and the type of conditioning. For example, rabbit eyeblink conditioning is most effective with a relatively brief 500-millisecond CS–US onset interval (the time between the onset of the CS and the onset of the US). For pigeon key-pecking, the optimal period is of the order of seconds. For rats learning a fear response, the optimal CS–US interval is longer still. Finally, food aversions with rats can develop even with CS–US intervals several hours in length (Mackintosh, 1983). This simply demonstrates that both between species and even within species these temporal parameters influence learning beyond the basic notion of temporal contiguity. One could argue that the animals have different causal models that guide their learning. In some sense this might be true. We still find it productive to argue that contingency remains the primary driver of learning, but there are differences between CSs and USs that influence how they will be learned. One of the reasons for this confidence is that in spite of the differences in optimal period, in all cases learning is dramatically interfered with if another stimulus occurs in the period between CS onset and US onset.

Research into animal conditioning has proceeded to develop real-time models of association formation (e.g., Schmajuk, 1997; Wagner & Brandon, 2001), precisely to deal with some of these issues. However, no such development has occurred in the field of human contingency learning (although see chaps. 7 and 8, this volume). It is possible of course that these variables are irrelevant for human learning. However, we already know that contingency learning in humans is sensitive to the delay between signal and outcome (Dickinson & Shanks, 1995). One of the goals of this chapter is to look at the influence of other temporal variables.

Because of the conceptual similarity between an animal acquiring a conditioned response on the basis of CS–US contingency and a human reasoner gauging the relationship between a cue and an outcome, the Rescorla–Wagner (1972) model of associative learning has been applied to human contingency learning. Similar to early animal associationists, researchers studying human contingency learning suggested that participants were simply sensitive to either the number of times that the signal and the outcome co-occurred or the rate at which the outcome occurred in the presence of the signal (Smedslund, 1963; Ward & Jenkins, 1965). We discuss one such study and illustrate using data from our lab how this original theoretical bias led to experiments that failed to test whether humans were sensitive to contingencies.
Researchers have devised a number of procedures for assessing human contingency learning. They can be categorized as involving either actively learning a response-outcome relationship or passively learning a signal-outcome relationship. In the prototypical case of the latter type, participants are required to learn a relationship between one signal and an outcome. The task for the participant is to report whether the signal is a reliable predictor of the outcome. Because the signal and outcome in these tasks are binary, there are four types of events that can occur: The signal and outcome occur together, either the signal or the outcome occur in the absence of the other, or neither event occurs. The four trial types can be represented in a contingency table (see Fig. 10.1). The underlying logic has been to present a sequence of trials and then ask participants to rate the strength of the relationship between the signal and the outcome. You might then be able to learn how accurate people were and the cognitive strategy they use by comparing judgments with various methods of combining the four cells of the table (e.g., Arkes & Harkness, 1983; Shaklee & Tucker, 1980). Judgment accuracy is defined by how the participants are able to integrate information consistent with a positive relationship, a negative relationship, or no relationship between the two events.

\[
\text{Delta } P = \frac{A}{(A+B)} - \frac{C}{(C+D)} = P(O|S) - P(O|-S)
\]

**FIG. 10.1.** 2 × 2 contingency table.
The accepted normative measure of the contingency between events in such a table is called Delta P (ΔP). ΔP is the difference between the conditional probability of an outcome (O) in the presence of the predictor signal (S), or P(O|S) and the probability of the outcome in the absence of the signal, P(O|−S) (Allan, 1980). A positive value of this difference indicates that the outcome occurs more frequently following the signal than in its absence, whereas a negative value indicates the converse. Positive values suggest a facilitatory causal relationship and negative ones a preventive or inhibitory cause. When the two probabilities are equal, no contingency exists between the two events.

To calculate the contingency, and the conditional probabilities, the frequencies of four different types of evidence or event conjunctions must be known. The probability of the outcome in the presence of the signal, P(O|S), requires knowing how often the two events have occurred together (Cell A) and how often the signal has occurred in the absence of the outcome (Cell B). The proportion of A cells relative to the sum of A and B cells provides the likelihood of the outcome in the presence of the signal. The second conditional probability, P(O|−S), requires knowing how often the outcome has occurred during the absence of the signal (Cell C) and how often the absence of the outcome has occurred in the absence of the signal (Cell D).

However, notice that this analysis ignores the temporal duration of the predictive signal, the duration of the outcome, the actual signal–outcome onset interval. It can code the length of time between signals by increasing the number of Cell D occurrences, but even this has its problems.

Logically each of these conjunctions of events is equally informative about the overall relationship, but Cell D poses problems for both the experimenter and the participant. The absence of both events provides positive information about a contingency but is difficult to define experimentally and indeed epistemologically (Nisbett & Ross, 1980). Given a finite period in which nothing has happened how can one determine how many conjunctions of no-signal and no-outcome have occurred? For example, because increasing frequencies of Cell D increase ΔP, if participants overestimate the number of times that neither event has occurred then their estimates should exhibit a positive bias.

The following sections illustrate how the notion of contingency has been useful for our understanding of human learning in these contingency paradigms. We then show how two aspects of the temporal relationship, which are difficult to define, can influence learning. This in itself is not novel (see also Shanks & Dickinson, 1987); however, we discuss how a failure to recognize this issue has contributed to two possibly erroneous conclusions in the contingency learning literature. The first is that judgment of instrumental contingencies is more influenced by the presence of the outcome than by its absence. The second is that people who exhibit mild symptoms of depression might be better judges of contingency than those without these symptoms.
ASSESSING CONTINGENCY LEARNING

Initial studies of human contingency learning implied that the pairing between signal and outcome exerted an inordinate influence on judgments relative to the other events of the contingency table. A number of studies in different areas of psychology showed that in the absence of an overall positive signal-outcome contingency [i.e., \( P(O|S) = P(O|\sim S) \)], participants often judged there to be a positive relationship when there was a high frequency of event pairings. This "illusory correlation" phenomenon has been observed in simulated medical diagnosis tasks (e.g., Smedslund, 1963), in the interpretation of psychodiagnostic tests (e.g., Chapman & Chapman, 1969), and in stereotype formation (Hamilton & Gifford, 1976). A similar finding has been reported in animal conditioning (Quinsey, 1971).

In each of these cases there are two potential problems with interpretation of participants' positive judgments of a putative zero contingency. The first involves assumptions about how participants use the measurement scale. The second involves assumptions about the perceived frequency of events. Participants' judgments might not transparently map onto the measurement scale imposed on them by researchers. If participants are trained with a noncontingent relationship and asked to make a judgment of the relationship on a scale of \(-100\) to \(+100\), a rating of \(0\) is generally the expected outcome. However, this assumes that participants align their subjective experience of a noncontingent relationship with the value \(0\) on the rating scale. Most studies have implicitly assumed that a positive judgment implies that the subjects are above psychological zero. A fairer test requires comparing these judgments with judgments from a range of other contingencies. The minimal evidence for sensitivity to the noncontingent relationship should be that participants judge all noncontingent relationships similarly, but different from positive and negative contingencies. Therefore, there should be a direct relationship between changes in contingency and changes in judgments, but not necessarily a direct mapping between the experimenter's programmed contingency and the dependent variable.

The second, somewhat related, problem is whether the contingency presented is really noncontingent. The assumption is that the experimenter is in the privileged position of being able to objectively measure and present the appropriate events that define a zero contingency. However, the experimenter must be able to assume that the four cells of the contingency table are equally under their control. Certainly a case can be made for the number of times two things are paired together (Cell A), or either event occurs alone (Cell C and Cell B), but even these events are crucially determined by the level of contiguity between the signal and the outcome. For instance, do the two events need to occur simultaneously or, as is usual, does the signal occur before the outcome and, if so, how long before? Is it possible that people will mistakenly consider some conjunctions to be individual pairings and hence overestimate
Cells B and C at the expense of Cell A? Or might they think some disjoint presentations to be conjunctions with the opposite effect? In addition Cell D, the absence of both events, is even more difficult to control (Shanks & Dickinson, 1987).

A participant's perception of the contingency between events can depart significantly from the experimenter's intended contingency if participants estimate the frequency of the nonoccurrence of both events in a manner different than programmed by the experimenter. For example, if they overestimate the frequency of Cell D this will reduce \( P(O|\neg S) \) and thus generate a positive bias. Thus, from the perspective of the experimenter two events may occur independently of each other. The experimenter has defined a task as noncontingent, but this experimenter contingency is based on assumptions about how the participant should partition time, and also assumes that the subject should use only a certain type of evidence for their decision.

For example, if participants overestimate the frequency of Cell D observations, then they may form the impression of a positive contingency. The participants' perceived contingency may be positive whereas the experimenter's is zero. Neither estimate is more "correct" than the other in any true sense, and therefore experimenters must be careful how they interpret differences between expected judgments and actual judgments. Evidence that judgments were positive following zero contingency training might say very little about learning noncontingent relationships. Importantly, this could indicate a failure of the experimenter to control the contingency rather than a failure of the participant to respond appropriately.

The first problem described relates to whether it is reasonable to expect that participants' judgments should map transparently onto the experimenter's scale. Therefore, does the possibility that participants judge a zero contingency positively on the experimenter's scale indicate that they have misinterpreted the evidence? Early work studying contingency learning concluded that they had. Smedslund (1963) claimed to show that participants were excessively reliant on Cell A events to inform their judgments, suggesting that they were not able to use all four types of events, and therefore were not naturally sensitive to contingencies. He suggested that his findings reflected a tendency to rate relationships as positively contingent based on the frequency of signal–outcome co-occurrences alone. We work through how contingency theory usefully helped develop our understanding of human learning before illustrating how its misinterpretation may have misled researchers.

In Smedslund's (1963) study, student nurses were presented with a deck of 100 cards; each card represented a patient (and one instance of a cell in the contingency matrix) and indicated the presence or absence of a symptom and the presence or absence of a disease. Smedslund designed the deck such that (a) 70% of the cards (the patients) showed the disease and (b) that the symptom was a poor predictor of the disease because the probability of the disease in the presence and in the absence of the symp-
tom was 0.69 and 0.72, respectively (see Table 10.1). Thus the disease was approximately as likely to occur in the presence as in the absence of the symptom. Smedslund reported that half of the student nurses “said there was a relationship [between the symptom and the disease] because the number of symptom–disease cards was the largest or was large” (p. 171). This was indeed the most frequent patient type (see the second column of Table 10.1).

On the basis of this finding, Smedslund concluded that humans were very poor reasoners about correlations, and that what little sense could be made of such information was driven entirely by consideration of the symptom–disease frequencies, a conclusion granted textbook legitimacy even now (e.g., Baron, 2000; Sutherland, 1992). However, Smedslund did not attempt to dissociate the impact of the disease base rate from the actual symptom–disease correlation. The idea that participants might judge the presented relationship as less predictive than one actually containing a positive contingency was not tested, and therefore requires that the participants’ perceptions map directly onto the measurement scale. Subsequent work in a number of laboratories using different procedures was able to show that participants could discriminate positive, zero contingencies, and negative contingencies, irrespective of the number of signal–outcome pairings (e.g., Dickinson et al., 1984; Wasserman et al., 1993).

Using Smedslund’s symptom–disease scenario, Vallée-Tourangeau, Hollingsworth, and Murphy (1998) presented participants with five different symptom–disease relationships (see Table 10.1). In two of

<table>
<thead>
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<th>Table 10.1</th>
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<tr>
<td>Frequency of the Four Trial Types in Smedslund (1963, Experiment 2) and in the Five Conditions of Vallée-Tourangeau, Hollingsworth, and Murphy (1998, Experiment 1)</td>
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<table>
<thead>
<tr>
<th></th>
<th>Smedslund</th>
<th>Vallée-Tourangeau, Hollingsworth, &amp; Murphy</th>
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<tbody>
<tr>
<td></td>
<td>Zero</td>
<td>Neg</td>
</tr>
<tr>
<td>Symptom-Disease</td>
<td>37</td>
<td>9</td>
</tr>
<tr>
<td>Symptom-No Disease</td>
<td>17</td>
<td>21</td>
</tr>
<tr>
<td>No Symptom-Disease</td>
<td>33</td>
<td>21</td>
</tr>
<tr>
<td>No Symptom-No Disease</td>
<td>13</td>
<td>21</td>
</tr>
<tr>
<td>P(Disease)</td>
<td>0.70</td>
<td>0.50</td>
</tr>
<tr>
<td>P(Disease</td>
<td>Symptom)</td>
<td>0.69</td>
</tr>
<tr>
<td>P(Disease</td>
<td>No Symptom)</td>
<td>0.72</td>
</tr>
<tr>
<td>ΔP</td>
<td>-0.03</td>
<td>-0.40</td>
</tr>
</tbody>
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these, the symptom-disease contingency was nonzero, either negative 0.4 or positive 0.4, whereas in the remaining three it was zero; that is, the disease was as likely to be observed in patients with the symptom as in those without. These three zero-contingency conditions differed in the disease base rate, either 30%, 50%, or 70%. Therefore, the disease occurred relatively more frequently for patients with and without the symptoms as the percentage increased.

The mean judgments in all five conditions are plotted in Fig. 10.2. Participants did indeed rate zero-contingency relationships as slightly positive and, as the disease base rate increased, they were more likely to rate the symptom-disease relationship positively. This is consistent with Smedslund's early work. However, high or low disease base rates did not irremediably blunt the participants' judgment accuracy. Despite the fact that the high-density zero contingency contained a higher disease base rate than the positive correlation condition (0.7 vs. 0.5), the mean judgment was significantly higher in the positive condition than in the high-base rate zero condition. Similarly, although the disease base rate was lower in the low base rate zero correlation condition than in the negative correlation condition (0.3 vs. 0.5), the mean judgment in the negative condition was significantly lower than in the low-density zero condition.

![Diagram](image)

**FIG. 10.2.** Symptom-disease correlation judgments in the five conditions of Vallée-Tourangeau, Hollingsworth, and Murphy (1998, Experiment 1). The disease base rate was 0.5 in both the negative- and positive-correlation conditions. The disease base rate in the three zero-correlation conditions was 0.30, 0.50, and 0.70, respectively.
Thus, unlike Smedslund's original experiment, our experiment enabled us to clearly document people's ability to discriminate zero from nonzero symptom-disease contingencies. The disease base rate did have a significant impact on judgments as revealed by the order of the mean judgments across the three zero-correlation conditions. It is this finding that lends credence to our thesis that Smedslund's results were driven by the very high disease base rate used in the zero symptom-disease correlation presented to his group of student nurses. The fact that in our study the three zero contingencies were judged differently suggests that contingency is not all that is being judged.

In summary, people are able to discriminate different levels of contingency, but judgments of zero contingencies sometimes seem to be different from the objective contingency programmed by the experimenter. There are a number of possible explanations for this departure. It may reflect a bias in processing the four different types of trial that provide evidence for the contingency (e.g., Baron, 2000). Alternatively, it is also possible that causal inferences are influenced by more than Ap. The outcome base rate is an integral part of this inferential process (see, Cheng's power probabilistic contrast theory, 1997). We now examine some of the data on base rate effects and attempt to show that some of these effects might be better explained with reference to a more precise mapping of experience onto the events of the contingency table.

**BASE RATE DEPARTURES FROM CONTINGENCY**

The base rate effect in contingency learning is the finding that judgments of similar contingencies seem to show influences beyond the contingency. To make matters even more confusing, there are two base rate effects reported in the literature that seem to relate to the type of task used by the experimenter. Passive tasks, like those used in Smedslund (1963) in which participants simply view a series of events, seem to result in judgments that increase with increasing outcome density, whereas tasks, in which a participant's instrumental behavior or response is the signal for the possible outcome, seem to result in weaker discrimination of different contingencies with increasing outcome frequency (Wasserman et al., 1993).

In one study we demonstrated the impact of the effect base rate within different correlation conditions (Vallée-Tourangeau, Murphy, Drew, & Baker, 1998). Using a virus-disease scenario, different contingencies were instantiated over a series of discrete trials (or "patients") much like Smedslund's task. We recorded judgments in two contingency conditions: zero and positive. At each level of contingency, the disease (outcome) base rate could be low at 0.25 (i.e., the overall probability of the disease was 25%), medium at 0.50, or high at 0.75. Terminal mean judgments in these six treatments are shown in the left panel of Fig. 10.3. The influence of the actual virus-disease contingency and the
disease base rates are clearly apparent. Thus, participants' judgments in the positive conditions were significantly more positive than judgments in the zero conditions. But, within each of the three levels of virus-disease contingency, the disease base rate significantly influenced judgments: Thus, the more frequent the overall likelihood of the disease the more positive the attribution of causal importance to the virus.

The influence of the effect base rate on judgments is a robust phenomenon. Earlier descriptions are found in the work of Alloy and Abramson (1979, with nondepressed participants), Allan and Jenkins (1980), Dickinson et al. (1983), Shanks (1985, 1987), and Baker et al. (1989). Commentators from opposite ends of the theoretical spectrum have attempted to explain the finding that the same contingency defined with different base rates often elicits different ratings. Some have suggested that it is a reflection of incomplete learning; that, is the different judgments reflect the different experience with the outcome but that with more experience judgments would eventually become more similar. Alternatively, Shanks has suggested that it may reflect a within-participants training artefact (Shanks, Lopez, Darby, & Dickinson, 1996). However, we have looked for this difference in our data and found no consistent evidence for this effect. Similarly, Shanks (1985) elegantly demonstrated that the effect base rate significantly influenced judgments in a between-subjects design, hence the cross-condition interference hypothesis offers little explanatory value.
Cheng (1997) has argued that the base-rate effect in zero-contingency conditions reflects either incomplete learning (the judgments are preasymptotic) or interference from nonzero correlation conditions in within-subjects design. However, in positive contingencies, Cheng’s power PC theory describes an extra causal learning process that attributes increased causal efficacy to signals that are related to higher outcome densities (see chap. 2, this volume, for a fuller discussion). A further feature of these density effects is that they are seemingly dependent on some unspecified aspect of the task. For instance, the outcome effect described by Vallée-Tourangeau, Murphy, Drew, and Baker (1998) is typical of other findings; however, using a quite different preparation without discrete trials, Wasserman and his colleagues (1993) have shown a completely opposite density effect.

THE INSTRUMENTAL LEARNING DENSITY EFFECTS

In an instrumental learning preparation, participants are asked to judge the contingency between an action they perform and a subsequent outcome. In the laboratory, that action might be pressing a key on a computer keyboard, and the outcome a brief presentation of a geometric figure on the monitor. Though many researchers had studied this type of learning using discrete trials in which the opportunity to respond was signaled, Wasserman used a free-operant procedure in which participants responded as often as they liked during a specified training session (Wasserman, Chatlosh, & Neunaber, 1983; Wasserman et al., 1993). This procedure was adapted from a standard contingency learning procedure used in instrumental conditioning with animals described by Hammond (1980). Hammond noticed the problem involved in programming a contingency for experimental subjects. He chose one method although it is not the only solution. By systematically mapping contingency judgments as a function of a wide range of experimenter programmed action-outcome contingencies, Wasserman et al. (1993) have reported an impressive degree of accuracy. Not only were the contingency judgments closely aligned with the actual degree of action-outcome contingency but they were also largely unaffected by the base rate of the outcome (Bennis & Mahler, 1985; Wasserman et al., 1983). The latter finding contrasts rather sharply with the robust impact of the effect base rate in noninstrumental contingency judgment tasks such as ours.

Wasserman et al. (1993) used the Rescorla–Wagner model to explain their results. They showed an impressive correspondence between judgments and predictions of the Rescorla–Wagner model by assuming that the parameter that codes learning on outcome trials was higher than the parameter coding learning on no-outcome trials. However, this is not the only set of parametric assumptions that produces this pattern. In particular, if one assumes that there is a bias in the interpretation of
the four cells of the contingency table in Wasserman's procedures, then this pattern of results can also be reproduced. There is good reason for positing that this might happen.

A particular feature of the Hammond schedule used by Wasserman is that, although the participant is unaware of the segmentation of time, the experimental period is segmented into 1-second bins. The conditional probabilities for the occurrence of the outcome in the presence and the absence of a response are mapped onto the bins. If the participant performs at least one response during any 1-second bin, then it is deemed a response and an outcome occurs at the end of the bin according to the probability of an outcome given a response, $P(O|R)$. If no response is made during the 1-second bin, then the outcome occurs with the probability $P(O|\neg R)$. One consequence of this procedure is that on average there is a .5-second delay between a response and an outcome. If the participant happens to respond at the start of a bin, then the outcome is delayed by 1 second; similarly, if the participant happens to respond near the end of the bin, then the delay is close to 0 seconds. This has three possible effects on the perception of contingency. The first is that responses that actually generate an outcome might be categorized as having not been paired with the outcome, especially if there is a 1-second delay. This is akin to reducing the impact of Cell A events. There is good evidence that even a relatively short delay between a signal and an outcome can seriously attenuate the perception of contingency (Dickinson & Shanks, 1995). In addition, outcomes that are nominally produced by responding might be categorized as not being contingent on responding. This is like increasing the number of Cell C events. Finally, because participants can respond as often as they like, they may respond more than once during a given 1-second bin, but still receive only one outcome presentation. This has the effect of increasing the number of Cell B events, in which a response is presented and no outcome occurs (Shanks & Dickinson, 1987). The overall effect of these factors is that the actual perceived contingency might be very difficult to establish. To the extent that judgments map onto estimates of contingency, the experimenter can be hopeful that this mapping is meaningful. However, any attempt to interpret departures from the estimates of contingency by resorting to auxiliary assumptions may be mistaken because the perceived contingencies might be different than the programmed one. We sought to explore whether the differences in density effects may reflect the attenuated contiguity in the Hammond schedule.

**TEST OF THE CONTIGUITY HYPOTHESIS OF DENSITY EFFECTS**

To test whether the attenuated contiguity present in Wasserman et al. (1993) might be responsible for the pattern of density effects observed in that study, we report here some unpublished work in which we exam-
ined contingency judgments with an instrumental learning methodology in six conditions analogous to the ones run in Vallée‐Tourangeau, Murphy, Drew, and Baker (1998). These conditions reflected the factorial combination of two levels of action–outcome contingency (zero, $\Delta P = 0$; and positive, $\Delta P = .5$) with three levels of outcome density (low at .25, medium at .5, high at .75). In the task, participants were asked to assess the causal importance of pressing the spacebar in producing the appearance of a geometrical figure on the computer screen. They could press the bar as many or as few times as they wished. As in any instrumental learning preparation, the experimenter did not control precisely the frequency and timing of delivery of the reinforcer (the outcome); rather, these were determined by the participants' behavior. The outcome would appear on the computer screen with probability $P(O|R)$ given an action and with a probability $P(O|\neg R)$ given the absence of an action after a certain amount of time elapsed. The difference between these two conditional probabilities determined the level of action–outcome contingency. The programmed conditional probabilities in the six conditions are shown in Table 10.2 along with the mean actual conditional probabilities experienced by the participants. In the three zero-contingency conditions, the outcome was as likely to occur following an action as following no action; that is, $P(O|R)$ equaled $P(O|\neg R)$. However, the overall probability of the outcome occurring varied from 0.25 to 0.75. In the three positive-contingency conditions, the outcome was programmed to occur more often following an action than following no action; that is, $P(O|R)$ was larger than $P(O|\neg R)$, and as in the zero-contingency conditions, the base rate of the outcome varied from 0.25 to 0.75. This experimental design enabled us to determine the degree of discrimination between two levels of action–outcome contingency and the impact of the outcome base rate on judgments.

**TABLE 10.2**

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<th>Programmed</th>
<th>Actual</th>
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Each of the six action–outcome contingencies was instantiated over a sequence of 40 sampling intervals whose maximum duration was 1 second. During that 1-second segment, the computer program monitored whether the spacebar had been pressed. If so, then the outcome would be shown on the screen immediately after the bar press with probability $P(O|R)$ for a quarter of a second, and then a new 1-second sampling interval would be initiated. The stronger contiguity of response and outcome pairings was a departure from the Hammond schedule used by Wasserman et al. If, at the end of the 1-second segment, no bar presses had been detected, then the outcome would be presented on the computer monitor with probability $P(O|\neg R)$ and a new 1-second segment was initiated. Participants were not informed of this segmentation, nor was it marked in any manner during the task. Rather, they pressed the spacebar and stared at a blank computer screen on which a geometrical shape sometimes appeared. In contrast to the 45-minute testing sessions with the procedure of Vallée-Tourangeau, Murphy, Drew, and Baker (1998), participants were exposed to each of the six conditions for at most 40 seconds. Hence the experiment took between 5 and 10 minutes to complete, including reading the task instructions and making judgments at the end of each condition.

Participants were exposed to all six treatments. To minimize cross-treatment interference, unique geometrical figures were assigned to different treatments. The treatment orders as well as the assignment of figures to treatments were partially counterbalanced across participants. Twenty-six psychology undergraduates received course credits for their participation.

The mean contingency judgments for the six conditions are plotted in the right-hand panel of Fig. 10.3. Contingency discrimination appeared very good. Judgments in the three positive conditions were much more positive than judgments in the three zero conditions. Unexpectedly, judgments in the three positive conditions were not as positive as those recorded in the positive conditions of Vallée-Tourangeau, Murphy, Drew, and Baker (1998). The average judgment in the three positive conditions was 29.9 in this study whereas it was 56.1 in Vallée-Tourangeau et al. In turn, judgments across the three zero conditions were more negative (overall mean of $-32.9$) than the judgments in the analogous zero-contingency conditions of Vallée-Tourangeau et al. (mean of 9.1). Of primary interest is the fact that participants discriminated between the two levels of contingency.

But what of the impact of the outcome base rate on judgments? In both levels of contingency, mean judgments in the low-base rate treatments were lower than in the high-base rate treatment, which suggests that the outcome base rate did influence judgments. But importantly this pattern of results was opposite to that shown by Wasserman et al. (1993). A two-way repeated measures analysis of variance (ANOVA) on these data supported these observations (all tests for this and subsequent experiments assume a .05 alpha level). The main effect of contin-
gancy was reliable, \( F(1, 50) = 65.1 \), but neither the main effect of base rate, \( F < 1 \), nor the interaction, \( F < 1 \), were reliable.

The potentially important difference between our findings, and Wasserman et al.'s finding that increased outcome density dampened judgments, was our improved contiguity between response and outcomes. In our procedure an outcome programmed to occur after a response did so immediately following the response and the 1-second sampling interval was reset to 0. Thus the contiguity between response and outcome was consistently perfect. As described earlier, with Wasserman et al.'s (1993) the high outcome base rates, that is, when \( P(O|R) \) is greater than .5, this degraded contiguity may contribute to a degraded perception of contingency. That is, any delay between a response and an outcome may sometimes lead reasoners to attribute the occurrence of the outcome not to their behavior, but rather to the absence of a response, thereby inflating the perception of \( P(O|\sim R) \). The higher the outcome base rate the larger the subjective perception of \( P(O|\sim R) \), and the more degraded the perception of contingency (see Shanks & Dickinson, 1987).

To test this hypothesis, we conducted a second study but this time directly compared the two experimental procedures. We tested participants with both the Hammond schedule and our revised version with stronger contiguity on the two positive \( \Delta P = .75 \) contingencies from Wasserman et al. (1993) that showed the biggest effect on the Hammond schedule. The two contingencies involved either low outcome density with the following two conditional probabilities for the likelihood of the outcome \( P(O|R) = .75 \) and \( P(O|\sim R) = 0; \Delta P = .75 - 0 = .75 \) or high outcome density \( P(O|R) = 1.0 \) and \( P(O|\sim R) = 0.25; \Delta P = 1.0 - 0.25 = .75 \). In Wasserman et al. (1993) these two contingencies elicited judgments corresponding to .65 and .51 respectively. Both judgements are lower than the actual \( \Delta P \), but these judgments are both higher than any judgment of a lower contingency, and therefore are perfectly in line with the notion of sensitivity to contingency. However, the crucial concern here is the fact that the higher outcome density elicited lower judgments (.51) than did the lower outcome density (.65).

In a within-subjects design, participants rated the effectiveness of their response in producing an outcome in the two positive-contingency conditions using our perfect contiguity procedure, and using Wasserman's original procedure. The order of the two contingencies within each contiguity procedure was counterbalanced, as was the degree of contiguity. The results are displayed in Fig. 10.4 and clearly show both outcome effects. With the original variable contiguity schedule, judgments were higher with the lower density contingency and with the modified, higher contiguity schedule, judgments of the higher density were higher. The statistical analysis supports this observation. The interaction between type of schedule and contingency was reliable, \( F(1, 31) = 14.60 \). These results confirm the quite fine sensitivity of hu-
FIG. 10.4. Mean action–outcome contingency judgments in positive contingency conditions ($\Delta P = .75$) as a function of level of contiguity (perfect or variable) and two levels of the outcome base rate (.375 in condition .75:0.0, .625 in 1.0/0.25).

man judgments to variations in contiguity (see also Shanks et al., 1989). They also suggest that the interpretation posited by Wasserman et al. (1993), involving the differential effectiveness of outcome and no-outcome trials as an explanation of the density effect, is incomplete or incorrect.

In summary, previous work had shown that people are quite accurate at judging contingencies. These judgments were relatively consistent with predictions from $\Delta P$, and an associative analysis based on the Rescorla–Wagner model. However, if one assumes that the experimenter has correctly manipulated the contingent relationship, then both models fail to predict the influence of outcome density. However, we have argued that the departures from this profile might most easily be accounted for by issues related to the contingency presentation rather than contingency learning per se. We have shown how modification of the temporal contiguity between responding and the outcome seems to elicit the positive base rate effect; judgments increase with increasing base rate.

Some researchers might conclude that this analysis is appropriate only for instrumental tasks in which there is an ill-defined intertrial interval (ITI), but this is incorrect. The same analysis can be applied to pre-
dictive tasks in which discrete trials are presented to the participant. Consider a standard signal-outcome scenario used to teach a positive contingency. The participant might be presented with trials representing patients either with or without some fictitious disease and virus labels. The presence or absence of the disease and virus might be fairly easy to parse, although the signal-outcome onset period is ignored. But, how is the participant meant to estimate the number of no-virus/no-symptom instances, Cell D from the contingency table? The experimenter might hope that the participant is using only the experience of patients described in the experimental protocol, but it is not too much of a stretch to assume that people have prior experience of the absence of viruses and the absence of diseases. Any causal scenario one might imagine might be similarly influenced.

Although we have discussed how manipulating the contiguity between signal and outcome can have important influences on judgments, we now show how varying the amount of context exposure can also influence judgments. We have already suggested that problems related to the presentation of the D cell in contingency tables (the absence of the signal and the absence of the outcome) may also contribute to affect judgments. In particular, increases in Cell D should produce a consequent increase in judgments because these events confirm that neither event or outcome occur by themselves. We now discuss one experiment that investigated the influence of Cell D exposure with respect to the depressive realism effect.

TESTING THE ROLE OF THE CELL D IN DEPRESSIVE REALISM

In addition to the general interest for human cognition that contingency learning has attracted, it has also been implicated in the study of individual differences. In particular, there is a long history of applying contingency analyses to the study of psychopathology. While studying conditioned reflexes in dogs, Pavlov (1927) noted the relation between learning conditional or contingent relationships and the development of emotional disturbances. However, Alloy and Abramson (1979) were the first researchers to imply that the contingency judgment paradigm with humans could be used as a tool to study individual differences including the symptoms of depression. Their experiments were designed to test predictions derived from theories that proposed that depressed people were characterized by a sense of a lack of control. They fail to properly evaluate the contingency between their behavior and its outcomes. This research was inspired in part by the learned helplessness theory of depression (Seligman, 1975), which argues that depressed people should be less able to learn response-outcome contingencies.

The experimental procedure employed by Alloy and Abramson involved an instrumental contingency learning task similar to the one
described by Wasserman et al. (1993) except that participants were not free to respond at any time during the training session, but had to restrict their response to particular signaled intervals. Participants were asked to learn the relationship between pressing a key and the illumination of a light. Strong differences were observed between normals and dysphorics in their judgments of zero contingencies that varied in terms of outcome density. In the low-base rate condition, the outcome occurred 25% of the time \( P(O) = .25 \) regardless of participants' pressing, whereas with the high base rate the outcome was presented 75% of the time \( P(O) = .75 \). These contingencies are the same as the .25BR zero and .75BR zero contingencies described in previous experiments. During trials, which were signaled by the occurrence of a yellow light lasting 3 seconds, participants were instructed to either press the key once or leave the key alone and the outcome (a green light) would or would not occur. Trials were separated by a variable 14-second interval (10–25 seconds). It was assumed that if subjects “realistically” assessed these contingencies there would be no differences in their judgments because, in spite of the density differences, both were zero contingencies. Moreover, it was implicitly assumed that the judgments if “realistic” should equal zero. Alloy and Abramson (1979) found that nondysphoric students' judgments increased with higher levels of outcome density. In contrast, students categorized as dysphoric, based on their score on a depression inventory, showed unchanged judgments in these two conditions. It is this increase in nondysphorics' judgments, which occurs as a function of higher levels of outcome density, that has been interpreted as an “optimistic” bias or a departure from “realism.” The lack of a density effect on the part of the dysphoric students was purported to be reflective of their tendency to be more realistic or accurate in their perceptions. Thus, the density effect, that we have argued may be a consequence of distorted perceptions of cell frequencies, is argued by Alloy and Abramson to be a consequence of unrealistic, but adaptive, optimism in nondysphorics.

Since Alloy and Abramson’s study, a number of replications of the depressive realism effect have been attempted. Much of the evidence suggests dysphoria influences judgments in situations in which the response does not control the outcome (zero contingencies) but the outcome occurs frequently; in these cases judgements are closer to zero—and therefore more accurate—than the judgments of nondysphorics (Alloy, Abramson, & Kossman, 1985; Benassi & Mahler, 1985; Martin, Abramson, & Alloy, 1984; Vasquez, 1987). This research has also shown that there is little difference between these two groups of participants when the contingencies to be judged are positive, suggesting that there is something special about the noncontingent conditions.

A number of explanations have been generated for the depressive realism effect. Alloy and Abramson (1979) suggested that conditions of zero contingency may be consistent with dysphoria and the expectations that it engenders concerning control over events in one's envi-
environment. This match between their expectations and the task allows them to accurately judge the two zero contingencies as being the same. In contrast, nondysphorics may have an optimistic bias that suggests they are much more in control than they actually are. As a consequence, some noncontingent situations are judged to be positive. Other proposals by Ackerman and DeRubeis (1991) are that motivation and perceptions of self, rather than expectations, drive the difference between the two groups. Self-esteem is somehow related to the person’s perceived instrumentality in controlling environmental events. Depression weakens this motivation to maintain self-esteem. Either or both motivation and expectation could contribute to the observed depressive realism effect.

However, returning to the associative perspective on human contingency learning, it is quite easy to predict more positive judgments with higher outcome density for zero contingencies if one simply assumes that, whereas the programmed contingency is intended to reflect a zero contingency, the participants perceive a positive contingency. This can happen simply if they overestimate the number of Cell D occurrences, that is the frequency of absences of the signal and the outcome. Even if every care is used by the experimenter to explicitly guide the participants, ITIs may also be construed as possible Cell D events. Controlling the perception of the absence of events and outcomes has been a particular problem in other contingency learning domains.

In animal conditioning, for example, in spite of evidence that rats learn a conditioned response on the basis of CS-US contingency, subsequent work has argued against this (c.f. Papini & Bitterman, 1990). For example, strong conditioning is sometimes found following zero-contingency training, or animals may be found to be insensitive to changes in contingency caused by introducing USs in the absence of the CS (Hallam, Grahame, & Miller, 1992; Jenkins & Shattuck, 1981; Quinsey, 1971). However, if the rats’ perception of the frequency of events in the contingency table differed from the experimenter’s programmed contingency, as we have suggested, then it might not be surprising that responding did not mirror the “objective” contingency. We have recently reported an experiment that showed strong discrimination between positive- and zero-contingency training with rats using a procedure in which the Cell D events were explicitly marked with a stimulus (Murphy & Baker, 2004). We showed, using an explicit contextual cue, how competition between contextual cues and the target signal predicted sensitivity to CS-US contingency. Therefore, it is important in any contingency learning task that the experimenter have adequate control over the likelihood of the outcome in the absence of the signal. The following experiment attempted to show the influence of the absence of the signal on contingency learning in dysphoric and nondysphoric students.

It is also worth noting that associative theories can model the effect. The Rescorla-Wagner model treats adding extra trials, in which the absence of the signal occurs without the outcome (Cell D), as increasing
the amount of context extinction. This decreases the context's associative strength, thereby allowing the signal to retain a stronger association with the outcome. Thus, adding either real or perceived Cell D events would be predicted to elicit stronger judgments of a contingency.

The two contingencies described by Alloy and Abramson were a low-density and a high-density zero contingency. The low-density zero contingency was constructed by presenting the outcome on 25% of the trials when the response occurred, and 25% of the trials when the response did not occur. If participants include some of the intertrial time in their processing of the information, in associative terms this will involve extra context extinction that will indirectly generate more associative strength to the response and, hence, a slight positive bias in judgments. From the perspective of $\Delta p$ this would be represented by extra Cell D experience, which would reduce the conditional probability of an outcome in the absence of the response, $P(O|R)$. This would increase the perceived $\Delta p$ and thus would also generate a bias. The limit of this bias would be .25 for the low-density contingency as large numbers of Cell D event would cause $P(O|R)$ to approach zero whereas as $P(O|R)$ would remain at .25. With the high-density zero contingency in which both unconditional probabilities are .75, the limit of this process would be a $\Delta p$ of .75 because $P(O|R)$ would be maintained at .75. A similar increased bias would also be predicted by the associative analysis. Therefore, by incorporating the $ITI$ both the $\Delta p$ contingency and the Rescorla-Wagner predictions suggest that judgments should exhibit a bias that increases with outcome density.

In fact, most of the successful depressive realism studies that we have found have involved training with somewhat longer $ITI$ time periods than is traditionally used in the field of learning (Shanks, 1985; Wasserman et al., 1993). Furthermore, at least one study that has been published that failed to replicate the depressive realism effect used relatively shorter $ITIs$ of less than 5 seconds (e.g., Dobson & Pusch, 1995). The following experiment sought to replicate both the presence and absence of the depressive realism effect by assuming that the crucial variable underlying the two findings was the difference in $ITI$.

Ninety-six participants were assigned to dysphoric ($n = 48$) and nondysphoric ($n = 48$) groups on the basis of their scores on the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). Scores of 8 or below were taken to indicate no depression. BDI scores were higher in the depressed groups than in the nondepressed groups [$F(1,81) = 133.61]$. Groups were successfully matched on gender, digit span and age, but differed on the National Adult Reading Test [$F(1,89) = 4.74]$ and years of education [$F(1,89) = 5.68$]. Therefore, subsequent data analysis included these factors as covariates to ensure that any variance in judgements attributable to these factors was removed from the analysis.

The experiment involved a $2 \times 2 \times 2$ (mood) $\times$ (ITI length) $\times$ (outcome density) fully factorial between-subjects design. Each participant made
judgments of only one contingency problem—a high-density zero-contingency \[ P(O|R) = P(O|\neg R) = .75 \] or a low-density zero-contingency \[ P(O|R) = P(O|\neg R) = .25 \]. The experimental conditions differed as to whether the ITI was short (3 seconds) or long (15 seconds) and the participants' mood—dysphoric or nondysphoric. Experimental events were presented via computer monitor.

Participants were asked to judge how much pressing of a button controlled a light switching on. They were further instructed that it was necessary to press the button on some occasions and not press it on an approximately equal number of occasions. Each trial was constructed such that there was a 3-second opportunity for the participants to make their response. This period was signaled by an on-screen message saying, "You may press the button now!" This was followed by a 2-second period, in which the light either switched on or remained off. During the ITI period, the unlit light bulb remained on the screen. After the contingency judgment task was completed, participants were debriefed.

The results confirmed the findings of Alloy and Abramson (1979): Using the long, 15-second ITI conditions, dysphoric participants showed little evidence of a density effect whereas the non-dysphoric group did; however, neither mood group exhibited an outcome density effect when the ITIs were short. The mean judgments of the contingency between button pressing and occurrence of the outcome are presented in Fig. 10.5.

![Figure 10.5](image_url)

**FIG. 10.5.** Mean judgment of control in dysphoric and nondysphoric participants as a function of short (S) and long (L) ITI and low (25%) and high (75%) outcome base rate.
A 2(ITU) × 2(density) × 2(mood) × 2(gender) fully factorial between subjects ANOVA confirmed these observations. The three-way interaction between length of ITI, outcome density (low, high) and mood (dysphoric, nondysphoric) was reliable \(F(1,78) = 4.27\). Further analysis of the three-way interaction showed that when ITIs were short, there was no reliable density by mood interaction \(F < 1\), but there was when ITIs were long, \(F(1,78) = 4.46\). Furthermore, there was no difference between mood groups in the low-density/long-ITI condition \(F < 1\), but as expected, nondepressed people’s judgments were higher than depressed people’s judgments in the high-density/long-ITI condition \(F(1,78) = 11.6\). Taken together these results support the hypothesis that the increase in contingency estimates found with the increase in outcome density is a function of the relative amounts of context extinction. The higher outcome density groups receive less context extinction and thereby perceive the contingency as more positive.

Unlike the interpretation offered by Alloy and Abramson (1979) our interpretation of the difference between dysphorics and nondysphorics suggests that judgments of zero contingencies may not easily inform us about perceptions of realism. Instead, programmed contingencies may elicit judgments that differ from experimenter expectations for a number of reasons including the segmentation of −O|−R experience. Although this work does not explain why there is a difference with dysphoric participants, it raises the possibility that some of it relates to contextual learning, and this requires further investigation. Our interpretation is also a new direction for the use of associative models, in particular the idea that contextual conditioning in human contingency learning is influenced by the ITI. This analysis also offers a potentially fruitful direction to advance our understanding of depression.

**SUMMARY AND CONCLUSIONS**

This work has attempted to show how the flexibility of associative models can account for outcome density effects thereby eliminating the need to invoke auxiliary assumptions concerning causal schemas, optimistic biases, or sensitivity to the reinforcing properties of the outcome. What the associative account does require in order to make accurate predictions is a more accurate representation of the associated events including the precise temporal relations between the variables. If the temporal resolution governing the segmentation of time is different from that predicted by the experimenter, or if the participant includes unpredicted events into the estimate of how often no outcome has occurred in the absence of the signal, then it will be difficult for the experimenter to interpret departures from predictions. Outcome density may reflect the additional processes that have been developed to account for judgments, but they may also reflect poor experimental control or understanding of how events participants deem useful for judging a contingent rela-
tionship. Our research has shown that associative theory is well able to account for seeming departures from predictions based on normative reasoning models by ensuring that both contiguity and contextual exposure are controlled.

REFERENCES


