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MODELS OF CLASSICAL CONDITIONING

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INTRODUCTION

Models of classical conditioning were preceded by more general explanatory ideas, which became progressively less vague. The early **ideas** of merit emerged from those researchers pioneering experimental procedures for studying conditioning phenomena in animals.

There is a tendency for the uninitiated to initially **visualise** associative conditioning as an entirely excitatory process, with increasing strength of association producing greater excitation. However, the apparent influence of an opposing inhibitory process assumed a role of major importance for **many** early researchers. The importance of inhibition in associative conditioning **was recognised** by **Pavlov** (1927) in his pioneering work, and indeed was responsible for most of his **ideas**. **Konorski** (1948) further formulated more specific concepts of inhibition, which have since become the basis of most recent models. **Konorski** regarded inhibition **as** an associative phenomenon which complemented excitatory associative phenomena, and proposed some possible models.

Konorski considered that both excitatory and inhibitory conditioning were mediated by the learning of connections between CS and US centres, having an **excitatory** or inhibitory (respectively) influence upon the activation of the US centres. In effect, the inhibitory connections determined the threshold level which excitatory inputs must exceed in order to activate the US centre. In other words, the net excitatory and inhibitory influences **are** summed. This is why **experimental** summation procedures use observed reductions in the efficacy of conditioned **excitators** in order to **infer the** strength of conditioned inhibitors, which on their own elicit no specific observable response. **Just as** the excitatory connections strengthen if **the** US centre **reinforcement** increases unexpectedly following CS presentation, **inhibitory** connections strengthen if US reinforcement is **less** than expected following CS presentation.

Konorski (1964) later abandoned **this** view, introducing "no-US" and "US" **gnostic** units **which** inhibited one another, **and** postulated only excitatory **conditionable** pathways between the **CSs** and these **gnostic** units. However,

Rescorla (1979) considered that this change was probably motivated more by **Konorski's** desire to integrate conditioned inhibition into his new framework, rather than to **provide** a better fit with experimental **data**. In any case, the newer model was behaviorally essentially identical to the first model which incorporated only US gnostic units. **Konorski's** original model remains the most influential for modern-day **researchers** developing **neuronal** models of classical conditioning.

While most of the basic concepts **were** shaped by the work of the early experimentalists, there has been an increasing trend in recent times for **mathematicians** and engineers to apply their problem solving skills to the basic **issue** of **classical** conditioning, and for experimental psychologists to rigorously define their models mathematically. Those researchers producing the most noteworthy models for classical conditioning have been prepared to address its considerable **behavioral complexity** (Rescorla and Wagner, 1972; Barto and Sutton, 1985; Klopff, 1987; Grossberg and Schmajuk, 1987, 1989; Gluck and Thompson, 1987; Kehoe, 1988). Most of this work has also **utilised** computer simulation to test **model** performance, and facilitate comparison with results from animal experiments. **That** work which is most relevant to the research to be described **will now be summarised critically**.

THE RESCORLA-WAGNER MODEL

The symmetry between excitatory and **inhibitory** rules of associative learning desired by Konoreki **was** exemplified by a **later** model which was, in **this respect**, completely symmetrical. **Two researchers** who had been **developing** separate theories of **Pavlovian** conditioning **decided** to collaborate **when** they **realised** that their differently expressed theories were **functionally** very similar. **The** result was **the Rescorla-Wagner** model (Rescorla and Wagner, 1972). This model was significant **because** it provided a more integrated theoretical **account** of associative conditioning phenomena than other preceding theories, and was clearly expressed in mathematical form. **Rescorla** and Wagner found that a formal mathematical specification of their verbally expressed formulations greatly assisted comparison between the model's performance and

experimental results, and also facilitated development of the model itself. Their model is described by the following equation:

$$\Delta V = a \cdot b \cdot (L - V) \quad [2-1]$$

Where: V = Compound associative strength (+ve=excitatory, -ve=inhibitory)
 ΔV = Change in the **associative** strength of a CS after each trial
 a = Learning rate associated with a CS ($0 \leq a \leq 1$)
 b = Learning rate associated with the US ($0 \leq b \leq 1$)
 L = Asymptotic strength of association supported by the US

The term $(L - V)$ in Equation [2-1] expresses the difference between the associative strength supported by the US, and the associative strength of the preceding **CS(s)**. In other words, the difference between the effect of actual reinforcement delivered and that expected. The associative strength of each CS is thus changed when a discrepancy between expectation and result is detected, **and** changed in such a way as to diminish this discrepancy. Terms a and b control the rate of learning, subject to variations in the salience of the CS, and the effectiveness of the US, respectively. Note that V can be either positive or negative, corresponding to an excitatory or inhibitory compound CS respectively, and that the same simple learning rule governs changes in the associative strength of both types of CS.

The main power of the **Rescorla-Wagner** model comes into **play** when compound **CSs** are employed. Terms ΔV , a , and b remain specific to **individual CSs**, but $(L - V)$ relates to the **discrepancy** between compound expectation and compound **reinforcement** delivered. **This** means that the change in **associative** strength of each CS component of the compound CS is dependent upon the total associative strength of the compound, which integrates a number of important empirical results. These include stimulus amplitude effects, acquisition of conditioned excitation and inhibition, extinction, overshadowing (except perhaps 1 trial overshadowing), compound conditioning effects (e.g., **blocking** and unblocking), and discriminative stimulus effects - **all** of which are defined in Rescorla and Wagner (1972).

Despite its success, the Rescorla-Wagner **model** suffers **many** limitations, the **most** serious being that it is a "trial-level" model. In other words, it only seeks to approximate the state of affairs at the end of each acquisition or extinction trial, and does not address the **intratrial** processes which produce the end of **trial** results. Nor does it address aspects of timing **within** each trial, **such as** the effect of ISI, or the **temporal** characteristics of the CS, US, CR, or UR. As such, it cannot reveal many of **the** underlying processes **capable** of producing associative learning, and can only approximate the gross results of several general **types** of experimental procedure.

As impressive **as** it **still** is, the breadth of behavior accounted for by this relatively **simple model** is by no **means** complete. Furthermore, **some** characteristics of the behavior that it does produce do not accord **well** with **empirical** results. For example, **the Rescorla-Wagner** model predicts that a conditioned inhibitor (CS-) will extinguish if presented alone. Experimental results indicate that **while** a conditioned **excitor** (CS+) will extinguish if presented without subsequent reinforcement, a CS- presented **alone** (without a simultaneously presented CS+ which expects more reinforcement than **actually** arrives) is not significantly affected (**Zimmer-Hart and Rescorla, 1974**). This vivid asymmetry in the behavior of conditioned **excitors and** inhibitors contradicts the complete symmetry of the Rescorla-Wagner model.

Also, none of the terms in **Equation [2-1]** relate specifically to direct CS or US characteristics, but are instead abstractly defined quantities somehow derived from them. For example, it is not specified **mathematically how** the learning rates **a** and **b**, associated with the CS **and** US respectively, are calculated. Similarly, **L** is **not** mathematically defined **in** terms of the US. It is only specified that **all** three variables increase with increases in **the** intensity of **their** respective stimuli.

Finally, the Rescorla-Wagner model predicts a strictly negatively accelerating acquisition curve, despite the common occurrence of sigmoidal acquisition curves (Spence, 1956; Mackintosh, **1974**).

THE BARTO-SUTTON NEURONAL MODEL

Barto and **Sutton** (1985) sought to tackle **intratrial** phenomena not addressed by the **Rescorla-Wagner** model (1972). They were particularly interested in supporting the anticipatory nature of the CR, and ISI dependency. Their **model** also responded to direct neuronal CS **and** US inputs, without using abstractly derived quantities like the **Rescorla-Wagner** model.

The **Barto-Sutton** model uses averaged previous CS input activation to gate (or enable) changes in the associative strength of the CS. **The** direction **and** extent of **change** is **then determined** by subsequent changes in output activity. Associative learning between a CS input **and** the US input is possible because **the** US **also activates** the model's output. Using changes in output **activity** to determine changes in **associative** strength **means** that a simple form of both higher order conditioning and sensory preconditioning are also automatically supported.

Conditioned inhibitors are prevented from extinguishing when presented alone, by **only** permitting non-negative output signals. This type of solution was **also** suggested by **Rescorla** (1979) to improve the **Rescorla-Wagner** model.

However, the **Barto-Sutton** model is excessively **sensitive** to the timing of CS offset, producing conditioned **inhibitors** in delayed conditioning paradigms when **exciters** have been **shown** to result, in the case where CS and US overlap significantly (**Klopf**, 1987). Also, like the **Rescorla-Wagner** model, it **still** produces only a negatively accelerated acquisition curve.

In general, the **main** achievement of **the Barto-Sutton** model **was** to demonstrate **that** fine grained **intratrial** phenomena could be tackled using a mathematically explicit **model**. However, its deficiencies invite further attempts to produce more robust and comprehensive models.

THE KLOPF NEURONAL MODEL

Klopf's (1987) recent drive-reinforcement neuronal model represents an **attempt** to further improve the modelling of the **intra-trial** processes underlying classical conditioning. It can be **considered as an extension** to the Barto-Sutton model, which in turn represents an attempt to temporally refine the Rescorla-Wagner model. Increments in, rather than **previous** activation of, CS input activity **are** correlated with subsequent changes in nodal output activity, **occurring** some time later.

In the abstract of his interim report, Klopf (1987) claims that "It is shown that the proposed neuronal **model** predicts the basic categories of classical conditioning phenomena including delay and trace conditioning, conditioned and unconditioned stimulus duration and amplitude effects, **partial** reinforcement effects, **interstimulus** interval effects including simultaneous conditioning, **second-order** conditioning, conditioned inhibition, extinction, reacquisition effects, backward conditioning, blocking, overshadowing, compound conditioning, **and** discriminative stimulus effects." However, examination of the text of his report **reveals** that the degree of fit between the models' behavior **and** empirical results is not quite as **impressive** as the abstract might suggest. Furthermore, being an extension of the Rescorla-Wagner model automatically means that **stimulus** amplitude effects, conditioned inhibition, extinction, overshadowing, **compound** conditioning and discriminative stimulus effects **are** already able to be accounted for.

The "partial reinforcement effects" referred to above do not include *the* **partial** reinforcement effect (PRE), in which the **rate** of extinction **following** **partially** reinforced acquisition is retarded, by comparison with that following fully reinforced acquisition. Instead, it refers **to** the reduced rate, and sometimes reduced asymptote, of conditioning resulting **from** partially reinforced acquisition (Klopf, 1987, p. 42). This phenomenon is far more **simply** accounted for than the **PRE**, and in fact is to be expected from most simple neuronal models of conditioning, since the presence of nonreinforced trials during acquisition Bowers the mean effect of reinforcement.

The "interstimulus interval effects" referred to above do in fact refer to the inverted U shaped curve expressing learned performance **as** a function of interstimulus **interval**. However, the shape is not attained by interacting quantities which can produce a smoothly changing relationship, **but** instead by an arbitrarily determined set of coefficients controlling the rate of learning over coarse **500ms** intervals (**Klopf**, 1987, p. 45). This type of "solution" does not really provide any insight into the type of mechanism which might be responsible for the behavior.

Finally, backward conditioning produces **conditioned** inhibition with **Klopf's** model (p. 57), which is by no means unanimously established empirically.

Mechanistically, Klopf's **neuronal** model **also** suffers two further deficiencies:

(i) If the CS and US terminate simultaneously, and the duration of each is progressively increased, the extent of negative feedback provided by offset of output signal which **occurs** at CS and US offset, quickly **diminishes** to zero. In these circumstances, **the** associative strength of the CS would keep on growing with each acquisition trial, never levelling off.

(ii) Klopf (1987) uses CS excitatory weight to gate changes in it, to produce both S shaped acquisition (p. 4) and faster **reacquisition** (p. 55). However, the faster reextinction **also** observed following the alternate **acquisition-extinction** sessions **which** produce more rapid reacquisition is not supported by this mechanism, **or** any other aspect of the model. Using CS excitatory weight to gate **its** own learning also necessitates some means of avoiding a zero excitatory **CS** value. Furthermore, this technique invariably produces **sigmoidal** acquisition curves when acquisition begins with a weakly excitatory CS, which while consistent with most subject-averaged **empirical** results, is not consistent with the results **from** all individual subjects (Spence, 1956, p. 65).