Role of Surprising Nonreward in Associative Learning

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Abstract Early experimental accounts of learning assumed that reinforcement strengthened associative bonds whereas nonreinforcement weakened them. Strengthening-weakening models explain the adjustment of nonmammalian species to downshifts in incentive magnitude, but fail to explain a mammalian phenomenon known as successive negative contrast (SNC). In SNC, exposure to incentive downshift leads to poorer performance than that of unshifted controls. SNC forced the development of new learning models emphasizing the acquisition of expectancies and the induction of emotional responses when expectancies are violated. Comparative research suggests a distinction between allocentric learning (tracking changes in the environment) and egocentric learning (remembering the emotional response to environmental changes). Recent research shows that egocentric learning in the SNC situation is modulated by the opioid system. Similar modulation is observed in situations involving physical pain, fear, and social exclusion. An evolutionary hypothesis about the evolution of egocentric learning mechanisms suggests deep connections between the brain circuits controlling fear, frustration, and possibly grief.

Introduction

What are the roles of reinforcement and nonreinforcement in learning? This question was first approached scientifically during the late years of the 19th century by those who introduced the study of learning into the laboratory (Boakes, 1984). An influential set of studies published by Thorndike (1898) looked at the acquisition of new skills using what were then novel learning techniques. Thorndike placed food-deprived cats inside a box that contained a latching mechanism that the animal could operate to open a door and reach a dish with food. The food was visible from inside this puzzle box, a fact that initially instigated many responses. Only one response, the one eventually acquired, was effective in activating the latch and opening the door. There was an interesting parallel between the development of a new skill during training trials and the Darwinian model of evolution based on the notions of character variation and natural selection according to reproductive fitness. From an initial stage of behavioral variation, food reinforcement selected those responses that activated the latching device. Later, Thorndike (1911) accounted for these findings in terms of his S-R theory of learning. The idea was very simple, although also counterintuitive. Placed in a situation with many stimuli (Ss), responses (Rs) tend to occur under the control of specific stimulus components (S→R₁, S₂→R₂, and so on). One of them is eventually followed by the operation of the latching device and access to food, a sequence represented in Figure 1. A notorious and counterintuitive feature of Thorndike’s S-R theory is that the food (technically called “incentive”) led to a change in the associative connection between S and R, without participating in it. In other words, Thorndike created a theory of learning with no encoding of the incentive event that triggered learning in the first place. Thorndike’s view also incorporated a rule concerning changes in associative
strength that occurred when incentives failed to occur (Figure 1). In cases when the incentive was unexpectedly omitted (i.e., surprising nonreward), the strength of the S→R link was assumed to decrease. This theory can thus be labeled the strengthening-weakening view of learning (S-W). An S-W animal is one that knows what to do in each situation — although it may be ignorant about the purpose of its behavior (Papini, 1998).

![Brain diagram](image)

**Figure 1.** A schematic representation of Thorndike's (1911) strengthening-weakening theory of learning. Notice that the feedback arrows influence the link between “stimulus” and “response,” not any of these individual representations. The outcomes of each response (“incentive” and “nothing”) are not encoded in the associative structure used to represent learning. Thus, a Thorndikean animal knows what to do, but not what is the goal of its behavior.

The S-W model of learning was dominant during the first half of the 20th century. In the hands of theorists like Hull (1943) and Guthrie (1952), parsimonious explanations of a wide variety of seemingly complex behaviors were proposed. One discovery would, however, undermine this view by demonstrating that incentives were, in fact, encoded into the associative structure formed during acquisition. Elliott (1928) observed that rats trained to run a complex maze for a highly preferred incentive (bran mash) exhibited a striking disruption of their behavior when unexpectedly switched to a less preferred incentive (sunflower seeds). Eventually, this became known as successive negative contrast (SNC). Interestingly, the degree of behavioral disruption in downshifted rats was significantly greater than that observed in a group exposed only to the less preferred incentive. Thus, although after the downshift both groups of rats were receiving the same incentive, their behavior was drastically different. The fact that the control group behaved steadily suggested that the disruption that followed incentive downshift could not be attributed to the use of an incentive so weak that it would not support acquired behavior. The lower incentive (sunflower seeds in this case) was acceptable, except when rats had previously been exposed to the higher incentive (bran mash). Besides, SNC was transient; that is, the deterioration dissipated across trials, thus providing evidence that the lower incentive had the ability to eventually support acquired behavior. Similar effects were later reported in experiments with monkeys (Tinklepaugh, 1928) and chimpanzees (Cowles & Nissen, 1937). These experiments yielded results consistent with Tolman’s (1932) view based on the idea that learning involves the acquisition of goal expectations. Because both antecedent stimuli and incentives can be treated as stimuli, this view became known as the S→S view of learning. SNC and similar effects demonstrated that learning could not be reduced to S→R associations because information about the incentive was incorporated into the associative structure. However, the Tolmanian animal was described as one that knows exactly what to expect, although it may not know how to get to the goal, other than in terms of general tendencies to approach and avoid a location. As Guthrie (1952, p. 143) eloquently put it, a Tolmanian, S→S rat may remain “buried in thought” unable to select the appropriate response.

**Surprising Nonreward: Data and Theory**

A traditional solution to the S→R vs. S→S choice is to incorporate both into a parallel-processing structure, as done in two-process learning theories (Amsel, 1958; Mowrer, 1960; Trapold & Overmier, 1972). An alternative view is provided by hierarchical
theories that emphasize R→S (or response-incentive) associations (Colwill & Rescorla, 1986). Of these accounts, Amsel’s (1958, 1992) frustration theory, developed as an extension of earlier views (e.g., Hull, 1934), is particularly relevant for present purposes because it was formulated to account for SNC and related phenomena. Figure 2 illustrates the SNC effect described previously, as well as the partial reinforcement extinction effect (PREE), a learning phenomenon also dependent on surprising reward loss. The PREE can be described as increased persistence in extinction after training with partial reinforcement (i.e., a random mixture of reinforced and nonreinforced trials), than after training with continuous reinforcement (i.e., every acquisition trial ends in food reinforcement). To explain these effects, Amsel (1992) proposed a theory based on three main stages described in theoretical terms in Figure 3.

The first stage involves the violation of an expectancy that occurs when the obtained incentive is of lesser value than the incentive expected on the basis of prior experience under similar conditions (Figure 3a). Thus, the procedure is termed surprising nonreward because the event was unexpected (i.e., better outcomes had occurred previously under similar conditions) and because it involved incentive loss. The loss may have been partial (as in incentive downshift situations) or complete (as in nonreinforced trials during partial reinforcement training). Surprising nonreward triggers an internal aversive state, called primary frustration, that has a variety of behavioral and physiological consequences (see Papini & Dudley, 1997). For example, surprising nonreward invigorates ongoing behaviors in a variety of situations (Dudley & Papini, 1995; Stout, Boughner, & Papini, 2003; Thomas & Papini, 2003). Additionally, primary frustration can support Pavlovian conditioning (the S→Rf connection in Figure 3a). By virtue of this new learning, stimuli present in the situation can activate the memory of primary frustration in an anticipatory fashion during subsequent trials (Figure 3b). The aversive hedonic tone of this conditioned internal response, called sec-

![Figure 2](image-url)

**Figure 2.** (a) Two groups of rats received access to sucrose solutions during 5-min long daily trials. For Group 32-4 (n = 6), a 32% sucrose solution was presented for 10 trials and followed by a 4% solution during the remaining 5 trials. For Group 4-4 (n = 7) the 4% solution was presented in each of 15 trials. During the postshift phase, both groups received the same 4% solution, yet their consummatory behavior is significantly different, as shown by a significant group by trial interaction, F(4, 44) = 6.25, p < 0.03. This provides evidence for cSNC. (These unpublished results were collected by Eugene Castro.) (b) Two groups of rats received a single trial per day in a lever-pressing situation. In Group CR (continuous reinforcement, n = 8), every acquisition trial ended with the delivery of 15 45-mg food pellets. In Group PR (partial reinforcement, n = 8), a random 50% of the trials ended in food delivery and the rest with no food delivery. Rats received 48 acquisition trials followed by 80 extinction trials in which no food was ever delivered. The latency to complete 10 lever presses was the dependent variable. Whereas groups did not differ during acquisition, a group by trial analysis indicated a significant interaction during extinction, F(1, 22) = 4.40, p < 0.05. This provides evidence for the PREE. (These unpublished results were collected by Steven Stout.)
(a) Aftereffects of surprising nonreward:

\[
S \rightarrow R_{app} \ldots \text{No reward} \ldots R_F
\]

\[R_F = \text{Primary frustration}\]

(b) Anticipatory effects:

\[
S \rightarrow R_{app} \ldots \text{Reward}
\]

\[e_F \rightarrow R_{avo}\]

\[e_F = \text{Secondary frustration}\]

(c) Persistence:

\[
S \rightarrow R_{app} \ldots \text{No reward} \ldots R_F
\]

\[e_F \rightarrow R_{app} = \text{Counterconditioning}\]

Figure 3. Mechanisms suggested by Amsel’s (1992) frustration theory to explain SNC and the PREE. (a) Primary frustration, \(R_F\), is an internal unconditioned state induced by the surprising omission or reduction of an incentive. Thus, \(R_F\) occurs only when there is a negative discrepancy between the expected, \(e_I\), and the obtained incentives, contingent on an approach response, \(R_{app}\). A Pavlovian association between prevailing stimuli and \(R_F\) is automatically established during this stage. (b) The stimulus acquires the ability to elicit ambivalent expectations of both the incentive, \(e_I\), and the frustrative response, \(e_F\). The latter, referred to as secondary frustration, induces a competing avoidance response, \(R_{avo}\). The competing expectations give rise to a psychological conflict that induces response competition. This anticipatory effect is designed to explain SNC. (c) If secondary frustration is occasionally paired with the incentive, as it happens during partial reinforcement training, then it loses its disruptive properties and becomes associated to the approach response. This mechanism, called counterconditioning, explains the PREE.

Secondary frustration \((e_F)\), coexists with the appetitive tone of the incentive expectancy, thus generating a conflict that results in response competition between approach and avoidance tendencies (Jones, 1970). The ensuing conflict explains the SNC effect, as well as some additional extinction effects. For example, administering a single trial per day, Hulse (1958) reported that running responses reinforced by large incentives extinguished faster than responses reinforced by small incentives. This so-called magnitude of reinforcement extinction effect (MREE) may be seen as paradoxical under the light of a theory based on the S-W mechanism. If the strength of the S→R connection is a direct function of reinforcer magnitude (Hull, 1943; Thorndike, 1911), and performance in extinction reflects mainly the strength of the associative bond, then extinction should be faster after acquisition with a small reward because the S→R association is presumably weaker. However, the MREE can be easily seen as a special case of SNC in which two groups reinforced by incentives of different
value are downshifted to a common lower value in extinction (only one group is downshifted in the case of SNC, but both groups get a common value in the postshift phase). In both SNC and MREE, the transition from a larger to a smaller incentive generates (1) primary frustration, (2) conditioned frustration, and (3) conflict, thus reducing persistence in extinction. Secondary frustration is also established during partial reinforcement training, where it is occasionally paired with the incentive, as shown in Figure 3b. Under some conditions, pairing an aversive stimulus with an appetitive consequence tends to modify the ability of the aversive stimulus to elicit avoidance behavior. This phenomenon, called counterconditioning, was first described by Pavlov (1927), who reported that shock—food pairings endowed the shock with the ability to provoke salivation in dogs. Thus, the occasional pairings between secondary frustration and food during partial reinforcement training reduce avoidance tendencies and link secondary frustration to the approach response (Figure 3c). Thus, counterconditioning is the mechanism that promotes persistence in extinction after chronically uncertain conditions of reinforcement in acquisition, including partial reinforcement (a mixture of reinforced and nonreinforced trials), partial delay of reinforcement (a mixture of immediate and delayed reinforcement), and variable magnitude of reinforcement training (a mixture of large and small reinforcements; for a review, see Amsel, 1992).

This theoretical framework has been successfully applied to a range of conditions involving changes in incentive parameters (see, e.g., Amsel, 1992; Daly, 1991; Daly & Daly, 1982). Of course, there are problems with this theoretical framework that have been pointed out in the literature (e.g., Mackintosh, 1974; Flaherty, 1996; Gray & McNaughton, 2000). A major limitation of this theory is that it applies well only to mammalian behavior.

**Comparative Research**

When learning experiments are carried out with species other than the typical laboratory animals (e.g., rats, mice, primates), the results show some impressive regularities. Instrumental training in nonmammalian vertebrates (birds, reptiles, amphibians, and bony fish) usually yields graded acquisition and extinction functions, as well as a variety of familiar effects. The similarities among vertebrates are so compelling that it has even been suggested that there are no major species differences in learning mechanisms, with the exception of humans (Macphail, 1982). While such a view may seem extreme, commonality in learning phenomena is far more striking than species divergence, a fact that provides an evolutionary basis for the view that basic learning phenomena are produced by a relatively small and general set of mechanisms, as suggested by the general-process view of learning (Papini, 2002a). One of the best sources of evidence for species divergence in learning mechanisms is provided by comparative research on the SNC, PREE, and MREE phenomena.

Figure 4 shows data from species assigned to three different vertebrate classes, all exposed to incentive downshifts in experiments designed after those that yielded evidence of the SNC effect in mammals. Consider, for example, the data presented in Figure 4a. Three groups of toads (*Bufo arenarum*) received training in a runway and were reinforced with access to water for approaching the goal box. Incentive magnitude was manipulated by allowing access to water during either 1.280 s (large) or 80 s (small). As shown by the two unshifted groups, these magnitudes were effective in producing different levels of instrumental performance. The difference in incentive magnitude can be easily verified by weighing the animals before and after the single daily trial (toads consume water by absorption through a patch of ventral skin). Notice, however, that a shift from a large to a small incentive induces a readjustment of performance, but yields no evidence of SNC. This pattern may be referred to as a “reversed SNC” effect. Similar reversed SNC results were obtained with turtles (*Chrysemys picta*, Pert & Bitterman, 1970; *Geoclemys reevesii*, Papini & Ishida, 1994), also in a runway situation, and with pigeons (*Columba livia*, Papini, 1997), in a
Figure 4. The results of analogous experiments reflecting the effects of downshifts in incentive magnitude in instrumental behavior in (a) toads (from Papini, Muzio, & Segura, 1995, Experiment 1; copyright by S. Karger A. G., reproduced with permission), (b) turtles (from Papini & Ishida, 1994; copyright by the Experimental Psychology Society, reproduced with permission), and (c) pigeons (from Papini, 1997, Experiment 1; copyright by the American Psychological Association, reproduced with permission). Groups were exposed to a transition (marked by the dashed line) from large to small incentive magnitudes (L-S), control groups received training either always with the small incentives (S), or always with the large incentives (L). In all cases, there was evidence that the different incentives magnitudes controlled behavior differentially and that the downshift affected behavior. However, behavior exhibit no hint of the SNC effect.

key-pecking situation (see Figures 4b and 4c). Also similar were the results obtained with bony fish (Carassius auratus) trained to swim in a straight alley for solid food (Lowes & Bitterman, 1967), or to consume a semisolid food solution delivered through a plastic target (Couvillon & Bitterman, 1985).

A similar picture emerges from a review of the MREE data. Fish, toads, turtles, and pigeons trained at a rate of one trial per day show graded acquisition and extinction, and provide evidence of magnitude discrimination. But persistence in extinction is higher after training with large incentives than with small incentives — a reversed MREE (Gonzalez, Holmes, & Bitterman, 1967; Papini, 1997; Papini & Ishida, 1994; Papini et al., 1995; Papini & Thomas, 1997; Pert & Bitterman, 1970; Thomas & Papini, 2003). Thus, SNC and MREE co-vary across species whenever they have been studied under spaced conditions of training (see Figure 5a). That is, they are either both present (rats) or both absent (fish, toads, turtles, and pigeons). Such co-variation is consistent with the hypothesis that these two learning phenomena are determined by the same mechanisms.

The PREE, however, exhibits a more complex distribution across species (see Figure 5b). First, evidence of the PREE has been reported in a variety of species when conditions of training involve short intertrial intervals.
Thus, the massed-trial PREE has been reported in goldfish (Gonzalez & Bitterman, 1967), toads (Muzio, Segura, & Papini, 1992), and turtles (Ishida & Kitamura, 1988). By way of comparison, the SNC effect failed to occur in goldfish and toads even when massed training conditions were used (Mackintosh, 1971; Schmajuk, Segura, & Ruidiaz, 1981). Second, when trials are widely spaced, as in the experiments reviewed previously, the typical outcome has been reversed PREEs, that is, greater persistence in extinction after training with continuous reinforcement, rather than partial reinforcement. Reversed PREEs have been reported in fish (Boitano & Foskett, 1968), toads (Muzio, Segura, & Papini, 1992, 1994), iguanas (Dipsosaurus dorsalis, Graf, 1972), and turtles (Gonzalez & Bitterman, 1962). Third, pigeons exhibit the spaced-trial PREE, whether in key pecking (Papini, Thomas, & McVicar, 2002) or runway performance (Thomas & Papini, 2003), even though analogous experiments have produced evidence of reversed MREE and SNC results. Thus, pigeons have provided the only available evidence that these effects are dissociable. This dissiciability was found within a single experiment (Thomas & Papini, 2003). Three groups of pigeons received

Figure 5. Distribution of the SNC and MREE (a) and of the PREE (b) among vertebrates. Data from spaced-trial experiments is presented (i.e., one trial per day). The phylogenetic trees presented here are based on paleontological data described in more detail in Papini (2002b). Dotted and dashed lines are inserted to reflect the evolutionary addition of mechanisms.
training in a runway apparatus for access to either 1 g or 5 g of mixed grain. A group that received continuous reinforcement with the large reward showed faster extinction than a group receiving 50% partial reinforcement (the PREE), but slower extinction than a third group that had been trained under continuous reinforcement with the small reward (reversed MREE). A fourth group, trained under continuous reinforcement, was trained only when a reinforced trial was scheduled for the partial animals (i.e., with number and distribution of reinforcements matched across groups). The PREE still emerged under these conditions. Additional evidence indicates that pigeons are also capable of developing increased persistence in extinction after exposure to variable magnitudes of reward (Thomas & Papini, 2003). If instead of administering a random series of rewarded and nonrewarded trials pigeons are exposed to a random series of large and small rewards, they exhibit increased persistence in extinction relative to either a group trained always with the large reward or one trained always with the average amount of the two rewards. Thus, the effects of reward uncertainty and magnitude, which tend to co-vary in other vertebrates, are dissociable in pigeons.

An Evolutionary Framework

In recent decades, the field of animal learning has been dominated by an approach that stresses the adaptive value of learned behavior (e.g., Domjan, 2005). Research inspired by this view has yielded substantial evidence of the contribution of learning mechanisms to individual reproductive success (see, e.g., Hollis, Pharr, Dumas, Britton, & Field, 1997). As information about learning mechanisms from research with different species accumulates, a complementary approach to that of adaptive significance becomes possible (Bitterman, 1975). By centering the analysis on learning mechanisms, rather than function, it is possible to develop a theory of the evolution of proximate mechanisms of learning that accounts for the actual variation in mechanistic phenotypes. Conventional knowledge suggests that evolution is an opportunistic process guided by conservative forces that fix some traits keeping them strikingly stable across taxa (homology) and by creative forces that promote alternative solutions to similar problems (divergence and homoplasy). Thus, the goal of an analysis of the evolution of learning mechanisms is to identify homologies, homoplasies, and divergence so as to develop a phylogenetic (i.e., historical) theory of learning.

In the past, this comparative analysis of learning mechanisms has remained at the level of behavior. However, increasing knowledge about the determinants of learning has revealed a nested set of at least four mechanistic levels organized hierarchically (Papini, 2002a). From top to bottom:

- **Psychological level**: Behavioral processes like anticipatory frustration (Amsel, 1992).
- **Neurobiological level**: Neural circuits that encode information, such as coincidence detectors in classical conditioning (Blair, Schafe, Bauer, Rodrigues, & LeDoux, 2001).
- **Neurochemical level**: Synaptic transmission, as in the NMDA receptor complex (Riedel, Platt, & Micheau, 2003).
- **Cell-molecular level**: Cellular processes involved in experience-dependent gene transcription, such as the cyclic adenosine-monophosphate (cAMP) pathway (Kandel & Abel, 1995).

A recognition that the concept of “mechanism” refers to several levels of analysis suggests a novel implementation of the three fundamental evolutionary concepts mentioned previously, namely, homology (phenotypic similarity attributable to common ancestry), homoplasy (phenotypic similarity attributable to common ecological pressures), and divergence (phenotypic differences). According to this hierarchical-levels framework:

- **Homology** is demonstrated when the same behavioral outcome observed in different species trained under analogous conditions can be shown to depend on the same learning mechanisms at all four levels of analysis.
- **Homoplasy** is demonstrated when the same
behavioral outcome observed in different species under analogous conditions can be shown to depend on different learning mechanisms at least in one level of analysis.

- Divergence is demonstrated when different behavioral outcomes in different species trained under analogous conditions can be shown to depend on different learning mechanisms at least in one level of analysis.

Although the explicit articulation of the hierarchical-levels framework is relatively novel (Papini, 2002a, 2002b), the idea is implicit in virtually all the research on learning since its inception. As noted elsewhere (Papini, 1998), Thordike’s view of learning in terms of what today would be called synaptic plasticity, thus bridging the behavioral and neurochemical levels mentioned above. The standard techniques of behavioral neuroscience provide insights into some of these levels. Thus, brain lesions and electrophysiological recordings tap on the neurobiological level, whereas drug manipulations highlight the role of neurochemical systems and cell-molecular mechanisms on synaptic plasticity. A similar reasoning is applied for the development of animal models of psychological disorders in humans (Carroll & Overmier, 2001). For an animal model to be applicable and useful, the underlying mechanisms of the model must be homologous with those operating in humans under similar conditions. Thus, establishing an animal model in psychology requires distinguishing homologous from homoplastic similarities in some learning paradigm. Bringing the evolutionary implications of the hierarchical-levels framework to the foreground can help organize research efforts and establish clear connections between comparative psychology and evolutionary biology.

Available information covers evidence from some of these mechanistic levels, but not all of them. One example is provided by the neurochemical analysis of the PREE in pigeons (Thomas & Papini, 2003). In one experiment, pigeons received runway training under a 50% partial reinforcement or continuous reinforcement schedule, and were treated with three different drugs carefully chosen for their known effects on the PREE in rats. These drugs were chloral hydrate (CDP), nicotine (Nic), and haloperidol (Hal), which reduce, enhance, and have no effect on the PREE in rats, respectively. CDP is a benzodiazepine anxiolytic that increases the affinity of the γ-amino butyric acid receptor (GABA) for the neurotransmitter GABA. GABA receptors are widely distributed in the telencephalon and diencephalon of both birds and mammals, and exhibit similar patterns of density that suggest conservative evolution of this system (Veeman, Albin, Richfield, & Reiner, 1994). The chronic administration of CDP during both acquisition and extinction eliminates the spaced-trial PREE in rats trained to run or to press a lever for food reinforcement (Feldon & Gray, 1981; McNaughton, 1984). In pigeons, however, CDP delayed the onset of extinction, but had no detectable effect on the PREE. Nic is a stimulant that selectively binds to nicotinic receptors, a subclass of cholinergic receptors. Nicotinic receptors are located also in relatively conserved areas, including the substantia nigra and the ventral tegmental area (Jarvik & Schnaider, 1992). In rats, Nic administration induced the PREE under conditions that failed to demonstrate any evidence of it in the saline groups (Grigoryan & Gray, 1996). In pigeons, however, Nic administration eliminated the PREE. Hal is an antipsychotic drug that binds predominantly to dopaminergic receptors, where it has an antagonistic action (Weiner & Molino, 1994). Dopaminergic receptors are conservatively distributed in the vertebrate central nervous system (Smeets & Gonzalez, 2000). In rats, Hal administration has no detectable effect on the PREE (Feldon, Katz, & Weiner, 1988; Feldon & Weiner, 1991). In contrast, pigeons treated with Hal exhibit no evidence of the PREE. This pattern of results provides supporting evidence for homoplasticity of PREE mechanisms among rats and pigeons (see summary in Table 1). A homoplastic hypothesis is consistent with two additional aspects of the available evidence mentioned previously,
Table 1. Drug Effects on the Runway PREE for Rats and Pigeons.

<table>
<thead>
<tr>
<th>Drug</th>
<th>System</th>
<th>Rats</th>
<th>Pigeons</th>
</tr>
</thead>
<tbody>
<tr>
<td>CDP</td>
<td>GABA</td>
<td>Eliminates</td>
<td>No effect</td>
</tr>
<tr>
<td>Nicotine</td>
<td>Acetylcholine</td>
<td>Enhances</td>
<td>Eliminates</td>
</tr>
<tr>
<td>Haloperidol</td>
<td>Dopamine</td>
<td>No effect</td>
<td>Eliminates</td>
</tr>
</tbody>
</table>

Note. See text for references.

namely, the dissociation of PREE and MREE in pigeons, but not in rats, and the taxonomic distribution of the PREE (see Figure 5b). The hypothesis of independent evolution of the mechanisms underlying the spaced-trial PREE is more parsimonious than the alternative phylogenetic account that pigeons and rats share the same mechanisms, but these were lost in turtles and iguanas, both of which showed reversed PREEs.

Papini (2002a, 2002b) suggested that the mechanistic components of a given learning phenomenon may behave in a modular fashion. Such a modular arrangement is implied by the role played in associative learning by some cell-molecular processes in distantly related phyla. For example, the cAMP pathway has been shown to modulate synaptic plasticity in mollusks, arthropods, and vertebrates (Dubnau & Tully, 1998; Kandel & Abel, 1995). Because these animals exhibit no homologies in the macroanatomical organization of the central nervous system (neurobiological level), the framework described above suggests the independent evolution of the circuits involved in associative learning (i.e., homoplasy). Thus, although learning may have evolved independently in distantly related phyla, they may share some cell-molecular mechanisms, such as the cAMP pathway involved in synaptic plasticity. A similar situation applies to the evolution of the eyes in mollusks, arthropods, and vertebrates, which despite being homoplastic in design, share some genes coding for pigments involved in light reception (Raff, 1996).

Mechanisms of cSNC in Mammals

Rationale

A theory of the evolution of SNC mechanisms in vertebrates requires a good understanding of such mechanisms in mammals. The major evolutionary outcome in the case of SNC is the potential divergence uncovered by comparative research. It is “potential,” and not actual, because a demonstration of divergence requires more than behavioral research — although behavioral evidence provides the starting point for an evolutionary analysis. Following the hierarchical-levels framework described above, one would proceed from the highest (psychological) to the lowest mechanistic level (cell-molecular). Such a theory would then provide a guide as to where to look for divergence in learning mechanisms among the vertebrates. Once the brain components of the basic circuit underlying a given learning phenomenon are identified, one can proceed by looking at the role of the homologous components on equivalent learning tasks in species from other taxonomic groups.

Amsel’s (1992) theory, depicted in Figure 3, provides a starting point as a description of the SNC mechanism at the psychological level. This account captures many of the behavioral properties of SNC (see Amsel, 1992; Daly & Daly, 1982; Flaherty, 1996; Papini, 2003), although it is by no means universally accepted. Part of the reason may be that the SNC phenomenon engages a variety of processes that go beyond the original set that frustration theory was designed to explain. Two main
insights were provided by frustration theory. First, it identified an unconditioned form of frustration, called primary frustration, originally postulated to account for the frustration effect (Amsel & Roussel, 1952), or greater invigoration of a dominant response immediately after surprising nonreward than after surprising reward. Later research (reviewed in Papini & Dudley, 1997) showed that surprising nonreward has, in fact, a variety of effects that could be attributed to the unconditioned emotional state of primary frustration. Second, the hypothesis that primary frustration supports the development of a conditioned form of frustration, called secondary frustration. Can this distinction stand the test of factors operating at lower mechanistic levels?

This question can be partially answered by studies focusing on consummatory SNC (or cSNC). Consummatory behavior also seems a priori a better model for comparing distantly related species than instrumental behavior. The distinction between consummatory and instrumental behavior was first introduced by Craig (1918), who argued that the former is more stereotypical and less flexible than the latter. Broadly speaking, consummatory behavior seems to share some fundamental properties across many species of vertebrates (including apprehending the prey with the mouth and swallowing it) that may provide analogous responses for analysis. Instrumental behavior, however, varies more markedly across species, from the sit-and-wait to the active group hunting strategies described in different species (Papini, 2002b). In the specific case of cSNC, although solid food has been used as the incentive (Pellegrini & Mustaca, 2000), the typical experiment involves access to sucrose solutions that are licked and swallowed by rats during brief 5-min long trials (Flaherty, 1996). The basic effect (see Figure 2a) is deceivingly simple. What appears to be a single process of consummatory suppression is, in fact, a complex process involving at least two sets of underlying mechanisms. The first involves the initial impact of the incentive downshift on consumption and appears in full strength during the first downshift trial (trial 11 in Figure 2a). The second source of consummatory suppression arises only after some experience with the downshifted solution and it reaches full strength during the second postshift trial (trial 12 in Figure 2a). It has been suggested (Papini, 2003; Papini, Wood, Daniel, & Norris, in press) that these two stages correspond to the primary and secondary frustration processes postulated by frustration theory.

**First vs. Second Postshift Trials**

Three sources of evidence demonstrate that the mechanisms underlying consummatory suppression after the first and second downshift trials are dissociable. The first source of evidence is provided by the selective action of anxiolytic drugs on the cSNC effect. For example, the administration of 0.75 and 1 g/kg (i.p.) of a 15% ethanol solution before the second postshift trial reduced the magnitude of the cSNC effect. With higher or lower doses, the anxiolytic effects disappeared. However, this attenuating effect was not present when ethanol was administered before trial 11 (Becker & Flaherty, 1982, 1983). A similar dissociation was found with a more selective drug, the benzodiazepine anxiolytic chlordiazepoxide (CDP). The anxiolytic effects of CDP (6-10 mg/kg, i.p.) were observed when administered before the second postshift trial, but not before the first postshift trial (Flaherty & Rowan, 1989; Flaherty, Grigson, & Rowan, 1986). Ethanol and CDP have an additive effect on cSNC; thus, marginally effective doses of these two drugs become effective when administered together (Becker & Flaherty, 1983). Furthermore, the reduction of cSNC by ethanol treatment can be reversed by the coadministration of drugs that interfere with the γ-aminobutyric acid (GABA) receptor, including the GABA antagonist picrotoxin (Becker & Anton, 1990), and the partial inverse agonist Ro 15-4513 (Becker & Hale, 1991). Other benzodiazepine anxiolytics have effects analogous to those of CDP (e.g., midazolam, 2 mg/kg, i.p.; Becker, 1986). The absence of a CDP effect on the first postshift trial appears to be related to insufficient experience with the downshifted solution. Thus, both CDP and diazepam do show an attenuat-
ing effect during the first downshift trial if the duration of this trial is extended beyond the typical 5 min (4 mg/kg, i.p.; Mustaca, Bentosella, & Papini, 2000). Interestingly, diazepam (30 μg/kg) infused into the amygdala, but not the hippocampus, also attenuates cSNC in rats (Liao & Chuang, 2003), providing one clue as to a possible site of action.

A second source of evidence is provided by opioids. Of particular interest here are the actions of opioids that selectively target the δ receptor subsystem. For example, administration of the selective agonist DPDPE (24 μg/kg, i.p.) before the first postshift trial, but not before the second, significantly attenuates cSNC (Wood, Daniel, & Papini, 2005). Furthermore, the selective antagonist naltrindole (1 mg/kg, i.p.) enhances cSNC when administered before the first postshift trial, but has no effect when administered before the second postshift trial (Pellegrini, Wood, Daniel, & Papini, 2005). Nonselective opioid receptor agonists (morphine) and antagonists (naloxone) affect cSNC whether they are administered before the first or second postshift trial (Pellegrini et al., 2005; Rowan & Flaherty, 1987). Furthermore, exposure to incentive downshift induces hypoalgesia in the hot plate when the latter is tested immediately after the second postshift trial, but has no effect on hot plate latencies when tested after the first postshift trial (Mustaca & Papini, 2005). Because the hot plate test measures pain sensitivity, this result suggests the selective activation of another opioid subsystem (μ receptors?) during the second postshift trial. Thus, different opioid receptor subsystems appear to be involved in the initial response to incentive downshift and the onset of the recovery process.

A third source of evidence is provided by the activation of the hypothalamic-pituitary-adrenal (HPA) axis. Plasma levels of the pituitary hormone ACTH and of the adrenal hormone corticosterone are elevated during the initial period of extinction (Davis, Memmott, MacFadden, & Levine, 1976; Romero, Levine, & Sapolsky, 1995). Similarly, the enhancement of extinction performance, referred to variously as the extinction burst or spike, is eliminated in adrenalectomized rats (Thomas & Papini, 2001). Both ACTH and corticosterone are generally considered to be markers of stress, but this term is usually vaguely defined. The dynamics of corticosterone in the cSNC situation provides an interesting insight because plasma levels are elevated during the second postshift trial, but not during the first trial (Flaherty, Becker, & Pohorecky, 1985). Given that consummatory behavior is typically suppressed in both trials, this differential effect suggests that what causes stress in the cSNC case is the emergence of an approach-avoidance conflict, not incentive downshift per se. Interestingly, corticosterone is also elevated before the start of the second postshift trial (Mitchell & Flaherty, 1998). Although the reasons for this elevation are unclear, one possibility is that stimuli associated with the initiation of the trial acquire the ability to induce an anticipation of the impending conflict situation that elevates corticosterone levels before the second postshift trial. This hypothesis assumes that the elements of the conflict are acquired during the first postshift trial, although the conflict itself may not be fully expressed at that time. This is consistent with the results of posttrial corticosterone administration. In one experiment (Bentosela, Ruetti, Muzio, Mustaca, & Papini, in press), corticosterone (3 mg/kg, s.c.) administered immediately after the first postshift trial significantly enhanced suppression during the subsequent four postshift trials. This enhancement was eliminated when corticosterone was administered 3 h after the first postshift trial, rather than immediately afterward, suggesting that the aversive learning taking place during this trial tends to decay in time.

Egocentric vs. Allocentric Learning

The mammalian learning effects emphasized thus far (SNC, MREE, and PREE) suggest that two learning mechanisms are at play in situations involving surprising changes in incentive conditions. This can be illustrated most clearly by the extinction effects. In the PREE, for example (see Figure 2b), a transition
to extinction is followed by a decrement in behavior to a new asymptote, as well as by differential rates of decrement. The asymptotic effect reflects the sensitivity of the learning mechanism to changes in incentive parameters that occur in the learning environment. Tracking such changes is one of the main functions of the conditioning process. Because these changes occur in the environment, these are said to be allocentric mechanisms, that is, processes tuned to capture environmental regularities. Thorndike’s S-W mechanism and Tolman’s S→S associations accomplish this task in a simple manner, by adjusting the strength of associative connections. Allocentric learning can thus be characterized by the strengthening and weakening of stimulus value dependent on the extent to which the stimulus is paired with any kind of consequent event (e.g., an incentive). In less technical terms, allocentric learning may be said to encode knowledge of the causal structure of events in the animal’s environment and, as a result, it may be described as a cognitive process.

The types of environmental change that can be tracked by the conditioning mechanism are limited by the sensory-perceptual capacity at any given time. If events are perceived, at least some animals will be capable of learning about them, as shown by such phenomena as higher-order conditioning (Macphail, 1982; Rescorla, 1980). However, some of these changes are trivial while others are significant. Significant changes tend to trigger an emotional evaluation with hedonic content. For the SNC situation, and following Amsel’s (1992) frustration theory, this idea has been phrased in terms of primary frustration (see Figure 3a), an emotional internal state induced when appetitive expectations have a higher incentive value than the actual events. In a fear conditioning situation, the presentation of an electric shock induces an internal state of physical pain that also has a significant hedonic component (Price, 2000). A second function of the conditioning process is to provide a mechanism for anticipating these significant emotional states. This type of learning is called egocentric learning because it reflects learning about the organism’s own emotional reaction to environmental change. In the PREE example, allocentric learning is responsible for the asymptotic effect of the extinction procedure (i.e., the ultimate decrease in behavior), whereas egocentric learning determines the differential rates of extinction (i.e., the speed of behavioral change).

The allocentric-egocentric dimension of learning was first suggested by comparative data of the type shown in Figure 4 (Papini, 2003). Species that exhibit a reversed SNC effect nonetheless show a proper discrimination of incentive levels (as demonstrated by preshift performance) and the ability to track changes in incentive magnitude (as demonstrated by the adjustment of behavior after incentive downshift). Thus, these species may be said to exhibit good evidence of allocentric learning, but no evidence of egocentric learning. A similar conclusion may be applied to the performance of infant rats in situations involving changes in incentive parameters (Amsel, 1992; Daly, 1991). However, it would be inaccurate to conclude that nonmammalian vertebrates show no evidence of egocentric learning in general. As mentioned above, fear conditioning provides a second type of egocentric learning, one that allows an organism to anticipate its emotional reaction to painful events and coordinate defensive responses. Unlike secondary frustration, fear seems to be quite general among vertebrates. For example, recent experiments demonstrate active avoidance learning in goldfish trained under spaced-trial conditions that eliminate the influence of carry-over effects and parallel the procedures used in experiments with appetitive incentives. In one experiment using a master-yoked design, goldfish controlling shock delivery by swimming over a barrier during the presentation of a warning stimulus exhibited better performance than yoked animals exposed to the same number and temporal distribution of shock independently of their behavior (Portavella, Salas, Vargas, & Papini, 2003). Moreover, this type of avoidance learning was shown to depend on the medial pallium, an area of the fish telencephalon that contains
structures homologous to the mammalian amygdala (Portavella, Torres, Salas, & Papini, 2004). Similarly, pigeons trained to peck at a key for food show suppression of key-pecking performance when it is punished with electric shock (Azrin, 1959). Such punished responding is alleviated by treatment with chlordiazepoxide and other benzodiazepine anxiolytics that enhance GABAergic synapses (Mansbach, Harrod, Hoffmann, Nader, Lei, Witkin, & Barrett, 1988). Thus, both goldfish and pigeons, neither one exhibiting SNC, share with mammals some of the components of the brain circuit responsible for fear conditioning, showing homologies at the neurobiological level (amygdala in goldfish) and neurochemical level (GABAergic system in pigeons). A more complete picture of these potential vertebrate homologies remains to be determined, but they suggest the working hypothesis concerning the evolution of egocentric learning mechanisms depicted in Figure 6.

An intriguing possibility (Papini, 2003) is that secondary frustration, the egocentric learning responsible for SNC, is supported by a set of mechanisms that evolved in early mammals or mammalian ancestors from the circuit responsible for fear conditioning. Knowledge about the genetic basis of both fear conditioning and SNC will be required to determine, for example, the extent of genetic similarities between these two phenomena (e.g., Stork, Stork, Pape, & Obata, 2001). Extensive genetic similarities would suggest a process of gene duplication analogous to that postulated to explain the evolution of a quadruplicate set of Hox genes in vertebrates, feathers in birds, and the eye’s lenses of vertebrates (Holland & Garcia-Fernandez, 1996; Raff, 1996). Gene duplication could help understand the extensive similarities between brain systems devoted to the control of physical pain, as that induced by peripheral electric shock, and psychological pain, as that induced by surprising nonreward or social exclusion (Eisenberger & Lieberman, 2004; Gray, 1987; Papini, Wood, Daniel, & Norris, in press). A good example of this similarity is provided by the role of the opioid system in cSNC reviewed above.

**Conclusions**

Although a phylogenetic approach to the evolution of egocentric learning was taken in this paper, the questions of adaptive value may still be raised. Whereas the protective value of fear conditioning suggests clear ties with fitness, what could be the adaptive value of secondary frustration? The mechanisms underlying secondary frustration, like all other phenotypic characters, have presumably evolved by a combination of ecological
pressures and existing preadaptations that could be co-opted into new functions (Papini, 2003; Stout, Muzio, Boughner, & Papini, 2002). A consequence of both primary and secondary frustration is that these are aversive states that tend to drive the rat away from the site where they have experienced a surprising reward omission. Thus, the adaptive value of these emotional states may reside in their ability to promote rapid change in both learning and performance, a process that fits Klinger’s (1975) notion of incentive disengagement. Frustration may be conceptualized as an internal state that facilitates a switch from responses that no longer yield incentives to a search mode that may result in the discovery of needed resources. If reinforcement is thought of as inducing a kind of attachment with the site or context in which it occurs, then frustration may be seen as an efficient way to break that attachment. Incentive disengagement may be particularly relevant to mammals given their high activity levels and metabolic rates.

Klinger (1975) also pointed out that incentive disengagement may help explain symptoms of depression induced by social loss. It is likely that Mesozoic mammals were mainly solitary, as are extant conservative species like the egg-laying monotremes, didelphid marsupials, and insectivores (Eisenberg, 1981). Thus, the evolution of mechanisms responsible for secondary frustration may have occurred in the context of feeding and foraging in largely solitary mammals (or their cynodont ancestors; Papini, 2002b). The evolution of mammalian sociality in several lineages creates new objects for attachment and their loss can have serious implications for fitness (MacDonald & Leary, 2005). The words “grief” and “sadness” are used in common language in reference to the emotional state triggered by social loss, as it happens when animals that have developed an attachment are separated. There are anecdotal reports of grief in chimpanzees (Goodall, 1986), but also extensive research on the behavioral and physiological effects of mother-infant separation in mammals (e.g., Levine, 2005). These studies show impressive parallels with the effects of surprising reward loss studied in situations involving food reinforcement (Papini & Dudley, 1997). It is tempting to speculate that grief and other emotions triggered by social loss share with frustration a similar incentive disengagement function, that is, to promote a switch to a search mode that may lead to the establishment of a new social bond. This hypothesis, tentatively represented in Figure 6, suggests deep links between three of the most fundamental aversive emotions that influence mammalian behavior.

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**Author's Note**

This article is based on the key-note address entitled “Reward Loss as Psychological Pain: Role of the Opioid System,” given by the author at the 65th Annual Meeting of the Japanese Society for Animal Psychology, Chiba, 2005.

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(2006. 1.14 受稿, 2006. 2.20 受理)