Brain, Behavior and Evolution

Brain Behav Evol 2003;62:83–95
DOI: 10.1159/000072439

Comparative Psychology of Surprising Nonreward

Mauricio R. Papini

Texas Christian University, Fort Worth, Tex., USA

Key Words
Allocentric learning · Egocentric learning · Frustration · PREE · SNC · Fear · Emotion

Abstract
The surprising or unexpected omission of an appetitive reinforcer has at least two effects: An allocentric effect according to which the organism updates knowledge about the environment, and an egocentric effect that allows the organism to learn about its own emotional reaction to the change. This egocentric effect (traditionally called frustration) is correlated to activation of the hypothalamic-pituitary-adrenal axis, can be modulated by treatment with anxiolytics, and is expressed in terms of behavioral changes that have an emotional component (e.g., agonistic behavior). It is hypothesized that all vertebrates share the mechanisms underlying the allocentric effect, but only mammals possess the mechanisms underlying the egocentric effect. It is further argued that frustrative mechanisms evolved in early mammals from those underlying fear conditioning.

Introduction

In the essay *On Tranquility of Mind*, Seneca recommends to a friend not to trust the promises of Fortune: 'Say "I will set sail unless something happens," and "I shall become praetor unless something hinders me," and "My enterprise will be successful unless something interferes." This is why we say that nothing happens to a wise man contrary to his expectations' [Book IX, 13, 2–3]. Accepting the possibility that the people, activities, and objects one loves and enjoys can be lost, become inaccessible, or lose their value attenuates the aversive impact of the loss. Stoics recommended starting the day by adjusting our expectations so as to cope with the uncertainties of life, an exercise called praemeditatio. Modern research shows that Seneca was right on target. Unless we are prepared for it, reward loss can have devastating effects.

Research on stressful life events indicates that many such events involve reward loss. In a widely used checklist, the Social Readjustment Rating Scale [Scully et al., 2000], death of a spouse, divorce, separation, jail term, death of a close family member, losing a job, and retirement, all among the top ten most stressful life events, involve some variety of reward loss. In addition, clinical research shows that separation from or loss of a loved one

KARGER

Fax + 41 61 306 12 34
E-Mail karger@karger.ch
www.karger.com

0006–8977/03/0622–0083$19.50/0

Accessible online at:
www.karger.com/bbe

Mauricio R. Papini
Department of Psychology
Texas Christian University, Box 298920
Fort Worth, TX 76129 (USA)
Tel. +1 817 257 6084, Fax +1 817 257 7681, E-Mail m.papini@tcu.edu

Table 1. Core postulates of Amsel's [1992] frustration theory

Phenomenon	Mechanism	Requisite	Example
Aftereffect	Primary frustration	Negative discrepancy between expectancy and current reward value	ROE
Anticipatory effect	Secondary frustration	Approach-avoidance conflict	SNC
Persistence	Counterconditioning	Moderate-to-extensive exposure to uncertain reward conditions	PREE

ROE = Reinforcement-omission effect (see text and Stout et al. [2002] for details). SNC: Successive negative contrast (see text and Mustaca et al. [2000] for details). PREE: Partial reinforcement extinction effect (see text and Thomas and Papini [2003] for details).

to death are often followed by affective disorders, disruption of autonomic function, changes in appetite, disruption of sleep patterns, general health deterioration, suppression of immune activity, and increased mortality [Bartrop et al., 1977; Schleifer et al., 1983; Stein and Trestman, 1990; Rando, 1993; Hall and Irwin, 2001]. From an evolutionary perspective, these data suggest that reward loss can have a negative impact on individual fitness [Archer, 2001]. However, clinical research cannot identify causes, but only point to correlations between various factors. For example, bereavement-related stress and depression are positively correlated with impairment of immune function, but these may be 'proxy measures for some other causal factor such as diet or other health behavior,' or, 'conversely, it might also be argued that immune system changes cause symptoms of depression and stress' [Hall and Irwin, 2001, p. 482]. Animal models that permit careful manipulation of reward loss parameters can contribute significantly to our understanding of the interconnection of these factors, but only if the underlying mechanisms are homologous. Consider, as an example, the similarity between human grief and mother-infant separation in other mammalian species. The mother of a newborn mammal provides rewards in the form of milk, warmth, contact comfort, and familiar olfactory cues. Separation of the infant mammal from its rewarding mother is followed by distress vocalizations, reduced social interactions, increased glucocorticoid levels, and reduced immune activity [e.g., Jordan et al., 1984; Coe et al., 1985; Reite and Capitanio, 1985; Suomi, 1991; Kling et al., 1992; Bailey and Coe, 1999]. These findings are clearly related to the results of clinical research mentioned previously, suggesting homology of mechanisms.

Stressful situations involving reward loss, from a person who loses a job to an infant monkey separated from its mother, share one feature: Expectancies are violated. As an animal learns to expect the occurrence of an appetitive event in a given situation, the violation of such expectancy by reward omission induces, first, new learning about a change in the environment (typically studied in the laboratory under the label of extinction), and, second, new learning about the organism's own emotional reaction to the environmental change (traditionally termed frustration) [Papini and Dudley, 1997]. Both of these effects require that reward loss is surprising or unexpected. In this paper I argue that the mechanisms underlying frustration are unique to mammals.

Adjustment to Surprising Nonreward in Mammals

The terminology used in the rest of the paper is derived from Amsel's [1992] frustration theory, developed explicitly to account for the behavioral effects of surprising nonreward (SN) in experimental situations. The term nonreward refers to the omission, reduction in magnitude, or quality degradation of an appetitive reinforcer (e.g., food, water). Nonreward is surprising if it occurs in the presence of signals previously paired with a reward of greater incentive value than the current one. The core of frustration theory involves three theoretical postulates (see table 1) [Amsel, 1992]. First, SN induces an internal state, called primary frustration, which has immediate consequences for the behavior of the organism, acting as an aversive reinforcer. The observable consequences of pri-

Table 2. Aftereffects of surprising nonreward reviewed in Papini and Dudley [1997]

Effect	Reward manipulation	Species
Corticosteroid release	Ext, iSNC, cSNC, MIS	Rats, monkeys
Decreased heart rate	Ext	Rats
Increased blood pressure	Barrier task	Humans
Frustration odors	Ext, iSNC, PR	Rats, wood rats, gerbils
Ultrasonic vocalizations	Ext	Rats
Crying, avoidance behavior	Ext, iSNC, barrier task	Humans
Increased locomotor activity	Ext, cSNC, Pavlovian	Rats
Increase in aggressive behavior	Ext, PR	Rats, pigs, humans
Scheduled-induced polydipsia	FT, iSNC	Rats
Increased eating	Sexual frustration	Rats
Suppression of drinking	cSNC	Opossums, rats, humans
Reinforcement-omission effect	Ext, PR, Pavlovian	Rats, opossums, humans
Escape from frustration	A+/B–, Ext, iSNC	Rats

See Papini and Dudley [1997] for relevant references and further details. A+/B–: Discrimination training. Barrier task: A previously rewarded response is prevented from occurring. Ext: Extinction. FT: Fixed-time schedule. cSNC: Consummatory successive negative contrast effect. iSNC: Instrumental successive negative contrast effect. MIS: Mother-infant separation. PR: Partial reinforcement. Pavlovian: Classical conditioning training.

mary frustration are called aftereffects. Second, the pairing of stimuli with primary frustration endows the stimuli with the ability to trigger an expectation of such aversive reinforcer, called secondary frustration. These conditioned or anticipatory effects result in interference with approach to the goal object. Third, the pairing of secondary frustration with reward results in the development of tolerance to the disrupting effects of SN through a process called counterconditioning. Counterconditioning implies a hedonic shift in the value of secondary frustration from aversive to appetitive that increases behavioral persistence in the face of reward loss. Each of these three postulates has received extensive empirical support from experiments involving mammalian species, although there are several unresolved issues. The next three sections provide a selective review of the evidence; more extensive reviews that include discussions of unresolved issues may be found in Macphail [1982], Amsel [1992], Flaherty [1996], Papini and Dudley [1997], and Bitterman [2000].

Aftereffects

'What is unexpected we count undeserved. And so we are mightily stirred by all that happens contrary to hope and expectation,' wrote Seneca [*On Anger*, Book II, 31, 1]. In modern terminology, the aftereffects of SN often involve emotional activation that has both behavioral and physiological correlates. Papini and Dudley [1997] reviewed the literature on aftereffects (summarized in table 2) and concluded that it supports the first theoretical postulate mentioned previously, namely, that the consequences of SN are hedonically aversive. Three lines of evidence suggest such a conclusion. First, SN promotes the acquisition of responses that allow the animal to escape from the site [Daly, 1974]. Second, SN triggers changes in agonistic behavior in social interactions [Tondat and Daly, 1972; Mustaca et al., 2000]. Third, SN leads to the invigoration of dominant responses [Amsel and Roussel, 1952; Dudley and Papini, 1995, 1997; Stout et al., in press], an effect that is vulnerable to amygdalectomy [Henke, 1977].

Anticipatory Effects

Seneca argued for a fundamental connection between expectations and the 'tranquility of mind.' When an animal expects an incentive of greater value than that of the incentive actually received, an aversive state of primary frustration is induced. The same stimuli that previously predicted a highly valued reward are now paired with primary frustration. As a result, such cues become ambiguous, activating opposing expectations of reward and frustration, and inducing an approach-avoidance conflict. During extinction of runway performance, rats exhibit a

pattern of behavioral hesitation as approach to, and avoidance of, the goal compete for expression [Jones, 1970]. The effects of secondary frustration require some minimum amount of experience with the new, impoverished incentive conditions. Extensive reviews of this topic are available [Amsel, 1992; Flaherty, 1996]. What follows is a selective description of the critical evidence supporting the second theoretical postulate, namely, that SN results in the conditioning of an aversive state of secondary frustration that interferes with previously acquired goal-approach tendencies.

Instrumental extinction is followed by glucocorticoid release in several species [Davis et al., 1976; Dantzer et al., 1980; Carbonaro et al., 1992; Lyons et al., 2000]. Relative to presession levels, corticosterone is elevated within 5 min of extinction onset and further increased 20 min into the first extinction session [Coe et al., 1983]. During the initial trials, extinction often facilitates behavior, an effect that is eliminated by adrenalectomy [Thomas and Papini, 2001]. Corticosterone is also elevated in a consummatory training situation in which consumption of a low-value sucrose solution is depressed in animals that had prior access to a high-value solution, relative to unshifted, low-value controls. This suppression of consumption, called consummatory successive negative contrast (cSNC), has been described in marsupials, rodents, and human infants [Vogel et al., 1968; Kobre and Lipsitt, 1972; Papini et al., 1988; Mustaca et al., 2000]. With 5-min-long sessions, plasma corticosterone levels are elevated on the second postshift session, but not on the first one [Flaherty et al., 1985; Mitchell and Flaherty, 1998].

Similarly, benzodiazepine anxiolytics (e.g., chlordiazepoxide, diazepam) typically reduce cSNC when administered on the second postshift session, but not on the first one [Flaherty et al., 1986]. Interestingly, repeated downshifts in sucrose concentration increase the size of cSNC on the first postshift session [e.g., Papini et al., 1988], suggesting that secondary frustration can be aroused in this first postshift session provided the animal has had previous downshift experience. Concomitantly, anxiolytics reduce contrast on the first postshift session after the animal has experienced several such downshifts [Flaherty et al., 1996], or when the first postshift session lasts more than 5 min [Flaherty et al., 1986; Mustaca et al., 2000].

One problem with the cSNC effect is that aftereffects and anticipatory effects cannot be clearly dissociated. However, the instrumental version of this effect (iSNC) allows for a clean assessment of anticipatory effects, especially if training involves one trial per day. Under such spaced-trial conditions, stimulus and memory traces from previous trials are minimized or eliminated, and the instrumental response is influenced predominantly or exclusively by current stimuli. iSNC occurs in rats exposed to a surprising shift in reward quality or magnitude [Elliott, 1928; Crespi, 1942]. In either case, the behavior of downshifted animals deteriorates after the first postshift trial, relative to the behavior of unshifted controls. A similar effect can be obtained if groups trained with either large or small reward magnitudes are shifted to extinction. Extinction is faster after acquisition training with large rewards than after small-reward training [Hulse, 1958], an effect known as the magnitude of reinforcement extinction effect (MREE). Notice that both the iSNC and MREE occur before the animal arrives at the goal and therefore reflect conflicting anticipatory tendencies to approach and avoid the goal site. iSNC is also disrupted by the chronic administration of the anxiolytic chlordiazepoxide [Rosen and Tessell, 1970], possibly because anxiolytics reduce approach-avoidance conflicts.

Behavioral Persistence

'Unimpaired prosperity cannot withstand a single blow; but he who has struggled constantly with his ills becomes hardened through suffering, and yields to no misfortune,' says Seneca [*On Providence*, Book I, 2, 6], in a statement that seems to foresee the partial reinforcement extinction effect (PREE), one of the most interesting and well-studied effects in the animal learning literature. The PREE is defined as greater resistance to extinction after acquisition under partial reinforcement than under continuous reinforcement. In fact, it is not just partial reinforcement, but any sort of reward inconsistency (e.g., large vs. small rewards, immediate vs. delayed rewards) that increases persistence in extinction [Amsel, 1992]. Several theories have been proposed to account for the PREE and, as will be shown later, it is possible that similar behavioral effects obtained under similar conditions are based upon different mechanisms. However, in mammals trained under widely-spaced practice conditions, the nature of the PREE is consistent with the third theoretical postulate of frustration theory mentioned previously, namely, that the counterconditioning of secondary frustration promotes goal approach [Amsel, 1992]. The following is a selected review of evidence.

There are several ways in which the frustrative consequences of SN can be reduced during acquisition so as to disrupt the PREE, including treatment with anxiolytics. A drug such as chlordiazepoxide should reduce the intensity

of the conflict induced by partial reinforcement training and thus disrupt counterconditioning. This should, in turn, reduce persistence in extinction after partial reinforcement training, thus eliminating the PREE. Such an outcome was reported in both runway and Skinner box situations [Iwahara et al., 1967; Feldon and Gray, 1981; McNaughton, 1984]. Chlordiazepoxide administered during both acquisition and extinction also increases extinction persistence in the continuously rewarded animals. Thus, chronic injections eliminate the PREE by both decreasing persistence in partial animals and increasing persistence in continuous animals [Feldon and Gray, 1981]. If partial reinforcement training leads to counterconditioning, then it should also attenuate the SNC effect, an effect that occurs in both the instrumental and consummatory situations [Mikulka et al., 1967; Pellegrini and Papini, 2002]. Furthermore, the effect of partial reinforcement on cSNC can be eliminated by the administration of chlordiazepoxide before the nonreinforced sessions during acquisition training [Pellegrini and Papini, 2002]. In this experiment, rats had access either to a 32% sucrose solution (reinforced) or to water (nonreinforced), in a random sequence, during 5-min long daily sessions, before being shifted to a 4% solution. This result is important because it eliminates the alternative account that the attenuation of contrast by partial reinforcement is caused by incentive averaging (i.e., partial reinforcement involves less reinforcement on average than continuous reinforcement) [see Flaherty, 1996].

The theoretical distinction between primary and secondary frustration is supported by evidence suggesting that different factors modulate these effects. In the double-runway situation, it is possible to administer partial or continuous reinforcement training in the first runway to different groups, followed by extinction in the first runway (all animals receive continuous reinforcement training in the second runway throughout the experiment). Using such a design, treatment with sodium amobarbital, a barbiturate with anxiolytic effects, eliminates the PREE observed in the first runway during extinction, but does not affect response facilitation (i.e., the reinforcement-omission effect, ROE, see table 1) observed in the second runway during partial reinforcement training [Gray and Dudderidge, 1971]. In addition, amygdalar lesions eliminate the ROE, but not the PREE [Henke, 1973; Henke and Maxwell, 1973], whereas septo-hippocampal lesions eliminate the PREE, but not the ROE [Swanson and Isaacson, 1967; Henke, 1973; Feldon et al., 1985; Lobaugh et al., 1985].

Surprising Nonreward in Fish, Amphibians, and Reptiles

As mentioned in the introduction, SN may induce new learning either about a change in the environment, or about the organism's own emotional reaction to that change. The first may be called an allocentric effect in the sense that the organism simply updates knowledge about the environment, whereas the second may be called an egocentric effect because the organism learns something about itself. The simplest allocentric rule for updating associative knowledge after SN is part of both classical and contemporary learning models [Thorndike, 1911; Hull, 1943; Rescorla and Wagner, 1972; Couvillon and Bitterman, 1985; Schmajuk, 1997], and it simply maintains that signal value is strengthened by reinforcement and weakened by nonreinforcement. To understand the significance of this strengthening-weakening rule, consider again the PREE. During extinction, approach behavior decreases faster in the continuous animals than in the partial animals until it is finally eliminated in both groups. According to the account of the PREE described previously, the first effect (differential extinction rates) is egocentrical and reflects increased persistence due to the counterconditioning of secondary frustration. However, the fact that approach responses are finally eliminated in both groups suggests that signal value has been updated according to the new environmental conditions (i.e., an allocentric effect).

Available evidence from spaced-trial experiments with osteichthyes fish, amphibians, and reptiles indicates that only the allocentric effects of SN are present in these animals [see Papini et al., 1995; Bitterman, 2000]. Thus, for example, a shift from a large to a small reward magnitude is followed by an adjustment of performance without deterioration, that is, without SNC. Similarly, extinction is slower after training with continuous reinforcement, than with partial reinforcement (i.e., a reversed PREE), and slower after training with large, rather than small, rewards (i.e., a reversed MREE). These results reflect the action of a simple strengthening-weakening rule. For example, relative to 100% reinforcement, 50% partial reinforcement should weaken signal value and thus result in faster extinction; similarly, a shift from a larger to a smaller reward magnitude should weaken signal value leading to an adjustment of performance without contrast.

In rats, the PREE is known to be disrupted by hippocampal lesions [Rawlins et al., 1980]. Such a disruption follows a pattern analogous to that observed with chlordiazepoxide treatment, that is, hippocampectomized rats

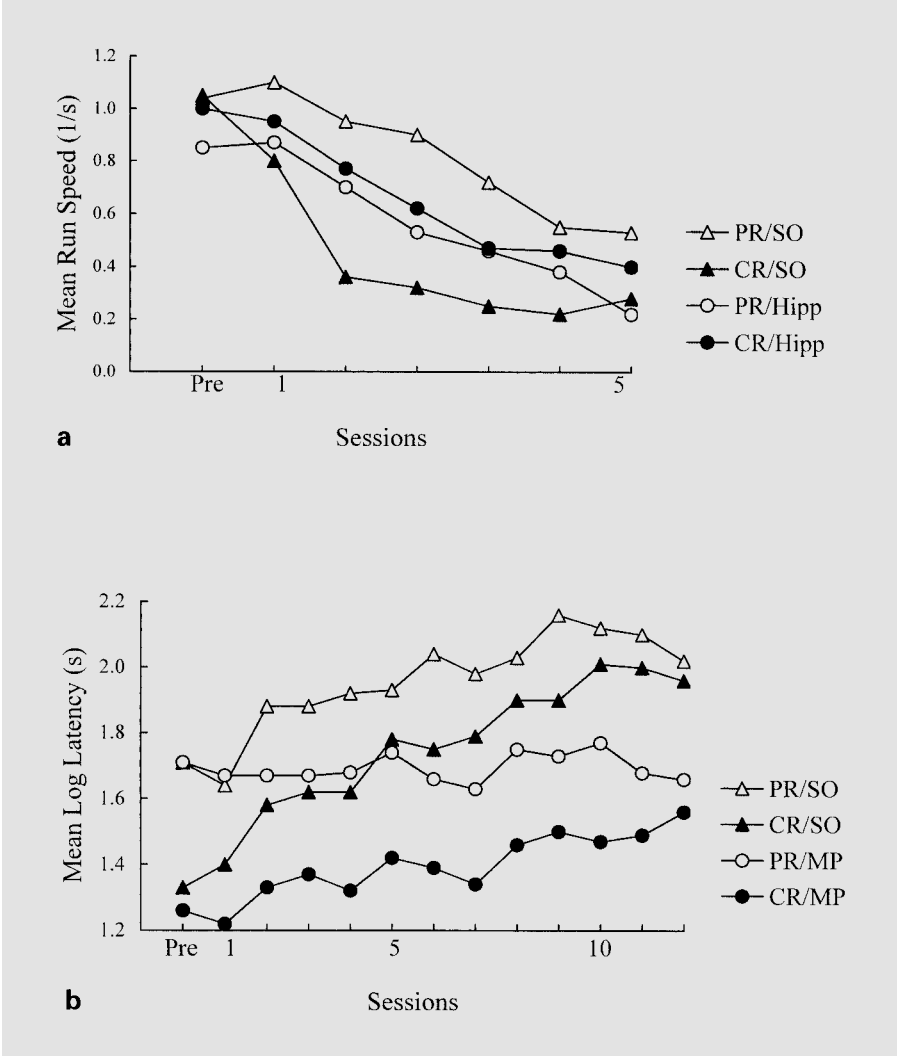

Fig. 1. a Terminal acquisition (Pre) and extinction performanc of rats with either hippocampal (Hipp) or sham (SO) lesions, and after training with either partial (PR) or continuous reinforcement (CR). Cortical controls are omitted for clarity. [From Rawlins et al., 1980, Exp Brain Res, copyright Springer-Verlag, reproduced by permission.] **b** Terminal acquisition and extinction performance of toads with either lesions in the medial pallium (MP) or sham operations, and after training with either partial or continuous reinforcement. [From Muzio et al., 1994, Behav Neural Biol, copyright Academic Press, reproduced by permission.] In both experiments, animals were trained in a runway, received one trial per day, and were reinforced with food (rats) or water (toads).

show less persistence in extinction after partial reinforcement, but more persistence after continuous reinforcement training (fig. 1a). Consistent with frustration theory's account of the PREE, such opposite effects of the same lesion suggest that the associative structure underlying learning under these two schedules is different (i.e., counterconditioning occurs only in the partially reinforced animals). In contrast with this pattern, and as shown in figure 1b, lesions of the amphibian medial pallium, a structure thought to be homologous to the mammalian hippocampal formation [Northcutt and Ronan, 1992], increase extinction persistence after both partial and continuous reinforcement [Muzio et al., 1993, 1994]. This result is consistent with a strengthening-weakening model according to which partial and continuous reinforcement schedules produce changes along a single dimension of signal value.

An Evolutionary Framework

The data reviewed so far suggest a working hypothesis that could be phrased as follows. First, all vertebrates share a basic strengthening-weakening, allocentric rule for associative learning that updates signal value as a function of environmental changes. Second, in addition, mammals evolved an egocentric mechanism that allows for learning about the organism's own frustrative reaction to environmental changes. This egocentric mechanism is vulnerable to anxiolytic treatment and to lesions in some

limbic structures. One way to think about the evolution of the egocentric mechanism in some putative mammalian ancestor is in terms of the addition of a new developmental stage in brain differentiation. In developmental evolutionary biology, changes in the sequence of developmental stages in descendant species, relative to their ancestors, are studied under the term heterochrony [see Papini, 2002a]. In one specific class of heterochronic phenomena, known as peramorphosis, new traits evolve in descendants, relative to their ancestors, as a result of either a shift in the onset of development of the trait to an earlier stage, an acceleration of the trait's developmental rate, or a general delay in sexual maturity. For example, selection for large body size may generate some developmental room for the evolution of novel structures. The horns and antlers of large hervibore mammals have presumably evolved by peramorphosis. Going back to the case of anticipatory frustration, evidence consistent with the view that its underlying mechanisms evolved by developmental addition comes from research with infant rats [Amsel, 1992]. Before postnatal day 12, infants are capable of learning but show only allocentric effects when exposed to SN situations. However, the PREE, MREE, and SNC emerge gradually between postnatal days 12 and 24, in correlation with the maturation of the hippocampal formation.

An interesting implication of the hypothesis that the egocentric mechanism evolved in early mammals relates to the status of SN phenomena in birds. Birds and mammals evolved from diapsid and synapsid early reptiles, respectively. These groups appear already differentiated in fossils from the upper Carboniferous, about 360 million years ago [Benton, 1990]. Interestingly, birds and mammals achieved a striking degree of convergence. Relative to reptiles, birds and mammals exhibit larger relative brain size, high metabolic rates, high activity levels, and relatively complex parental care behavior, among other features [Papini, 2002a]. One may, therefore, expect to find similar learning phenomena, but based upon different underlying mechanisms. For comparative purposes, learning mechanisms may be defined as processes hypothesized to explain learning phenomena (e.g., PREE, SNC). Comparative psychologists have traditionally framed such hypothetical processes in such terms as primary and secondary frustration, as I have used them previously. But these psychological terms must ultimately correlate with underlying physiological processes at, at least, three levels of analysis. From the highest to the lowest, these levels are the neurobiological (e.g., neural networks in specific brain areas), neurochemical (e.g., synaptic plasticity), and cell-molecular (e.g., second-messenger systems). If a given learning phenomenon, say the PREE, is present in two species and it is shown to respond in the same manner to a set of manipulations at all three levels of analysis, then this would constitute evidence of homology of mechanisms. However, if a similar learning phenomenon occurs in two species but responds differently to the same treatment at any one of these three levels, then the phenomenon could be considered homoplasic [Papini, 2002b]. The effect of SN on the behavior of birds provides a preliminary illustration of this framework.

Until recently, only one experiment had been published on the effects of SN under spaced training in an avian species, the pigeon *(Columba livia)*, and it provided clear evidence of the PREE [Roberts et al., 1963]. Since the spaced-trial PREE, SNC, and MREE were characterized by their covariation in any species in which they had been studied, it seemed appropriate to predict that pigeons would also exhibit the latter two effects. However, spaced-trial experiments revealed a dissociation of these effects in pigeons, with clear evidence for the PREE, but no hint of SNC and a reversed MREE [Papini, 1997; Papini and Thomas, 1997; Papini et al., 2002; Thomas and Papini, 2003]. One interpretation of this dissociation is that the pigeon PREE is based on mechanisms that are different from those causing the seemingly analogous mammalian effect [see Thomas and Papini, 2003, for additional interpretations].

There are conflicting results in experiments studying the aftereffects of nonreward in birds. For example, unexpected food omissions trigger aggressive behavior in pigeons and hens [Azrin et al., 1966; Terrace, 1971; Haskell et al., 2000; Rodenburg et al., 2002], as they do in mammals [see Papini and Dudley, 1997]. Moreover, pigeons trained in an A+/B– discrimination rapidly learn to peck at a key that has as its only consequence to turn off the B stimulus [Terrace, 1972], a result analogous to escape from frustration in rats [Daly, 1974]. In contrast, primary frustration appears to play no role in the ROE, or higher response level immediately after nonreward than after reward in a partial reinforcement situation. The ROE has been described in both rats and pigeons [e.g., Dudley and Papini, 1995; Papini and Hollinsworth, 1998], but a major problem of interpretation has been to determine whether response is facilitated after nonreward (as predicted by frustration theory), or depressed after reward (a postconsummatory effect). Analysis of the time course of the aftereffects of reward and nonreward shows that, unlike in rats, the pigeon ROE is entirely attributable to a postconsummatory effect [Stout et al., 2002]. Thus, whether the aftereffects described in avian species, in-

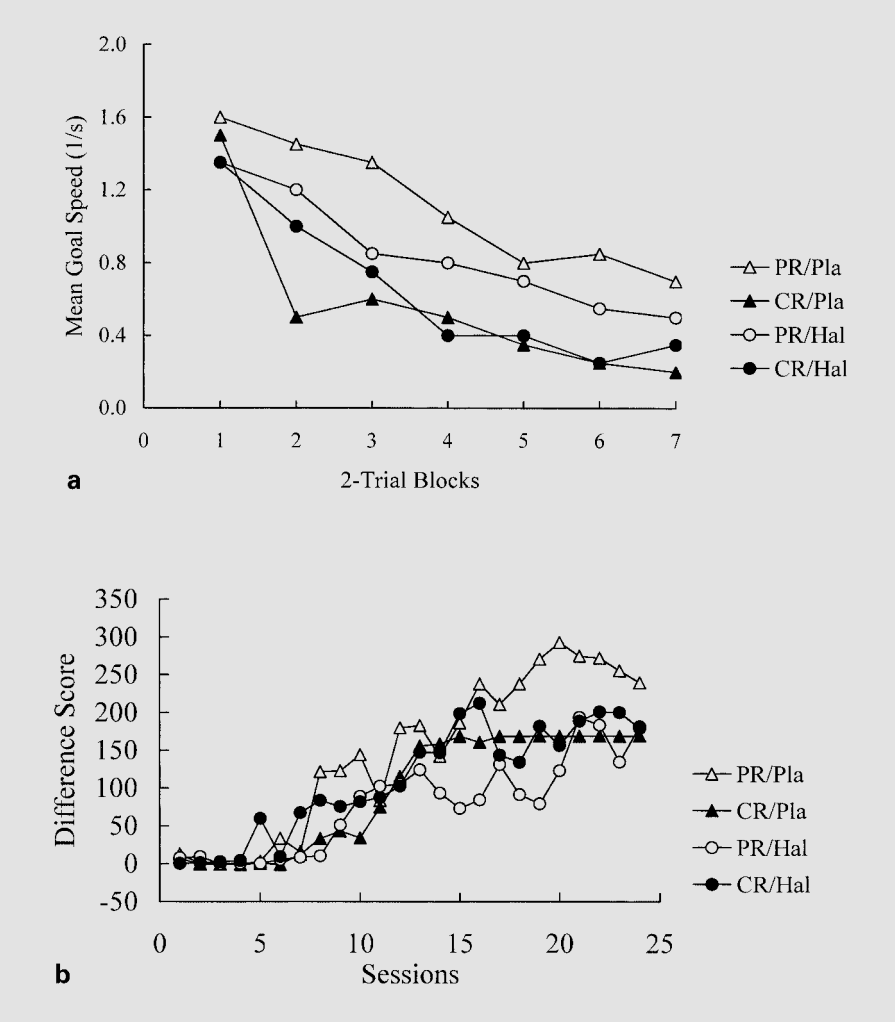

Fig. 2. a Extinction performance of rats treated with haloperidol (Hal; 0.1 mg/kg), or placebo (Pla), and after training with either partial (PR) or continuous reinforcement (CR). [From Feldon et al., 1988, Psychopharmacology, copyright Springer-Verlag, reproduced by permission.] **b** Extinction performance of pigeons treated with haloperidol (0.1 mg/kg) or placebo, after training under partial or continuous reinforcement. The difference scores plotted as a dependent variable are computed by subtracting the latency for each pigeon in each extinction trial from the latency obtained for that pigeon in the last acquisition trial. [From Thomas and Papini, 2003, Learn Motiv, copyright Academic Press, reproduced by permission.] In both experiments, animals were trained in a runway, received one trial per day, and were reinforced with solid food.

cluding extinction-induced aggression and escape from the B– stimulus of a discrimination, result from the engagement of the same mechanisms giving rise to analogous aftereffects described in mammals awaits a detailed comparative analysis. The case of the ROE suggests that similar behavioral phenomena may be based on different underlying mechanisms.

A more direct assessment of the mechanisms underlying adjustment to SN involves drug manipulations. Recent research on the PREE illustrates this approach [Thomas and Papini, 2003]. In rats, the PREE is known to be modulated by some drugs, but not by others. For example, chlordiazepoxide (a benzodiazepine anxiolytic) eliminates the PREE [e.g., McNaughton, 1984], whereas haloperidol (predominantly a dopamine antagonist) does not interfere with its development [Feldon et al., 1988; Feldon and Weiner, 1991]. Pigeons trained in a runway situation and receiving daily treatment with these drugs during both acquisition and extinction (similar to rats in the cited experiments), demonstrated a different pattern of results. Unlike the case with rats, chlordiazepoxide did not eliminate the PREE in pigeons, but haloperidol did. The rat and pigeon results for haloperidol are presented in figure 2.

Frustration, Pain, and Fear

'A strong emotion does not arise except a desire fails to attain its object [frustration], or an aversion falls into what it would avoid [pain-fear],' said Epictetus [Book III, 2, 3]. Contemporary psychologists recognize the parallel

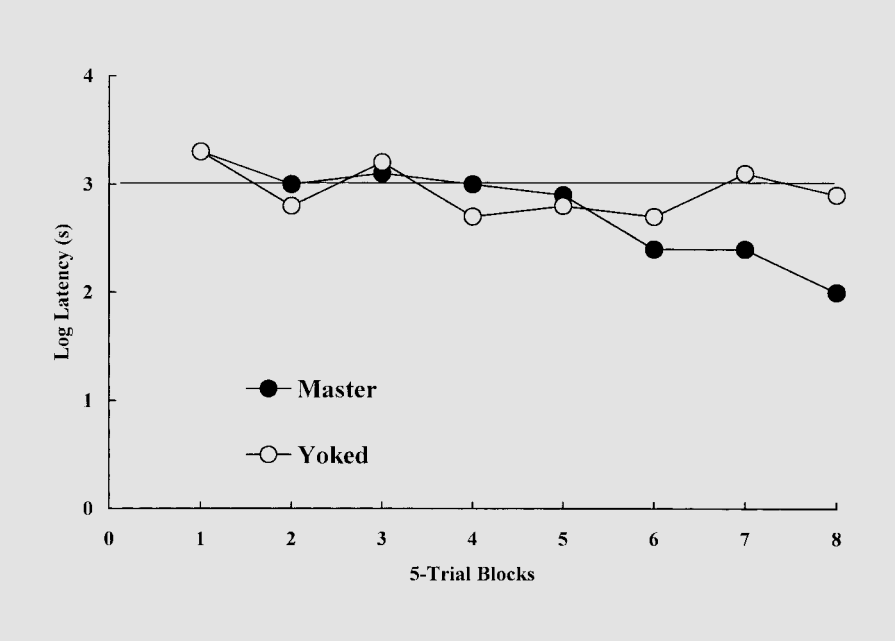

Fig. 3. Avoidance performance of two groups of goldfish receiving a single trial per day (intertrial interval: 24 h), in a master-yoked design. Master animals were trained to swim over a barrier during the presentation of a green light to avoid shock. In the absence of a response, the light and shock overlapped during 20 s. Latencies below 20 s (marked by a solid line) indicate successful avoidance behavior. Yoked goldfish were matched in terms of the amount and temporal distribution of shocks with master goldfish, but their responses had no scheduled consequences [from Portavella et al., in press].

functions played by the removal of an appetitive reinforcer and the administration of an aversive reinforcer on associative learning [Papini, 2002a]. Removing an appetitive reinforcer or administering an aversive reinforcer contingent on an instrumental response tends to suppress that behavior and results in the acquisition of an expectancy (secondary frustration and fear, respectively) that can be described as egocentric. Fear may be defined as an expectation of impending pain activated by a signal previously paired with an aversive event such as pain. In a sense, fear is to pain what secondary frustration is to primary frustration, an analogy captured by the so-called fear = frustration hypothesis [Gray and McNaughton, 2000]. This simple idea gives rise to at least two predictions related to the material covered in this paper, for which there is some supporting evidence.

The first prediction suggests behavioral phenomena based on pain and primary frustration should be modulated by a similar set of factors. Consistent with this idea is the evidence that opioid-receptor agonists, which play a key role in the reduction of pain, also reduce cSNC during the first session after a downshift in reward magnitude [Rowan and Flaherty, 1987; Castro, 2000]. First-session performance arguably reflects the effects of primary frustration on consummatory behavior. It would be interesting to take an individual-difference approach to this problem and determine whether, for example, there is a positive correlation between the size of first-session cSNC and some index of pain sensitivity (e.g., pain threshold). A study of individual differences has yielded evidence on the connection between fear conditioning and cSNC. Flaherty et al. [1998] exposed rats to four different behavioral tests, including fear conditioning and cSNC, and determined the extent to which performance in these situations was correlated. A factor analysis indicated that whereas the amount of freezing behavior during fear conditioning did not load on first-session consummatory performance, it related significantly with second-session performance. This result is consistent with the assumption that second-session consummatory performance reflects predominantly the effects of secondary frustration.

The second prediction of the fear = frustration hypothesis is that comparative research on fear and secondary frustration should show a similar species distribution. There is, however, some evidence suggesting that this may not be correct. For example, goldfish are capable of truly instrumental avoidance learning [e.g., Overmier and Papini, 1986], which, according to current learning theory, is based on the reduction of an internal response of fear induced by the warning signal [Gray and McNaughton, 2000]. In fact, goldfish given avoidance training at a rate of a single trial per day show the development of avoidance behavior when compared to yoked controls, as shown in figure 3 [Portavella et al., in press]. This result is

striking in view of the repeated failure to find evidence of spaced-trial PREE, MREE, and SNC in fish [Bitterman, 2000; Papini, 2002b], and it suggests that the mechanisms underlying fear conditioning may be phylogenetically older than those supporting secondary frustration.

Consider the implications of this hypothesis. It is commonly assumed that fear is related to self-preservation either in connection to predatory encounters or in intraspecific contests [Stamps, 1998]. The evolution of structures that correlate with an active mode of life in early chordates (e.g., tail and paired lateral muscles of Cambrian chordates such as *Pikaia*), and of structures that might have played an antipredatory role (e.g., the armors of Paleozoic agnathan and placoderm fish) suggest that predatory pressures were of considerable importance [Papini, 2002a]. As a result, the egocentric brain mechanisms responsible for fear may have been established in early chordates, a hypothesis consistent with the relative degree of conservation of fear-related learning phenomena in vertebrates. Unlike the case with fear, the selective pressures encouraging the evolution of the egocentric brain mechanisms underlying secondary frustration may not have been particularly strong in early chordates. One possible such pressure could relate to the energy requirements of animals with a relatively high metabolic rate, as might have been the case with Mesozoic mammals, relative to more conservative vertebrates. Following this argument, Stout et al. [2002, p. 255] suggested that the adaptive function of frustrative mechanisms is to facilitate a switch 'from previously successful responses that no longer work, to new responses than may bring the animal in contact with needed resources.' This may be akin to Klinger's [1975] notion of incentive disengagement. Mesozoic mammals may have possessed a set of characters that preadapted them to evolve disengagement mechanisms based on an emotional response. First, the relatively large olfactory bulbs of Mesozoic mammals suggest they were dependent on olfaction for foraging [Jerison, 1973; Kielan-Jaworowska, 1986]. Second, in extant mammals there are direct connections between the olfactory system and limbic structures involved in fear conditioning, including the amygdala. The size of the olfactory bulbs in extant insectivores (i.e., the most conservative placentals) correlates positively with the size of the centro-medial amygdala, but not with the size of nonlimbic structures such as the vestibular and cochlear nuclei [Barton and Harvey, 2000]. Furthermore, the amygdala is part of a network that participates both in fear conditioning [Blair et al., 2001] and in the adjustment to SN [Henke, 1977; Becker et al., 1984]. High metabolic rates are also likely to have been present in Mesozoic birds, but it is assumed that their predominantly visual foraging style preadapted them for a nonemotional resolution of the incentive disengagement problem [see Stout et al., 2002]. If it is indeed the case that fear, but not frustration, is present in bony fish, and if indeed the brain circuits and neurochemical mechanisms underlying fear and secondary frustration overlap considerably [Gray and McNaughton, 2000], then the egocentric mechanisms underlying secondary frustration could be seen as evolving from those underlying fear by a combination of gene duplication and co-option. Gene duplication and co-option have been postulated to explain such cases as the quadruplication of Hox genes in vertebrates, the evolution of feathers in birds, and the evolution of the enzymes involved in the development of the eye's lenses [Holland and Garcia-Fernandez, 1996; Raff, 1996]. The challenge now lies in designing appropriate experimental tests of this hypothesis.

Concluding Comments

A traditional view holds that the evolution of vertebrates is characterized by relatively stable motivational-emotional processes (largely viewed as plesiomorphic, that is, as primitive characters) and increased intellectual complexity (largely viewed as apomorphic, or derived characters). For example, Nissen [1958, p. 204] argued that species may be compared along the lines of 'receptor, effector, integrative, and motivational mechanisms,' concluding that 'the most significant phylogenetic differences (...) are found in the dimension of central integration or cognitive capacities.' Jerison [1973] argued that encephalization in birds and mammals was driven by selective pressures related to the internal representation of environmental stimuli, which he called biological intelligence. MacLean [1990] suggested a distinction between three major neural sections, namely, the reptilian complex (diencephalic and rhombencephalic areas), involved in maintenance activities and basic displays; the paleomammalian formation (limbic system), involved in basic emotional reactions, including fear; and the neomammalian formation (isocortex and thalamus), playing a major role in the connection of the organism to its environment (including human language), and in goal-directed behavior. Even Macphail [1982], who suggested that all vertebrates share the same basic set of learning mechanisms, noted as an exception that human language provides for qualitatively different cognitive abilities.

The research reviewed in this article suggests a more complex picture. On the one hand, some emotions (e.g., fear) may be more primitive than others (e.g., frustration). On the other hand, if the egocentric mechanisms underlying frustration turn out to be unique to mammals, as hypothesized here, then the relatively larger mammalian brains may have also made it possible for these animals to experience a wider emotional spectrum. Part of this spectrum relates to the issue of grief mentioned in the introduction. Grief is a relatively long-lasting emotional state that arises from the loss of a loved one (i.e., a conspecific with whom the organism has formed an attachment). To be more specific, a parallel could be drawn between grief and the extinction of appetitive behavior. According to this idea, grief is the emotion induced by cues previously associated with the presence of a conspecific that is no longer around. It seems likely that primitive mammals were mostly solitary, as this is true for extant conservative species [Eisenberg, 1981], so that the evolution of the mechanisms underlying secondary frustration must have occurred in the context of foraging for food, water, and other similar resources. In the case of grief, the lost object has a social dimension because the attachment is with a conspecific, rather than with a site where food was previously found. The sharing of similar effects in situations involving food omission and social separation (see introduction for references) suggests that the mechanisms underlying grief may have evolved from those underlying secondary frustration in mammals displaying complex social behavior (e.g., primates). In such a case, it is tempting to speculate that the function of frustration and grief may be one and the same, namely, to promote incentive disengagement from a source of reward (a site or a conspecific) that is no longer available. These speculative ideas are offered as an impetus for careful experimental and comparative analyses.

Acknowledgement

The author wishes to thank Steven Stout, Brian Thomas, Robert Boughner, and Dawn McVicar for valuable comments on an earlier version of this paper. They also contributed to the research reviewed in this paper, together with Alba Mustaca, Ruben Muzio, Santiago Pellegrini, Mariana Bentosela, Tom Dudley, Pete Hollingsworth, Gene Castro, Manuel Portavella, and Cosme Salas.

References

Amsel A (1992) Frustration Theory. New York: Cambridge University Press.

Amsel A, Roussel J (1952) Motivational properties of frustration: I. Effect on a running response of the addition of frustration to the motivational complex. J Exp Psychol 43:363–368.

Archer J (2001) Grief from an evolutionary perspective. In: Handbook of Bereavement Research (Stroebe MS, Hansson RO, Stroebe W, Schut H, eds), pp 263–283. Washington, DC: American Psychological Association.

Azrin NH, Hutchinson RR, Hake DF (1966) Extinction-induced aggression. J Exp Anal Behav 9:191–204.

Bailey MT, Coe CL (1999) Maternal separation disrupts the integrity of the intestinal microflora in infant rhesus monkeys. Devel Psychobiol 35: 146–155.

Barton RA, Harvey PH (2000) Mosaic evolution of brain structure in mammals. Nature 405:1055–1058.

Bartrop RW, Lockhurst E, Lazarus L, Kiloh LG, Penny R (1977) Depressed lymphocyte function after bereavement. Lancet 1:834–836.

Becker HC, Jarvis M, Wagner G, Flaherty CF (1984) Medial and lateral amygdala lesions differentially influence contrast with sucrose solutions. Physiol Behav 33:707–712.

Benton MJ (1990) Vertebrate Paleontology. London, UK: Unwin Hyman.

Bitterman ME (2000) Cognitive evolution: A psychological perspective. In: The Evolution of Cognition (Heyes C, Huber L, eds), pp 61–79. Cambridge, MA: MIT Press.

Blair HT, Schafe GE, Bauer EP, Rodrigues SM, LeDoux JE (2001) Synaptic plasticity in the lateral amygdala: A cellular hypothesis of fear conditioning. Learn Mem 8:229–242.

Carbonaro DA, Friend TH, Dellmeier GR, Nuti LC (1992) Behavioral and physiological responses of dairy goats to food thwarting. Physiol Behav 51:303–308.

Castro EA (2000) Role of opioids in consummatory successive negative contrast. Unpublished Master's Thesis, Texas Christian University.

Coe CL, Stanton ME, Levine S (1983) Adrenal responses to reinforcement and extinction: Role of expectancy versus instrumental responding. Behav Neurosci 97:654–657.

Coe CL, Wiener SG, Rosenberg LT, Levine S (1985) Endocrine and immune responses to separation and maternal loss in nonhuman primates. In: The Psychobiology of Attachment and Separation (Reite M, Field T, eds), pp 163–200. New York: Academic Press.

Couvillon PA, Bitterman ME (1985) Analysis of choice in honeybees. Anim Learn Behav 13: 246–252.

Crespi LP (1942) Quantitative variation of incentive and performance in the white rat. Am J Psychol 55:467–517.

Daly HB (1974) Reinforcing properties of escape from frustration aroused in various learning situations. Psychol Learn Motiv 8:187–231.

Dantzer R, Arnone M, Mormede P (1980) Effects of frustration on behaviour and plasma corticosteroid levels in pigs. Physiol Behav 24:1–4.

Davis H, Memmott J, MacFadden L, Levine S (1976) Pituitary-adrenal activity under different appetitive extinction procedures. Physiol Behav 17:687–690.

Dudley RT, Papini MR (1995) Pavlovian performance of rats following unexpected reward omissions. Learn Motiv 26:63–82.

Dudley RT, Papini MR (1997) Amsel's frustration effect: A Pavlovian replication with control for frequency and distribution of rewards. Physiol Behav 61:627–629.

Eisenberg JF (1981) The Mammalian Radiations. An analysis of trends in evolution, adaptation, and behavior. Chicago, IL: University of Chicago Press.

Elliott MH (1928) The effect of change of reward on the maze performance of rats. Univ Calif Pub Psychol 4:19–30.

Epictetus (2000) Discourses (Oldfather WA, trans), Classical Loeb Collection, Vol. 218. Cambridge, MA: Harvard University Press.

Feldon J, Gray JA (1981) The partial reinforcement extinction effect after treatment with chlordiazepoxide. Psychopharmacology 73: 269–275.

Feldon J, Weiner I (1991) Effects of haloperidol on the multitrial partial reinforcement extinction effect (PREE): Evidence for neuroleptic drug action on nonreinforcement but not on reinforcement. Psychopharmacology 105:407–414.

Feldon J, Katz Y, Weiner I (1988) The effects of haloperidol on the partial reinforcement extinction effect (PREE): Implications for neuroleptic drug action on reinforcement and nonreinforcement. Psychopharmacology 95:528–533.

Feldon J, Rawlins JNP, Gray JA (1985) Fornix-fimbria section and the partial reinforcement extinction effect. Exp Brain Res 58:435–439.

Flaherty CF (1996) Incentive Relativity. Cambridge, UK: Cambridge University Press.

Flaherty CF, Becker HC, Pohorecky L (1985) Correlation of corticosterone elevation and negative contrast varies as a function of postshift day. Anim Learn Behav 13:309–314.

Flaherty CF, Clarke S, Coppotelli C (1996) Lack of tolerance to contrast-reducing actions of chlordiazepoxide with repeated reward reductions. Physiol Behav 60:645–652.

Flaherty CF, Greenwood A, Martin J, Leszczuk M (1998) Relationship of negative contrast to animal models of fear and anxiety. Anim Learn Behav 26:397–407.

Flaherty CF, Grigson PS, Rowan GA (1986) Chlordiazepoxide and the determinants of negative contrast. Anim Learn Behav 14:315–321.

Gray JA, Dudderidge H (1971) Sodium amylobarbitone, the partial reinforcement extinction effect and the frustration effect in the double runway. Neuropharmacology 10:217–222.

Gray JA, McNaughton N (2000) The Neuropsychology of Anxiety. An enquiry into the functions of the septo-hippocampal system. 2nd Edition. Oxford, UK: Oxford University Press.

Hall M, Irwin M (2001) Physiological indices of functioning in bereavement. In: Handbook of Bereavement Research (Stroebe MS, Hansson RO, Stroebe W, Schut H, eds), pp 473–492. Washington, DC: American Psychological Association.

Haskell M, Coerse NCA, Forkman B (2000) Frustration-induced aggression in the domestic hen: The effect of thwarting access to food and water on aggressive responses and subsequent approach tendencies. Behaviour 137:531–546.

Henke PG (1973) Effects of reinforcement omission on rats with lesions in the amygdala. J Comp Physiol Psychol 84:187–193.

Henke PG (1977) Dissociation of the frustration effect and the partial reinforcement extinction effect after limbic lesions in rats. J Comp Physiol Psychol 91:1032–1038.

Henke PG, Maxwell D (1973) Lesions in the amygdala and the frustration effect. Physiol Behav 10:647–650.

Holland PWH, Garcia-Fernandez J (1996) Hox genes and chordate evolution. Dev Biol 173: 382–395.

Hull CL (1943) Principles of Behavior. New York: Appleton-Century-Crofts.

Hulse SH (1958) Amount and percentage of reinforcement and duration of goal confinement in conditioning and extinction. J Exp Psychol 56: 48–57.

Iwahara S, Nagamura N, Iwasaki T (1967) Effect of chlordiazepoxide upon experimental extinction in the straight runway as a function of partial reinforcement in the rat. Jpn Psychol Res 9: 128–134.

Jerison HJ (1973) Evolution of the Brain and Intelligence. New York: Academic Press.

Jones EC (1970) A correlational study of pre- and postgoal competing responses. Psychon Sci 21: 25–27.

Jordan TC, Coe CL, Patterson J, Levine S (1984) Predictability and coping with separation in infant squirrel monkeys. Behav Neurosci 98: 536–560.

Kielan-Jaworowska Z (1986) Brain evolution in Mesozoic mammals. In: Vertebrates, Phylogeny, and Philosophy (Flanagan KM, Lillegraven JA, eds), pp 21–34. Laramie, WY: University of Wyoming Press.

Kling A, Lloyd R, Tachiki K, Prince H, Klimenko V, Korneva E (1992) Effects of social separation on immune function and brain neurotransmitters in cebus monkeys *(C. apella)*. Ann New York Acad Sci 650:257–261.

Klinger E (1975) Consequences of commitment to and disengagement from incentives. Psychol Rev 82:1–25.

Kobre KR, Lipsitt LP (1972) A negative contrast effect in newborns. J Exp Child Psychol 14:81–91.

Lobaugh NJ, Bootin M, Amsel A (1985) Sparing of patterned alternation but not partial reinforcement effect after infant and adult hippocampal lesions in the rat. Behav Neurosci 99:46–59.

Lyons DM, Fong KD, Schrieken N, Levine S (2000) Frustrative nonreward and pituitary-adrenal activity in squirrel monkeys. Physiol Behav 71:559–563.

MacLean PD (1990) The Triune Brain in Evolution. New York: Plenum Press.

Macphail EM (1982) Brain and Intelligence in Vertebrates. Oxford, UK: Clarendon Press.

McNaughton N (1984) Effects of anxiolytic drugs on the partial reinforcement effect in runway and Skinner box. Q J Exp Psychol 36B:319–330.

Mikulka PJ, Lehr R, Pavlik WB (1967) Effect of reinforcement schedules on reward shifts. J Exp Psychol 74:57–61.

Mitchell C, Flaherty CF (1998) Temporal dynamics of corticosterone elevation in successive negative contrast. Physiol Behav 64:287–292.

Mustaca AE, Bentosela M, Papini MR (2000) Consummatory successive negative contrast in mice. Learn Motiv 31:272–282.

Muzio RN, Segura ET, Papini MR (1993) Effects of lesions in the medial pallium on instrumental learning in the toad *(Bufo arenarum)*. Physiol Behav 54:185–188.

Muzio RN, Segura ET, Papini MR (1994) Learning under partial reinforcement in the toad *(Bufo arenarum)*: Effects of lesions in the medial pallium. Behav Neural Biol 61:36–46.

Nissen HW (1958). Axes of behavioral comparison. In: Behavior and Evolution (Roe A, Simpson GG, eds), pp 183–205. New Haven, CT: Yale University Press.

Northcutt RG, Ronan M (1992) Afferent and efferent connections of the bullfrog medial pallium. Brain Behav Evol 40:1–16.

Overmier JB, Papini MR (1986) Factors modulating the effects of teleost telencephalon ablation on retention, relearning, and extinction of instrumental avoidance behavior. Behav Neurosci 100:190–199.

Papini MR (1997) Role of reinforcement in spaced-trial operant learning in pigeons *(Columba livia)*. J Comp Psychol 111:275–285.

Papini MR (2002a) Comparative Psychology. Evolution and development of behavior. Upper Saddle River, NJ: Prentice Hall,.

Papini MR (2002b) Pattern and process in the evolution of learning. Psychol Rev 109:186–201.

Papini MR, Dudley RT (1997) Consequences of surprising reward omissions. Rev Gen Psychol 1:175–197.

Papini MR, Hollingsworth PR (1998) Role of nonreinforcement in the fixed-interval performance of pigeons. Psychon Bull Rev 5:84–90.

Papini MR, Mustaca AE, Bitterman ME (1988) Successive negative contrast in the consummatory responding of didelphid marsupials. Anim Learn Behav 16:53–57.

Papini MR, Muzio RN, Segura ET (1995) Instrumental learning in toads *(Bufo arenarum)*: Reinforcer magnitude and the medial pallium. Brain Behav Evol 46:61–71.

Papini MR, Thomas BL (1997) Spaced-trial operant learning with purely instrumental contingencies in pigeons *(Columba livia)*. Int J Comp Psychol 10:128–136.

Papini MR, Thomas BL, McVicar DG (2002) Between-subject PREE and within-subject reversed PREE in spaced-trial extinction with pigeons. Learn Motiv 33:485–509.

Pellegrini S, Papini MR (2002) Partial reinforcement attenuates consummatory successive negative contrast. Abst Psychon Soc 7:56.

Portavella M, Salas C, Vargas JP, Papini MR (in press) Implication of telencephalon in spaced-trial avoidance learning in the goldfish *(Carassius auratus)*. Physiol Behav.

Raff RA (1996) The Shape of Life. Genes, development, and the evolution of animal form. Chicago, IL: University of Chicago Press.

Rando TA (1993) Treatment of Complicated Mourning. Champaign, IL: Research Press.

Rawlins JNP, Feldon J, Gray JA (1980) The effects of hippocampectomy and of fimbria section upon the partial reinforcement extinction effect in rats. Exp Brain Res 38:273–283.

Reite M, Capitanio JP (1985) On the nature of social separation and social attachment. In: The Psychobiology of Attachment and Separation (Reite M, Field T, eds), pp 223–255. New York: Academic Press.

Rescorla RA, Wagner AR (1972) A theory of Pavlovian conditioning: Variations in the effectiveness of reinforcement and nonreinforcement. In: Classical Conditioning: II. Current research and theory (Black AH, Prokasy WF, eds), pp 64–99. New York: Appleton-Century-Crofts.

Roberts WA, Bullock DH, Bitterman ME (1963) Resistance to extinction in the pigeon after partially reinforced instrumental training under discrete-trials conditions. Am J Psychol 76: 353–365.

Rodenburg TB, Zimmerman PH, Koene P (2002) Reaction to frustration in high and low feather pecking laying hens. Behav Proc 59:121–129.

Rosen AJ, Tessel RE (1970) Chlorpromazine, chlordiazepoxide and incentive shift performance in the rat. J Comp Physiol Psychol 72: 257–262.

Rowan GA, Flaherty CF (1987) The effects of morphine in the consummatory contrast paradigm. Psychophramacology 93:51–58.

Schleifer SJ, Keller SE, Camerino M, Thornton JC, Stein M (1983) Suppression of lymphocyte stimulation following bereavement. J Am Med Assoc 250:374–377.

Schmajuk N (1997) Animal Learning and Cognition. Cambridge, UK: Cambridge University Press.

Scully J, Tosi H, Banning K (2000) Life event checklists: Revisiting the Social Readjustment Rating Scale after 30 years. Educ Psychol Measur 60:864–876.

Seneca LA (1996) On providence. In: Moral Essays, Vol. I (Basore JW, trans), Classical Loeb Collection, Vol. 214. Cambridge, MA: Harvard University Press.

Seneca LA (1996) On anger. In: Moral Essays, Vol. I (Basore JW, trans), Classical Loeb Collection, Vol. 214. Cambridge, MA: Harvard University Press.

Seneca LA (1996) On tranquility of mind. In: Moral Essays, Vol. II (Basore JW, trans), Classical Loeb Collection, Vol. 254. Cambridge, MA: Harvard University Press.

Stamps J (1998) Territoriality. In: Comparative Psychology (Greenberg G, Haraway MM, eds), pp 761–770. New York: Garland Publishing.

Stein M, Trestman RL (1990) Psychiatric perspectives of brain, behavior, and the immune system. In: Immunologic Mechanisms in Neurologic and Psychiatric Disease (Waksman BH, ed), pp 161–169. New York: Raven Press.

Stout SC, Boughner RL, Papini MR (in press) Reexamining the frustration effect in rats: Aftereffects of surprising reinforcement and nonreinforcement. Learn Motiv.

Stout SC, Muzio RN, Boughner RL, Papini MR (2002) Aftereffects of the surprising presentation and omission of appetitive reinforcers on key-pecking peformance in pigeons. J Exp Psychol: Anim Behav Proc 28:242–256.

Suomi SJ (1991) Primate separation models of affective disorders. In: Neurobiology of Learning, Emotion, and Affect (Madden J IV, ed), pp 195–214. New York: Raven Press.

Swanson AM, Isaacson RL (1967) Hippocampal ablation and performance during withdrawal of reinforcement. J Comp Physiol Psychol 64: 30–35.

Terrace HS (1971) Escape from S-. Learn Motiv 2: 148–163.

Terrace HS (1972) By-products of discrimination learning. Psychol Learn Motiv 5:195–265.

Thomas BL, Papini MR (2001) Adrenalectomy eliminates the extinction spike in autoshaping with rats. Physiol Behav 72:543–547.

Thomas BL, Papini MR (2003) Mechanisms of spaced-trial runway extinction in pigeons. Learn Motiv 34:104–126.

Thorndike EL (1911) Animal Intelligence: Experimental studies. New York: Macmillan.

Tondat LM, Daly HB (1972) The combined effects of frustrative nonreward and shock on aggression between rats. Psychon Sci 28:25–28.

Vogel JR, Mikulka PJ, Spear NE (1968) Effects of shifts in sucrose and saccharine concentrations on licking behavior in the rat. J Comp Physiol Psychol 66:661–666.