SPONTANEOUS RECOVERY OF CONSUMMATORY SUCCESSIVE NEGATIVE CONTRAST

by

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Introduction

Consummatory successive negative contrast (cSNC) is the abrupt downshift of consummatory behavior by an experimental group after experiencing a downshift to a lower incentive value. Consummatory behavior in downshifted animals falls below the level of a control group that has received the lower incentive during a period of acquisition (Vogel, Mikulka, & Spear, 1968). In these experiments, the incentives are usually sucrose solutions of different concentrations (e.g., 32% and 4% solutions). The consummatory behavior of drinking during acquisition and the avoidance of the incentive after the downshift may be viewed as examples of Pavlovian conditioning. In this framework, approach and drinking are examples of appetitive conditioned responses, whereas the avoidance behavior is an example of an aversive conditioned response (Wasserman, Franklin, & Hearst, 1974; Papini & White, 1994). Given these similarities, one may ask whether a standard Pavlovian phenomenon, such as spontaneous recovery, would be present in the cSNC situation.

Spontaneous recovery (SR) occurs when an extinguished response to a conditioned stimulus recovers part of its former strength after a resting period without further stimulation. SR occurs in a variety of Pavlovian conditioning situations (Rescorla, 2004a), including aversive-to-appetitive counterconditioning (Bouton & Peck, 1992). In phase one, Bouton and Peck used aversive conditioning, pairing a stimulus (a tone) with an aversive stimulus (footshock). Pairing the tone shortly before the footshock elicited freezing behavior from subjects. In the second phase, they conducted appetitive training, pairing the tone with an appetitive stimulus (food pellets). After many such appetitive pairings, the tone elicited appetitive head jerking behavior. This is the experiment's counterconditioning phase. In phase 3, after a resting period, subjects were again presented with the tone and observed to see which behavior occurred. Did subjects display those behaviors seen during the aversive conditioning or those seen during more recent

appetitive counterconditioning? Bouton and Peck found that behavior seen during aversive training, freezing, increased in frequency after the resting period, despite appetitive training having occurred more recently.

Aversive-to-appetitive counterconditioning and cSNC have common procedural characteristics that make them *analogous* (although not identical). First, counterconditioning and cSNC share an aversive hedonic component. The initial learning about this aversive component is called downshift in cSNC and aversive conditioning in the counterconditioning situation. Second, both counterconditioning and cSNC share a period wherein the subject's initial aversive response diminishes and appetitive responses come to predominate. In the cSNC situation this is called recovery. During the course of the remaining contrast trials, the subject's interaction with the lower incentive increases. In counterconditioning, appetitive responses appear when the same stimuli that earlier predicted the painful stimuli later predicts an appetitive stimuli. There is also one key difference. cSNC involves a preliminary training phase called preshift training, during which the animal comes to expect a highly preferred reward. The counterconditioning procedure has no such component. The reason for this asymmetry lies in the different conditions that induce frustration and pain. Whereas frustration requires the acquisition of a reward expectancy before its violation causes emotional arousal, pain is immediately arousing even without previous training.

Although these paradigms share procedural aspects, possible similarities have not been explored in detail. SR is known to occur in counterconditioning, but it is unknown whether it would occur within the cSNC paradigm. The aim of this proposal is to answer the following question: Could a resting period interpolated after the recovery from cSNC induce the SR of the cSNC effect? First, however, let us look at cSNC.

Consummatory Successive Negative Contrast

The cSNC effect is part of a larger set of behavioral phenomena called incentive contrast effects, a group of effects induced by shifts in the quantity or quality of reward (Mackintosh, 1974; Flaherty, 1996; Williams, 1997). Researchers study incentive contrast phenomena in consummatory situations using three different types of procedures. First is the successive contrast procedure. This is the procedure described in the introductory paragraph (Vogel et al., 1968). Second is the anticipatory contrast procedure. In anticipatory contrast, the reward is shifted once within the session, from a target reward to an alternative reward. The target reward, usually a saccharin solution, is followed by the alternative reward, usually a sucrose solution. In this paradigm, the data of interest concerns changes in target reward intake as the animal receives repeated experience with the target/alternative pairing. Researchers evaluate the effect of the target/alternative pairing on reward intake in comparison to animals that receive a target/target reward pairing or an alternative/alternative reward pairing (Flaherty & Checke, 1982; Flaherty, 1996). Third is the simultaneous contrast procedure. Simultaneous contrast may be seen as an extension of anticipatory contrast. In simultaneous contrast, the reward shifts multiple times during the session, switching between the target and alternative rewards repeatedly (Flaherty & Avdzej, 1974; Flaherty & Largen, 1975).

In contrast procedures there are two different ways in which the reward can be shifted. The reward can be downshifted, that is, shifted from a more preferred to a less preferred reward, or upshifted, that is, from a less preferred to more preferred reward. These procedures are called respectively negative and positive contrast. The rest of this document focuses on the cSNC preparation.

Procedure for cSNC

Vogel et al. (1968) first used the technique that has become the standard methodology for the investigation of cSNC effects (see also Flaherty, 1996). In this procedure, subjects are assigned to two groups, an unshifted control group (Group 4→4) and a downshifted experimental group (Group 32→4). Subjects are placed into conditioning boxes and a sucrose solution is presented from a sipper tube that the subject can lick. The amount of time that a subject spends in contact with the sipper tube (goal tracking time), the frequency of licking, or the amount of fluid intake are the usual dependent variables. In a typical experiment, during acquisition, Group 4→4 receives a 4% sucrose solution and Group 32→4 receives a 32% sucrose solution. The acquisition or preshift phase continues for a number of trials, usually between 10 and 20 trials, each one lasting 5 min and conducted approximately 24-h apart. After the preshift trials in the conditioning boxes, a change is implemented for Group 32→4, but not for Group 4→4. On the first trial after the preshift phase, Group 32→4 receives a 4% solution, instead of the 32% solution received during preshift trials. The change from 32% solution to 4% solution persists for the remaining trials of the experiment, usually between 2 and 7. These are referred to as the postshift trials.

Typically, this procedure produces the following results that illustrate basic properties of cSNC (see Figure 1). First, during the preshift trials (1-10), the subjects show a steady rate of acquisition, in both groups. Typically (but not always), rats exposed to the 32% solution exhibit a higher performance than those exposed to the 4% solution. Second, on the first postshift trial, trial 11, when Group 32→4 is downshifted from a 32% solution to a 4% solution, licking behavior declines sharply and falls below that of Group 4→4. Third, for each trial after the first postshift trial (12-15), Group 32→4 begins to recover normal levels of consummatory behavior until there is no discernible difference in the licking behavior of the experimental and control

groups. Group 32→4's drop in performance below Group 4→4 on the first postshift trial and subsequent recovery, known as the cSNC effect, indicates that the change in behavior is not simply an adjustment to the 4% level (Flaherty 1996).

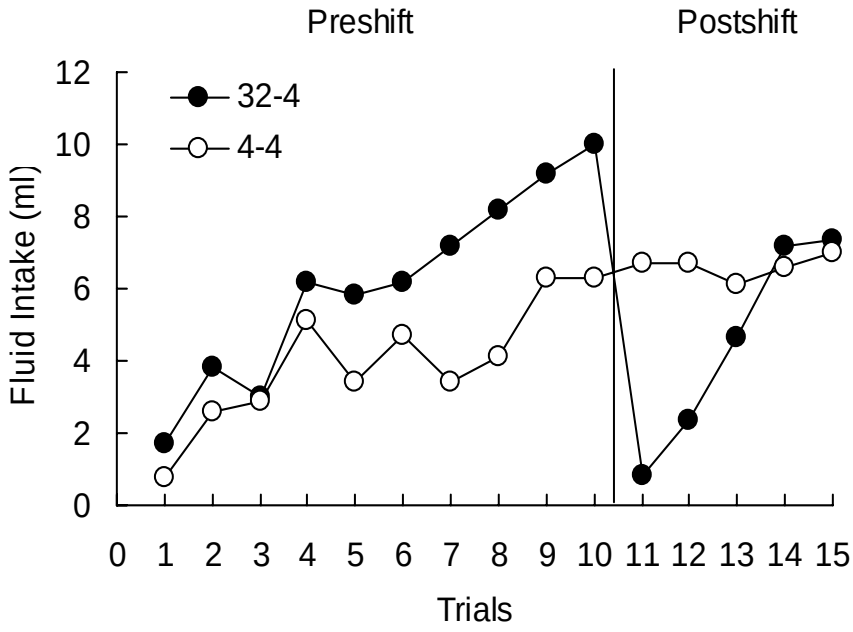

Figure 1. Illustration of the basic properties of cSNC. During preshift trials (1-10) Group 32→4 receives 32% sucrose solution, while Group 4→4 receives 4% sucrose solution. During the postshift trials (12-15) both groups receive 4% sucrose solution (taken from Papini, in press).

Theories of the cSNC Effect

There have been several attempts to explain the cSNC effect, namely Group 32→4's consummatory behavior falling below Group 4→4's consummatory behavior. Some look toward search components (e.g., Elliot 1928) or emotional reactions to explain contrast effects (Amsel, 1992; Tinkelpaugh, 1928). These ideas were incorporated in two recent hypotheses, Flaherty's (1996) multistage hypothesis and Wood et al.'s (2005) frustration hypothesis.

Flaherty's multistage hypothesis incorporates both search and emotional aspects into an explanation of cSNC. According to the multistage hypothesis, rats develop a representation of the 32% solution, established through a conditioning process. The initial taste of the sucrose functions to retrieve the representation of the solution concentration. It is not the training context that activates the representation because evidence indicates that altering the context during the shift in reward does not eliminate contrast (Flaherty, Hrabinski, & Grigson, 1990).

The 32→4 reward downshift starts a sequence of psychological processes. First, there is detection of the change in solution. The subject has its expectations violated when it detects the new 4% solution. Then it evaluates the change. Next, it reacts to the new solution by searching for the missing 32% solution instead of performing the consummatory behavior. According to the multistage hypothesis, activation of this search process is the mechanism responsible for consummatory suppression. The subject rejects the downshifted incentive as a result of searching.

Then, beginning perhaps after an interval of 5-min from the initial encounter with the downshifted reward, there is the activation of a stress response. While the stress response characterizes recovery and develops during the initial encounter with the downshifted reward, it does not play a role until after the initial 5-min period, at which time there is a detectable elevation of plasma corticosterone (Flaherty, Becker, & Pochorecky, 1985). When sufficient

elevation of plasma corticosterone is achieved recovery mechanisms activate. Among these mechanisms is a GABAergic (gamma-aminobutyric acid) circuit that promotes recovery from negative contrast. This is supported by evidence that only after at least 5 min of exposure to the downshifted solution until this interval elapses, GABA-dependent anxiolytics become effective in reducing contrast effects. Before this interval, GABA-independent anxiolytics, such as sodium amobarbital, can alleviate the contrast effects (Flaherty, Becker, & Driscoll, 1982). The activated GABAergic circuit inhibits a system that suppresses consummatory activity, having the net effect of promoting consummatory activity. For the remainder of the postshift trials, contrast will dissipate through the replacement of the preshift sucrose representation with the postshift representation, along with the action of a GABAergic circuit, reducing the stress-related aspects of the reward shift (Flaherty, 1996).

An alternative theory of cSNC, proposed by Wood et al. (2005), suggests a mechanism for consummatory suppression different from Flaherty's search induction, called primary frustration. Similarly to Flaherty's multistage hypothesis, rats develop a representation of the 32% solution, established through a conditioning process during the acquisition trials, the initial taste serving to retrieve the representation of the solution concentration. Again similar to Flaherty's hypothesis, the 32→4 reward downshift starts a sequence of psychological processes. Both Flaherty and Wood et al. propose that the initial processes are detection followed by rejection. However, while Flaherty suggested that rejection reflects search of the missing incentive, Wood et al.'s hypothesis suggests that the violation of high incentive expectancy induces an emotional response, called primary frustration that leads to both search and consummatory suppression (rejection).

The rationale behind Wood et al.'s argument is provided by Amsel's (1992) frustration theory. In frustrative conditioning procedures (of which cSNC is an example), there are two

types of responses to reward downshift. Frustration theory defines the initial, unconditioned response to reward downshift as primary frustration, and the later, conditioned response to reward downshift as secondary frustration. When applied to cSNC, frustration theory implies that the subject's initial response to reward downshift may be predominantly dependent on primary frustration and recovery from contrast that follows may depend largely upon the approach-avoidance conflict induced by reward consumption (approach) and secondary frustration (avoidance).

Wood et al.'s hypothesis integrates emotional and search components to explain several behavioral properties of cSNC, paralleling several elements of Flaherty (1996). However, it explains in more detail, the depression of consummatory behavior below that of the control group on the first postshift day. According to Wood et al.'s hypothesis, the mechanism responsible for consummatory suppression on trial 11 is primary frustration. In primary frustration, the subject has its expectations violated when it detects the new 4% solution. This causes an aversive emotional reaction that inhibits consummatory responding and induces searching for the missing 32% solution. After the initial 5 min, secondary frustration builds to a critical level such that GABA-dependent drugs act to alleviate consummatory suppression. The opioid system is also related to both the initial reaction to the downshift and the recovery that follows.

A consideration of these hypotheses of the cSNC phenomenon sets a theoretical stage. However, the present proposal is not designed to choose between these alternatives, but to expand the empirical base of the cSNC effect. Let us now turn to basic aspects of Pavlovian conditioning and extinction, and then to a more detailed description of SR in the counterconditioning situation.

Spontaneous Recovery

Pavlovian Conditioning and Extinction

SR is a basic phenomenon of Pavlovian conditioning (Pavlov, 1927). In Pavlovian or classical conditioning, an initially novel stimulus that elicits little more than an orienting response, is paired with a stimulus that elicits a strong response. After several such pairings the novel stimulus begins to elicit a new response. The initially novel stimulus is called the conditioned stimulus (CS). The response that comes to be made to the CS as the result of the pairing process is called the conditioned response (CR). The stimulus that elicits the strong response with little or no explicit raining is called the unconditioned stimulus (US) and the response that occurs to that stimulus is called the unconditioned response (UR).

In a classic demonstration, Pavlov (1927) noted that salivation (UR) occurred after the presentation of food to the mouth of the dog (US). This demonstrates the unconditional power of the food on the dog's salivation. In his next demonstration, Pavlov presented the US shortly after presenting the initially novel stimulus of a metronome. After several trials of pairing the metronome with the presentation of food, the sound of the metronome elicited the same degree of salivation as the food itself. The metronome came to serve as a CS capable of eliciting the CR of salivation from the dog (Pavlov, 1927).

In the same way that an animal, such as the dog in Pavlov's demonstrations, shows learning about the signaling properties of the CS, an animal can also learn that a CS no longer signals the US. This is called extinction. One aspect of extinction is that the term is used in three different senses: as a procedure, as a result, and as an explanation (Rescorla, 2004a). First, in a procedural sense, extinction refers to the technique of omitting the US after the presentation of the CS. The CS is presented alone so that it fails to signal the outcome. Secondly, as a result, extinction refers to the decrease in the CR that occurs when the US is omitted. Thirdly, in an

explanatory sense, extinction acts as an intervening variable explaining the observed CR deterioration after a CS is presented alone. Another aspect of extinction is that the response decrement appears to reflect new learning, rather than simply forgetting of the CS-US association (Bouton, 2004; Domjan, 2003; Rescorla, 2004a). This idea is supported by the presence of various extinction phenomena, including renewal, rapid reacquisition, reinstatement, and SR (Bouton, 2004). The next section considers SR in detail.

Spontaneous Recovery after Extinction

SR is an extinction phenomenon that supports the hypothesis that extinction involves new learning. If extinction could be explained as forgetting of the CS-US association, then interpolating a resting period should not lead to response recovery. The fact that SR occurs suggests that extinction does not result in the complete elimination of the CS-US association.

In the classic demonstration of SR, Pavlov (1927) and his associates trained dogs to salivate, and then shifted the CS to extinction. After extinction was complete, a resting period was introduced without further presentations of the CS. When the CS was presented again, after the resting period, the salivary response showed a substantial level of recovery. Data from two of Pavlov's experiments are presented in Figure 2. Pavlov (1927, p. 58) suggested that fully formed conditioned reflexes "invariably and spontaneously" regain their strength after extinction. Since Pavlov, several experimental procedures have been used by researchers to demonstrate SR (Rescorla, 2004a).

One type of procedure outlined in Table 1a (and illustrated in Figure 2) shows the minimum design needed to demonstrate SR. In this procedure, one group of subjects receives CS-US pairings during acquisition, then CS-only presentations in extinction, and then a resting period is introduced. Lastly, the CS is presented again in a test similar to the extinction phase. SR

is said to have occurred if there is an increase in the CR during the final test, relative to the end of extinction training.

Table 1 also shows the standard within-subject and between-subject procedures to demonstrate SR. In the standard within-subject procedure (Table 1b), subjects receive CS-US pairings with two different CSs (A+, B+) during an acquisition phase and undergo an extinction phase with both CSs (A-, B-). After a short resting period, A is tested for SR and, after a longer resting period, B is tested for SR. Researchers measure the CR developed by each of the CSs. SR occurs if there is a greater CR to B than to A. In the standard between-subject procedure (Table 1c), two groups (1, 2) receive a single CS, during acquisition (A+). Both groups then undergo an extinction phase (A-) contemporaneously. After a short resting period, group 1 is tested for SR. After a longer rest period, group 2 is tested for SR. Researchers measure the CR developed by each group. SR occurs if there is greater CR in group 2 than in group 1.

Table 1 also summarizes alternative within-subjects and between-subjects procedures used to study SR. Researchers use the alternative design when it is important to show that the absolute time at which SR testing occurs does not play a role in the degree of recovery, testing all groups for SR at the same time. In the alternative within-subjects design (Table 1d), one group of subjects are trained with two CS-US pairings (A+, B+), during an acquisition phase. In the second phase, one CS undergoes extinction training (A-), while the other CS (B) is untested. In the next phase, the CSs receive opposite treatment. Finally, both CSs (A, B) test for evidence of differential recovery. SR occurs if A elicits more CR than B. In the alternative between-subjects procedure (Table 1e), training is the same except that a single CS (A) is assigned to independent groups (Rescorla, 2004a).

These procedures revealed four empirical properties of SR (Rescorla, 2004a). First, SR increases in a negatively accelerated fashion over resting time. Evidence indicates that there is

more recovery as the length of the resting period between extinction and testing increases. For example, Robbins (1990) trained pigeons on a signal-tracking task using four different keylights, followed by extinction trials. Robbins tested for SR after resting periods of 15 min, 24 h, 48 h, and 168 h, finding a greater number of pigeons demonstrated recovery at each successive testing interval. Similarly, Quirk (2002) found increasing recovery of conditioned freezing as post-extinction time increased; testing occurred at intervals ranging from 15 min to 336 h. Evidence also indicates the amount of recovery across resting periods follows a negatively accelerated function. Negative acceleration refers to diminishing change of the SR effect with increasingly longer resting periods (Quirk, 2002; Robbins, 1990).

The second empirical property of SR detailed by Rescorla (2004a) is the incomplete nature of recovery. Even when subjects appear to have complete recovery in the initial testing trial, recovery is fleeting. There is a rapid decrease of CR after the initial SR, compared to the longer more gradual decrease of CR seen in original extinction trials, indicating that recovery is less than complete. If there were complete recovery, then the rate of extinction following SR would be similar to the rate of original extinction.

Third, SR declines with repeated extinction. This most commonly appears as a reduced amount of recovery from day to day after given multiple extinction trials. An example of this decline was demonstrated using a magazine-approach task using rat subjects (Rescorla, 2004a). After training an approach response to a magazine (CR) containing food pellets signaled by noise (CS), rats underwent repeated daily extinction sessions that tested for SR. During each one of the sessions, the CS was presented 8 times, but the magazine did not contain food. Between extinction sessions, a resting period occurred in the form of single presentation of a different Pavlovian association (light→food). With each successive extinction session (noise→no food), SR of an approach response decreased. In a similar task, Rescorla (2004b) trained rats to make

an approach response to a magazine containing food pellets, followed by extinction training. After a 48-h resting period, rats underwent 8 CS-alone (extinction) trials. In the initial trial, the rats showed SR of the approach response. In the next 7 trials, the approach response was again extinguished. After another 120-h resting period, the rats underwent another 8 CS-alone trials. Again, in the initial trial, rats showed SR of the approach response. However, the amount of SR was less than the original SR, illustrating the point that SR declines with repeated extinction.

Fourth, SR can be brought under stimulus control. Learning processes, such as extinction are subject to stimulus control (Rescorla, 2004a). For example, stimuli presented during nonreinforcement of a trained excitatory CS acquire inhibitory properties. Similarly, extinction may be administered in a new context. When the context is again changed after extinction, the presentation of the CS induces responding again (Bouton, 1991). Researchers have extended these findings to SR. For example, Brooks (2000) trained appetitive responding to a tone (T). During extinction, an extinction cue (EC) occurred immediately prior to T. A keylight (Y) served as an EC preceding T. For the testing phase, Brooks interpolated a 6-day RP, and then divided the animals into groups in order to test for SR of appetitive responding, based on responding to T. One group received EC before the respective appetitive stimuli (Y→T-), while another group received no EC before the appetitive stimuli. Brooks found that compared to the no-EC group, the group that had received EC had reduced SR. Therefore, explicit stimuli present during extinction of an excitatory CS have the ability to diminish SR if presented during a testing phase. This supported the notion that SR can be brought under stimulus control.

Table 1

Designs Used to Demonstrate Spontaneous Recovery.

		Phase			
Design	Group	1	2	3	4
(a) Minimum	1	A+	A-	RP	A?
(b) Standard within	1	A+	A-	Short RP	A?
	1	B+	B-	Long RP	B?
(c) Standard between	1	A+	A-	Short RP	A?
	2	A+	A-	Long RP	A?
(d) Alternative within	1	A+	A-	RP	A?
	1	B+	RP	B-	B?
(e) Alternative between	1	A+	A-	RP	A?
	2	A+	RP	A-	A?

Note: RP = resting period, A and B represent conditioned stimuli, + = presentation of unconditioned stimuli, - = no unconditioned stimuli presented (from Rescorla, 2004a).

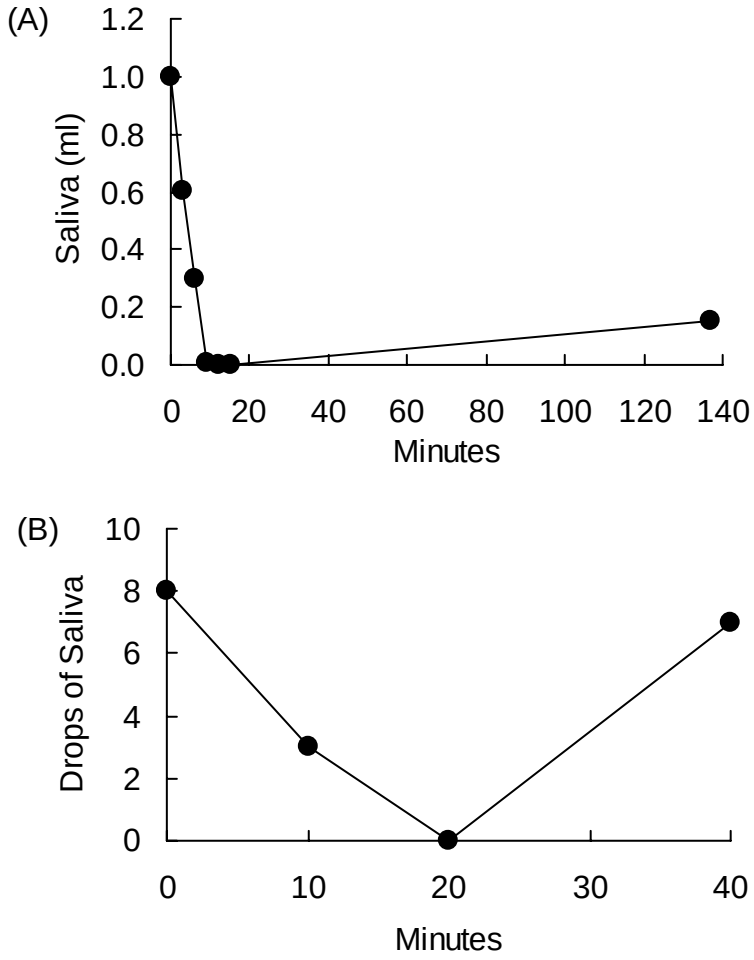

Figure 2. Examples of spontaneous recovery. Animals recovered conditioned salivatory response to a previously extinguished CS after a resting period (adapted from Pavlov, 1927).

Spontaneous Recovery after Counterconditioning

A survey of the literature shows that evidence for SR in the counterconditioning paradigm is limited. However, available results indicate that an extinguished CR can reemerge in aversive-to-appetitive situations. Bouton and Peck's (1992) study, whose results were briefly discussed earlier, will now be addressed in greater detail to enable discussion of parallels between cSNC and counterconditioning. They used a variation of the standard between-subject procedure, substituting counterconditioning for extinction (procedure outlined in Table 2). In the experiment, after a period of pretraining, groups A and B (interference conditions) experienced a CS (300-Hz tone presented for 30-s) shortly before an aversive US (0.6-mA scrambled footshock presented for 0.5-s) during phase 1. These trials occurred over the course of three days in 90 minute sessions, during which they received a six tone→shock pairings. They measured CS-elicited freezing behavior. Groups C and D (noninterference condition) underwent the pretraining phase but did not receive aversive conditioning. Counterconditioning occurred next, in phase 2. In phase 2, all groups (interference and noninterference) received five daily 50-min sessions of appetitive conditioning. Each session consisted of four tone→food pellet pairings. They measured CS-elicited head jerking behavior (an indicator of appetitive conditioning). Phase 2 is analogous to extinction; at the end of this phase interference animals no longer displayed freezing behaviors. In phase 3, animals were tested for SR using CS-alone presentations, measuring CS-elicited freezing and head jerking to see which behavior occurred more. After a one day resting period, B and D were tested for SR. After a 28 day resting period, A and C were tested for SR. Bouton and Peck predicted if aversive-to-appetitive counterconditioning is similar to extinction, then group A should show SR (displaying more behaviors seen during the aversive conditioning than those seen during more recent appetitive counterconditioning). In agreement with their predictions, Bouton and Peck found group A showed an increase in behavior seen

during aversive training, i.e. freezing, increased in frequency after the resting period, despite appetitive training having occurred more recently.

To explain SR of aversive-to-appetitive counterconditioning, Bouton (1993) looked at several situations in both human and nonhuman animals, and proposed a memory interference hypothesis. According to Bouton, interference paradigms, such as counterconditioning, refer to associative learning situations where an animal learns information at one point that conflicts with information learned at another point. Usually the significance of CS changes during the course of the experiment, signaling a different US at different time points. This results in the learning about the CS in one phase interfering with performance appropriate to the other CS in another phase. The interference can be *retroactive,* when phase 2 learning interferes with performance appropriate to phase 1, or it can be *proactive*, when phase 1 learning interferes with performance appropriate to phase 2.

Aversive-to-appetitive counterconditioning is a process involving proactive and retroactive interference on performance (Bouton, 1993; Bouton & Peck, 1992). In phase 1, the CS signals an aversive US, leading to appropriate responses, such as freezing. During phase 1, a single association is retrieved, consisting of tone→e_{Shock} (where e_{Shock} refers to the expectation of shock). In phase 2, the CS signals an appetitive US, leading the animal to perform appetitive responses. During phase 2, three associations are retrieved: tone→e_{Shock}, an excitatory association that elicits freezing behavior; tone−●e_{Shock}, inhibitory association that interferes with freezing behavior; tone→e_{Food}, an excitatory association that elicits appetitive responding, such as head jerking behavior. Retrieval of the tone→e_{Food} association results in increases in head jerking, while simultaneously conflicting with aversive responding when the tone→e_{Shock} association is retrieved. The net result is an increase in appetitive behavior and a decrease in aversive behavior.

Outlined in Figure 3 is the explanation of SR of aversive-to-appetitive counterconditioning with the addition of phase 3 after a resting period (Bouton, 1993; Bouton & Peck, 1992). In this third phase, two memories are retrieved: tone→e_{Shock} association, an excitatory association that elicits freezing behavior; and tone→e_{Food} association, an excitatory association that elicits appetitive responding, such as head jerking behavior. Retrieval of the tone→e_{Food} association results in increases in head jerking, while simultaneously conflicting with aversive responding when the tone→e_{Shock} pairing is retrieved. However, during the resting period the inhibitory pairing, tone−●e_{Shock} is weakened, as inhibitory associations are more easily forgotten over time (Pavlov, 1927). Because the inhibitory association is weakened, the net result is an increase in aversive behavior, causing SR. In this account, SR occurs because inhibitory associations fail to be retrieved while excitatory associations are readily retrieved.

Table 2

Bouton and Peck (1992) Aversive-to-Appetitive Counterconditioning Design.

Group	Phase 1	Phase 2	Resting period	Phase 3
A	Aversive	Appetitive	28 days	Test for SR
B	Aversive	Appetitive	1 days	Test for SR
C	-----	Appetitive	28 days	Test for SR
D	-----	Appetitive	1 days	Test for SR

Note: Aversive = Tone→footshock pairings, Appetitive = Tone→food pellet pairings, SR = Spontaneous recovery.

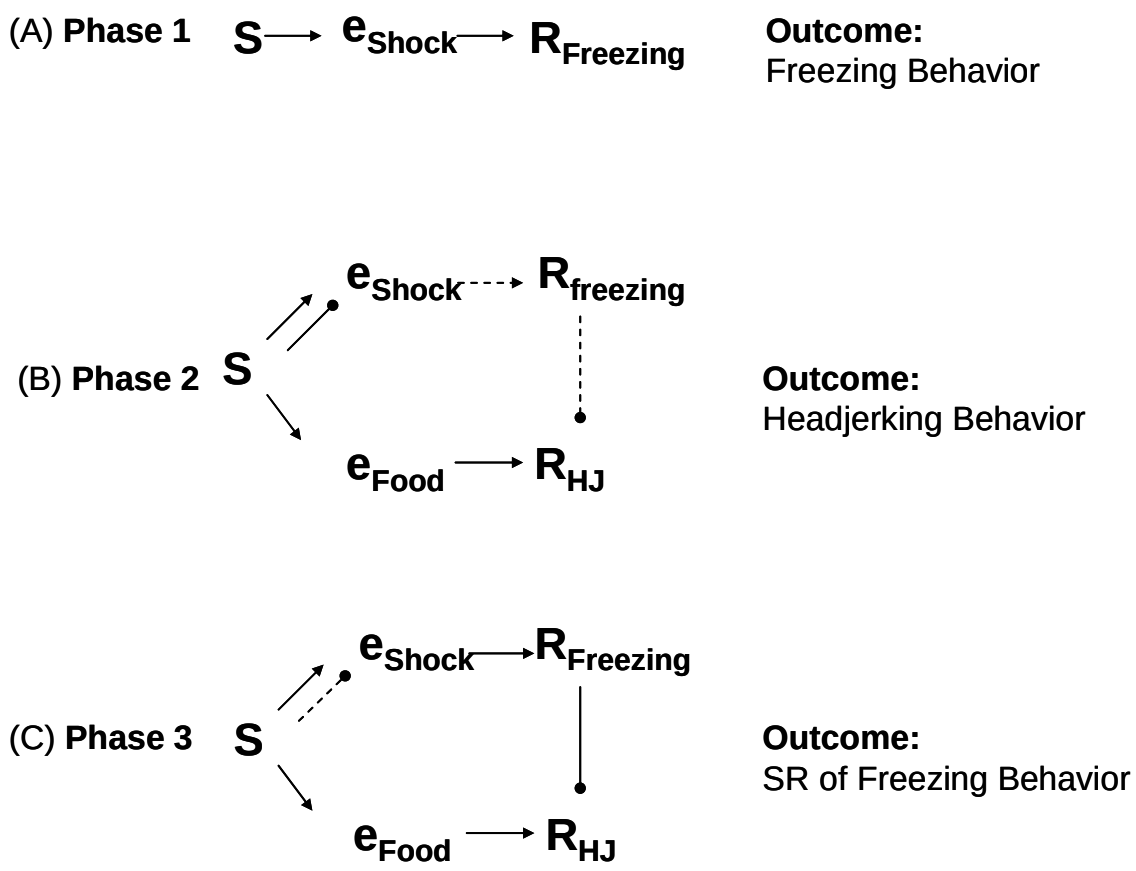

Figure 3. Diagram of Bouton and Peck's (1992) memory retrieval explanation of SR of aversive-to-appetitive counterconditioning. (A) In phase 1, during aversive conditioning, the conditioned stimulus (S) elicits expectations of footshock (e_{shock}), evoking a freezing response ($R_{freezing}$). The outcome is freezing. (B) In phase 2 during appetitive conditioning, in addition to e_{shock}, S elicits expectations of food e_{food}, evoking a head jerking response (R_{HJ}) and an expectation of no shock, via an inhibitory association, S−●e_{shock}. The net outcome is head jerking behavior. Between phase 2 and phase 3, a 28-d resting period is interpolated. (C) In phase 3, the inhibitory association, S−●e_{shock}, reduces over the course of the resting period. Therefore the animal retrieves e_{shock}, and this leads to SR of freezing behavior.

Parallels Between Aversive Conditioning and cSNC

Bouton and Peck's (1992) counterconditioning preparation and the typical cSNC procedure share common procedural characteristics (summarized in the introduction). Additionally, the subject's responses to these similar procedures have common elements. One such way that the responses are similar is the common involvement of an aversive component to generate these responses. The fear component developed by Bouton and Peck's tone→footshock pairings and the frustration component developed by cSNC are rooted in similar brain systems. Gray (1987) argued that fear and frustration are anticipatory responses to conditioned aversive stimuli that depend on brain systems that show extensive overlap. This became known as the fear = frustration hypothesis. In typical fear conditioning procedures, a signal predicts the onset of painful stimuli. Pain is unconditioned and fear is conditioned. Papini (2003) extended Gray's (1987) fear = frustration hypothesis by introducing Amsel's (1992) theory, arguing that the parallel between fear and frustration should extend to the unconditioned states that support them, namely, pain (since fear is usually generated by the administration of electric shocks) and primary frustration.

The analogy between cSNC and fear conditioning is supported by pharmacological evidence. For example, anxiolytics and opioid drugs alter Pavlovian fear conditioning and cSNC effects. Benzodiapezine tranquilizers, including chlordiazepoxide, flurazepam, and midazolam, have been shown to reduce to cSNC when administered on the second postshift day (Becker & Flaherty, 1983; Becker, 1986; Flaherty, 1990; Flaherty, Becker, Checke, Rowan, & Grigson, 1992). These results are analogous to those found using midazolam in fear conditioning situations. Santos, Gárgaro, Oliveira, Masson, and Brandão (2005) found that rats administered midazolam showed reduced startle reactions when placed in a moderate fear conditioning situation compared to saline controls. Szyndler, Sienkiewicz-Jarosz, Maciejak, Siemiatkowski,

Rokicki, Członkowska, and Płaźnik (2001) reported that rat subjects treated with midazolam showed significantly reduced freezing reactions when placed back into the experimental chamber 24 h after experiencing contextual fear conditioning. Another benzodiapezine, diazepam has been shown to eliminate contrast effects in mice (Mustaca, Bentosela, & Papini, 2000) and in rats when infused into the amygdala (Liao & Chuang, 2003). These results mirror those found in fear conditioning situations. For example, diazepam reduces the anticipatory fear-induced potentiated startle (Pietraszek, Sukhanov, Maciejak, Szyndler, Gravius, Wisłowska, Płaźnik, Bespalov, & Danysz, 2005). In these situations, anxiolytics seem to be acting on anticipatory or conditioned components. In cSNC, anxiolytics may be reducing the approach-avoidance conflict. In fear conditioning, anxiolytics reduce fear-potentiated startle, and freezing reactions after conditioning in a contextual fear situation.

Drugs acting on the opioid system, can also affect both Pavlovian fear conditioning and cSNC effects. The exogenous opioid drug, morphine, administered during the postshift trials reduces cSNC on trial 12 (Rowan & Flaherty, 1987). Similarly, morphine has been shown to reduce conditioned fear in the fear-potentiated startle paradigm when administered prior to training (Davis, 1979), and to induce amnesia of contextual fear conditioning when administered after training (McNally & Westbrook, 2003b). In both scenarios, morphine affects the period of recuperation wherein the subject adjusts to the new situation, recovering from cSNC and extinguishing conditioned fear. The δ-opioid receptor agonist drug, D-Ala2-N-MePhe4,Gly-ol (DPDPE) effectively reduces cSNC during the first postshift trial, but not during subsequent trials (Wood et al., 2005). This is similar to the findings reported by Fanselow, Calcagnetti, and Helmstetter (1989). In their experiment, DPDPE and two other selective δ-opioid receptor agonists produced high levels of conditioned analgesia compared to saline controls in a formalin test. In both scenarios, the δ-opioid agonists affected the initial encounter with an aversive

hedonic component, attenuating primary frustration in cSNC effects and inducing greater levels of antinociception in the formalin test.

Strengthening the analogy, pharmacological evidence also indicates that opioid antagonists have a similar effect in cSNC and fear conditioning. For example, cSNC research suggests the contrast reducing effects of morphine can be blocked with the administration of the non-selective antagonist, naloxone (Rowan & Flaherty, 1987). Pellegrini, Wood, Daniel, and Papini (2005) reported that naxolone, administered at 2 mg/kg before trials 11 and 12 has a robust and long lasting effect, enhancing contrast not only in those two trials, but also in the three remaining postshift trials. Furthermore, they reported naltrindole, a selective δ-opioid receptor antagonist, reduces contrast on the first postshift trial. Expectedly, this is the opposite effect of DPDPE on trial 11. Likewise, in fear conditioning, naloxone reduces extinction from auditory fear conditioning (McNally & Westbrook, 2003a), contextual fear conditioning (Fanselow, 1981) and fear-potentiated startle (Davis, 1979). In both instances, opioid antagonists affect the subjects' recovery period, reducing both recovery from cSNC and extinction of fear conditioning. Studies using another non-selective opioid antagonist, naltrexone, have similar effects in contextual fear conditioning situations, reducing extinction (Helmstetter & Fanselow, 1987). These results show extensive parallels between the aversive component of aversive-to-appetitive counterconditioning and the downshift of cSNC.

Experiment

The procedural and response parallels existing between aversive-to-appetitive counterconditioning and cSNC go hand-in-hand, suggesting their overall *analogous* nature. This analogy serves as a guidepost, directing research to investigate further similarities. The following experiment is one example. To the best of my knowledge, the SR of cSNC has not been addressed before, making the rationale to conduct such an investigation mainly an empirical

question. If there is SR within a cSNC preparation, its presence would strengthen the analogy. The experiment was conducted using the standard between-subjects design described in Table 1c. Rats received training in two conditions: 32→4 vs. 4→4. Once recovery from cSNC was complete, rats in each contrast condition were randomly assigned to 24-h, 96-h, or 336-h resting periods. At the end of the resting periods, all rats received 2 additional trials of access to the 4% solution. It was predicted that downshifted subjects tested after 96-h and 336-h resting periods, but not after a 24-h resting period, will demonstrate SR, operationalized as consummatory suppression on trial 19 in groups that had been exposed to the 32→4 incentive downshift. It was also predicted that SR will be greater at 336-h than at the 96-h resting period.

Method

Subjects

Sixty males from either Harlan (Indianapolis, Indiana) or the TCU animal vivarium, approximately 90 days old at the start of the experiment, served as subjects. Rats were housed in the TCU vivarium under a 12:12-h light:dark cycle (lights on at 07:00 h) and were deprived of food to an 81-84% of the free-food weight. Water was continuously available in each individual cage. Animals were trained during the light phase of the daily cycle. All subjects served as saline control animals for other cSNC studies and received preshift and postshift training as part of those experiments.

Apparatus

Training was conducted in four conditioning boxes (MED Associates, St. Albans, VT) constructed of aluminum and Plexiglas and measuring 29.3 cm in length, 21.3 cm in height, and 26.8 cm in width. The floor was made of steel rods 0.4 cm in diameter and 1.6 cm apart running perpendicular to the feeder wall. A bedding tray filled with corncob bedding was placed below the floor to collect fecal pellets and urine. Against the feeder wall was an elliptical hole 1-cm

wide, 2-cm high, and 4 cm from the floor. A sipper tube, 1 cm in diameter, was inserted through this hole. When fully inserted, the sipper tube was flush against the wall. A computer located in an adjacent room controlled the presentation and retraction of the sipper tube, and detected contact with the sipper tube by way of a circuit involving the steel rods in the floor. Each conditioning box was placed in a sound-attenuating chamber that contained a houselight, a speaker to deliver white noise, and a fan for ventilation. Together, the speaker and fan produced noise with an intensity of 80.1 dB (SPL, Scale C).

Procedure

Training lasted for a total of 20 daily trials. Each rat was assigned to one of the four conditioning boxes and always trained in that box. The order of training of the 4-rat squads varied across days. After each trial, conditioning boxes were cleaned with a damp paper towel, feces removed, and bedding material replaced as needed. During trials, the houselight, white noise, and fan were on constantly. The 20 trials were divided into a preshift phase (10 trials), a postshift phase (8 trials), and a SR phase (2 trials). Rats were randomly assigned to one of two groups during the preshift and postshift phases (shifted, unshifted). After the postshift trials, but before the SR testing trials, the shifted rats were matched in terms of their performance and randomly assigned to one of the three different resting periods: 24-h, 96-h, and 336-h. The unshifted controls were assigned likewise.

Table 3 outlines the experiment's design. For the three 32→4 groups (32/336, 32/96, 32/24), the 10 preshift trials involved access to a 32% sucrose solution (w/w, prepared by mixing 32 g of commercial sugar for every 68 g of distilled water); the 8 postshift trials and 2 SR trials involved access to a 4% solution (w/w, 4 g of sugar for every 96 g of distilled water). The three 4→4 groups (4/336, 4/96, 4/24) received the 4% sucrose solution in all 20 trials.

Each trial started with a variable pretrial interval of 30 s (range: 15–45 s). At the end of this interval, the sipper tube was automatically presented. A trial started when a rat contacted the sipper tube. The trial lasted 5 min. Retraction of the sipper tube was followed by a posttrial interval averaging 30 s (range: 15–45 s). The dependent variable was the cumulative amount of time in contact with the sipper tube, measured in 0.05-s units and labeled *goal tracking time*.

Table 3

SR of cSNC Experiment Design

Group	Preshift	Postshift	Resting Period	SR Test
32/24 (n = 9)	32%	4%	24 h	4%
4/24 (n = 10)	4%	4%	24 h	4%
32/96 (n = 9)	32%	4%	96 h	4%
4/96 (n = 10)	4%	4%	96 h	4%
32/336 (n = 9)	32%	4%	336 h	4%
4/336 (n = 10)	4%	4%	336 h	4%

Note: Preshift = Trials 1-10, Postshift = Trials 11-18, SR Test = Trials 19-20.

Results

Group Measures

Figure 3A shows the overall results, in terms of goal tracking time as a function of trial. Two rats assigned to the 32→4 were eliminated for failing to show any performance decrement on trial 11. In the absence of response suppression, there would be no basis to expect SR. An independent one-way analysis of variance (ANOVA) performed on goal tracking times on trial 11, comparing each of the groups showed significant differences across groups, $F(5, 57) = 12.953$, $p < 0.01$. LSD multiple comparisons revealed significant differences between 32/24 vs. 4/24, 32/96 vs. 4/96, and 32/336 vs. 4/336, ps < 0.01. This provides evidence of a cSNC effect. An independent one-way ANOVA performed on goal tracking times on trial 18 $F < 1$. This provides evidence of recovery from cSNC. Two downshifted rats failed to show contrast. They were removed from the experiment, making the final subject number 58.

Figure 3B shows goal tracking ratios (trial 11/trial 10, trial 19/trial 18). A ratio of one indicates goal tracking time was equal in both trials, a ratio less than one indicates that goal tracking time in the second trial was less than the first trial in the pair, and a ratio greater than one indicates goal tracking time in the second trial was greater than first trial. These ratios were used to quantify the amount of consummatory suppression observed in trial 11 and trial 19 by comparing those trials to the trial that preceded them. These ratios were subjected to independent one-way ANOVAs followed by LSD multiple comparisons. The trial 11/trial 10 one-way ANOVA showed significance differences across groups, $F(5, 57) = 25.720$, $p < 0.01$. The LSD multiple comparisons revealed significant differences between the following groups: 32/24 vs. 4/24, 32/96 vs. 4/96, 32/336 vs. 4/336, ps < 0.01. This shows that 32% solution groups had significantly greater consummatory suppression on trial 11 than their 4% solution counterparts. Also important, the multiple comparisons showed that the 32% groups were not significantly

different from each other, nor were the 4% groups significantly different from each other. However, the trial 19/trial 18 ratio one-way ANOVA did not show any significant group difference, $F(5, 57) = 1.587$, $p > 0.17$. This statistical analysis, fails to support the first prediction, that there would be SR after 96-h and 336-h resting periods. As a consequence, the second prediction, SR at 336-h would be greater than SR at 96-h, also failed to be supported. An independent-sample t-test failed to reveal a difference between 96-h and 336-h trial 19/ trial 18 ratios, $t(17) = -1.574$, $p > 0.13$. Evaluating the data in this manner fails to support either experimental prediction.

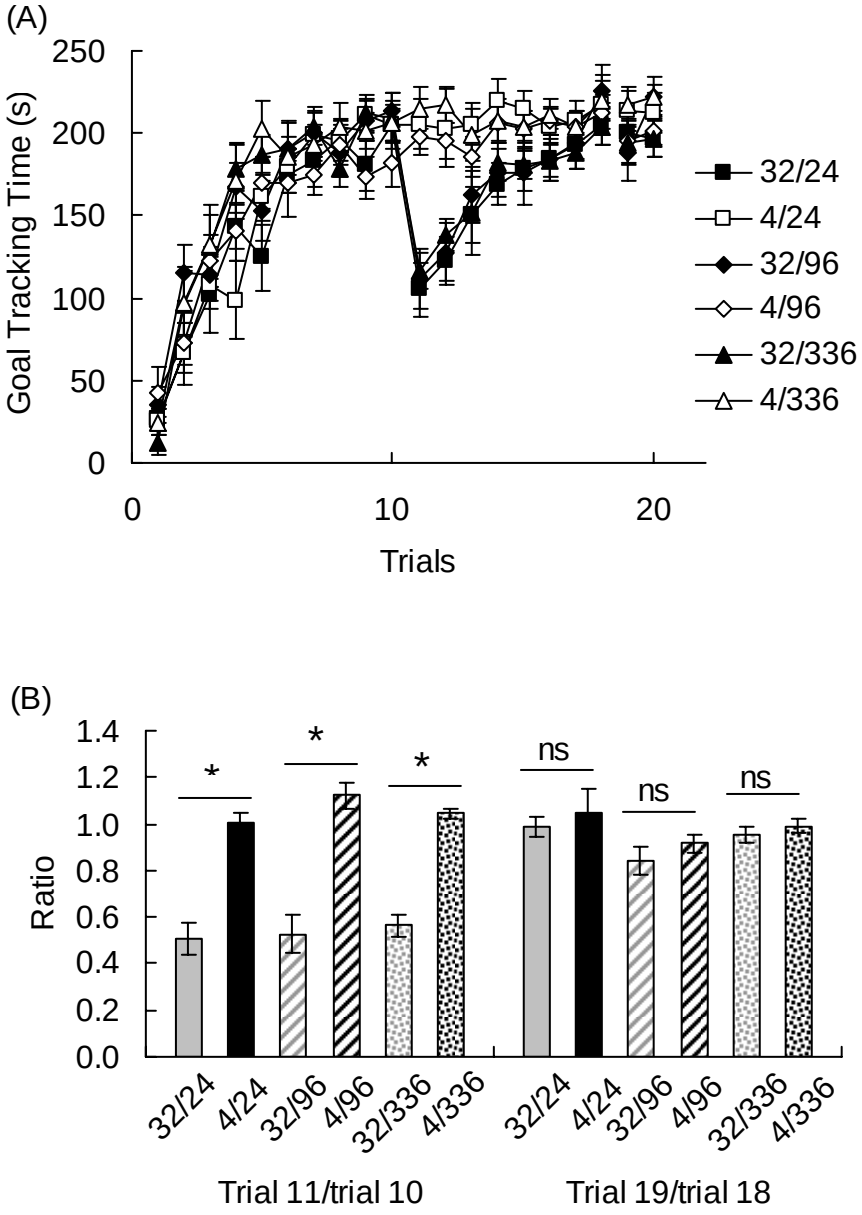

Figure 4. Group measures of spontaneous recovery of cSNC. (A) Shows overall results, in terms of goal tracking time as a function of trial. (B) Shows goal tracking ratios (trial 11/trial 10, trial 19/trial 18). A ratio of one indicates goal tracking time was equal in both trials, a ratio less than one indicates that goal tracking time in the second trial was less than the first trial in the pair, and a ratio greater than one indicates goal tracking time in the second trial was greater than first trial. Horizontal lines indicate pairwise comparsions (LSD post-hoc tests). *, $p < 0.05$. ns, nonsignificant.

Individual Differences

However, it was observed that performance during the SR test was variable across subjects. A median split was calculated on the data to evaluate the contribution of individual differences to SR. Each 32→4 condition (32/24, 32/96, 32/336) was divided into two subgroups based on their trial 19/trial 18 ratio, placing subjects into either a high consummatory suppression group (H) or a low consummatory suppression (L). If there is SR in some individual subjects then one should expect to see the following results when evaluating the data using a median split: In the 96-h condition, H_{96} should be significantly lower than both L_{96} and 4/96 in trial 19/trial 18 performance; and in the 336-h condition, H_{336} should be significantly lower than both L_{336} and 4/336 in trial 19/trial 18 performance. Importantly, there should not be any statistical differences between groups in the 24-h condition.

Figure 4 shows trial 19/18 goal tracking ratios for all conditions, comparing them to counterparts in the same resting period. Ratios were subjected to independent one-way ANOVAs, followed by LSD multiple comparisons, comparing groups within a resting period. Figure 4A shows the 24-h condition. The ANOVA showed no effect, $F < 1$. In agreement with the prediction, H_{24} does not differ from L_{24}, $p > 0.25$, H_{24} does not differ from 4/24, $p > 0.28$, nor does L_{24} differ from 4/24, $p > 0.77$. Figure 4B shows the 96-h condition. The ANOVA showed an effect, $F(2, 19) = 7.370$, $p < 0.01$. In agreement with the prediction, H_{96} is significantly lower than L_{96}, $p < 0.01$, and it is significantly lower than 4/96, $p < 0.01$. L_{96} does not differ from 4/96, $p > 0.361$. Figure 4C shows the 336-h condition. The ANOVA did not show an overall effect, $F(2, 17) = 3.269$, $p > 0.06$. However, in agreement with the 336-h predictions, H_{336} is significantly lower than L_{336}, $p < 0.032$, and it is significantly lower than 4/336, $p < 0.048$. Additionally, L_{336} does not differ from 4/336, $p > 0.497$. This statistical manipulation revealed individual differences in SR. Some individuals showed more SR than others. However, the

second prediction, SR at 336-h would be greater than SR at 96-h, remains unsupported using the median split manipulation. An independent samples t-test failed to show differences between H_{96} and H_{336}, $t(7) = -2.338$, $p > 0.051$, failing to show that SR increases as resting period increases.

It was decided to evaluate the individual differences results with respect to other variables. In other words, are the individual differences in SR an artifact of the data or do they relate to some other aspect of the experiment in a systematic way? In addressing this question, I looked at two aspects of the experiment: initial contrast and recovery from contrast.

Measurements of initial contrast (trial 11/trial 10 ratios) were subjected to independent one-way ANOVAs, followed by LSD planned comparisons for 24-h, 96-h, and 336-h resting periods. Within the resting periods there should be differences between the 4→4 group and 32→4 groups, but there should no differences between the H and L groups. As expected, the 24-h resting period ANOVA showed an $F(2, 17) = 19.468$, $p < 0.01$. 4/24 is significantly different from H_{24}, $p < 0.01$, and L_{24}, $p < 0.01$, and H_{24} and L_{24} do not differ from each other, $p > 0.22$. Also in agreement with the prediction, the 96-h resting period ANOVA showed an effect, $F(2, 19) = 19.594$, $p < 0.01$. 4/96 is significantly different than H_{96}, $p < 0.01$, and L_{96}, $p < 0.01$, and H_{96} and L_{96} do not differ from each other, $p > 0.15$. The same pattern holds true for the 336-h resting period. The ANOVA showed an effect, $F(2, 17) = 44.115$, $p < 0.01$. 4/336 is significantly different than H_{336}, $p < 0.01$, and L_{336}, $p < 0.01$, and H_{336} and L_{336} do not differ from each other, $p > 0.09$. These results indicate that the H and L in the 96-h and 336-h were not different from each with respect to their initial degree of consummatory suppression seen during the downshift.

The data were evaluated for recovery from initial contrast using Pellegrini et al.'s (2005) procedure based on trials 11 and 12, when most of the change is observed. Trial 12 – trial 11 difference scores were subjected to independent one-way ANOVAs, followed by LSD planned comparisons for all resting periods. Within the 24-h resting period groups, it was expected they

not show any recovery differences. Figure 5a shows the 24-h group. The ANOVA showed no effect, $F(2, 17) = 0.427$, $p > 0.660$. Planned comparisons showed no significant differences between L_{24} and H_{24}, between L_{24} and 4/24, ps > 0.05. However, within 96-h and 336-h resting period there were expected differences. If there are differences in initial recovery from contrast (one group recovering faster than another), then 32→4 groups should differ from each other and the fast recovery group should also differ significantly from the 4→4 control. Figure 5b shows the 96-h group. The ANOVA showed an effect, $F(2, 19) = 6.886$, $p < 0.01$. Planned comparisons showed a significant difference between L_{96} and H_{96}, $p < 0.012$. Planned comparisons also showed a significant difference between L_{96} and 4/96, $p < 0.01$. These results indicate faster recovery from contrast for L_{96} compared to H_{96}. Figure 5c shows the 336-h group. The ANOVA showed an effect, $F(2, 17) = 5.998$, $p < 0.013$. Planned comparisons showed a significant difference between L_{336} and H_{336}, $p < 0.019$. Planned comparisons also showed a significant difference between L_{336} and 4/336, $p < 0.01$. These results indicate faster recovery from contrast for L_{336} compared to H_{336}. Overall, this shows that those individuals demonstrating high amounts of SR showed slower recovery from initial downshift than those individuals demonstrating low amounts of SR. This indicates that individual difference data in the 96-h and 336-h groups correlates with the initial recovery after the downshift. Rats that exhibited evidence of SR were more likely to have experienced slow recovery from contrast, compared to rats that exhibited no evidence of SR.

To further examine the relationship between degree of SR and recovery from contrast both downshifted groups from 96-h and 336-h were collapsed into one group. A Pearson product-moment correlation indicated that consummatory suppression (trial 19/ trial 18) was significantly and positively correlated to recovery (trial 12 – trial 11), $r(17) = 0.541$, $p < 0.05$

(see Figure 6). These results support notions about individual differences in SR. Moreover, these results show that SR varies as a function of recovery from cSNC.

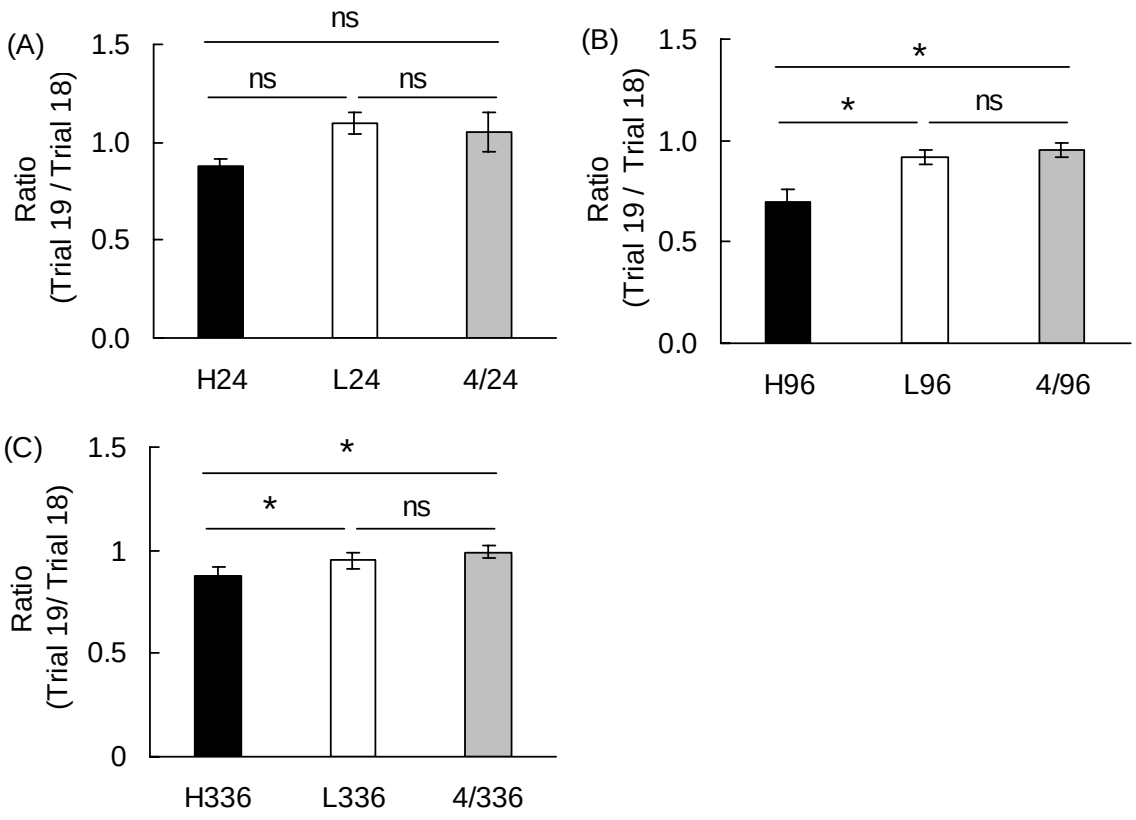

Figure 5. Median split measures of spontaneous recovery of cSNC. Within each resting period, a median split was conducted on the trial 19/trial 18 goal tracking ratios, dividing downshifted animals as high spontaneous recovery (H) and low spontaneous recovery (L). These scores were compared to the unshifted animals within a resting period. (A) Shows median split data for the 24-h resting period. (B) Shows median split for 96-h resting period. (C) Show median split for the 336-h resting period. Horizontal lines indicate pairwise comparisons (LSD post-hoc tests). *, $p < 0.05$. ns, nonsignificant.

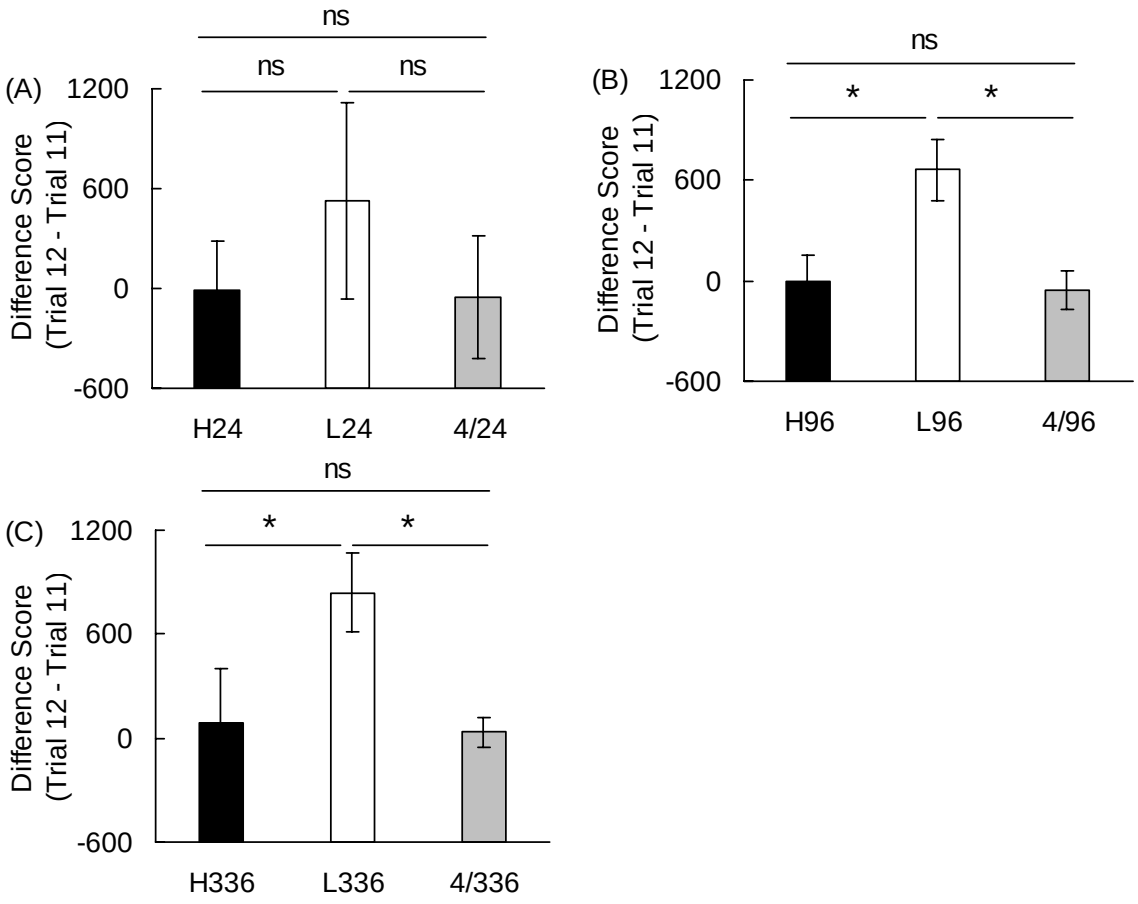

Figure 6. Differences in recovery from initial contrast between groups within resting period. The data were evaluated for recovery from initial contrast using Pellegrini et al.'s (2005) procedure based on trials 11 and 12, when most of the change is observed. Within each resting period, groups H, L, and unshifted were compared based on differences in recovery from initial contrast (trial 12 – trial 11). Horizontal lines indicate pairwise comparsions (LSD post-hoc tests). *, $p < 0.05$. ns, nonsignificant.

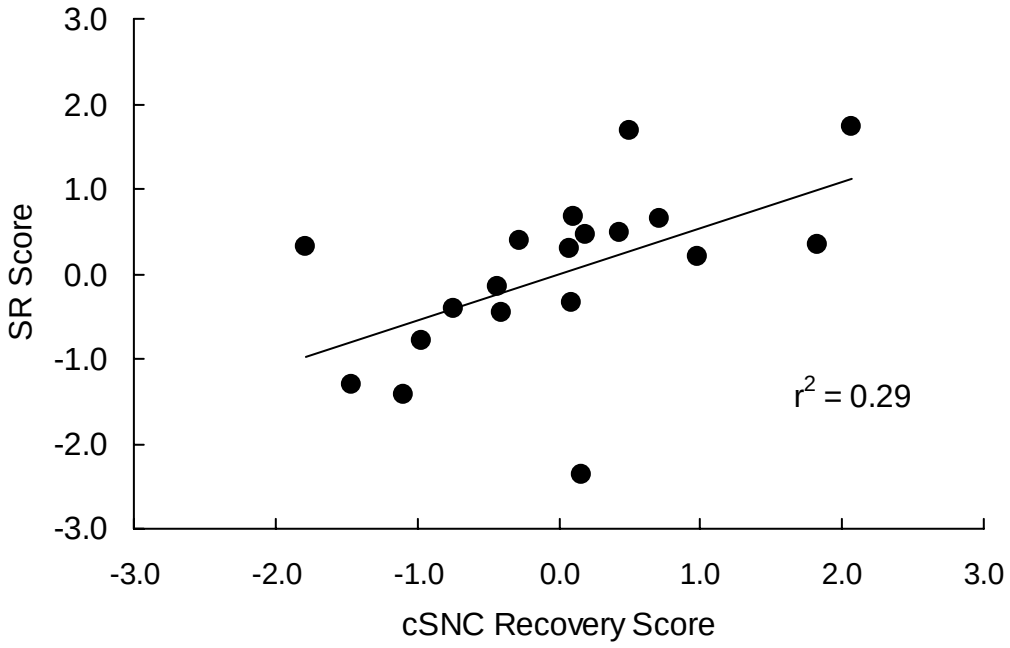

Figure 7. Correlation between SR and recovery from cSNC. A Pearson product-moment correlation indicated that SR (trial 19/ trial 18) was significantly and positively correlated to recovery from cSNC (trial 12 – trial 11) in 96-h and 336-h resting periods. A low SR score indicates high SR, while a high SR indicates low SR. Ratio and differences scores were converted to z-scores.

Discussion

An experiment was conducted to investigate the occurrence of spontaneous recovery of cSNC using the standard between-subject design (see Table 1c). Rats received training under two conditions: 32→4 vs. 4→4. Once recovery from cSNC was complete, rats in each contrast condition were randomly assigned to 24-h, 96-h, or 336-h resting periods. At the end of the resting period, all rats received 2 additional trials of access to the 4% solution. Overall, results failed to show significant differences in consummatory suppression during tests for SR (trial 19) at 96-h and 336-h resting periods and accordingly failed to show greater SR at 336-h compared to 96-h. However, using a median split to group downshifted subjects as high spontaneous recovery (H) or low spontaneous recovery (L) in terms of the performance on trial 19 revealed three findings: (1) H_{96} and H_{336} subjects showed significant consummatory suppression compared to L_{96} and L_{336}, and to unshifted controls; (2) L_{96} and L_{336} subjects showed significantly faster recovery (trial 12 – trial 11) from initial contrast than their respective H counterparts; and (3) a significant positive correlation was observed between degree of consummatory suppression on trial 19 and recovery from contrast (trial 12 – trial 11). These results indicate that SR may occur in cSNC with individual differences corresponding to initial recovery from contrast playing a role.

Individual differences have been shown to contribute to behavior in cSNC, such as recovery. Individual differences in opioid sensitivity play a role in recovery from cSNC (Pellegrini et al., 2005). In the first phase, Pellegrini and colleagues used the cSNC paradigm to classify animals as either fast-recovery or slow-recovery. This was accomplished in the following manner: First rats were placed into quadruplets, matched by trial 11 performance; second, a difference score for goal tracking was calculated for each rat by subtracting trial 12 from trial 11, assessing speed of recovery; and lastly, in each quadruplet, the two rats with the

highest trial 12 – trial 11 score were classified as fast-recovery animals and the two rats with lower scores were classified as slow-recovery animals. After full recovery from contrast, all subjects were placed back on free food for 10 days. In the second phase, all animals underwent an activity test in a runway to evaluate opioid sensitivity. Fifteen minutes prior to testing, half of the fast-recovery and half of the slow-recovery animals were administered naloxone (2 mg/kg, i.p.). The remaining half received saline injections. Pellegrini and colleagues found that slow-recovery individuals administered saline showed less habituation to the activity runway than either of the fast-recovery groups. However, the slow-recovery animals administered naloxone showed greater habituation to the activity runway. From these results, they concluded that individual differences in the opioid system may moderate recovery from cSNC.

Based on the effects of traditional anxiolytic drugs on cSNC, Flaherty (1996) argued cSNC serve as an anxiety model. Evidence indicates that individual differences play a role in other anxiety-related behaviors. For example, Ho, Eichendorff, and Schwarting (2003) demonstrated that performance in the elevated plus-maze (EPM) predicted performance on other anxiety-related tasks, such as object-burying and two-way avoidance. Rats were initially screened using the EPM. Based on the time spent in the open arms, the animals were divided into two subgroups with either “low” or “high” anxiety (LA or HA) levels. In the EPM, the percentage of time spent on, and the number of entries into open arms were lower in HA than in LA rats. In the object burying task, HA rats showed more burying behavior of Tabasco coated marbles, and in the active avoidance task, they showed slower acquisition of avoidance learning and higher escape latency as compared to LA rats.

Using this framework, Borta, Wöhr, and Schwarting (2006) demonstrated systematic differences between HA and LA rats in auditory fear conditioning. Researchers used an EPM to assess levels of anxiety. After a single day of testing in the EPM, animals were ranked by their

relative time spent on the open arms of the maze. Animals above or below the median were termed as rats with either high or low open arm time. Because, high open arm time indicated low anxiety and low open arm time indicated high anxiety, high open arm time rats were LA rats and low open arm time rats were HA rats. Several days later, animals underwent fear conditioning. Testing was performed on 3 consecutive days. On the first day, rats habituated to the shock chamber. On the second day each rat was exposed to six CS-US (tone→shock) pairings. On the third day, each rat was tested for freezing to the tone. It was found that HA animals spend significantly more time freezing during the second half of the test session than LA animals and more likely to emit ultrasonic vocalizations indicative of fear/anxiety (22 kHz). The HA rats showed greater fear conditioning than their LA counterparts, further showing the importance of individual differences in anxiety. Together these individual-difference studies are consistent with the current findings, providing evidence for individual differences in cSNC and other anxiety-related tasks.

One possible explanation for individual differences in SR of cSNC could be differences in memory retrieval for the contrast event. This explanation has been proposed for the analogous behavioral phenomenon of aversive-to-appetitive counterconditioning (see Figure 3). While there are several explanations that have been proposed for SR (see Rescorla, 2004a, for a review), the memory retrieval explanation best fits the present study. In this account, SR occurs because the inhibitory association fails to be retrieved while excitatory associations are readily retrieved. In a similar fashion, one might conceive of SR of cSNC as a result of a memory retrieval process (as shown in Figure 8). During the initial downshift event the CS (sipper tube) is paired with an aversive US (primary frustration), leading the animal to suppress drinking response. Over the course of phase 1 (trial 11 in this case), the primary frustration creates one CS-US association, sipper tube→$e_{Frustration}$. In phase 2 (trials 12-18), during recovery from contrast, the CS signals an

appetitive US (4% sucrose solution), leading the animal to perform approach responses. During recovery, three CS-US associations are retrieved: sipper tube→$e_{Frustration}$, an excitatory association that elicits avoidance behavior; sipper tube−●$e_{Frustration}$, an inhibitory association that interferes with avoidance behavior; and sipper tube→$e_{4\%}$, an excitatory association that elicits appetitive responding, such as drinking. Retrieval of the sipper tube→$e_{4\%}$ and sipper tube−●$e_{Frustration}$ associations results in increases in goal tracking, while simultaneously conflicting with avoidance responding when the sipper tube→$e_{Frustration}$ is retrieved. The net result is an increase in drinking behavior and a decrease in avoidance behavior.

In the SR test phase (Figure 8C), only two memories are retrieved: sipper tube→$e_{Frustration}$, an excitatory association that elicits avoidance behavior; and sipper tube→ $e_{4\%}$, an excitatory association that elicits drinking. Retrieval of the sipper tube→ $e_{4\%}$ association results in increased goal tracking, while simultaneously conflicting with aversive responding when the sipper tube→$e_{Frustration}$ association is retrieved. However, during the resting period the inhibitory pairing, sipper tube−●$e_{Frustration}$ weakens, as inhibitory pairings are more easily forgotten over time. Because there is no inhibitory pairing suppressing avoidance behavior (allowing for strong retrieval of the aversive memory), the net result is an increase in avoidance behavior, causing SR.

Individual differences in this retrieval process may occur due to differences in opioid sensitivity. H subjects may have retrieved the initial event more successfully than L counterparts, caused by individual differences in the opioid system, which has been shown to play a role in recovery from cSNC (Pellegrini et al., 2005). Studies of the opioid system and memory retrieval have concluded that mu and delta antagonists and kappa agonists enhance memory retrieval for aversive events. Studying memory retrieval and the opioid system using passive avoidance in mice, Ilyutchenok and Dubrovina (1994) found that after a 21-day resting period, mice injected

with either naloxone, ICI 174,864 (mu and delta antagonist), or dynorphin (kappa agonist) showed greater mean step through latency, indicating greater memory retrieval, relative to saline controls. Taking these memory retrieval results in conjunction with Pellegrini et al.'s (2005) results showing that slow recovery animals exhibit a higher sensitivity to the nonselective opioid-receptor antagonist naloxone, and the current experiment's finding that H animals show slower recovery, one might argue the following explanation for SR of cSNC. Individuals experiencing high sensitivity to naloxone and slow recovery from cSNC (trial 12 – trial 11) can retrieve aversive memories of the downshift event more efficiently than individuals that are less sensitive to naloxone and recover faster from cSNC during SR test trials via memory retrieval differences of the inhibitory association, sipper tube$-\bullet e_{Frustration}$.

However, there is an alternative explanation for individual differences in SR of cSNC. It is possible that differences in SR of cSNC are a result of individual differences in memory consolidation, not retrieval. All subjects within a resting period may have retrieved their memory of the event equally well, but the content of the memory may have varied, causing differences in consummatory suppression. H subjects may have consolidated the initial event as more aversive than L counterparts, caused by individual differences in the opioid system, which has been shown to play a role in recovery from cSNC (Pellegrini et al., 2005).

The opioid system has been shown to regulate memory for aversive events. McNally and Westbrook (2003) showed that posttrial injections of naloxone, retarded extinction of fear conditioning, leading them to conclude that opioid receptors modulate memory of aversive events. Similarly Wood, Norris, and Papini (2006) administered injections after trial 11 of the kappa agonist U-50,4886H or saline in the cSNC situation. Downshifted animals administered 3 mg/kg of U-50 recovered more slowly from contrast than saline animals or those administered 1 mg/kg of U-50. From these results, it was concluded that kappa agonists enhanced consolidation

of the aversive downshift event. Taking these memory consolidation results in conjunction with Pellegrini, et al. (2005) results and the current experiment's finding that H animals show slower recovery, one might argue for a similar hypothesis to that outlined above, except that the critical mechanism would be individual differences in the consolidation of the aversive memory of the incentive downshift episode (a "bigger" memory for the aversive event).

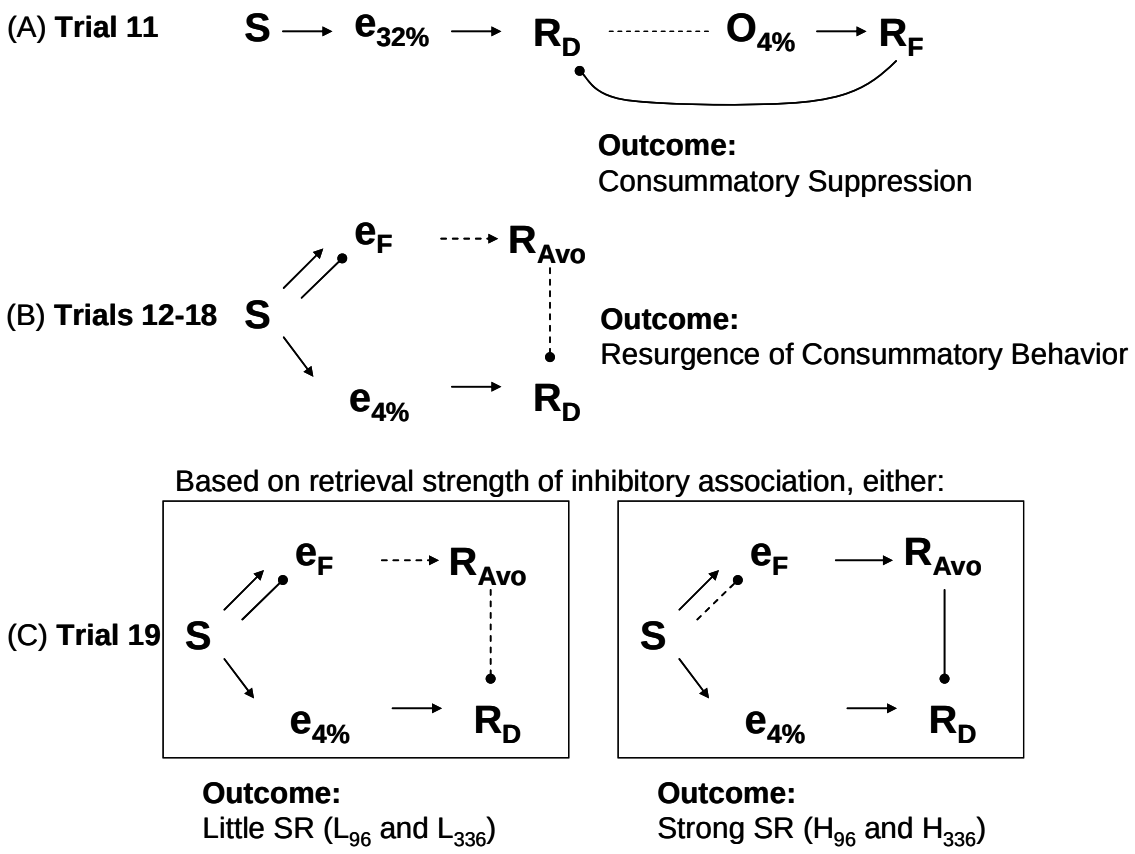

Figure 8. Diagram applying Bouton and Peck's (1992) memory retrieval explanation to SR of cSNC. (A) In trial 11, the conditioned stimulus (S) elicits primary frustration (R_F) from the mismatch between $e_{32\%}$ and $O_{4\%}$. The outcome is consummatory suppression. (B) In trials 12-18 during recovery, S elicits expectations of frustration (e_F), S elicits expectations of reward $e_{4\%}$, evoking a drinking response (R_D) and an inhibitory expectation, S−●e_F. The net outcome is resurgence of consummatory behavior. After trial 18, a resting period is interpolated. (C) In trial 19, some rats strongly retrieve the inhibitory association, S−●e_F. These rats show little SR. However, other rats do not strongly retrieve S−●e_F. It reduces over the course of the resting period. Therefore these animals fail to retrieve S−●e_F, allowing e_F to be more successfully retrieved and consummatory suppression to SR. Proposed opioid action is on the inhibitory association, S−●e_F.

Future Directions

The current experiment contained four weaknesses, which are explored here along with other possible future directions. First, several subjects were used as saline controls in other cSNC experiments. Such subjects received i.p. saline injections at various times throughout the experiment. Some were injected before the initial downshift trial (trial 11), some subjects were injected after the initial downshift, and still others were injected prior to trial 12. While an evaluation of the data did not reveal any relationship between receiving a saline injection and individual performance of SR, further investigation of SR of cSNC should be done with noninjected rats.

A second experimental weakness was the exclusion of female subjects. Flaherty (1996) reviewed research suggesting that sex made no difference with respect to cSNC. Flaherty concluded that sex differences do not occur in cSNC, but may be revealed in recovery from contrast under special circumstances such as septal lesions or shifts to sucrose-quinine solutions. However, some unpublished data from our laboratory along with evidence from opioid research suggests otherwise. In our lab, female subjects have shown more erratic consummatory behavior with more variability in goal tracking time measures.

With respect to sex differences in the opioid system, Klein, Popke, and Grunberg (1998) showed that male rats are more susceptible to the effects of the opioid drug naloxone than females. Researchers exposed male and female Wistar rats to 10 min of mild, unpredictable footshock stress. These were compared to male and female rats in a no-stress condition. Following stress or no-stress conditions, researchers injected subjects with naloxone or saline. In males, naloxone induced freezing following stress but had no effect on freezing following no-stress. But in females, regardless of the condition, naloxone did not affect freezing. From these results Klein, et al. suggested that sex differences may exist with respect to the role of

endogenous opioids under stress. These results show that future research into SR of cSNC should take sex differences into consideration. Once the SR of cSNC has solidified as a phenomenon, future research should seek to incorporate female subjects.

A third experimental weakness was to match groups on the number of postshift trial, rather than their performance. As exemplified by the trial 12 – trial 11 recovery score, it is apparent that some subjects recover faster from contrast than others, taking fewer trials to fully recover than those who recover more slowly from contrast. The fast recovery animals may have shown less SR because they underwent more extensive counterconditioning than the slow recovery animals. This is based on Rescorla's (2004a) third empirical property of SR: SR declines with repeated extinction, commonly appearing as a reduced amount of SR after multiple extinction trials. In a similar fashion, fast recovery animals may have shown less SR because they were given excessive aversive-to-appetitive counterconditioning during the postshift phase. In the future, instead of matching rats in terms of number of postshift trials, each downshifted individual animal should be assessed on its own. One possible measure of recovery that may serve valuable in this purpose could be using a master-yoked design. Each downshifted animal could be yoked to an unshifted animal. When the master animal's goal tracking time recovers to some minimum percentage of the yoked animal's goal tracking time, the master animal is considered recovered and placed into its respective resting period. After waiting its appropriate resting period, both master animal and yoked animals undergo SR test trials. Or one could also vary number of recovery trials on a group basis. In this design, conditions would vary with respect to the number of recovery trials based on group averages, while factors such as resting period remained constant.

A fourth experimental weakness was the limited number of resting periods. The current experiment focused on three resting periods, 24, 96, and 336 h. These may not have been the

optimal resting periods for observing SR of cSNC. Traditionally, SR in extinction situations increases over time. Therefore, one might observe greater SR of cSNC using a longer resting period. However, it is not reasonable to expect that SR should increase indefinitely. Flaherty's (1996) cSNC review showed that contrast is eliminated if a 336-h resting period is interpolated between the last preshift trial and the first postshift trial. Additionally in the current experiment, the 336 hour resting period did not induce more SR than a 96 hour resting period. Using group averages did not reveal any significant differences in consummatory suppression between 96-h and 336-h resting periods. Moreover, while approaching significance, using median splits also failed to reveal significant differences. Future research should address this issue by using resting periods between 48 h to 336 h to better plot the time course of SR.

Future research can gain greater understanding of SR of cSNC with the following manipulations. First, research should increase the sample size to see if the recovery score (trial 12 – trial 11) predicts SR. The current experiment classified subjects as either H or L suppressors. Using this classification, we were able to reveal that L_{96} and L_{336} subjects recovered faster (trial 12 – trial 11) from initial contrast than their respective H counterparts. Moreover, a significant positive correlation between degree of consummatory suppression on trial 19 and recovery from contrast (trial 12 – trial 11) was observed. However, these are post hoc analyses. A stronger argument for the impact of recovery from contrast on SR could be made if recovery (trial 12 – trial 11) were used to predict SR performance.

Second, one could evaluate the memory retrieval/consolidation explanations using various opioid drugs. Future research could alter SR with pharmacological manipulations on trial 19. A future experiment could utilize opioid drugs to study memory retrieval using the following design: First, categorize subjects as either fast recovery or slow recovery animals based on trial 12 – trial 11. Second, prior to SR test trials, inject half the animals with one of the opioid drugs,

such as naloxone; the other half receiving saline injections. A successful pattern of results would show that slow recovery naloxone animals show significantly greater SR of cSNC than their saline counterparts. An alternative experiment to investigate the memory retrieval/consolidation explanations using opioid drugs draws from Wood, Norris, and Papini (2006). Recall that these researchers administered posttrial 11 injections of the kappa agonist, U-50,4886H or saline in the cSNC situation. Downshifted animals administered 3 mg/kg of U-50 recovered more slowly from contrast than saline animals or those administered 1 mg/kg of U-50. From these results, it was concluded that kappa agonists enhanced consolidation of the aversive downshift event. A future experiment could utilize the following design to study memory consolidation: (1) following the initial downshift event, match subjects for trial 11 performance; (2) administer posttrial 11 injections of U-50; (3) allow the animals to fully recover from cSNC; and (4) test the animals for SR of cSNC. A successful pattern of results would show that animals treated with U-50 recover more slowly from initial contrast (an indicator of strong memory consolidation) and show greater SR of cSNC during subsequent test trials.

Summary

An experiment was conducted to investigate occurrence of SR of cSNC. Rats received training under two conditions: 32→4 vs. 4→4. Once recovery from cSNC was complete, rats in each contrast condition were randomly assigned to 24-h, 96-h, or 336-h resting periods. After the resting period, rats received 2 additional trials of access to the 4% solution. Results failed to show significant differences in consummatory suppression during tests for SR (trial 19) at 96-h and 336-h resting periods and accordingly failed to show greater SR at 336-h compared to 96-h. Using a median split to group downshifted subjects as high (H) or low (L) in terms of the performance on trial 19 revealed that H_{96} and H_{336} subjects showed significant consummatory suppression compared to L_{96} and L_{336}, and to unshifted controls. Second, L_{96} and L_{336} subjects

showed significantly faster recovery (trial 12 – trial 11) from initial contrast than their respective H counterparts. Third, there was a significant positive correlation between degree of consummatory suppression on trial 19 and recovery from contrast (trial 12 – trial 11). Based on the results indicating that SR may occur in cSNC with individual differences corresponding to initial recovery from contrast playing a role, in conjunction with other cSNC studies which have looked at opioid sensitivity, possible explanations involve differences in memory retrieval and memory consolidation, mediated by the opioid system.

References

Amsel, A. (1992). *Frustration theory: An analysis of dispositional learning and memory,* Cambridge, UK: Cambridge University Press.

Becker, H. C. (1986). Comparison of the effects of the benzodiazepine midazolam and three serotonin antagonists on a consummatory conflict paradigm. *Pharmacology Biochemistry and Behavior,* **24**, 1057-1064.

Becker, H. C., & Flaherty, C. F. (1983). Chlordiazepoxide and ethanol additively reduce gustatory negative contrast. *Psychopharmacology,* **80**, 35–37.

Borta, A., Wöhr, M., & Schwarting, R. K.W. (2006). Rat ultrasonic vocalization in aversively motivated situations and the role of individual differences in anxiety-related behavior. *Behavioural Brain Research,* **166**, 271–280

Bouton, M. E. (1991). Context and retrieval in extinction and in other examples of interference in simple associative learning. In L. Dachowski and F. Flaherty (Eds.) *Current topics in animal learning: Brain, emotion, and cognition* (pp. 25-33). Hillsdale, NJ: Lawrence Erlbaum.

Bouton, M. E. (1993). Context, time, and memory retrieval in the interference paradigms of pavlovian learning. *Psychological Bulletin,* **114**, 80-99.

Bouton, M. E. (2004). Context and behavioral processes in extinction. *Learning and Memory,* **11**, 485-494.

Bouton, M. E., Peck, C. A. (1992). Spontaneous recovery in cross-motivational transfer (counterconditioning). *Animal Learning and Behavior,* **20**, 313-321.

Brooks, D. C. (2000). Recent and remote extinction cues reduce spontaneous recovery. *Quarterly Journal of Experimental Psychology,* **53B**, 25-58.

Crespi, L. R. (1942). Quantitative variation of incentive and performance in the white rat. *American Journal of Psychology,* **40***,* 467-517.

Davis, M. (1979). Morphine and naloxone: Effects on conditioned near as measured with the potentiated startle paradigm. *European Journal of Pharmacology,* **54***,* 341-347.

Domjan, M. (2003). *The principles of learning and behavior, 5th edition.* Belmont, CA: Wadsworth.

Elliot, M. S. (1928). The effect of change of reward on the maze performance of rats. *University of California Publications in Psychology,* **4***,* 19-30.

Fanselow, M. S. (1981). Naloxone and Pavlovian fear conditioning. *Learning and Motivation,* **12***,* 398-419.

Fanselow, M. S., Calcagneti, D. J., & Helmstetter, F. J. (1989). Delta opioid antagonist, 16-Me Cyprenorphine, selectively attenuates conditional fear- and DPDPE-induced analgesia on the formalin test. *Pharmacology Biochemistry and Behavior,* **32***,* 469-473.

Flaherty, C. F. (1990). Effects of anxiolytics and antidepressants on extinction and negative contrast. *Pharmacology and Therapeutics,* **46**, 309–320.

Flaherty, C. F. (1996). *Incentive relativity.* Cambridge, UK: Cambridge University Press.

Flaherty, C. F., & Avdzej, A. (1974). Bidirectional contrast as a function of rate alteration of two sucrose solutions. *Bulletin of the Psychonomic Society,* **4***,* 505-507.

Flaherty, C. F., Becker, H. C., Driscoll, C. (1982). Conditions under which amobarbital sodium influences consummatory contrast. *Physiological Psychology,* **10***,* 122-128.

Flaherty, C. F., Becker, H. C., Checke, S., Rowan, G. A., & Grigson, P. S. (1992). Effect of chlorpromazine and haloperidol on negative contrast. *Pharmacology Biochemistry and Behavior,* **42***,* 111-117.

Flaherty, C. F., Becker, H. C., & Pochorecky, L. (1985). Correlation of corticosterone elevation and negative contrast varies as a function of the postshift day. *Animal Learning and Behavior,* **13**, 309-314.

Flaherty, C. F., & Checke, S. (1982). Anticipation of incentive gain. *Animal Learning and Behavior,* **10**, 177-182.

Flaherty, C. F., Hrabinski, K., & Grigson, P. S. (1990). Effect of taste context and ambient context changes on successive contrast. *Animal Learning and Behavior,* **18**, 271-276.

Flaherty, C. F., & Largen, J. (1975). Within-subjects positive and negative contrast effects. *Journal of Comparative and Physiological Psychology,* **88**, 653-664.

Gray, J. A. (1987). *The psychology of fear and stress.* Cambridge, UK: Cambridge University Press.

Helmstetter, F. J., & Fanselow, M.S. (1987) Effects of naltrexone on learning and performance of conditional fear-induced freezing and opioid analgesia. *Physiology and Behavior,* **39**, 501-505.

Ho, Y., Eichendorff, J., & Schwarting, R. K. W. (2003). Individual response profiles of male Wistar rats in animal models for anxiety and depression. *Behavioural Brain Research,* **136**, 1-12.

Ilyutchenok, R. Y., Dubrovina, N. I. (1994). Memory retrieval enhancement by kappa opioid agonist and mu, delta antagonists. *Pharmacology Biochemistry and Behavior,* **52**, 683-687.

Klein, L. C., Popke, E. J., & Grunberg, N. E. (1998). Sex differences in effects of opioid blockade on stress-induced freezing behavior. *Pharmacology Biochemistry and Behavior,* **61**, 413–417.

Liao, R., & Chuang, F. (2003). Differential effects of diazepam infused into the amygdala and the hippocampus on negative contrast. *Pharmacology Biochemistry and Behavior,* **74***,* 953-960.

Mackintosh, N. J. (1974). *The psychology of animal learning.* Oxford, UK: Academic Press.

McNally, G. P., & Westbrook, R. F. (2003a). Opioid receptors regulate the extinction of Pavlovian fear conditioning. *Behavioral Neuroscience,* **117***,* 1292-1301.

McNally, G. P., & Westbrook, R. F. (2003b). Temporally graded, context-specific retrograde amnesia and its alleviation by context preexposure: Effects of postconditioning exposures to morphine in the rat. *Journal of Experimental Psychology: Animal Behavior Processes*, **29***,* 130-142.

Mustaca, A. E, Bentosela, M., & Papini, M. R. (2000). Consummatory successive negative contrast in mice. *Learning and Motivation,* **31***,* 272-282.

Papini, M. R. (in press). Role of surprising nonreward in associative learning. *Japanese Journal of Animal Psychology.*

Papini, M. R. (2003). Comparative psychology of surprising nonreward. *Brain, Behavior and Evolution,* **62***,* 83-95.

Papini, M. R., & White, N. (1994). Performance during signals of reward omission. *Learning and Motivation,* **25***,* 45-64.

Pavlov, I. P. (1927). *Conditioned Reflexes.* Oxford, UK: Oxford University Press.

Pellegrini, S., Wood, M., Daniel, A., Papini, M. R. (2005). Opioid receptors modulate recovery from consummatory successive negative contrast. *Behavioural Brain Research,* **164**, 239-249.

Pietraszek, M., Sukhanov, I., Maciejak, P., Szyndler, J., Gravius, A., Wisłowska, A., Płaźnik, A., Bespalov, A., & Danysz, W. (2005). Anxiolytic-like effects of mGlu1 and mGlu5 receptor antagonists in rats. *European Journal of Pharmacology,* **514***,* 25-34.

Quirk, G. J. (2002). Memory for extinction of conditioned fear is long-lasting and persists following spontaneous recovery. *Learning and Memory,* **9***,* 402-407.

Rescorla, R. A. (2004a). Spontaneous recovery. *Learning and Memory,* **11***,* 501-509.

Rescorla, R. A. (2004b). Spontaneous recovery varies inversely with the training-extinction interval. *Learning and Behavior,* **32***,* 401-408.

Robbins, S. J. (1990). Mechanisms underlying spontaneous recovery in autoshaping. *Journal of Experimental Psychology: Animal Behavior Processes,* **16***,* 235-249.

Rowan, G. A., & Flaherty, C. F. (1987). The effects of morphine in the consummatory contrast paradigm. *Psychopharmacology,* **93***,* 51-58.

Santos, J. M., Gárgaro, A. C., Oliveira, A. R., Masson, S., & Brandão, M. L. (2005). Pharmacological dissociation of moderate and high contextual fear as assessed by freezing behavior and fear-potentiated startle. *European Neuropsychopharmacology,* **15***,* 239-246.

Szyndler, J., Sienkiewicz-Jarosz, H., Maciejak, P., Siemiatkowski, M., Rokicki, D., Członkowska A., & Płaźnik, A. (2001). The anxiolytic-like effect of nicotine undergoes rapid tolerance in a model of contextual fear conditioning in rats. *Pharmacology Biochemistry and Behavior,* **69***,* 511-518.

Tinkelpaugh, O.L. (1928). An experimental study of representative factors in monkeys. *Journal of Comparative Psychology,* **8***,* 197-236.

Vogel, J. R., Mikulka, P. J., & Spear, N. E. (1968). Effects of shifts in sucrose and saccaharine concentrations on licking behavior in the rat. *Journal of Comparative and Physiological Psychology,* **66***,* 661-666.

Wasserman, E. A., Franklin, S. R., & Hearst, E. (1974). Pavlovian appetitive contingencies and approach versus withdrawal to conditioned stimuli in pigeons. *Journal of Comparative and Physiological Psychology,* **86***,* 616-627.

Williams, B. A. (1997). Varieties of contrast: A review of *Incentive relativity* by Charles Flaherty. *Journal of the Experimental Analysis of Behavior,* **68***,* 133-141.

Wood, M. D., Daniel, A. M., & Papini, M. R. (2005). Selective effects of the δ−opioid receptor agonist DPDPE on consummatory successive negative contrast. *Behavioral Neuroscience,* **119**, 446-454.

Wood, M., Norris, J., & Papini, M. (2006). *Administration of the kappa opioid receptor agonist U-50,488H after the first downshift trial prolongs recovery from consumatory successive negative contrast.* 52nd Annual Meeting of the Southwestern Psychological Association: Austin, TX.

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	Norris, J., Dougan, J. (2004). *Effects of disruptive events on time-place learning in rats.* 2004 Convention for the International Association for Behavioral Analysis: Boston, MA.
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ABSTRACT

SPONTANEOUS RECOVERY OF CONSUMMATORY SUCCESSIVE NEGATIVE CONTRAST

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Spontaneous Recovery (SR) refers to the reemergence of an extinguished conditioned response after a resting period. SR occurs in a variety of Pavlovian situations, but remains to be shown in the consummatory successive negative contrast (cSNC) situation. Results failed to show SR at 96-h and 336-h resting periods and accordingly failed to show greater SR at 336-h compared to 96-h. However, a median-split procedure to group downshifted subjects as high spontaneous recovery (H) or low spontaneous recovery (L) in the SR trial, revealed: (1) H_{96} and H_{336}, but not H_{24}, showed significant consummatory suppression compared to L and unshifted controls; (2) L_{96} and L_{336} showed significantly faster recovery from initial contrast than their respective H counterparts; and (3) A significant positive correlation between degree of SR on trial 19 and recovery from contrast. Results indicate any demonstration of SR in cSNC must take into account individual differences in consummatory behavior.