Deepened Extinction of Conditioned Suppression in Rats and Implications for the Treatment of Behavioral Disorders

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Extinction has been both highly researched and highly implemented in the treatment of addictions and psychological disorders. Although the stability of extinction has been in question, research continues to explore the extinction of drug cues in animal models that may be more successfully applied to the treatment of drug addiction in humans (Tobena, Fernandez-Teruel, Escorihuela, Nunez, & Zapatin, 1993). Many theories and models of the mechanisms of extinction exist, however, the basis of extinction involves the weakening of previously learned associations between cues and stimuli by presenting the cues without the stimuli (Myers & Carlezon, 2010). This has been applied to the treatment of drug addictions by seeking to weaken the association between drug cues and drug cravings or drug-seeking behaviors, feared stimuli and avoidance behaviors in disorders such as phobias and obsessive compulsive disorder, as well as using extinction to decrease inappropriate behaviors in developmental disorders such as autism spectrum disorders.

The effectiveness of extinction as a treatment for drug addictions has been weakened because of the tendency of extinction to be rather context-specific (Tobena, et al., 1993), limiting the likelihood that such treatments would generalize across all contexts and increasing the likelihood for relapse. However, drug-associated stimuli play an important role in drug addiction and thus the association between drug cues and drug-related stimuli and drug cravings are an important area to target for the treatment of drug addictions. This experiment is part of several experiments conducted under a single grant to further investigate procedures that will more effectively weaken the association between drug cues and the drug.

It is believed that drug-associated stimuli increase drug cravings and urges and increase the risk of relapse when presented with such stimuli (Perkins, 2009). Studies have shown that stimuli repeatedly presented with rewarding drugs begin to increase dopamine levels in the brain

when presented without the drugs (Volkow, et al., 2006). The stimuli become associated with the rewarding drug after repeated pairings such that the drug-associated stimuli can cause the same effects in the brain that drug use has been shown to cause. Since dopamine is a neurotransmitter that is believed to be involved in the rewarding and reinforcing effects of drug use, the ability of drug-associated stimuli to similarly increase dopamine levels is an indication of the role drug-associated stimuli may play in drug use and addiction (Volkow, et al., 2006). Dopamine is also involved in the prediction of reward, which may be an indicator for the mechanism by which drug cues increase drug-seeking behavior. Thusly, the increases in dopamine after exposure to drug cues in individuals with drug addictions would be associated with increases in drug cravings. (Volkow, et al., 2006) Accordingly, exposure to drug-related stimuli has been shown to elicit drug-craving and drug-seeking behaviors (Conklin & Tiffany, 2002).

This highlights an important area for further research into treatments for drug addiction, since drug-related cues can greatly hinder treatment if they cause relapse by way of eliciting drug cravings (Marlies, Ingmar, Peter, Wim van den, & Vincent, 2007). Breaking the association between drug cues and the drug may work to prevent relapse risk when presented with drug stimuli, such as in cue-exposure treatment. Cue-exposure is a basic application of response extinction, based on the classical conditioning model of learning. The conditioned stimulus, drug cues, through repeated pairings with the unconditioned stimulus, the drug, eventually is able to elicit the conditioned response, even when presented on its own. Cue-exposure involves breaking the association through repeated unreinforced exposures to drug-associated stimuli in order to break the contingency between the drug cues and behavioral response, such as drug cravings or drug-seeking behaviors (Conklin & Tiffany, 2002). By breaking this contingency, the conditioned stimuli (drug cues) no longer predict drug use and therefore do not elicit the

conditioned drug response (Marlies, et al., 2007). In respect to treatment of drug addiction, cue-exposure therapy usually entails exposure to drug cues such as drug paraphernalia or imaging situations typical of drug use (Conklin & Tiffany, 2002).

The development of treatments for and ongoing research into drug addiction is dependent upon greater understanding of drug relapse and reinstatement of drug-seeking behaviors. Theories of extinction and conditioning provide a base for the mechanisms of learning, such as how associations between drug cues and drug seeking behaviors are created, as well as the mechanisms of how to weaken or unlearn associations. An understanding of how we make associations is a crucial factor in determining how to approach breaking associations or creating new associations, which can then provide a starting point to developing effective clinical treatments for disorders such as drug addictions. Although there are many theories of extinction, the two main conflicting classes of extinction theories involve whether or not extinction is a result of forgetting or whether it is a result of new associations becoming more salient (Falls, 1998).

Extinction in itself is a form of learning that involves weakening of the associations between cues and the events they predict (Myers & Carlezon, 2010). The basic procedure of extinction involves non-reinforced presentation of the conditioned stimulus so that the CS is no longer predictive of an event and thus does not elicit the conditioned response. The first class of extinction theories assumes that extinction is a destruction of the original learning (Bouton, 2007). This perspective of extinction posits the idea that extinction involves unlearning of the previous associations between cues and the events that they predict (Delamater, 2004). The original learning of the association between cues and the events they predict is erased. The

association is not simply weakened under this theory, but it is erased such that that the CS cannot elicit the conditioned response.

The second class of theories takes a different approach and argues that the original learning is not erased or destroyed, but is inhibited by new and conflicting associations (Falls, 1998). Under this perspective, the associations are preserved, but new learning occurs, and the new associations become more salient than the original learning, which is inhibited and suppressed. The new associations learned during extinction become inhibitory stimulus-response associations (Delamater, 2004).

In order for the first class of extinction theories to be true, it would be impossible to see relapse of conditioned responses. However, extinction research indeed shows that relapse does occur, and such research has uncovered four potential mechanisms of relapse that occur after extinction of original learning, including reinstatement, renewal, recovery, and reacquisition (Bouton, 2002). Reinstatement involves recovery of the conditioned response upon exposure to the unconditioned stimulus on its own. Renewal is the recovery of the conditioned response that occurs when the context is changed following extinction. Recovery occurs over time and involves recovery of the conditioned response upon presentations of the CS after a passage of time following extinction (Bouton, 2002). Finally, reacquisition involves recovery of the conditioned response upon paired presentation of the CS-US following extinction (Bouton, 2007).

According to the first class of extinction theories that posit the idea of extinction causing unlearning, these relapse phenomena could not be observed. If unlearning was indeed the case, presentations of either or both the conditioned or unconditioned stimulus would not produce recovery of the conditioned response, because the associations would have been unlearned and

the conditioned stimulus would no longer be predictive of any event. However, these phenomena do occur, indicating that extinction does not erase previous learning. The existence of relapse phenomena provide support for the idea that original associations are preserved but inhibited and extinction results in the formation of inhibitory associations. Pavlov was the first scientist to hypothesize about the formation of inhibitory associations as opposed to unlearning. His ideas were based on the observation of spontaneous recovery of responding even after much passage of time between extinction and testing, which led him to the conclusion that previously learned associations were preserved during and despite extinction. He instead hypothesized that the mechanism underlying the elimination of responding after extinction was due to the formation of inhibitory associations between the CS and the US (Delamater, 2004).

Although the phenomena of response relapse provides a mechanism for the disappearance of conditioned responding, it does not necessarily explain how the new association becomes more salient and is able to replace the original learning. Context may play a large role in determining which association takes precedence and in how the inhibitory associations are formed. It has been suggested that the context itself may serve as a cue that is predictive of the meaning of the conditioned stimuli. If the second model of extinction is correct, then the conditioned stimulus has two meanings by way of its multiple associations. One meaning may predict one outcome, and after extinction, the conditioned stimulus may predict another outcome, that is, the lack of the original outcome (Bouton, 2007).

Context may likely serve as the cue indicating which outcome is to be predicted by the conditioned stimulus and thus determine which association will be activated. If the conditioned stimulus is presented in the extinction context, the extinction context may serve as a cue that in this context, conditioned responses will go unreinforced. Thus, presentation of the conditioned

stimulus in the extinction context will activate the inhibitory association and conditioned responses will cease. However, if the conditioned stimulus is presented in any context other than the extinction context, then the inhibitory association will not be activated, and we will see renewal of conditioned responding elicited by the conditioned stimulus once again. Interestingly, only the inhibitory association requires context in order to be activated, unlike the excitatory association. Conditioning is less affected by changing contexts than is extinction (Bouton, 2007).

One specific theory of extinction that has been influential in the field of behavioral psychology is the Rescorla-Wagner Model of Learning. A key characteristic of the Rescorla-Wagner model is the element of surprise, which it states is crucial and necessary in order for learning to occur. If the unconditioned stimulus is not surprising, then according to the Rescorla-Wagner model, no associative learning will occur (Bouton, 2007). Accordingly, it assumes that the greatest amount of learning will occur in earlier trials of conditioning because the unconditioned stimulus is more surprising in the beginning trials (Renner, 2004).

As the association between the conditioned stimulus and the unconditioned stimulus increases in strength, the unconditioned stimulus will become less surprising and will be predicted by the conditioned stimulus. Once the conditioned stimulus consistently predicts the unconditioned stimulus, no more learning can occur. This also assumes that there is a maximum or upper limit of learning and that less learning occurs during later trials as the upper limit is reached (Miller & Barnet, 1995). Thus, there will be greater increases in associative strength from one trial to the next in the first few trials. As conditioning progresses, associative strength increases less and less until the upper limit is reached and the unconditioned stimulus ceases to be surprising (Bouton, 2007).

The Rescorla-Wagner model assumes that associative strength and the change in strength from one trial to the next can be predicted and determined using an equation. This equation, $\Delta V = \alpha\beta(\lambda - V)$, integrates the factors deemed important to learning, including predictive value (V), the upper limit of learning (λ), and the salience of both the conditioned stimulus (α) and the unconditioned stimulus (β). The change in predictive value and associative value is a function of salience and how surprising the unconditioned stimulus is. The term $\lambda - V$ tells us how surprising the unconditioned stimulus is by indicating the difference between the unconditioned stimulus and that predictive value of the conditioned stimulus. If the unconditioned stimulus is greater than the predictive value of the conditioned stimulus, then there is greater surprise, and thus greater learning as well. As conditioning progresses, the conditioned stimulus becomes a greater predictor of the unconditioned stimulus. The difference between the unconditioned stimulus and the predictive value of the conditioned stimulus decreases, and thus the change in associative strength from one trial to the next will decrease (Miller & Barnet, 1995). In the event that the presentation of two conditioned stimuli are compounded, then the sum of the predictive values of each stimulus determines the extent to which the unconditioned stimulus will be presented (Bouton, 2007).

The Rescorla-Wagner model also provides a model of extinction using the same equation, except that under extinction, the repeated presentations of a conditioned stimulus without an unconditioned stimulus is essentially equivalent to conditioning with an unconditioned stimulus of zero intensity (Bouton, 2007). According to this model, conditioned inhibition and conditioned excitation are exact opposites, with positive values indicating excitatory CS-US associations and negative values indicating inhibitory associations (Miller & Barnet, 1995). The change in associative strength will decrease from one trial to the next as the

conditioned stimulus loses its ability to predict the unconditioned stimulus. Eventually, associative strength will reach zero since the conditioned stimulus eventually no longer predicts the unconditioned stimulus at all and will cease to elicit the conditioned responses. However, this model also makes the unique assumption that since conditioned inhibition and conditioned excitation are on opposite ends of a spectrum of associative strength, that they cannot coexist. This contrasts with the models that argue that a conditioned stimulus may have multiple associations, both excitatory and inhibitory depending on which association is activated.

The characteristics of surprise and discrepancy in expectations are also key elements in the theory behind deepened extinction. Deepened extinction is based heavily in the error-correction elements of the Rescorla-Wagner model of extinction. As already mentioned, new learning ceases to occur when the unconditioned stimulus is preceded by a conditioned stimulus that has been strongly conditioned to stably predict the US (Rescorla, 2006). Error-correction models of extinction are similar to the Rescorla-Wagner model of extinction in that there is an error, or a discrepancy, between what is expected upon presentation of the conditioned stimulus and what actually occurs. In the Rescorla-Wagner model, this discrepancy between the unconditioned stimulus that is anticipated to occur and the unconditioned stimulus that actually occurs is noted as the amount of surprise upon presentation of the unconditioned stimulus. The correction of the error between the unconditioned stimulus anticipated and actually received indicates the increasing associative strength of the conditioned stimulus to predict the unconditioned stimulus (Rescorla, 2006).

Extinction occurs similarly by creating a greater discrepancy when the conditioned stimulus is no longer reinforced by the unconditioned stimulus. In the beginning extinction trials, the conditioned stimulus still holds strong associative power and will predict the unconditioned

stimulus. However, in extinction trials, the conditioned stimulus is no longer followed by the unconditioned stimulus. Thus, a greater error is generated when an unconditioned stimulus is expected and anticipated by the conditioned stimulus but not is not received. Eventually, the increased error signals will reduce the associative power of the conditioned stimulus, just as it enhanced the associative power in conditioning trials, until the conditioned stimulus loses its associative power and no longer predicts the unconditioned stimulus (Rescorla, 2006).

In deepened extinction, the intent is to generate an even greater enhanced error signal that will cause the conditioned stimulus to lose its associative power in predicting the unconditioned stimulus. Studies supporting this idea have shown that excitation after extinction trials enhances the effect that nonreinforcement has on the associate power of a conditioned stimulus (Rescorla, 2006). Deepened extinction ties in theories of inhibitory associations formed during extinction as well as the phenomena of the relapse of responding. Since deepened extinction involves the reinstatement of responding, it assumes that the learning still exists but is inhibited by the extinction context.

Deepened extinction induces reinstatement of responding by presenting the unconditioned stimulus during extinction trials, although non-contingent with the conditioned stimulus. Assuming that the original learning is inhibited in the extinction context, the presentation of the unconditioned stimulus should activate the excitatory association, and thus conditioned responding to the conditioned stimulus should be reinstated. This creates an enhanced error signal since the excitation activates excitatory responding, but extinction still continues and the conditioned stimulus goes unreinforced. The presentations of the unconditioned stimulus reactivate the association between the conditioned stimulus and the

unconditioned stimulus and thus the conditioned stimulus will likely cause anticipation for the unconditioned stimulus.

Since the presentations of the unconditioned stimulus are non-contingent, however, no reinforcement will be received for responding in the deepened extinction trials. The enhanced error signals are created when the conditioned stimulus once again causes anticipation of the unconditioned stimulus but no such reinforcement is received. This enhances the discrepancy between what is anticipated and expected and what is actually received. In this way, deepened extinction intends to further reduce the associative strength of the conditioned stimulus by continually generating error signals. In normal extinction, the error signals are reduced as the errors are corrected and ability of the conditioned stimulus to predict the unconditioned stimulus decreases as conditioning continues. In deepened extinction however, the error signals are re-generated to further reduce the association between the conditioned stimulus and the unconditioned stimulus and thus reduce the ability of the conditioned stimulus to predict the unconditioned stimulus (Rescorla, 2006). Under deepened extinction, we would expect to see even less responding to the conditioned stimulus because it has lost its associative power even further than in normal extinction trials.

Deepened extinction may also be used in conjunction with compound stimulus presentation, another method of enhancing the effects of extinction by enhancing the error signal created during extinction trials. In stimulus compound presentations, a conditioned stimulus that has undergone extinction trials on non-reinforcement is presented with another stimulus that creates a greater error signal and thus enhances the effects of extinction on the first stimulus (Rescorla, 2006). In reference to conditioned suppression procedures, we would expect to see more responding after deepened extinction or compound stimulus presentations because the

conditioned stimuli should no longer signal an aversive unconditioned stimulus. Studies have indeed shown that in such procedures, compound stimulus presentation of an extinguished exciter with another extinguished excitatory conditioned stimulus elicits more responding (that is, studies show less suppression of responding, as expected). The nonreinforcement of a compound presentation of extinguished stimuli was shown to decrease the associative strength between the stimuli and the unconditioned stimuli they previously signaled. This resulted in less suppression of behavior in the presence of the conditioned stimuli and eliminated the spontaneous recovery of responding seen when each stimuli was extinguished separately (Rescorla, 2006).

Deepened extinction is an important aspect of the drug reinstatement model, which is used specifically to research and understand the mechanisms underlying the relapse of drug-seeking behaviors that is commonly seen in animals in the laboratory and in humans undergoing treatment for addictions (Shaham, Shalev, Lu, de Wit, & Stewart, 2003). In the context of drug use and drug addictions, "reinstatement of drug seeking" is defined as the relapse of behaviors that were previously reinforced by the actual drug in response to drug-related stimuli even after such stimuli have undergone extinction (Shaham, et al., 2003). Deepened extinction under the drug reinstatement model seems to hold promise as a model of treatment to further prevent relapse of drug-seeking behaviors in drug addicts.

Drug-seeking behaviors tend to be resistant to extinction and relapse is often seen elicited in response to a host of different drug-related stimuli (Epstein, Preston, Stewart, & Shaham, 2006). Cues such as drug and drug-associated stimuli environments and stressors have been known to induce relapse and recovery of conditioned responding in rats, especially cocaine-related stimuli (Shaham, et al., 2003). Even when these drug-related cues are repeatedly

presented and unreinforced, such as in drug exposure treatments, studies have still that they are resistant to extinction and maintain their ability to elicit drug-seeking behaviors, even after long periods of time (Ciccocioppo, Sanna, & Weiss, 2001).

This clearly presents a problem for the treatment of drug addictions if drug-related stimuli and environmental factors still pose a risk of inducing relapse. In humans, the first several months provide the greatest risk of relapse, although relapse may occur after even longer periods of time (Ciccocioppo, et al., 2001). In order to develop more effective means of treatment for drug addictions, we must figure out more stable methods of extinguishing drug-related cues that are less resistant to extinction so that such treatments may have greater success in preventing relapse. Deepened extinction may hold a possible solution to such problems by enhancing the extinction effects of non-reinforcement. By potentiating the error signals during extinction, deepened extinction may weaken the association between drug-related stimuli and drug-seeking behaviors to the point that such stimuli hold less associative power and thus have a reduced ability to cause relapse of drug-seeking behavior.

The ability of non-contingent priming presentations of excitatory stimuli to reinstate conditioned responding has been consistently demonstrated and supported in similar studies. Previous studies have shown that priming injections of drugs after drug-associated stimuli have undergone extinction have indeed elicited a reinstatement of drug-seeking behaviors. Non-contingent presentations of reinforcing stimuli, including food and self-administered priming drug injections such as cocaine, amphetamine and heroin, reinstate the drug-seeking behaviors associated with the respective drugs. Interestingly, studies have also found that exposure to stressful stimuli such as shocks, bright lights or loud tones have caused reinstate of previously reinforced behavior (Shaham, et al., 2003). Such studies highlight the importance of developing

methods to reduce the associative strength between conditioned stimuli and the reinforcers they previously signaled to eliminate spontaneous recovery.

One goal of this study to examine different methods of using deepened extinction to enhance the extinction of conditioned stimuli since previous studies have failed to show significant results. Five preliminary experiments have investigated the use of deepened extinction to enhance the extinction of stimuli previously reinforced by either cocaine or food. These experiments failed to demonstrate the ability of deepened extinction to enhance the extinction of a stimulus and to eliminate spontaneous recovery. In each of the preliminary experiments, the results either showed higher rates of responding in the presence of the stimuli that experienced deepened extinction or showed no significant difference between the rates of responding in the deepened extinction and normal extinction groups (see Figures 1-3).

This purpose of this current study is to further investigate the ability of deepened extinction to better extinguish the ability of conditioned stimuli to anticipate an unconditioned stimulus and thus eliminate recovery of responding. This study utilized the drug-reinstatement model and deepened extinction to induce reinstatement of conditioned responding by priming with non-contingent presentations of the unconditioned stimulus. The goal was to ask whether the priming presentations of an aversive stimulus and the following compounded presentation of two extinguished stimuli would generate an enhanced error signal and reduce the conditioned suppression in the presence of the stimuli. Rats were presented with stimuli consisting of a light, tone and fan that were terminated in shock until the rats ceased responding in the presence of the light. After the rats experienced initial extinction of all of the stimuli, they were given non-contingent priming shocks at the beginning of additional trials during which only the tone or fan experienced extinction. A final test of the presentation of either the tone, fan, or the tone or fan

compounded with the previously extinguished light evaluated the relative amounts of extinction that occurred from the deepened extinction trials.

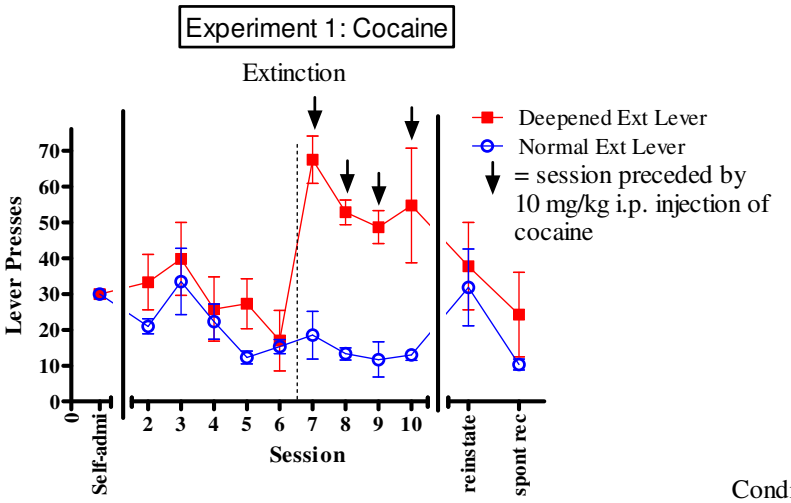

Figure 1

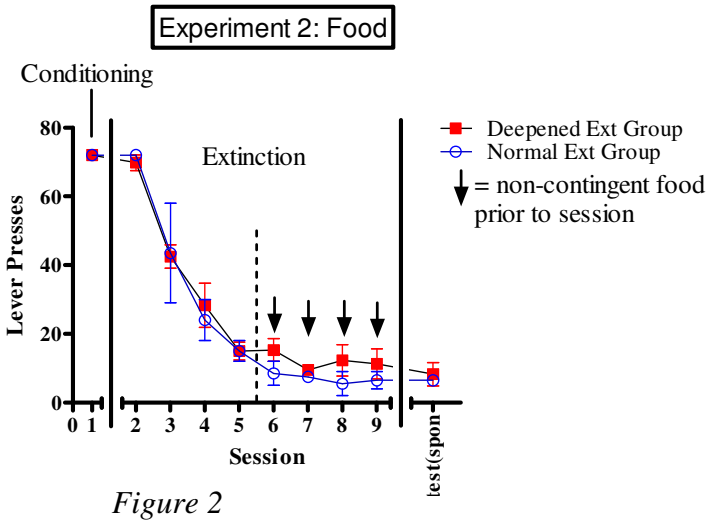

Figure 2

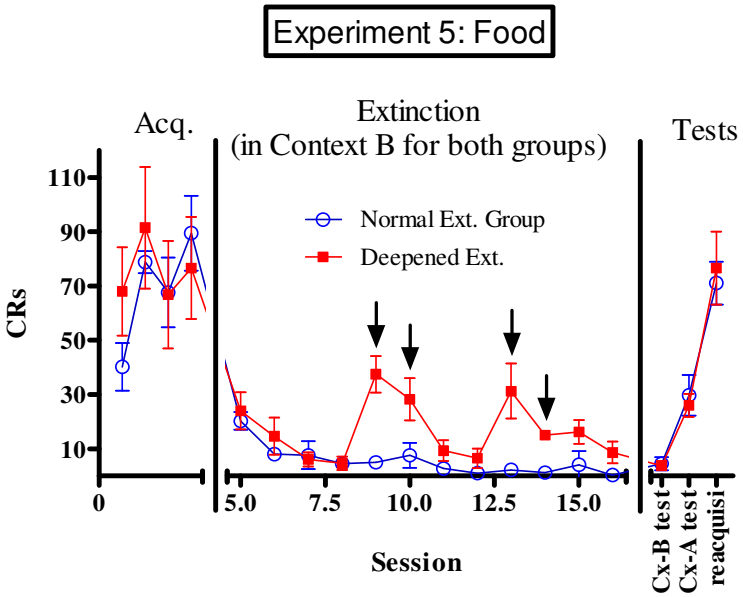

Figure 3

Method

Subjects and Apparatus

The subjects were 6 male adult Long-Evans. Subjects were placed on a food-deprivation diet that kept them at 85% of their ad-lib weights throughout the experiment. Free-feeding weights ranged from a minimum of 361 grams to a maximum of 415 grams. They had previously participated in an experiment in different apparatus' and with different stimuli, and thus previously conditioning should not interfere with the present experiment. Subjects lived in a colony room, which had a controlled 12 hour light/dark cycle (light was turned on at 8:00 am until 8:00 pm). They were housed individually in stainless steel cages and given unlimited access to water in their home cages. Subjects were given approximately 15 grams of food after daily conditioning sessions to maintain their weights at 85% of the ad-lib weights.

The apparatus consisted of six operant chambers measuring 8 in x 8 in x 8 in. The sidewalls of each chamber were white translucent plexiglass and the ceiling was clear plexiglass with several holes to circulate air; the front and rear walls were aluminum. The floor of each chamber was constructed of stainless steel bars .0125 inches in diameter and spaced .625 in apart. The bars of the grid floor were attached to shockers and scramblers that could deliver a 0.5-mA, 0.5-s shock that was subsequently adjusted for stimuli to cause suppression but not total disruption of behavior. Inside each chamber was a 2 in wide response lever, 0.75 in from the right of the rear wall and 2.5 in from the floor of the chamber. To the left of the lever was a feeder, located on the rear well, .5 in from the left, which delivered 45 mg food pellets (Research Diets). Mounted on top of the ceiling of each chamber was a fan, 4.5 inches in diameter and operated at 15 v DC. When turned on, the fan blew air through holes in the chamber ceiling to provide a moving air stimulus. Also mounted on each side of the chamber were 2 25-w tubular

frosted white light bulbs operated at 100 v AC. Light bulbs were mounted 3.75 in away from the side walls on the outside of the chamber and could be lit to provide a light stimulus during the dark sessions. Each chamber was housed in a sound attenuation chest (Weiss, 1970). A speaker was mounted on the ceiling of the sound attenuation chest, approximately 6-8 in above the apparatus that permitted the presentation of a 4000-Hz tone measuring approximately 80-dB.

Procedure

Magazine and Response Training. Initially, the rats received one magazine training session during which food pellets were delivered on a fixed ratio (FR) schedule of 1 in conjunction with a fixed time (FT) schedule of 2-min. Rats were trained to press the lever for a pellet, which was delivered contingently after each response. Rats also received non-contingent pellets on average every 2 minutes. The rats were then switched to a FR schedule of reinforcement during which rats received food pellets contingent upon lever-pressing in sessions that were approximately 45 minutes long. FR values gradually increased from FR1 to FR10 over several sessions that varied for each subject based on individual rates of responding.

Baseline Training. Once rats showed regular responding under the FR10 schedule, they were switched to a VI60 schedule of reinforcement in which the first lever press after an average of 60 seconds (range: 2-s to 240-s) since the last reinforcer was reinforced by delivery of a food pellet. The VI60 schedule continued for 12 sessions, each lasting approximately 45 minutes long.

Habituation. During the next two sessions, rats were presented with 3 30-s presentations of each the light, tone and fan. Every 4.5 min on average (range: 2.5-min to 6.5-min) rats were presented with one of the three stimuli for 30-s. The order of the stimuli was randomized in blocks of 3. In each block, each stimulus was presented once in a random order. Each session

lasted approximately 45 minutes and the VI60 schedule continued throughout the habituation sessions. During these first 2 sessions, stimuli were not followed by a shock in order to familiarize the rats with the stimuli so that the stimuli would not be aversive and cause a disruption of responding during the fear conditioning phase.

Fear Conditioning. Over the next several sessions, the rats were once again presented with 3 30-s presentations of the light, tone, and fan, with approximately 4.5 min in between presentations over the course of each approximately 45 min session. Stimuli were similarly presented in blocks of 3, with the order of the 3 stimuli randomized within each block. Each 30-s presentation of the stimuli was immediately followed by a .5-s, .5-mA shock. If suppression of responding was not evident, the shock intensity or duration was increased on an individual basis; similarly if there was a disruption of responding, the shock intensity or duration was decreased to maintain uninterrupted responding in the absence of the stimuli. The conditioned suppression was assessed in terms of the ability of the stimuli to disrupt lever-pressing. This was calculated by a suppression ratio of CS/(CS+Pre-CS), where CS is the response rate during the 30-s presentation of the stimulus and Pre-CS is the response rate in the 30-s period prior to the presentation of the stimulus. To better understand this ratio, a result of 0.5 indicates that the stimulus caused no disruption of responding during the stimulus, and a result of 0.0 indicates that the stimulus caused total suppression of responding. The criterion for progressing to the next phase of the experiment was a suppression ratio of ≤ 0.25 for each stimulus on 2 out of 3 consecutive days. The VI60 schedule of reinforcement continued throughout fear conditioning.

Extinction. After the criterion of ≤ 0.25 suppression ratio was met, rats received extinction with all three stimuli. Extinctions were identical to the habituation sessions in which rats were on a VI60 schedule of reinforcement and were presented with 3 30-s presentations of the stimuli in

blocks of three. The 30-s trials were not terminated with a shock. The extinction criterion was defined as at least three sessions with suppression ratio of ≥ 0.33 under the light and tone in the last session.

Deepened Extinction. Over the next 7 days, the rats underwent the second phase of extinction in which the rats received deepened extinction with either the fan or tone. The light did not receive either deepened or normal extinction during this phase. For each rat, one stimulus was selected to receive deepened extinction, and for whichever stimulus did not receive deepened extinction, the rats received normal extinction. The stimulus selected to receive deepened extinction was counterbalanced across subjects, such that some receive deepened extinction with the fan (and thus normal extinction with the tone) and others received deepened extinction with the tone (and normal extinction with the fan). The order of the stimuli presented was also counterbalanced across subjects such that some rats received the deepened extinction sessions first and then the normal extinction session, whereas other rats received normal extinction sessions first, followed by the deepened extinction sessions. Thus, there were four possibilities: deepened extinction with the fan followed by normal extinction with the tone, normal extinction with the tone followed by deepened extinction with the fan, deepened extinction with the tone followed by normal extinction with the fan, and normal extinction with the fan followed by deepened extinction with the tone.

The deepened extinction phase of conditioning consisted of 3 sessions of either normal or deepened extinction with either the fan or tone, followed by one session of VI60 as a baseline recovery session, and then 3 more sessions of either normal or deepened extinction with the stimulus that was not presented in the first three sessions. In the deepened extinction sessions, the rat received 3 0.5-s, 0.5-mA shocks every 5 minutes at the beginning of the session in the

absence of any stimuli. The sessions consisted of 6 trials of the one stimulus selected to experience deepened extinction, presented for 30-s with an average of 4.5 min between presentations. The stimuli were not followed by a shock.

The normal extinction phase was the same as the deepened extinction phase, but without the priming shocks at the beginning of the session. In the normal extinction sessions, the rats were presented with either the fan or tone for 30-s with an average of 4.5-min between trials for 3 sessions. The stimuli were not followed by a shock nor did the rat receive any shocks at the beginning of the session. The 3 normal extinction sessions were also followed by one session of VI60. All sessions lasted approximately 45 min and a VI60 schedule of reinforcement continued throughout extinction.

Test. After receiving the deepened extinction and normal extinction trials, rats received one test session to test the ability of stimulus compound presentation to enhance the extinction of the suppressed behavior. Rats were presented with four stimuli: the fan alone (F), the tone alone (T), the fan and the light presented together (FL), and the tone and the light presented together (TL). The rats received 4 30-s presentations of each stimuli, with the intertrial interval fixed at 90-s. The stimuli were presented in blocks of four such that in each block, each stimulus was presented once. The stimuli were also presented such that over the course of the four blocks, each stimulus was presented in each possible position (1^{st}, 2^{nd}, 3^{rd}, and 4^{th}) (See Figure 4).

Figure 4

Order of the Presentation of the Stimuli in Each Block of the Test Session

1st	2nd	3rd	4th
F	FL	T	TL
TL	F	FL	T
FL	T	TL	F
T	TL	F	FL

F- Fan, T-Tone, FL- Fan and Light, TL-Tone and Light

Results

Magazine and Response Training. Magazine and response training proceeded quickly. The rats were trained on a fixed ratio and variable interval schedule initially for an average of 1.7 sessions before lever-pressing was consistent enough to switch to the next phase. In the next phase of response training, the rats were placed on a FR schedule of reinforcement, the values of which were gradually increased to FR10. It took an average of 3.5 sessions for rats to reach the criteria of consistent responding at a value of FR10.

Baseline Training. Baseline lever-press training proceeded smoothly as well. It took 12 sessions of VI60 training to establish consistent responding. By the final day of VI training, the rats had mean responses per minute of 28.65.

Habituation. Rats were presented with three 30-s presentations of each stimulus in order to familiarize them with the stimuli so that the stimuli themselves would not cause a disruption of behavior during conditioning trials. Each rat experienced two sessions of habituation. In the

final habituation session, stimuli produced almost no suppression, as can be seen by mean suppression ratios that all exceed 0.4 (See Figure 5).

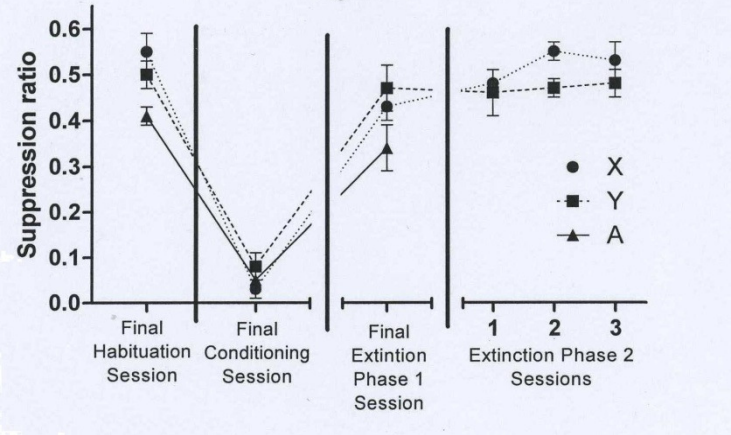

Figure 5

Fear Conditioning. Fear conditioning trials produced conditioned suppression in response to all three stimuli. By the final day of conditioning, all three stimuli produced almost total suppression of responding as seen in Figure 1 by mean suppressions ratios below 0.1. On average, the suppression ratio overall was 0.053 (light= 0.042, tone= 0.032, fan= 0.057). It took an average of 6.8 trials to reach the set criterion of ≤ 0.25 suppression ration for each stimulus on 2 of 3 consecutive days. The mean responses per minute on the final day of conditioning for the entire session was 19.57. The average response rate in the pre-CS period (the 30 second period preceding presentation of the stimuli) was 19.12 (light= 21.67, tone= 17.78, fan= 18.23), and the average response rate during presentation of the stimuli was 0.69 (light= 0.67, tone= 0.68, fan= 0.72).

Extinction. Over the course of extinction trials, all three stimuli demonstrated a loss in their ability to produce suppression, with the tone extinguishing slightly quicker than either the light or the fan. Extinction is represented as Extinction Phase 1 in Figure 1, and the loss of ability

to produce suppression is shown by suppression ratios that exceed 0.3. It took an average of 4.6 extinction sessions to reach the set criterion of at least 3 sessions on extinction, with the last session having a ratio of ≥ = 0.33 for the fan and the tone. The mean responses per minute on the final day had increased to 38.78. The mean response rate in the pre-CS period on the final day increased to 41.0 (light= 41.46, tone= 43.46, fan= 38.54), and the mean response rate during presentation of the stimuli increased to 21.57 (light= 23.74, tone= 39.62, fan, 25.08). The overall suppression ratio was 0.41 (light= 0.34 with an SEM of 0.05, tone= 0.48 with an SEM of 0.05, fan= 0.4 with an SEM of 0.3).

Deepened Extinction. In the deepened extinction trials, the rats experienced three sessions each of deepened extinction with either the fan or tone and three sessions of normal extinction for the stimulus that did not experience deepened extinction. The deepened extinction condition, represented as X in Figure 5, did cause resuppression on the first trial, but this effect was reduced over the next several trials to show less suppression than the normal extinction group.

Test. As seen in Figure 6, deepened extinction is represented as X, normal extinction is represented as Y, and the light stimulus is represented as A (AX is the compounded presentation of the light and the deepened extinction stimuli, AY is the compounded presentation of the light and the normal extinction stimuli). When the stimulus compounding sessions began and the light stimulus was added, there was a reinstatement of suppression, but the AX condition showed less suppression than the AY condition ($p = 0.06$) as shown by the slightly higher suppression ratio. When the single stimuli were presented as either deepened or normal extinction, there was less suppression than when presented in compound with the light ($p < 0.05$). Thus, regardless of whether the single stimuli had experienced regular or deepened extinction, the compounded stimuli produced more suppression of behavior than the single stimuli.

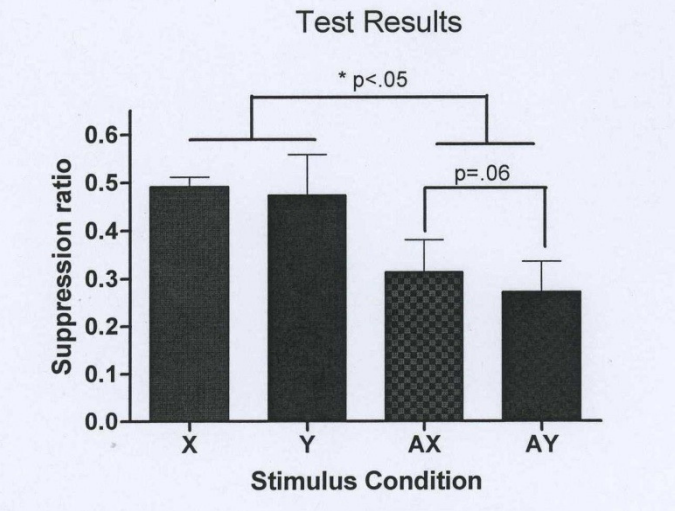

Figure 6

Discussion

This preliminary study shows promising results for the pursuit of further investigation into this area of interest. Although it was not clinically significant, this study did find that the compound stimulus presentation with the deepened extinction stimuli showed less suppression of behavior than with the normal extinction stimuli with a p value of 0.06, just out of the range of statistical significance. However, we did find with significance that the single stimuli produced less suppression than the compounded stimuli, with a p value of < 0.05. The results also showed that the deepened extinction condition overall produced less suppression of behavior than did the normal extinction stimuli.

Since this was a preliminary study, a small *n* was used, which may account for the marginally significant results. However, even with a small group of subjects, some significant results were found, and other results that were just outside of the criteria for significant results. Replicating this study with a larger group of subjects may indeed yield more significant results that will allow for the further development of treatment of drug addictions. If in further studies,

deepened extinction continues to show better ability to extinguish conditioned responding with greater clinical significance, it will hold major implications for the development of treatments for drug addictions. Based on the findings of this preliminary study, it is possible that priming patients with the drug while exposing them to drug related cues, stimuli, or environments may result in better, more thorough extinguishing of the stimuli than regular extinction treatments currently in practice.

While the results of this study are intended for application to the treatment of drug addictions, they also may have implications for the treatment of other disorders, including several behavioral disorders. Behavioral disorders such as phobias and anxiety disorders are often treated with extinction-based therapies that seek to eliminate the association between the fear- or anxiety-inducing stimuli and the resulting fear or anxious responses. Autism, another behavioral disorder, is also commonly treated with extinction-based therapies that extinguish inappropriate behavior through non-reinforcement and positive reinforcement of acceptable and productive behaviors. Disorders such as these can be particularly debilitating to live with and thus continuing to research more effective means of treatment can help give sufferers of these disorders brighter possibilities for a better quality of life. The results of these studies may have implications for the use of deepened extinction in treatments for disorders such as autism, phobias, and obsessive-compulsive disorder.

Autism

Autism is not a single disorder, but a group of related disorders on a spectrum, known as the pervasive developmental disorders (PDDs), characterized by deficits in social interaction, communication, and fixed repetitive interests or behaviors. Autistic disorder is one of the most

severe of the PDDs because it contains disabilities in all three areas that developmental deficits appear (Thompson, 2007, pp. 20-21). According to the Diagnostic and statistical manual of mental disorders (DSM-IV-TR), in order to be diagnosed with autistic disorder, individuals must show deficits in all three areas.

Deficits in social interactions may include impairment in the use of nonverbal behaviors such as eye contact, facial expression and body language, failure to share interests and excitements with others, and failures to develop social relationships (American Psychiatric Association. & American Psychiatric Association. Task Force on DSM-IV., 2000, pp. 20-23). Studies have also shown that social interaction symptoms also involve a reduced capability for empathy (Greimel, et al., 2010) and impairment not just in the use of nonverbal behaviors, but also difficulty understanding and responding to emotions and facial expressions in others (Baron-Cohen, 2009). The deficits in the abilities to process and recognize emotions inhibit the ability to communicate and interact socially with others because individuals with autistic disorder may react inappropriately to social cues (Rump, Giovannelli, Minshew, & Strauss, 2009).

Communication impairments are often demonstrated by the delay in the development of or the failure to develop spoken language, difficulty starting or maintaining conversations with others, or the repetitive use of stereotyped language (American Psychiatric Association. & American Psychiatric Association. Task Force on DSM-IV., 2000, pp. 20-23). Some children fail to ever develop spoken language, but may still develop receptive language, the ability to understand language (Ritvo, 2006, pp. 33-39). Children with autistic disorder may also demonstrate impairments in symbolic thinking and language, and thus much of their speech is a result of very concrete thinking or echoes previously heard speech.

Language in this stage, known as the Echo/Labeling/Concrete Phase is learned simply through rote memorization and words exist just as labels (Ritvo, 2006, pp. 33-39). A great example of the concrete thinking and the deficits in symbolic language can be seen in the movie *Rain Man*. In one scene, the main character, a man with high-functioning autism was asked by a doctor to calculate several multiplication problems. He was able to compute the numbers rapidly, but had more difficulty with word problems which involve symbolic thinking. He was unable to answer how much money he would have left if he had a dollar and spent fifty cents because it went beyond the concrete thinking involved in addition or multiplication and involved actual meaning and symbolic thinking (Levinson, 1988). The failure to develop symbolic thinking also affects the lack of eye-contact because facial expressions do not hold symbolic meaning for those with autistic disorders, therefore they do not look for eye contact because it lacks the meaning that eye contact can have for those without such deficits (Ritvo, 2006).

The third area associated with autistic disorder is often characterized by abnormally intense preoccupations with a limited area of interest, inflexibility with regards to specific routines and repetitive behaviors such as hand-flapping (American Psychiatric Association. & American Psychiatric Association. Task Force on DSM-IV., 2000, pp. 20-23). These deficits may be associated with delays in sensory-motor development that result in children with autistic disorder that affects the brain regulation of sensitivity (Ritvo, 2006, pp. 29-33).

Since autistic disorder is often characterized by impairment in processing symbolic meaning, the salient features of a situation for a normal individual and for an autistic individual are drastically different. While a normal individual may attend to facial expressions and body language, an autistic individual is much more likely to attend to more concrete, observable behavioral cues. Normal children may have the ability the adapt their behavior based off of

emotional cues from others such as anger or shame, but children with autism have difficulty reading and understanding such subtle emotional cues (Rump, et al., 2009). Thus behavioral therapies have been shown to be successful in treating symptoms and behaviors typical of individuals with autism (Schreibman, 2000), likely because behavior therapies involve observable, concrete aspects of behavior.

Behavior therapy is one of the more prevalent therapies used to treat autism and involves the principles of extinction to reduce the frequency of bad or inappropriate behaviors. Extinction is used to eliminate such inappropriate behaviors through non-reinforcement, essentially ignoring the behaviors, and focusing on positively reinforcing the appropriate, desired behaviors. Positive social reinforcers, such as hugs or candy, are used to reward and increase the desired behaviors and can be determined by what works best for each individual (Ritvo, 2006, p. 105). A program is designed based on the reinforcers that are best for the individual as well as the most appropriate schedule, which depends on how long individuals can sustain their attention for (Ritvo, 2006).

An important part of designing the program is gaining the approval of the parents and involving them in the behavior program (Ritvo, 2006, p. 105). Therapists cannot work with children at all hours every day, and therefore it is important for parents to be involved in the behavior therapy in order for the therapy to be successful. It is possible that the lack of involvement of parents may result in establishing stimulus control under which the therapist may signal that one type of behavior is appropriate and will result in reinforcement and the parents and others signal that inappropriate behaviors may still be reinforced. Under such a situation, the behavior therapy would not likely to be successful since the therapy would not be generalized to any context outside of the specific therapy context (Thompson, 2007, p. 99).

Generalization is an important aspect to successful behavior modification, but is usually best approached incrementally. In the beginning stages of behavior therapy stimulus control is usually desired, and introducing new settings and contexts will occur later, once skills have been proficiently acquired. In order to develop stimulus control, it is often recommended that the therapy setting be unique, and specific to the therapy purpose (and not used for any other purpose). New settings and individuals may be slowly introduced one at a time until behaviors are consistently demonstrated in each new context. Conducting therapy in different contexts encourages generalizations across all situations that a child may encounter, which will makes it more likely that the child will display appropriate behaviors in all situations, and not be limited to certain contexts, such as the therapy context (Thompson, 2007, p. 99). Generalization is also a goal of deepened extinction in respect to the treatment of drug addiction. The hope is that the extinction of undesired behaviors such as drug-seeking behaviors, and in the case of autism a range of behaviors including repetitive, stereotyped behaviors, will generalize to other cues so that the recovery of behavior is much less likely.

Another area of autism that can be handled by use of extinction is the inflexibility and adherence to rituals and routines. Children with autism often react poorly to a change in routine because such an occurrence breaks the predictable pattern that they have become used to, and throws their world into disconcerting disarray (Thompson, 2007, p. 64). Extinction may be used in this context by attempting to remove the aversiveness of a change in routine. It is helpful if parents prepare a child for the events that are going to happen, so that the change in routine does not completely destroy the predictability of a situation. Surrounding the child with familiar and comforting cues, such as a doll or music, may also ease the transition. The extinction occurs as

the child is exposed to the changed routine repeatedly without anything aversive happening, until the change in routine no longer elicits a reaction from the child (Thompson, 2007, p. 64).

This is also one area in which deepened extinction may be applied to further extinguish aversive stimuli such as a change in routine. Adjusting a child to new situations and changes in routine can use deepened extinction to increase the error signal generated and thus encourage more learning and further break the association between a stimulus and a response. An autistic child may first be exposed to normal extinction, repeatedly being exposed to a changed routine until the change is no longer aversive and thus does not elicit strong reactions from the child. If the child is then presented once again with cues signaling that the original routine will occur, he may again expect the same predictable routine that was previously replaced in salience with the new one. If however, the original routine does not occur as expected, a greater error signal will be generated. Based on the preliminary studies of deepened extinction, priming the child with cues that indicate the predictable routine will occur that are not actually followed by the predicted event make it more likely that the next time the child is faced with such cues, he will no longer expect the same outcome. This may make the child more flexible to adapting to new routines if deepened extinction eliminates the potential for cues to signal a specific, predictable occurrence.

Phobias

Phobic disorder is defined by the DSM-IV-TR as a persistent, excessive, or unreasonable fear of a specific object or situation which results in anxiety and avoidance of the object or situation (American Psychiatric Association. & American Psychiatric Association. Task Force on DSM-IV., 2000, pp. 432-456). The anxiety response produced by exposure to the feared stimulus

may take the form of a panic attack, which is characterized by symptoms associated with activation of the sympathetic nervous system, including increased heart rate, sweating or trembling, difficulty breathing, nausea or dizziness, as well as feelings of intense fear or dread (Root, 2000, p. 3). The person is able to recognize that the fear is excessive or unreasonable, however the avoidance and anxiety associated with the feared situation or object interferes significantly with relationships, jobs, and day-to-day life (American Psychiatric Association. & American Psychiatric Association. Task Force on DSM-IV., 2000, pp. 432-456). Anticipatory anxiety is also often seen in phobic disorders in which the possibility of exposure to the situation or object causes fear and anxiety and results in avoidance of any possible situations in which the person might encounter the feared object (Goodwin, 1983, p. 31; Root, 2000).

The three major phobic disorders include simple phobia, social phobia, and agoraphobia. Simple phobias are characterized by an excessive and irrational fear of a specific object or situation and tend to be less disabling because the object or situation can often be easily avoided. Some simple phobias can be more easily avoided than others, but when the phobic object or situation is common enough that it cannot be avoided, interference with daily life can occur. The anxiety and panic are only elicited by the specific object or situation. Simple phobias are extremely common and people can develop phobias to almost any situation or object. Common phobias include flying in airplanes, dentists, doctors, heights, and closed spaces (Goodwin, 1983, p. 32).

Social phobias are characterized by a persistent, excessive, and irrational fear of being stared at or exposed to scrutiny by others, and thus results in avoidance of situations in which this may occur (Beck, Emery, & Greenberg, 2005, p. 36). Social phobias involve the fear of being ridiculed or embarrassing one's self that may result from such scrutiny. Common social

phobias include a fear of public speaking, using public bathrooms, vomiting, or being touched (Goodwin, 1983, pp. 44-54). While many people have experienced stage fright or nervousness before a speaking engagement or performance, the difference between a normal amount of anxiety that can actually aid performance and a social phobia is the amount of anxiety caused and the avoidance that usually causes interference and inconvenience (Goodwin, 1983, pp. 44-45).

Agoraphobia regards to more broad fears than do the simple or social phobias, which more often involve fear of a specific situation or object. Agoraphobia usually involves multiple phobias that can apply to multiple situations, such as fear of crowded or confined places, public transportation, being home alone or being far from home. Public transportation such as buses or trains can become crowded and tunnels and bridges can feel confining which eliminates the possibility of escape, while being away from home or in places removes the individual from comfort, help or other things needed or not readily available (Goodwin, 1983, p. 56). Agoraphobia often causes more disruption of daily activities and life because common activities that are not as easily avoided become increasingly avoided, and since they are more general situations, avoidance of any crowded areas such as elevators, bridges, public transportation, even busy streets may occur (Beck, et al., 2005, p. 36). Agoraphobics also tend to be generally anxious, even when not presented with the fearful situations, unlike those with simple or social phobias (Goodwin, 1983, p. 57).

Phobias are often treated with desensitization, a behavior therapy which involves exposure to feared stimuli in a controlled environment with no aversive outcome, in the hopes that the feared stimuli will lose its associative power to predict a similarly feared outcome such as a panic attack. Desensitization is often conducted in a systematic fashion, starting with less

feared stimuli at lower exposures, such as visualizing initially as opposed to actual exposure. First the phobia must be identified, and then the therapist works with the patient to create a fear hierarchy, a list of aspects relating the phobia ranging from that which are least feared to that which are most feared. The patient is next instructed to visualize situations involving the least fearful stimuli related to the phobia until it no longer provokes anxiety. A key feature of this approach is reciprocal inhibition, which takes advantage of the fact that fear and relaxation are incompatible emotions. Reciprocal inhibition involves pairing the visualization of the feared stimulus with a pleasurable or relaxing experience, because since relaxation is incompatible with fear, the patient may be exposed to feared stimuli without experiencing fear (Goodwin, 1983, p. 108).

As the patient becomes desensitized to the less feared stimuli, it is possible to move up the fear hierarchy and start introducing more and more anxiety-provoking stimuli. Once the visualization of the elements on the fear hierarchy no longer produce anxiety, it is possible to start exposing the patient to real-life situations in which the fear stimuli might be encountered. Another related treatment is known as flooding, or extreme exposure therapy, which skips the visualization and feared hierarchy and involves extreme exposure to the phobic stimuli themselves (Goodwin, 1983, p. 111).

Another important element of desensitization is that exposure to the stimuli cannot be brief, but must be sustained for a sufficient amount of time for the patient to learn that anxiety or anything disastrous will not occur (Root, 2000, pp. 61-62). If the person is removed while anxiety is high, it continues to reinforce the avoidance because the avoidance also reduces the anxiety. By sustaining the exposure to the phobic stimuli, the panic subsides on its own without avoidance (Goodwin, 1983, p. 111). It is important to maintain exposure to the stimuli because

the anxiety response is often anticipatory, caused by the anticipation of seeing the feared stimuli. Thus, the anxiety response is elicited before the individual is even exposed to that which he finds fearful. The individual must be exposed to the stimuli itself and be able to recognize that nothing aversive will occur in order to extinguish the associative powers of the stimuli.

Systematic desensitization is an extinction-based treatment in that it involves repeated exposure to fear stimuli without being negatively reinforced by avoidance or without being followed by an aversive event. After repeated exposure without being followed by the aversive unconditioned stimulus or event, the feared stimulus that has become associated with that event loses its associative power and no longer predicts the aversive event. However, because avoidance of the phobic stimuli is so powerfully reinforcing and maintains the phobia itself, it can be difficult to find those willing to face their fears and go through exposure therapy (Goodwin, 1983, p. 111).

Deepened extinction may also be applied to the treatment of phobias, although just like exposure therapies, it would require willing patients to go through such a procedure. Deepened extinction would also prolong the treatment of phobias, although if successful, it would more successfully extinguish the anxiety response to phobic stimuli and produce more generalized results. The phobic stimuli would still need to experience extinction through some form of therapy, such as extreme exposure therapy or systematic desensitization until it no longer provokes the anxiety response. Under a controlled environment and in the presence of a trained professional, deepened extinction could be carried out by inducing anxiety in the patient (Walker, 1949).

After the patient is primed with the anxiety response and the anxiety has once again subsided, the patient should once again be exposed to the phobic stimuli. Feeling the anxiety

response and then being exposed to the phobic stimuli should cause the patient to once again expect the phobic stimuli to cause such an anxiety response. As already mentioned, this will generate a greater error signal when the anxiety response does not occur. Throughout the course of the therapy, the association between the phobic stimuli and the anxiety responses that they once elicited will be weakened. Based on the results of this study and of previous studies, because these phobic stimuli will have experienced deepened extinction, the likelihood that they will cause a relapse of avoidance behavior or anxiety in the future will be greatly reduced.

Obsessive-Compulsive Disorder

Obsessive-Compulsive disorder is a disorder related to such anxiety disorders as agoraphobia and the simple and social phobias. It is characterized by either obsessions or compulsions that are recognized as unreasonable by the individual, causes distress and anxiety, and interfere with normal functioning and relationships (American Psychiatric Association. & American Psychiatric Association. Task Force on DSM-IV., 2000, pp. 462-463).

An obsession is a persistent, intrusive, and repetitive thought, image or impulse that causes distress and are recognized by the individual as being unreasonable (Mavissakalian, Turner, & Michelson, 1985, p. 2). An individual who experiences obsessions actively attempts to resist them and clear his mind of the thoughts, but obsessions are difficult to control, and although the individual might try suppress obsessions with other thoughts or actions, (American Psychiatric Association. & American Psychiatric Association. Task Force on DSM-IV., 2000, pp. 462-463) suppression can often increase the experienced distress (Mavissakalian, et al., 1985; Root, 2000, p. 10). Obsessions are more than normal, common worries, but are extreme, disruptive and unwelcome. They most commonly involve dirt and contamination, aggression,

orderliness of objects, sex, and religion, and can take the form of ideas, images, beliefs or fears (Goodwin, 1983, pp. 78-79; Mavissakalian, et al., 1985, p. 2).

Compulsions can often be the results of obsessions, actions taken to suppress the intrusive and distressing obsessions. Compulsions are repetitive behaviors or mental acts such as hand washing or counting that are performed in response to obsessions or in adherence to a rigid set of rules that must be followed in order to avoid an unrealistic yet feared event (Root, 2000, p. 10). Although the compulsions seek to suppress and reduce the distress caused by obsessions or anticipation of some feared event, they are excessive, not realistically connected to the events, and actually cause even more distress. Compulsions are voluntary rituals, but are accompanied by an intense, powerful urge that is extremely difficult to ignore, although attempts to resistant are commonly associated with compulsions. The urge to engage in these behaviors is an internal pressure (Mavissakalian, et al., 1985, pp. 2-3). Compulsions can be understood as a type of avoidance behavior, because the individual believes that engaging in these acts will prevent some feared, aversive event or terminate obsessive thoughts, however irrational this may be.

Similar to phobic disorders, those suffering from obsessive-compulsive disorder recognize that their obsessions or compulsions are unreasonable and excessive, yet are still difficult to manage. Additionally, the disorder causes disruption in normal daily functioning and in relationships, however, people with obsessive-compulsive disorder are often reluctant to seek treatment because they are embarrassed by their condition (Root, 2000, p. 10).

Exposure therapies are commonly used to treat obsessive-compulsive disorders in a manner very similar to the treatment of phobic disorders. In context of obsessive-compulsive disorder, exposure therapies seek to eliminate the ability of obsessive cues to elicit anxiety. As a result, compulsive behaviors will also become eliminated because they are no longer necessary

to reduce the anxiety and distress caused by the obsessive thoughts or images and thus will no longer have reinforcing properties. Exposure therapies such as systematic desensitization target the obsessive symptoms of obsessive-compulsive disorder and assume that reducing these symptoms will eliminate that need for compulsions as well (Mavissakalian, et al., 1985, p. 61).

Systematic desensitization is carried out in the same way as was discussed in the treatment of phobic disorders. A hierarchy of anxiety-provoking stimuli or cues is constructed ranging from the least feared to the most feared. A relaxed state is then induced in the individual, which may be accomplished through pharmacological means, by visualizing something pleasant, or any means that induces a deep relaxation or pleasurable state. Since relaxation and pleasure are incompatible with fear, it allows the individual to be presented with the anxiety-provoking stimuli without actually inducing the anxiety response. The patient is then presented with the items listed in the fear hierarchy, beginning with the least feared and moving up the hierarchy as each item ceases to produce the anxiety response (Mavissakalian, et al., 1985, p. 61).

Response prevention is used with exposure therapy to directly target compulsive behaviors or actions. It still involves the confrontation or presentation of anxiety-provoking stimuli but seeks to block the avoidance of the anxiety response in the form of compulsive behaviors. Response prevention involves blocking or preventing the compulsive behaviors, and allows the individual to recognize that nothing catastrophic or aversive occurs if the behaviors are not performed. Since compulsions are maintained by their reinforcing properties by reducing the anxiety caused by obsessions or by avoiding the feared events that they predict will happen if they do not engage in such compulsions, response prevention allows the patient to realize that nothing aversive will happen if they do not perform the behaviors (O'Donohue & Fisher, 2009,

pp. 445-451). The anxiety response will subside naturally and avoidance will be unnecessary because the patient can recognize that nothing realistically will happen.

Both response prevention and exposure therapies are extinction-based treatments and present the aversive stimuli without being followed by an aversive event or without allowing avoidance. Once the patient experiences the feared stimuli for a sufficient amount of time without anything catastrophic happening, the association between the feared stimuli and the events they predict will weaken, and the patient may be able to recognize that the fears are unrealistic (O'Donohue & Fisher, 2009, pp. 445-451). Once again, these treatments involve repeated exposure to aversive events, situations or stimuli without being paired with the predicted aversive event, until the stimuli no longer predict such occurrences.

Deepened extinction may also be applied to the treatment of obsessive-compulsive disorders in conjunction with exposure therapies and response prevention. It is likely to prolong the treatment process, but as with phobias, if it more successfully extinguishes anxiety and compulsions is response to obsessive or compulsive cues, then it may be beneficial and worth further investigation. Exposure therapies and response prevention would be conducted prior to deepened extinction, but deepened extinction will be used to generate a greater error signal than was generated during the beginning of exposure therapy. Deepened extinction would involve inducing the anxiety response in the patient in a controlled environment and in the presence of a trained professional (Walker, 1949).

Similar to the application of deepened extinction to the treatment of phobic disorders, after priming the patient with the anxiety response and allowing the anxiety to subsided, the patient should once again be exposed to the phobic stimuli. Inducing the anxiety at the beginning of deepened extinction should remind that patient of the uncomfortable feeling of anxiety and

panic and evoke a reinstatement of avoidance and anxiety response elicited by the original feared stimuli. The patient may once again expect the obsessive or compulsive cues to predict an anxiety response or a catastrophic event, causing them to engage once again in compulsive and avoidant behaviors. Once again, a greater error signal will be generated when nothing aversive happens when the compulsive behaviors are prevented during response prevention, and throughout the course of the therapy, the association between the obsessive and compulsive cues and the responses and events they predict will be weakened. As the results of this study and previously conducted studies, the obsessive and compulsive cues that have experienced deepened extinction are less likely to cause a relapse of avoidance and compulsive behavior upon later exposure to such cues.

Finally, although the results of this study did not reach the criteria for statistical significance, they did indeed indicate that deepened extinction reduces the associative power between stimuli and the events or responses that they predict. Stimuli that experienced deepened extinction produced less suppression of behavior during the test phase as expected. The results were significant enough to warrant further investigation, and with changes such as a larger group of subjects, may yield more significant results in the future. The findings of these preliminary studies are promising for future research as well as for the treatment of behavioral disorders and addictions. Deepened extinction may be a crucial and welcome addition to already existing behavioral treatments for drug addictions and disorders such as autism, phobias, and obsessive-compulsive disorders by more thoroughly extinguishing the stimuli that elicit unwanted behaviors such as drug-seeking or avoidance behaviors. Deepened extinction procedures may improve the effectiveness of the treatments of these disorders by reducing the possibility that the

cues that originally elicited that unwanted response will cause a relapse in the future, thus allowing for a greater possibility of recovery.

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