Extinction Learning

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Synonyms
Inhibitory learning

Definition
Extinction learning refers to the gradual decrease in response to a conditioned stimulus that occurs when the stimulus is presented without reinforcement. The term “extinction” was first used by Ivan Pavlov in reference to his observation that the conditioned response to a cue that predicted food delivery decreased and eventually disappeared when food no longer followed the cue. In Pavlovian fear conditioning, a previously neutral stimulus comes to elicit a fear response through pairing with an aversive reinforcer, such as an electric shock or a loud noise. During extinction, a new association with the stimulus is learned that inhibits the expression of the original fear memory. Extinction learning serves as the foundation of exposure therapy, which is commonly used to treat pathological fear.

Theoretical Background
Russian scientist Ivan Pavlov first documented the phenomenon of extinction in his seminal classical conditioning experiments (Pavlov 1927). In these studies, dogs were repeatedly given food following the presentation of a tone. After several such pairings, the dogs began to salivate at the presentation of the tone. Pavlov referred to the tone as the conditioned stimulus (CS) and the food as the unconditioned stimulus (US). The innate salivation response to food was termed the unconditioned response (UR), while salivation in response to the tone was referred to as the conditioned response (CR). Once the conditioned response had been learned, repeated presentations of the tone without subsequent food delivery led to a gradual decrease in salivation that Pavlov referred to as the “extinction” of the conditioned response.

While early theories hypothesized that the conditioned response is unlearned during extinction, experimental evidence indicates that the original association between the CS and the US remains intact. Following extinction, several manipulations can bring about the reemergence of the CR (see Bouton 2004, for a review). Conditioned responding typically returns after the mere passage of time, an occurrence known as “spontaneous recovery.” Exposure to the US following extinction causes “reinstatement” of the CR. Presentation of the CS outside of the context in which extinction learning takes place leads to “renewal” of the CR. In light of these behavioral phenomena, current theories propose that extinction learning involves the formation of a new inhibitory association with the CS that competes with the original excitatory association for expression.

The brain mechanisms supporting extinction learning have primarily been investigated in studies examining the extinction of conditioned fear (see Quirk and Mueller 2008, for a review), as the neural processes underlying fear learning are relatively well understood. During fear conditioning, neurons in the lateral nucleus of the amygdala encode the association between the CS and the US, which controls the expression of the CR via projections to the central nucleus of the amygdala. The initial acquisition of extinction learning involves molecular changes within the basolateral amygdala. During the several hours following acquisition, the extinction memory undergoes ▶ consolidation, during which plasticity in the basolateral amygdala, ventromedial prefrontal cortex, and the hippocampus serve to stabilize the memory. Following consolidation, retrieval of the extinction memory elicits increased activity in the ventromedial prefrontal cortex, and the hippocampus serve to stabilize the memory. Following consolidation, retrieval of the extinction memory elicits increased activity in the ventromedial prefrontal cortex, which projects to the intercalated cell masses of the amygdala that in turn inhibit central nucleus output. Projections from the hippocampus to the vmPFC and the basolateral amygdala modulate fear expression based on whether the context predicts threat or safety.

The persistence of fear in the absence of any imminent threat is a hallmark of anxiety disorders. Impaired ability to acquire or retrieve extinction learning may underlie such pathological anxiety. Exposure therapy, in which individuals are exposed to stimuli associated with previous trauma in a safe context, attempts to treat anxiety disorders by fostering fear extinction. In addition, research in animals demonstrating the
enhancement of extinction through the administration of various pharmacological agents suggests a promising avenue for improving current therapies (see Anderson and Insel 2006, for a review). Recent research in animal models and humans suggests that when extinction learning occurs shortly after the retrieval of a fear memory, this new learning may disrupt the reconsolidation of the original fear memory leading to a persistent reduction in fear.

**Important Scientific Research and Open Questions**

At present, little is known about the factors that give rise to differences among individuals in extinction learning and retention. Recent exploratory research suggests that variation in brain structure, genetic polymorphisms that influence molecular learning processes, and experimental factors ranging from maternal care to chronic stress all contribute to variation in extinction. Further research into the origins of such individual differences may elucidate what renders a person vulnerable to or resilient against anxiety disorders.

The extinction of appetitive associations has not been widely studied to date. Preliminary research suggests that the vmPFC and the amygdala also play a role in appetitive extinction (see Quirk and Mueller 2008, for a review); however, the involvement of additional regions involved in reward processing (e.g., striatum) remains to be clarified. Reward-related classical and instrumental conditioning processes are thought to support the development and maintenance of drug addiction. A better understanding of the mechanisms underlying the extinction of appetitive associations may yield novel approaches to treating substance abuse disorders.

**Cross-References**

- Classical Conditioning, Anticipatory Learning
- Fear Conditioning in Animals and Humans
- Internal Reinforcement Hypothesis
- Memory Consolidation and Reconsolidation
- Pavlovian Conditioning

**References**


**Extraversion**

Extraversion is a personality dimension characterized by sociability, enthusiasm, risk taking, and needing the company of others. This dimension has been described in several models of personality including Eysenck's personality theory and the Big Five factor model.

**Extraversion, Social Interaction, and Affect Repair**

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**Synonyms**

Affect regulation; Personality; Self-regulation; Socialization

**Definition**

Extraversion is one of the Big Five personality traits (extraversion, neuroticism, agreeableness, conscientiousness, and openness to experience) used to encompass human personality. Extraversion is comprised of affective, social, and responsivity components with extraverts (vs. introverts) experiencing more positive affect, engaging in social interaction to a greater degree, and showing higher levels of reward responsivity. Affect repair is a form of affect regulation in which individuals attempt to decrease levels of negative (i.e., sadness, anxiety) and increase or maintain levels of positive (i.e., happiness, enthusiasm) affect.