Maize Books Michigan Publishing

Sign-Tracking and Drug Addiction

Arthur Tomie; Jonathan Morrow

DOI: http://dx.doi.org/10.3998/mpub.10215070

Permissions

Chapter 2: Sign-Tracking Model of the Addiction Blind Spot

Arthur Tomie,*[a,b] Peter Jeffers,[a] and Barbara Zito[c]

a Department of Psychology, Rutgers University, New Brunswick, NJ 08901

[b] Center of Alcohol Studies, Rutgers University, New Brunswick, NJ 08901

[c] ZT Enterprises LLC, Cranbury, NJ 08512

Corresponding Author:

Arthur Tomie, PhD, Associate Professor, Department of Psychology and Center of Alcohol Studies, Rutgers—The State University of New Jersey, New Brunswick, NJ 08903; e-mail: tomie@psych.rutgers.edu; phone: (848) 445-8885; fax: (848) 445-3500.

Chapter 2: Sign-Tracking Model of the Addiction Blind Spot

Arthur Tomie,*[a,b] Peter Jeffers,[a] and Barbara Zito[c]

Abstract

No one sets out to become an addict. Drug use begins voluntarily, but somehow, through repetition and ritual, drug-taking becomes unstoppable. The most obvious characteristic of addiction is that drug use takes on a life of its own, but how and why this happens remains a mystery. The voices of addicts serve only to deepen the mystery. Many tell us that their drug use escalated, even as they were trying very hard to keep it under control. Despite repeated failed attempts to maintain self-control, when addiction closed in, they were stunned, because all along they were certain that they could quit drug use if they really wanted to. There is something mysterious and stealthy about the drug addiction process that allows addiction to prey upon the unsuspecting. Based upon our research, we have concluded that there is an addiction blind spot in the form of a psychological scotoma, which enables the gradual erosion of self-control to proceed unrecognized. Due to this blind spot the loss of self-control occurs in the background, at a preconscious level, without awareness. The addiction blind spot obscures the loss of self-control that fuels the transition from social, recreational, and voluntary drug use into the realm of the habitual and automatic drug-taking of the drug abuser.

Keywords: autoshaping; drug addiction; misbehavior; reflex; self-control; sign-tracking

Introduction

Addiction nosologists (Babor, 1995; Jellinek, 1960) and addiction researchers (Barker & Taylor, 2014; Corbit & Janak, 2016; de Wit et al., 2012; Tiffany, 1990; Tiffany & Conklin, 2000) have long noted that repeated drug use sets the stage for the gradual progression from voluntary and intended drug use into reflexive and poorly controlled drug abuse. Our hypothesis is that repeated drug use induces Pavlovian sign-tracking of drug-taking, which accounts for the increase in vulnerability to drug cues that accompanies the habitual and automatic drug-taking of the drug abuser (Corbit & Janak, 2016). The sign-tracking model (STM) has previously been presented in a number of theoretical reviews (Tomie, 1995, 1996; Tomie, Badawy, & Rutyna, 2016; Tomie, Grimes, & Pohorecky, 2008; Tomie & Sharma, 2013) where we have proposed that the development of sign-tracking conditioned response (CR) performance of drug-taking provides a unified account of prominent features of drug abuse, including escalation into excessive drug-taking and the loss of control of drug-taking.

In the present chapter, we expand on the role of sign-tracking to account for the blind spot in the drug addiction process. The drug addict is in the dark, unaware that sign-tracking has developed and is gradually robbing him or her of his or her free will. The addiction blind spot is evidenced by their lack of awareness that their actions have become triggered and reflexive due to sign-tracking. They remain confident in their ability to control their drug use, even as they lose their grip. While treatment specialists have made reference to the addiction blind spot (Formica, 2012; Reich, 2015), their focus was on relapse and the ego-hypertrophic overconfidence that encourages risk-taking. In contrast, our focus is on a much earlier phase of the drug addiction process. It is when the user chooses to use drugs again and again, that this ritualized experience leads to the development of sign-tracking, which is a form of drug-taking that the user cannot control. We propose that the development of sign-tracking of drug-taking goes unnoticed because the reflexive use of drugs closely resembles and passes for voluntary drug-taking. Thus, the naïve user is oblivious to the emergence of sign-tracking of drug-taking, as sign-tracking integrates seamlessly with ongoing voluntary drug use. This is the addiction blind spot, where drug-taking is slipping out of control and the user is unable to see it.

Sign-tracking is especially likely to go undetected because of representational and cognitive momentum (Hubbard, 2015; Miura 1990), the tendency to see what we expect to see and to see it as that which we have seen all along. The user has already established a history of performing voluntary acts of intended drug-taking and continues to see it that way. When sign-tracking CR performance of drug-taking does emerge, the user will perceive it as just another ordinary routine act of voluntary and intended drug-taking. In other words, sign-tracking is simply overlooked because it is camouflaged as voluntary drug use and is virtually invisible, allowing sign-tracking of drug-taking to hide in plain sight.

The following discussion will focus on these fundamental questions: What is sign-tracking? What role does sign-tracking play in the compulsive use of drugs? How does sign-tracking contribute to the blind spot, that is, to the misguided belief of the drug abuser that they are in control of their drug-taking?

Sign-tracking is a form of Pavlovian conditioning. Pavlov's dogs learned to salivate to the tone conditioned stimulus (CS) that signaled food unconditioned stimulus (US). During sign-tracking procedures, the subject learns to react to the presentation of the object CS that signals the delivery of the food US as though the object CS was the actual food US (Brown & Jenkins, 1968). For example, when food is the US, the subject will approach the lever object CS, contact the lever object CS, and then lick, gnaw, and chew (i.e., "consume") the lever object CS. However, regardless of what the subject does, the food reward US is delivered. This is important to the understanding of sign-tracking. Even though lever-pressing for food reward is a widely employed operant reward training procedure, sign-tracking is not a voluntary goal-directed operant response (Locurto, 1981; Tomie, Brooks, & Zito, 1989), and therefore does not serve the instrumental purpose of acquiring the food reward US. It is important to note that sign-tracking may be induced during operant reward training procedures and may resemble operant responding, but sign-tracking induced during operant procedures does not increase operant goal-directed behavior; rather, sign-tracking increases non-instrumental performance of instrumental-like responding.

It bears repeating that sign-tracking is an acquired Pavlovian reflex. It is a complex sequence of directed skeletal-motor responses that are conditioned to be triggered by the object CS and, in addition, to be performed regardless of the intention of the subject (D. Williams & H. Williams, 1969; for reviews, see Herrnstein & Loveland, 1972; Locurto, 1981; Locurto, Terrace, & Gibbon, 1976, 1978). For a brief video showing the acquisition of sign-tracking in a laboratory rat, see http://www.youtube.com/watch?v=x38b0R6TZxM.

Herein lies the crux of the matter, the erroneous presumption of the drug abuser, who all along thought that quitting was simply a matter of deciding to do so. The drug abuser is baffled and confused because the actions of sign-tracking of drug-taking, which are reflexive and involuntary, were mistaken for and misconstrued as the actions of voluntary and intended drug-taking (Tomie & Sharma, 2013). Figure 2.1, using alcohol drinking as an example, illustrates how voluntary operant drug-taking brings about the development of sign-tracking CR performance of drug-taking.

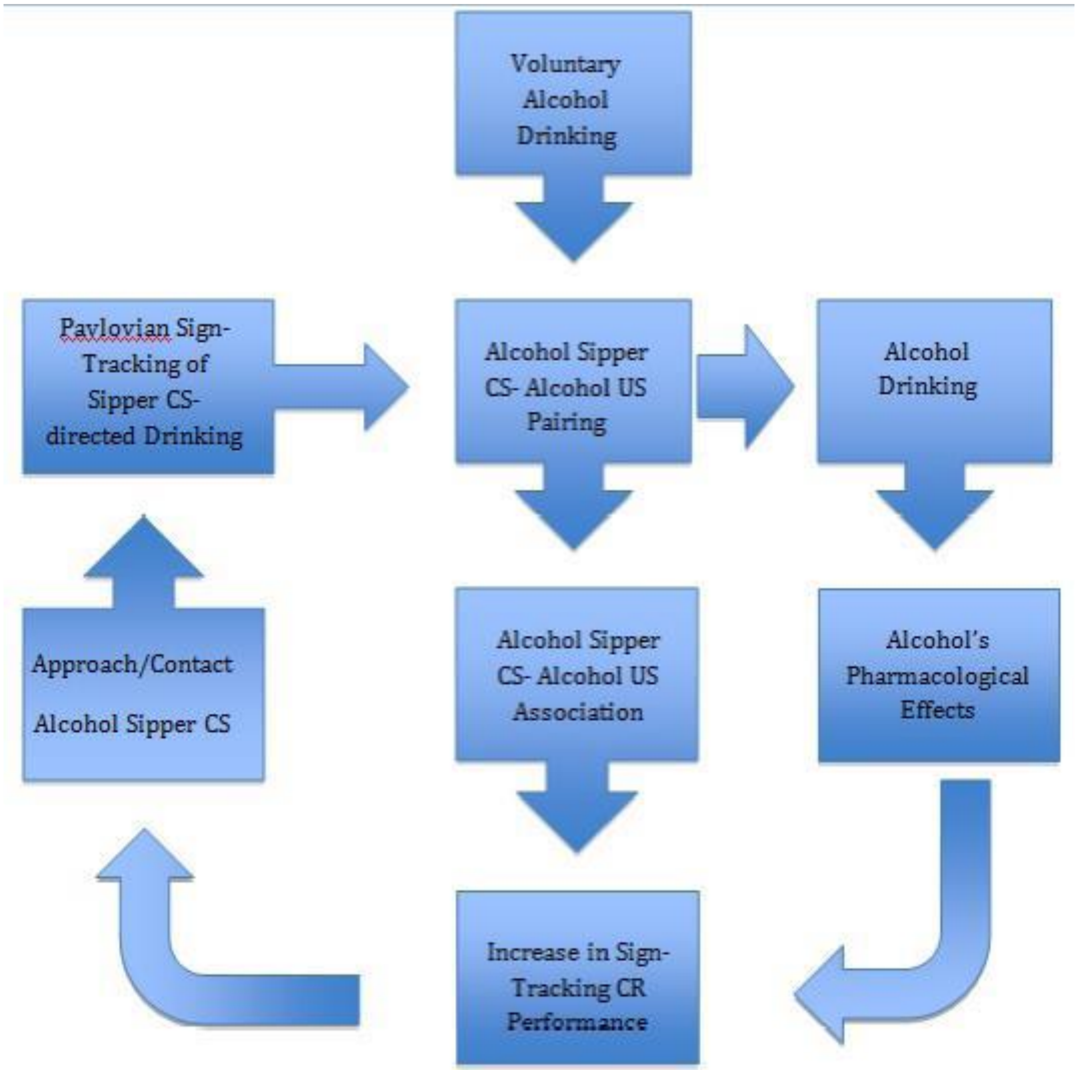

Figure 2.1. Sign-Tracking Model (STM) using the example of voluntary alcohol drinking that induces Pavlovian sign-tracking CR performance of alcohol drinking (Tomie & Sharma, 2013). Note that alcohol's pharmacological effects serve to further increase sign-tracking CR performance (Tomie, Cunha, Mosakowski, Quartarolo, Pohorecky, & Benjamin, 1998; Spoelder, Flores Dourojeanni, de Git, Baars, Lesscher, & Vanderschuren, 2017).

STM emphasizes the role of the tool used by humans as a conduit to aid in consuming the drug (Tomie, 1995, 1996). For example, through repetitions of drug-taking, the cocktail glass CS comes to signal the impending drug reward US. In this way, repeated voluntary acts of alcohol drinking from the cocktail glass CS provide the user with numerous Pavlovian CS-US pairings, which are conducive to the induction of Pavlovian sign-tracking CR performance. What does the topography, the sequence of physical movements, of sign-tracking CR performance of alcohol drinking look like?

Consider the case of the alcohol user who is in the early stages of exhibiting sign-tracking of triggered, reflexive, and automatic alcohol drinking. He comes home after a stressful day, walks straight to his home bar, and begins the well-practiced routine of pouring a drink. He reaches for his favorite bottle of bourbon, takes the tumbler glass from the cabinet and as he drops the ice cubes into the glass, he hears the familiar clink of the ice hitting the glass. He then pours the bourbon over the ice cubes and proceeds to bring to his lips his favorite drink, bourbon on the rocks. This ritual is performed over and over again. How does this physical process, the actual preparation of and consumption of the drink, differ from his bourbon-drinking prior to the acquisition of the triggered, reflexive, and automatic habit of doing it? The problem is, it doesn't. This is exactly how he made his drink before he developed the habit, and due to the development of sign-tracking, it's exactly how he will make his drink now and in the foreseeable future. In other words, the voluntary action and the reflexive action appear the same. Despite the fact that the subject's routine of pouring and consuming the drink has become reflexive and is now performed mindlessly and automatically, nothing in his actual skeletal-motor response sequence appears any different from before, when it was all performed as a voluntary and intended action. For this reason, drinking beyond what was intended is likely to be misinterpreted as "I changed my mind," indicating that I believe that I am still in control of my drinking. Note that the physical movements of sign-tracking of alcohol drinking and the physical movements of voluntary intended alcohol drinking appear to be identical. Sign-tracking of drug-taking, therefore, is inconspicuous, easily overlooked, and virtually invisible because sign-tracking of drug-taking looks very much like voluntary operant drug self-administration.

As alcohol use is repeated and loss of control of alcohol drinking begins to develop, voluntary acts of alcohol drinking will continue to take place as before. But now, in addition, due to the acquisition of sign-tracking CR performance, the drinker will absent-mindedly have a drink without intending to do it. This is not the same as deciding to have a drink. This is a triggered reflex. This is the first sign of the emergence of sign-tracking of alcohol drinking. Because the topography of the sign-tracking CR closely resembles the topography of voluntary drug-taking, the unconcerned user may drink excessively, having more than intended, but remain oblivious as to why. As sign-tracking develops further, alcohol drinking gradually becomes more reflexive, automatic, involuntary, mindless, thoughtless, and more difficult to stop. This is because, due to the strengthening of Pavlovian conditioning, the mere presence of the cocktail glass is now better able to trigger sign-tracking of alcohol drinking, resulting in reflexive drinking of the alcoholic beverage in the cocktail glass. Due to sign-tracking, an ongoing episode of alcohol drinking will be more difficult to stop, but the underlying cause of the actions of continued drinking will be overlooked. Having another drink will be seen as a voluntary and intended action that is regrettable, but correctable, an unfortunate consequence of a bad decision. This optimistic view allows the abuser to cling to the mistaken belief that they are in control of their drug-taking when they are not. In this way, they remain blind to the erosion of their self-control and continue to slide down the slippery slope into the pit of drug addiction.

The complaints of the confused drug addict are telling. "I was blind-sided," "I never saw it coming," "How did this happen to me?" Their words convey that they feel confused and cheated because they were unaware that their drug-taking would become unstoppable even when they were trying very hard to quit. Many addicts feel that they plummeted into the pit of addiction without fair warning because they were unaware that their control of their drug-taking was slipping away. This problem is addressed by Levitch, Marcinkowski-Paulis, and Tomie (this volume), who found that storytelling about sign-tracking significantly boosted awareness of the loss of self-control and improved understanding of how the loss of self-control contributes to the development of drug addiction.

Conduits

It should be noted that humans typically employ a tool as a conduit (i.e., tooter, bong, cigarette, syringe, capsule) to assist in the self-administration of abused drugs (i.e., cocaine, marijuana, tobacco, heroin, prescription pain-killers). In addition, humans typically consume alcohol from a container (i.e., cocktail glass, ale mug, beer bottle) employed as a conduit to assist in drinking alcoholic beverages. An unintended role of the conduit, therefore, is to allow humans to self-administer drugs in a manner that is perfectly suited to the development of sign-tracking CR performance of drug-taking. Moreover, and crucial to the understanding of the blind spot, the conduit provides a common target at which both forms of drug-taking (voluntary operant drug-taking and reflexive sign-tracking of drug-taking) are directed. As noted earlier, the form of the sign-tracking CR resembles the form of the operant response performed when the subject decides to have a drink. Their shared common target, at the cocktail glass, serves to bring them together, merging them into a single unified stream of responding. In this way, the conduit triggers excessive use but, at the same time, camouflages sign-tracking, creating the blind spot that allows the user to believe that their excessive use of drugs is by choice. Thus, the user remains overconfident, unconcerned, and oblivious, even as drug-taking becomes increasingly difficult to control.

Using a conduit to self-administer a drug plays a major role in the creation of the addiction blind spot. This is because repeatedly using the conduit to take the drug provides precisely the protocol that will lead to the development of sign-tracking of drug-taking. The reflexive and impulsive use of drugs is then able to masquerade as voluntary drug-taking when in fact it is due to sign-tracking. Using the conduit as the drug-taking tool allows the conduit to act as a drug cue, and this serves to obscure the distinction between voluntary and reflexive drug use. This provides the opportunity for sign-tracking to slip by unnoticed, so that the user fails to recognize that they are not acting by choice but losing control of their drug-taking.

CAM and Masking

When the conduit also serves as a drug cue, then the conduit provides a common target for sign-tracking of drug-taking and operant drug self-administration. To be clear, in the language of the animal learning literature, the conduit is the response manipulandum that the subject must contact in order to obtain the drug’s rewarding effects. The response manipulandum, therefore, serves as the target at which the operant drug self-administration response is directed. Examples of the manipulandum in the animal learning laboratory are the pecking key in the operant chamber of the pigeon or the response lever in the operant chamber of the rat. The conduit induces sign-tracking to the extent that the conduit is a reward cue, that is, when the conduit CS predicts the rewarding effects of the drug US. In the animal learning laboratory, examples of the reward cue are the sound of a tone emanating from a loudspeaker, which signals that the reward is available for performing the instrumental response. Other examples are the color (i.e., wavelength) of a light stimulus projected onto the pigeon's pecking key, or the illumination of a small light located inside or just above the rat's response lever. Of particular interest are arrangements where the conduit serves as the response manipulandum and, in addition, as the reward cue. Under such conditions, the conduit provides for Cue-At-Manipulandum (CAM), an arrangement that induces the performance of sign-tracking CRs that have often been mistaken for the operant responses that induced them (Tomie, 1995, 1996).

Consider the case where the conduit is the cocktail glass employed to self-administer an alcoholic beverage. The operant alcohol drinking response is directed at the cocktail glass that serves the role of the instrumental response manipulandum. The subject must contact the cocktail glass in order to obtain alcohol's rewarding effects. The cocktail glass conduit may also serve the function of a reward cue when the cocktail glass is positively correlated with the availability of the drug's rewarding effects. Under these conditions, the cocktail glass provides for CAM and allows sign-tracking of drug-taking to develop and co-mingle with operant drug-taking. Thus, the conduit that provides for CAM induces sign-tracking that is targeted at the same location as the operant drug-taking response. In this way, the conduit serves as a tool that allows sign-tracking to masquerade as operant drug-taking.

CAM and the Blind Spot

The animal learning literature reveals that operant procedures that provide for CAM induce sign-tracking CRs that resemble the operant responses that induced them (for reviews, see Hearst & Jenkins, 1974; Schwartz & Gamzu, 1977; Tomie, 1995, 1996). Remarkably, this resemblance was so striking to the naked eye that specifically trained scientific observers could not tell them apart (Schwartz, 1975; Schwartz, Hamilton, & Silberberg, 1975; Schwartz & Williams, 1972). In addition, during CAM procedures both response forms are directed at the same location, therefore, the induced sign-tracking CRs have been mistaken for and added to the frequency counts of operant responses (for reviews, see Hearst & Jenkins, 1974; Schwartz & Gamzu, 1977). Thus, the experimental scientific evidence reveals that when the operant response manipulandum provides for CAM, then sign-tracking CRs are masked to pass for operant responding. The masking by CAM is so effective that it was revealed only by rigorous experimental analysis in the animal learning laboratory.

For many years, learning scientists were perplexed by what was presumed to be the excessive levels of operant responding that developed during CAM procedures. Laboratory experiments examined the effects of manipulating the location of the cue with respect to the manipulandum or, alternatively, the cue's correlation with the reward. These studies revealed that excessive operant-like responding was not observed in these non-CAM control conditions. Moreover, it was discovered that the excessive operant-like responding varied with conditions known to be conducive to the induction of sign-tracking CRs. Investigators concluded that CAM recruits sign-tracking CRs that are indistinguishable from and additive with ongoing operant responding (Hearst & Jenkins, 1974; Schwartz & Gamzu, 1977). To detect the presence of sign-tracking, innovative experimental analyses of the effects of CAM arrangements were required before scientists who study learning discovered the presence of this blind spot. In this way, scientists discovered that sign-tracking CRs had long been mistaken for voluntary operant responses, and the mistaken identity created the false impression that operant responding was being performed to excess (Tomie, Brooks, & Zito, 1989). Note that the CAM arrangements that produce this masking effect, this blind spot, are analogous to those experienced by humans during drug-taking.

Non-CAM Controls

CAM procedures produce higher rates of operant-like responding than do non-CAM control procedures (Tomie, 1995, 1996). Signal-key studies reveal that this effect is due to the induction of sign-tracking CRs which, during CAM procedures, add to frequency counts of operant responding. In signal-key studies, for example, pigeons peck an operant response key (manipulandum) for food reward. In CAM procedures, the reward cue, a green keylight, is projected onto the operant manipulandum key. Non-CAM controls receive similar procedures except the green keylight reward cue is projected onto another key (signal key) located at a distance from the operant key (Hearst & Gormley, 1976; Keller, 1974; McSweeney, Dougan, & Farmer, 1986; Schwartz, 1975). These pigeons peck the operant key, as is required to obtain the reward, but at a reduced rate, relative to the CAM group. The non-CAM controls also pecked the signal key where the green keylight predictive of the food reward was projected. They did this even though pecking the signal key did not serve the purpose of procuring the food reward. Thus, signal-key studies reveal that when operant procedures provide for CAM, sign-tracking CRs are induced, and they are targeted at the operant response key, yielding higher rates of operant-like responding relative to non-CAM controls. The scientific literature reveals that the naked eye cannot differentiate sign-tracking CRs from operant responses, and rigorous experimental analysis in the animal learning laboratory is required to distinguish between them.

Another non-CAM control procedure is the non-differential reward training schedule (for reviews, see Dunham, 1968; Rachlin, 1973; Terrace, 1972). In these studies, pigeons peck an operant key (manipulandum) for food reward. The manipulandum key is illuminated either by a green light or a red light, which alternate sequentially. In the first phase, the non-CAM control procedures are in effect. The food reward is equally likely when pigeons peck the green keylight or the red keylight. In the second phase, the schedule in effect during the green keylight remains as before, but the red keylight now signals that an extinction schedule is in effect. The green keylight is a reward cue; therefore, CAM procedures are in effect, and the rate of responding directed at the green keylight increases dramatically.

For many years, this effect, called “positive behavioral contrast,” was interpreted as elevated operant responding, even though the higher rates of operant-like responding did not produce more food rewards. It is a well-documented property of time-based interval schedules of reward that the number of rewards delivered is largely independent of the rate of responding (Ferster & Skinner, 1957). The non-CAM control procedures favored a different interpretation of the positive behavioral contrast effect. They revealed that the excessive pecking directed at the green keylight was due to the signal value of the green keylight. This is consistent with an interpretation based on the induction of sign-tracking CRs that were simply misconstrued as operant responses. It should be noted that positive behavioral contrast during CAM procedures have also been reported in other species, including rats (Allison, 1976; Atnip, 1985; Freeman, 1971; Higa & McSweeney, 1987; Jensen & Fallon, 1973; Karpicke, Christoph, Peterson, & Hearst, 1977; Peterson, Ackil, Frommer, & Hearst, 1972) and goldfish (Bottjer, Scobie, &

Wallace, 1977). These data reveal that sign-tracking CRs have often been counted as operant responses and that non-CAM control procedures have been required to distinguish between them.

Preposterous Imposters

CAM induces sign-tracking of non-instrumental performance of instrumental-like responding. This type of effect may bedevil the drug abuser who intends to refrain from drug use but is instead triggered to have yet another. In this way, CAM may induce mistake-prone, erroneous, and unintended behavior that appears to be an operant or instrumental response but is instead a sign-tracking response. The unwelcome behavior is actually a well-disguised imposter. The actions are not voluntary, they are reflexive. Some examples of the sign-tracking CR posing as an error-prone operant response include the misbehavior effect and the feature-learning effect.

The "misbehavior of organisms" was first reported by professional animal trainers, Keller and Marian Breland, who successfully applied Skinnerian reinforcement contingencies in the training of thousands of animals in a variety of tasks (K. Breland & M. Breland, 1961, 1966). They did, however, experience some rather perplexing instances where things did not go according to plan. In a typical example, raccoons were trained to pick up wooden coins and deposit them through a slot into a small metal box for food reward. Though initially things went well, with further training the raccoons began to experience problems. They seemed unable to let go of the coins, spending several minutes handling them with their forepaws and "rubbing them together in a most miserly fashion" (K. Breland & M. Breland, 1961). The raccoons often dipped the coins into the slot only to pull them out again. In the end, the coins were chewed, licked, scratched, clawed, rubbed, and washed, but rarely deposited. Remarkably, the actions of the raccoons made it appear as if they were trying to clean a morsel of food. Further training only made matters worse, until the project was reluctantly abandoned. Other abandoned projects attempted similar training with rats, pigs, squirrel monkeys, chickens, turkeys, otters, porpoises, and whales. This "misbehavior" should not be construed as the distraction of an animal that has lost interest in eating, because increasing the animal's hunger merely intensifies this effect (K. Breland & M. Breland, 1961, 1966). For video of raccoons exhibiting misbehavior due to sign-tracking, see: https://tailoftheraccoon.com/the-integrated-reward-system/.

Procedures conducive to misbehavior provide for CAM. The coin serves as the reward cue and as the instrumental response manipulandum. Misbehavior is revealed by the development of a prohibited response that occurs excessively and persists despite contingent non-reinforcement. Misbehavior is an instance where CAM induces mistake-prone, erroneous, and unintended behaviors that appear to be operant or instrumental responses, when, in reality, the responses are actually due to sign-tracking, the well-disguised imposter. It should be noted that the misbehaving raccoons resemble drug abusers whose intention to refrain is thwarted by their triggered actions to have yet another. They are unable to stop themselves. They are repeatedly stymied by their inability to control themselves, as they are reflexively triggered to approach, contact, and "consume" the object that has been paired with the reward.

The feature-learning effect provides another instance where the intrusion of sign-tracking induced error-prone responding during operant procedures. In a typical study, the subject responds by touching the stimulus display, which is the response manipulandum. The S+ display is a red dot on a green background. The S- display is the same, except without the red dot. This is the feature-positive discrimination, which yields excellent accuracy. The feature-negative procedure reverses the displays, so that the red dot, the distinguishing feature, is on the S-. Although the stimulus displays used in the two discrimination tasks are equally distinguishable from one another, subjects in the feature-negative condition make far more errors (Bitgood, Segrave, & Jenkins, 1976; Norton, Muldrew, & Strub, 1971; Sainsbury, 1971).

Analysis of the location of responding supports a sign-tracking interpretation (Crowell & Bernhardt, 1979; Hearst & Jenkins, 1974). Feature-positive subjects respond to the S+ display by touching the red dot, thereby recording a correct response and earning the reward. Feature-negative subjects, on the other hand, respond to their S+ display by touching the green background. Apparently, the pairing of the green display with the reward induces sign-tracking CRs, leading to incorrect responding on S- trials. Some children expressed frustration upon performing the error, because they knew better, but responded without thinking (personal communication, H. Strub). This suggests that feature-negative errors were induced by sign-tracking CRs posing as operant responses. These sign-tracking CRs are preposterous imposters that served to sabotage the intention of the subject, which was to produce accurate discrimination performance.

CAM and the Alcohol Sipper

In the animal laboratory, voluntary operant alcohol self-administration is observed when a rat drinks an alcohol solution from a sipper tube. Note that the sipper tube is the response manipulandum, a tool employed as a conduit to assist in the drinking of the alcoholic beverage. The positive contingency between the sipper tube CS and alcohol (i.e., CAM arrangement) will elicit sign-tracking CR performance of sipper CS-directed alcohol drinking, and the alcohol drinking due to sign-tracking will add to operant alcohol drinking, resulting in elevated levels of alcohol intake relative to non-CAM controls. Studies that vary the contingency between the sipper CS and alcohol US show that alcohol intake varies directly with the positive contingency between the sipper CS and alcohol US (Tomie, Gittleman, Dranoff, & Pohorecky, 2005; Tomie, Miller, Dranoff, & Pohorecky, 2006). Note that the elevated alcohol drinking, presumably due to sign-tracking, is indistinguishable from operant alcohol self-administration.

Sign-tracking CR performance is sensitive to the positive contingency between the CS and the US, which varies as a function of the ratio of the duration of the non-CS (i.e., sipper CS retraction) periods relative to the duration of the CS presentation (i.e., sipper CS insertion) periods (Balsam & Gibbon, 1988; Gallistel & Gibbon, 2000; Jenkins, Barnes & Barrera, 1981). Intermittent sipper procedures improve the covariation between the sipper CS and the alcohol US by increasing the amount of time during the daily drinking session that the sipper CS and, consequently, the alcohol US, are both absent. The CAM hypothesis predicts that elevated sign-tracking of alcohol drinking will be induced by extending the periods of time during the daily drinking session when the sipper CS is retracted from the drinking chamber. This schedule increases the contingency between the sipper and the alcohol. An intermittent schedule of availability of the alcohol sipper CS (Intermittent Sipper procedure) provides for repeated insertions and retractions of the alcohol sipper CS such that the alcohol sipper CS is removed from the drinking chamber for the majority of the drinking session. In a Continuous Sipper procedure, fixed position alcohol drinking tubes provide for continuous availability of the alcohol sipper CS during the entire duration of the drinking session. Remarkably, it has been reported in several studies that the Intermittent Sipper procedure induced more alcohol drinking than the Continuous Sipper procedure, even though the Continuous Sipper procedure made the alcohol solution available to the rats for a much longer time (Tomie, et al., 2005; Tomie, et al., 2006).

Similar effects of intermittent sipper procedures versus continuous sipper procedures have been reported in studies of home cage alcohol drinking. Rats provided with continuous access to the alcohol sipper in their home cage drink less alcohol per day than rats deprived of access to the alcohol sipper, and consequently, access to the alcohol solution, on some of the days (Brancato, Plescia, Lavanco, Cavallaro, & Cannizzaro, 2016; Carnicella, Ron, & Barak, 2014; Loi et al., 2010; Peris, Rhodes, McCullough, Aramini, & Zharikova, 2015; Sabino, Kwak, Rice, & Cottone, 2013; Simms, et al., 2008; Simms, Nielsen, Li, & Bartlett, 2013; Wise, 1973). This effect has also been reported in mice (Melendez, 2011). This suggests that the effects of the contingency between sipper CS and alcohol US are evident across a broad range of alcohol drinking procedures.

CAM and Problem Drinking

STM predicts that, due to CAM-induced sign-tracking, excessive and poorly controlled alcohol drinking (i.e., problem drinking) will vary directly with the positive contingency between the conduit CS and alcohol US. Consider the following scenario. Suppose that your favorite drink is a double martini, extra dry, with two olives and an onion, served in a lead crystal cocktail glass. Suppose further that all of the alcohol that you drink is consumed in this way. Under these conditions, your alcohol drinking repertoire is extremely narrow. While this does simplify the ordering of drinks (i.e., “I’ll have the usual”), the narrow drinking repertoire is an alcohol drinking style that, according to STM will encourage problem drinking, due to the development of sign-tracking. This is because alcohol’s rewarding effects are only experienced after the cocktail glass has been experienced, making the cocktail glass an excellent cue that is highly predictive of alcohol reward. The cocktail glass will, therefore, readily elicit sign-tracking, resulting in reflexive acts of automatic and unintended alcohol drinking. Therefore, due to sign-tracking the narrow drinking repertoire will be associated with binge episodes of excessive alcohol drinking and elevated rates of problem drinking.

Addiction nosologists studying the progression into alcoholism have confirmed the longitudinal trend toward the narrowing of the drinking repertoire (Cottler, Phelps, & Compton, 1995; Jellinek, 1960; McCreary, 2002). Eventually, alcohol is consumed only in the form of a favorite alcoholic beverage that is served only in a particular form of glassware. This alcohol drinking style eventually becomes a ritual that has been noted to presage the nosological progression into more frequent episodes of excessive and poorly controlled alcohol drinking (Jellinek, 1960). The link between the narrowing of the drinking repertoire and the subsequent onset of problem drinking is consistent with the hypothesis that the CAM arrangement, and, consequently, the induction of sign-tracking, contributes to poorly controlled alcohol drinking in humans.

Additional evidence consistent with the CAM hypothesis is provided by cultural anthropologists who noted higher rates of problem drinking in Northern Europe, as compared to Mediterranean Europe, even though the per capita consumption of alcohol in those regions is comparable (de Lint, 1973; Heath, 1987). While there are many cultural aspects of alcohol drinking that may contribute to these regional disparities in problem drinking rates, it should be noted that the types of glassware used to consume alcoholic beverages differ across regions and in accordance with predictions by the CAM hypothesis. Northern European cultures have widely adopted a style of drinking alcohol almost exclusively from specialized containers, including lead crystal stemware, goblets, and flutes, as well as metal ware in the form of beer mugs and ale steins.

This style of drinking is in contrast to Mediterranean Europe, where alcohol is often consumed from common everyday glassware of the sort typically used to consume water or other non-alcoholic beverages (Levin, 1990). The common glassware is a poor alcohol cue because it is often present even though alcohol's effects are not, and, therefore, the common glassware is less likely to elicit sign-tracking. The link between specialized glassware and elevated rates of problem drinking is consistent with the hypothesis that CAM-induced sign-tracking contributes to the loss of control of alcohol drinking in humans.

If problem drinking in humans is partially due to CAM-induced sign-tracking of alcohol drinking, then an effective therapeutic remedy would be to reduce the cue value of the glassware used to consume alcoholic beverages. For example, consider the effects of pouring your favorite alcoholic beverage into a soup bowl. The soup bowl, that presumably has never been used to drink an alcoholic beverage, will be unlikely to trigger sign-tracking of alcohol drinking, so that the alcohol consumed will be limited to that which was intended. Another remedy to correct excessive drinking would consist of using your favorite specialized alcohol glassware to drink non-alcoholic beverages, such as milk, soft drinks, fruit juice, and vegetable smoothies. This practice will reduce the cue value of that glassware as a signal for alcohol reward, making your favorite specialized alcohol glassware less effective as a trigger. In addition, by using common tools unrelated to alcohol drinking, the entire ritual of consumption would be disrupted, thereby making the process much less familiar and enjoyable. In summary, evidence from studies of cross-cultural drinking styles reveals that problem drinking rates vary directly with the cue value of the container used to consume alcoholic beverages. This is consistent with the CAM hypothesis and suggests that problem drinking in humans, that has been interpreted as voluntary alcohol drinking performed to excess, is in part due to sign-tracking of alcohol drinking. This reflexive and excessive use of alcohol is camouflaged by CAM to pass for operant alcohol self-administration. Thus, a good recipe for the induction of problem drinking in humans is CAM.

Addiction Scotoma

A visual scotoma is a blind spot in the visual field, due to a floater, a cataract, or other physical obstruction in the eye. A mental or psychological scotoma is a blind spot in our perception, in the way we view reality. It is, therefore, in a sense, a denial of reality. The denial of reality is a losing proposition that pits our perception against what is true. This is a battle that we can never win. In any dispute with reality, we must lose eventually (Paul, 2017). Such is the case with drug addiction, where turning a blind eye toward the loss of self-control can only make matters worse. The question remains, why is the addiction blind spot so prevalent? Why are we unable to realize that we are losing control of our drug-taking? Part of the problem is our tendency to see things as they were before. Early in the process, when drug use was initiated, drug-taking was strictly voluntary and intended, so that these repetitions of voluntary acts of operant drug-taking provided us with a presented pattern. Factors related to representational momentum and cognitive momentum favor forward projected displacements that continue this presented pattern (Freyd, 1987; Hubbard, 2010, 2015, 2017; Miura, 1990). With regard to drug-taking, the repetitions of voluntary acts of operant drug-taking clearly provide the presented pattern, so that additional instances of drug-taking are likely to be seen as the mere continuation of this previously established pattern. Due to cognitive momentum, the user is biased to see drug-taking as voluntary and intended, regardless of whether the drug-taking was an operant or a sign-tracking CR. The previously well-established presented pattern of voluntary and intended drug-taking influences the user to see sign-tracking but mistake it as more of the same. We are blind to the influence of sign-tracking because of the strong expectation formed by the previously presented pattern of voluntary drug use.

When it comes to recognizing we are losing control of our drug-taking, another part of the problem, is the “illusion of control,” our inclination to overestimate our ability to exercise control over events in our lives (Chapin & Coleman, 2009; Gouveia & Clark, 2001). The illusion of control is consistent with a pervasive optimistic bias that fuels the ego-hypertrophic overconfidence that causes the user to fail to internalize risk (Kendler, Prescott, Myers, & Neale, 2003; Leyton & Stewart, 2014). The overconfident user believes that the risk of losing control of drug-taking is something that may happen to others, but it will never happen to them. They are in denial of how much is at risk. The illusion of control biases the user to see themselves as always in control of their drug-taking, and this is the case even when there is evidence that suggests self-control failure. Due to the overconfidence instilled by the illusion of control, the user’s perception will be biased so as to be highly resistant to the possibility they may lose control of their drug-taking.

To explain this overconfidence, Kahneman (2011) has introduced a concept that he labels What You See Is All There Is (WYSIATI). According to this theory, when the mind draws a conclusion, it deals primarily with what it knows. These are the phenomena that the mind has already observed, that is, Known Knowns. In the case of addiction, the Known Knowns are the previously performed acts of drug-taking, which were uniformly voluntary and intended. Thus, the mind concludes that acts of drug-taking are voluntary and intended actions. The mind rarely considers Known Unknowns, phenomena that it knows to be relevant but about which it has no information. An example of Known Unknowns is when the mind has been warned of triggers, or has heard of addiction, but lacks understanding of how they actually work. Finally, the mind is completely oblivious to the possibility of Unknown Unknowns, which are unknown phenomena of unknown relevance, such as sign-tracking. Thus, early on, according to WYSIATI, the pattern of voluntary and intended drug-taking establishes the Known Knowns as the set of possible exemplars of drug-taking forms. It follows, therefore, that all acts of drug-taking that you witness appear to be of this sort, that is, intended and voluntary. This is so because, with respect to drug-taking, intended and voluntary drug-taking are perceived as all there is.

A psychological scotoma is a mental activity in which one locks on to one idea and excludes all others. For example, the idea that I am in control of my drug-taking is locked in, while all other possibilities are locked out. In this way, I protect what I wish to maintain as my truth, even if it is not true. The addiction blind spot, the psychological scotoma, that leads the user to be unable to recognize that they are losing control of their drug-taking, is likely to develop because many of the predisposing factors that favor the development of the scotoma are present. For example, (1) representational momentum and cognitive momentum favor forward projected displacements that continue the presented pattern of well-controlled drug-taking, and; (2) the illusion of control biases the ego-hypertrophic and overconfident user to overestimate their control of their drug-taking, while failing to internalize the risk of losing self-control, and; (3) because of WYSIATI, the user sees voluntary and intended drug-taking and concludes that, going forward, voluntary and intended drug-taking is all there is.

These psychological factors relate to the processing and filtering of the mind's representations of reality. They do not act alone. They interact with the physical properties of the drug use environment (drug rewards) that produce operant drug-taking through the use of a conduit, which, in turn, induces sign-tracking of drug-taking, so that voluntary and reflexive drug-taking responses resemble each other and are targeted at the same location (CAM). The addiction blind spot, therefore, is created by the confluence of several factors that work together to blur the acuity necessary to distinguish reflexive acts of drug-taking from those that are intended.

The blind spot increases the risk of addiction by concealing the evidence from the user that they are losing control of their drug-taking, while simultaneously, increasing their use of the drug. The remedy for this situation is to develop tools to boost the user's awareness of and ability to recognize the loss of self-control, and how the loss of self-control of drug-taking contributes to the development of drug addiction. Levitch, Marcinkowski-Paulis, and Tomie (this volume) report that telling stories about sign-tracking is an effective tool for boosting awareness of self-control and how the loss of self-control relates to drug addiction in 9th–12th grade students. Although psychological scotoma obscures the detection of these factors, our objective is to arm the casual drug user with knowledge of sign-tracking which will reveal the stealthy aspects of the drug addiction process. By boosting awareness of the difference between voluntary operant drug-taking as opposed to reflexive sign-tracking of drug-taking, the drug user will be able to more quickly recognize the early indications that freedom of choice is slipping away and is being replaced by an automatic and reflexive form of drug use.

References

- Allison, J. (1976). Contrast, induction, facilitation, suppression, and conservation. Journal of the Experimental Analysis of Behavior, 25(2), 185–198. doi:10.1901/jeab.1976.25–185.
- Atnip, G. W. (1985). Positive contrast in the rat with the stimuli on the operanda. Psychological Record, 35(4), 583–588.
- Babor, T. (1995). The DSM-IV field trial for substance use disorders: Major results. Brief commentaries on this article by Cottler et al. pp. 59–69. Commentary no. 2: The road to DSM-IV: Confessions of an erstwhile nosologist. Drug & Alcohol Dependence, 38(1), 75–79. doi:10.1016/0376–8716(94)01092-Y.
- Balsam, P. D., & Gibbon, J. (1988). Formation of tone—US associations does not interfere with the formation of context—US associations in pigeons. Journal of

Experimental Psychology: Animal Behavior Processes, 14(4), 401–412. doi:10.1037/0097–7403.14.4.401.

- Barker, J. M., & Taylor, J. R. (2014). Review: Habitual alcohol seeking: Modeling the transition from casual drinking to addiction. Neuroscience & Biobehavioral Reviews, 47, 281–294. doi:10.1016/j.neubiorev.2014.08.012.
- Bitgood, S., Segrave, K., & Jenkins, H. (1976). Verbal feedback and the feature-positive effect in children. Journal of Experimental Child Psychology, 21(2), 249–255. doi:10.1016/0022–0965(76)90038–2.
- Bottjer, S., Scobie, S. R., & Wallace, J. (1977). Positive behavioral contrast, autoshaping, and omission responding in the goldfish (Carassius auratus). Animal Learning & Behavior, 5(4), 336–342. doi:10.3758/BF03209575.
- Brancato, A., Plescia, F., Lavanco, G., Cavallaro, A., & Cannizzaro, C. (2016). Continuous and intermittent alcohol free-choice from pre-gestational time to lactation: Focus on drinking trajectories and maternal behavior. Frontiers in Behavioral Neuroscience, 10(31). doi:10.3389/fnbeh.2016.00031.
- Breland, K., & Breland, M. (1961). The misbehavior of organisms. American Psychologist, 16(11), 681–84. doi:10.1037/h0040090.
- Breland, K., & Breland, M. (1966). Animal behavior. New York, NY: Macmillan.
- Brown, P., & Jenkins, H. (1968). Auto-shaping of the pigeon's key-peck. Journal of the Experimental Analysis of Behavior, 11(1): 1–8.
- Carnicella, S., Ron, D., & Barak, S. (2014). Intermittent ethanol access schedule in rats as a preclinical model of alcohol abuse. Alcohol, 48(3), 243–252. (Special Issue on Animal Models of Excessive Alcohol Consumption: Recent Advances and Future Challenges). doi:10.1016/j.alcohol.2014.01.006.
- Chapin, J., & Coleman, G. (2009). Optimistic bias: What you think, what you know, or whom you know? North American Journal of Psychology. 11(1), 121–132.
- Corbit, L. H., & Janak, P. H. (2016). Habitual alcohol seeking: Neural bases and possible relations to alcohol use disorders. Alcoholism: Clinical & Experimental Research, 40(7), 1380–1389. doi:10.1111/acer.13094.
- Cottler, L., Phelps, D., & Compton III, W. M. (1995). Narrowing of the drinking repertoire criterion: Should it have been dropped from ICD-10? Journal of Studies of Alcohol, 56(2), 173–176. doi:10.15288/jsa.1995.56.173.
- Crowell, C., & Bernhardt, T. (1979). The feature-positive effect and sign-tracking behavior during discrimination learning in the rat. Animal Learning & Behavior, 7(3), 313–317. doi:10.3758/BF03209675.
- de Lint, J. (1973). The epidemiology of alcoholism: The elusive nature of the problem, estimating the prevalence of excessive alcohol use and alcohol-related mortality, current trends and the issue of prevention. Toronto: Addiction Research Foundation of Ontario.
- de Wit, S., Watson, P., Harsay, H., van de Vijver, I., Ridderinkhof, K., & Cohen, M. (2012). Corticostriatal connectivity underlies individual differences in the balance between habitual and goal-directed action control. Journal of Neuroscience, 32(35), 12066–12075. doi:10.1523/JNEUROSCI.1088–12.2012.
- Dunham, P. (1968). Contrasted condition of reinforcement: A selective critique. Psychological Bulletin, 69(5), 295–315. doi:10.1037/h0025690.
- Ferster, C. B., & Skinner, B. F. (1957). Schedules of reinforcement. New York, NY: Appleton-Century-Crofts.

- Formica, M. J. (2012, May 15). Addiction’s blind spot: Constancy, overconfidence and conscious recovery. Psychology Today. Retrieved September 15, 2017, from www.psychologytoday.com/blog/enlightened . . . /201205/addiction-s-blind-spot.
- Freeman, B. (1971). The role of response-independent reinforcement in producing behavioral contrast effects in the rat. Learning & Motivation, 2(2), 138–147. doi:10.1016/0023–9690(71)90004-X.
- Freyd, J. (1987). Dynamic mental representations. Psychological Review, 94(4), 427–438. doi:10.1037/0033–295X.94.4.427.
- Gallistel, C. R., & Gibbon, J. (2000). Time, rate, and conditioning. Psychological Review, 107(2), 289–344. doi:10.1037/0033–295X.107.2.289.
- Gouveia, S., & Clarke, V. (2001). Optimistic bias for negative and positive events. Health Education, 101(5), 228–234. doi:10.1108/09654280110402080.
- Hearst, E., & Gormley, D. (1976). Some tests of the additivity autoshaping theory of behavioral contrast. Animal Learning & Behavior, 4(2), 145–150. doi:10.3758/BF03214025.
- Hearst, E., & Jenkins, H. M. (1974). Sign-tracking: The stimulus-reinforcer relation and directed action. Austin, TX: Psychonomic Society.
- Heath, D. B. (1987). Anthropology and alcohol studies: Current issues. Annual Review of Anthropology, 16, 99–120.
- Herrnstein, R., & Loveland, R. (1972). Food-avoidance in hungry pigeons, and other perplexities. Journal of the Experimental Analysis of Behavior, 18(3), 369–383. doi:10.1901/jeab.1972.18–369.
- Higa, J., & McSweeney, F. (1987). Behavioral contrast in rats when qualitatively different reinforcers are used. Behavioral Processes, 15(2–3), 131–142. doi:10.1016/0376–6357(87)90002–7.
- Hubbard, T. (2010). Approaches to representational momentum: Theories and models. In R. Nijhawan, & B. Khurana (Eds.), Space and time in perception and action (pp. 338–365). Cambridge University Press. doi:10.1017/CBO9780511750540.
- Hubbard, T. (2015). The varieties of momentum-like experience. Psychological Bulletin, 141(6), 1081–1119. doi:10.1037/bul0000016.
- Hubbard, T. (2017). Toward a general theory of momentum-like effects. Behavioural Processes, 141(1), 50–66. doi:10.1016/j.beproc.2017.02.019.
- Jellinek, E. (1960). The disease concept of alcoholism. New Haven, CT: College and University Press in Association with Hill House Press, New Brunswick, NJ.
- Jenkins, H. M., Barnes, R. A., & Barerra, F. (1981). Why auto-shaping depends on trial spacing. In C. M. Locurto, H. S. Terrace, & J. Gibbon (Eds.), Autoshaping and conditioning theory (pp. 255–284). New York, NY: Academic Press.
- Jensen, C., & Fallon, D. (1973). Behavioral aftereffects of reinforcement and its omission as a function of reinforcement magnitude. Journal of the Experimental Analysis of Behavior, 19(3), 459–468. doi:10.1901/jeab.1973.19–459.
- Kahneman, D. (2011, October 19). Don’t blink! The hazards of confidence. New York Times. Retrieved November 4, 2017, from www.nytimes.com/2011/10/23/magazine/dont-blink-the-hazards-of-confidence.html.
- Karpicke, J., Christoph, G., Peterson, G., & Hearst, E. (1977). Signal location and positive versus negative conditioned suppression in the rat. Journal of Experimental Psychology: Animal Behavior Processes, 3(2), 105–118. doi:10.1037/0097–7403.3.2.105.

- Keller, K. (1974). The role of elicited responding in behavioral contrast. Journal of the Experimental Analysis of Behavior, 21(2), 249–257. doi:10.1901/jeab.1974.21–249.
- Kendler, K., Prescott, C., Myers, J., & Neale, M. (2003). The structure of genetic and environmental risk factors for common psychiatric and substance use disorders in men and women. Archives of General Psychiatry, 60(9), 929–937. doi:10.1001/archpsyc.60.9.929.
- Levin, J. (1990). Alcoholism: A biopsychosocial approach. New York, NY: Hemisphere Publishing Corp.
- Levitch, E. A., Marcinkowski-Paulis, S. E., & Tomie, A. (2018). Telling stories about sign-tracking boosts awareness of loss of self-control related to drug use. In Sign-tracking and drug addiction. Ann Arbor, MI: Maize Books.
- Leyton, M., & Stewart, S. (Eds.). (2014). Substance abuse in Canada: Childhood and adolescent pathways to substance use disorders. Ottawa, ON: Canadian Centre on Substance Abuse.
- Locurto, C., Terrace, H., & Gibbon, J. (1976). Autoshaping, random control, and omission training in the rat. Journal of the Experimental Analysis of Behavior, 26(3), 451–462. doi:10.1901/jeab.1976.26–451.
- Locurto, C., Terrace, H., & Gibbon, J. (1978). Omission training (negative automaintenance) in the rat: Effects of trial offset. Bulletin of the Psychonomic Society, 12(1), 11–14. doi:10.3758/BF03329610.
- Locurto, C. M. (1981). Contributions of autoshaping to the partitioning of conditioned behavior. In C. M. Locurto, H. S. Terrace, & J. Gibbon (Eds.), Autoshaping and conditioning theory (pp. 101–135). New York, NY: Academic Press.
- Loi, B., Lobina, C., Maccioni, P., Fantini, N., Carai, M., Gessa, G., & Colombo, G. (2010). Increase in alcohol intake, reduced flexibility of alcohol drinking, and evidence of signs of alcohol intoxication in sardinian alcohol-preferring rats exposed to intermittent access to 20% alcohol. Alcoholism: Clinical &Experimental Research, 34(12), 2147–2154. doi:10.1111/j.1530–0277.2010.01311.x.
- McCreary, D. R. (2002). Binge drinking in adulthood: The influence of gender, age, and beverage exclusivity. International Journal of Men's Health, 1, 233–245.
- McSweeney, F., Dougan, J., & Farmer, V. (1986). Simple schedule and signal-key multiple schedule responding and behavioral contrast. Bulletin of the Psychonomic Society, 24(1), 88–90. doi:10.3758/BF03330512.
- Melendez, R. (2011). Intermittent (every-other-day) drinking induces rapid escalation of ethanol intake and preference in adolescent and adult C57BL/6J mice. Alcoholism: Clinical & Experimental Research, 35(4), 652–658. doi:10.1111/j.15300277.2010.01383.x.
- Miura, T. (1990). Active function of eye movement and useful field of view in a realistic setting. In R. Groner, G. d'Ydewalle, & R. Parham (Eds.), From eye to mind: Information acquisition in perception, search, and reading (pp. 119–127). North-Holland, England: Oxford Press.
- Norton, G., Muldrew, D., & Strub, H. (1971). Feature-positive effect in children. Psychonomic Science, 23(4), 317–318. doi:10.3758/BF03336129.
- Paul, G. (2017). How psychological scotoma keeps you trapped in addiction. Posted November 16, 2017. Retrieved November 30, 2017, from www.hope-rehab-center-thailand.com › Posts ›Blog › Addiction, Personal Development.

- Peris, J., Rhodes, N., McCullough, B., Aramini, R., & Zharikova, A. (2015). Intermittent high-dose ethanol exposure increases ethanol preference in rats. Journal of Studies on Alcohol and Drugs, 76(1), 165–173. doi: 10.15288/jsad.2015.76.165.
- Peterson, G. B., Ackil J. E., Frommer, G. P., & Hearst, E. (1972). Conditioned approach and contact behavior toward signals for food or brain-stimulation reinforcement. Science, 177(4053), 1009–011. doi:10.1126/science.177.4053.1009.
- Rachlin, H. (1973). Contrast and matching. Psychological Review, 80(3), 217–234. doi:10.1037/h0034325.
- Reich, C. (2015, October 15). Understanding the blind spot in addiction. Therapy Insider. Retrieved November 14, 2017, from www.therapyinsider.com/FeedItem/Understanding-the-Blind-Spot-in-Addiction/.
- Sabino, V., Kwak, J., Rice, K. C., & Cottone, P. (2013). Pharmacological characterization of the 20% alcohol intermittent access model in sardinian alcohol-preferring rats: A model of binge-like drinking. Alcoholism: Clinical & Experimental Research, 37(4), 635–643. doi:10.1111/acer.12008.
- Sainsbury, R. S. (1971). Effect of proximity of element on the feature-positive effect. Journal of the Experimental Analysis of Behavior, 16(3), 315–325. doi:10.1901/jeab.1971.16–315.
- Schwartz, B. (1975). Discriminative stimulus location as a determinant of positive and negative behavioral contrast in the pigeon. Journal of the Experimental Analysis of Behavior, 23(2), 167–176. doi:10.1901/jeab.1975.23–167.
- Schwartz, B., & Gamzu, E. (1977). Pavlovian control of operant behavior: An Analysis Of Autoshaping and Its Implications for Operant Conditioning. In W. K. Honig, & J. E. R. Staddon (Eds.), A handbook of operant behavior (pp. 76–108). Englewood Cliffs, NJ: Prentice Hall.
- Schwartz, B., Hamilton, B., & Silberberg, A. (1975). Behavioral contrast in the pigeon: A study of the duration of key pecking maintained on multiple schedules of reinforcement. Journal of the Experimental Analysis of Behavior, 24(2), 199–206. doi:10.1901/jeab.1975.24–199.
- Schwartz, B., & Williams, D. (1972). Two different kinds of key peck in the pigeon: Some properties of responses maintained by negative and positive response-reinforcer contingencies. Journal of the Experimental Analysis of Behavior, 18(2), 201–216. doi:10.1901/jeab.1972.18–201.
- Simms, J. A., Nielsen, C. K., Li, R., & Bartlett, S. E. (2013). Intermittent access ethanol consumption dysregulates CRF function in the hypothalamus and is attenuated by the CRF- R1 antagonist, CP-376395. Addiction Biology, 19(4), 606–611. doi:10.1111/adb.12024.
- Simms, J. A., Steensland, P., Medina, B., Abernathy, K. E., Chandler, L. J., Wise, R., & Bartlett, S. E. (2008). Intermittent Access to 20% Ethanol Induces High Ethanol Consumption in Long—Evans and Wistar Rats. Alcoholism: Clinical & Experimental Research, 32(10), 1816–1823. doi:10.1111/j.1530–0277.2008.00753.x.
- Spoelder, M., Flores Dourojeanni, J. P., de Git, K. C. G., Baars, A. M., Lesscher, H. M. B., & Vanderschuren, L. J. M. J. (2017). Individual differences in voluntary alcohol intake in rats: Relationship with impulsivity, decision making and Pavlovian conditioned approach. Psychopharmacology, 234(14), 2177–2196. doi:10.1007/s00213-017-4617-6.

- Terrace, H. S. (1972). By-products of discrimination learning. In G. H. Bower (Ed.), The psychology of learning and motivation (pp. 195–265), Vol 5. New York, NY: Academic Press.
- Tiffany, S. T. (1990). A cognitive model of drug urges and drug-use behavior: Role of automatic and nonautomatic processes. Psychological Review, 97(2), 147–168. doi:10.1037/0033–295X.97.2.147.
- Tiffany, S. T., & Conklin, C. A. (2000). A cognitive processing model of alcohol craving and compulsive alcohol use. Addiction, 95(Suppl. 2), S145–S153.
- Tomie, A. (1995). CAM: An animal learning model of excessive and compulsive implement assisted drug-taking in humans. Clinical Psychology Review, 15(3), 145–167. doi:10.1016/0272–7358(95)00005-A.
- Tomie, A. (1996). Locating reward cue at response manipulandum (CAM) induces symptoms of drug abuse. Neuroscience & Biobehavioral Reviews, 20(3), 505–535. doi:10.1016/0149–7634(95)00023–2.
- Tomie, A., Badaway, N., & Rutyna, J. (2016). Sign-tracking model of loss of self-control of drug-taking. In Substance Abuse. Open access ebook. Avid Science: Berlin. Retrieved April 10, 2016, from http://www.avidscience.com/wp-content/uploads/2016/04/SA-16-01_April-10-2016.pdf .
- Tomie, A., Brooks, W., & Zito, B. (1989). Sign-tracking: The search for reward. In S. B. Klein & R. R. Mowrer (Eds.), Contemporary learning theory: Pavlovian conditioning and the status of traditional learning theory (pp. 191–223). Hillsdale, NJ: Lawrence Erlbaum.
- Tomie, A., Cunha, C., Mosakowski, E. M., Quartarolo, N. M., Pohorecky, L. A., & Benjamin, D. (1998). Effects of ethanol on Pavlovian autoshaping in rats. Psychopharmacology, 139(1–2), 154–159.
- Tomie, A., Gittleman, J., Dranoff, E., & Pohorecky, L. A. (2005). Social interaction opportunity and intermittent presentations of ethanol sipper tube induce ethanol drinking in rats. Alcohol, 35(1), 43–55. doi:10.1016/j.alcohol.2004.11.005.
- Tomie, A., Grimes, K. L., & Pohorecky, L. A. (2008). Behavioral characteristics and neurobiological substrates shared by Pavlovian sign-tracking and drug abuse. Brain Research Reviews, 58(1), 121–135. doi:10.1016/j.brainresrev.2007.12.003.
- Tomie, A., Miller, W. C., Dranoff, E., & Pohorecky, L. A. (2006). Intermittent presentations of ethanol sipper tube induce ethanol drinking in rats. Alcohol & Alcoholism, 41(3), 225. doi:10.1093/alcalc/ag1002.
- Tomie, A., & Sharma, N. (2013). Pavlovian Sign-Tracking Model of alcohol abuse. Current Drug Abuse Reviews, 6(3), 201–219.
- Williams, D., & Williams, H. (1969). Auto-maintenance in the pigeon: sustained pecking despite contingent non-reinforcement. Journal of the Experimental Analysis of Behavior, 12(4), 511–520. doi:10.1901/jeab.1969.12–511.
- Wise, R. (1973). Voluntary ethanol intake in rats following exposure to ethanol on various schedules. Psychopharmacology, 29(3), 203. doi:10.1007/BF00414034.