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Social facilitation of polydipsia as an animal model of compulsive behavior

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Social Facilitation of Polydipsia as an Animal Model of Compulsive Behavior

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Dedication

I dedicate this project to my parents, Suhad Hashem and Ameed Eluri, sisters, Areeg and Seren Eluri, brothers, Hazem and Tarik Eluri, and my husband, Enad Mahmoud.

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Abstract

Behavior excesses are a key feature in many psychiatric diagnoses. Obsessive-compulsive disorder (OCD), in particular, is almost entirely defined in terms of behavior excesses. Although much research has been conducted on OCD treatment, very little research has focused on understanding how these compulsive behaviors are acquired. The few theories advanced to explain the etiology of OCD compulsions have significant limitations. The purpose of this study is to test social facilitation as a potential mechanism through which compulsive behaviors are acquired, via an animal model. Schedule-induced polydipsia (SIP) was employed as the behavior of interest because there is empirical support indicating it as an animal model of compulsive behavior. The fundamental issue was to determine if naïve rats exposed to rats that drank reliably would (1) show elevated rates of drinking as a result of the exposure, and then (2) acquire SIP more rapidly than rats without that exposure. Twenty-four male Sprague-Dawley rats were randomly designated to be (1) drinking model rats, (2) drinking naïve rats, (3) feeding control model rats, (4) feeding control naïve rats, (5) social contact control model rats, (6) social contact control naïve rats, and (7) naïve control rats. SIP was established in the drinking model rats using a fixed-time 60-second schedule of food delivery (FT-60) with water available. Once stable drinking occurred, the models and their matched naïve rat were placed in the same experimental chamber to determine if drinking in the naïve rat would be socially facilitated. Strong individual differences in drinking by the naïve rats were observed. However, the overall indications were that social facilitation may play a role in enhancing the acquisition of SIP and that social facilitation may be a factor in the development of compulsive behavior.

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Chapter 1: Introduction

Behavior excesses are a key feature in many psychiatric diagnoses, such as pervasive developmental disorders (PDD), tic disorders, and obsessive-compulsive disorder (Bodfish, Symons, Parker, & Lewis, 2000; Scahill et al., 2006). Obsessive-compulsive disorder (OCD) is a particularly salient example of this in that OCD is almost entirely defined in terms of behavior excesses. Specifically, OCD is characterized by (1) the presence of either obsessions or compulsions, or both, (2) recognition by the sufferer, if not a child, that the obsessions or compulsions are excessive, and (3) causes marked distress, is time-consuming, or interferes with a person's routine (DSM-IV-TR, 2000). The definition of OCD under the forthcoming DSM-V will be essentially the same. Obsessions are defined as "recurrent or persistent thoughts, impulses, or images that are experienced as intrusive and inappropriate and that cause marked anxiety or distress" (DSM-IV-TR, 2000). Compulsions are "repetitive behaviors or mental acts used to prevent or reduce anxiety or distress, not to provide pleasure or gratification" (DSM-IV-TR). Obsessions and compulsions are behavioral excesses by definition. OCD has been found to occur in less than 2.5% in the population. There are no reported gender differences in incidence in adult onset OCD. However, childhood onset OCD tends to be more common in boys.

Although compulsions are defined as repetitive behaviors, some researchers believe that this conceptualization is not sufficient to describe the range of repetitive behaviors observed in humans and other organisms (Turner, 1999). Because of this, Turner (1999) attempted to differentiate repetitive behavior into two categories labeled "lower-level" and "higher-level" behaviors. Lower-level behaviors are characterized by repetition of movement (e.g., tics, stereotyped movements, and self-injurious behavior). Higher-level behaviors are

characterized by more complex responses (e.g., insistence on sameness, repetitive language, and circumscribed interests). Although this conceptualization helps clarify two distinct classes of repetitive behavior, it remains unclear what is meant by a "compulsion," beyond repetition, especially since repetitive behaviors are evident in other psychiatric diagnoses such as impulse control disorders, somatoform and eating disorders, and neurological and developmental disorders. Therefore, a compulsion is usually considered to be an excessive expression of a voluntary behavior that results in physical or psychological harm to the individual. Examples include hand-washing, counting, hoarding, ordering, substance abuse, excessive eating, polydipsia, and self-injury. It is important to note that repetitive involuntary movements, especially those associated with typical physiological reactions such as ingestion (Pavlov, 1927; Rusiniak, Hankins, Garcia, & Brett, 1979) pain (Ulrich & Azrin, 1962; Williams & Eichelman, 1971), and those that are induced by repeatedly presented stimuli, are excluded from this definition because they are usually considered adaptive, or at least not harmful.

As mentioned previously, at some point during the course of OCD, the individual must become aware that the obsessions or compulsions are excessive and maladaptive. However, self-awareness is not required for diagnosing OCD in children because they are believed to lack the cognitive ability to understand that these behaviors are excessive and potentially troublesome. This lack of awareness of their OCD is a critical point, because many individuals with less sophisticated or impaired cognitive functioning, regardless of age, may not be aware that some of the behaviors that they engage in are excessive and nonfunctional. It is certainly also possible for people of average intelligence to simply be oblivious to the magnitude and deleterious effects of their own behavior. In any case, for

individuals with no speech, awareness in the conventional sense may not even be a possibility. In addition, individuals with diminished cognitive ability may also not be aware that they are engaging in these acts or may not be able to articulate their reasons for engaging in these behaviors. That is, there seems to be no justification for concluding that highly repetitive maladaptive behavior in those with severe developmental disabilities or traumatic brain injuries, who cannot communicate, are not true compulsions. Moreover, the requirement that the behavior be harmful is both a diagnostic and an ethical convention. The same controlling variables and neurological processes are in place whether the behavior becomes troublesome or not or whether the person recognizes it is not normative. In any case, while the excessive behavior that defines OCD might be recognized by the sufferer, we need not assume that the behavior arises from a rational adaptive process or that its proximal or distal causes are known by the sufferer. Similar conclusions are obviously the case for compulsive behavior in non-humans. Therefore, the awareness and harm criteria may not be useful or necessary in defining these behaviors, or OCD, in general.

Theories on the Development of OCD

There have been many attempts to explain the development of compulsive behaviors (e.g., Beck, 1976; Freud, 1920; Neale & Oltmanns, 1988; Pavlov, 1927; Stein et al. 1999). The theories include, but are certainly not limited to, conceptualizations arising from the psychodynamic, neurobiological, cognitive, and learning perspectives. Each of these major perspectives will be reviewed as a potential explanation for the etiology of compulsive behavior and will include an examination of their limitations. Additionally, some social learning factors that may contribute to the acquisition of compulsive behavior will be discussed and evaluated.

Psychodynamic theories. Psychodynamic theories regarding the etiology of psychopathology have developed particularly from Freud's (1915, 1920, 1933) writings about the psychosexual stages of development (i.e., oral, anal, phallic, latency, and genital). Freud (1920) believed that psychopathology can stem from a disruption in the progression from one psychosexual stage to another during childhood and that the lack of successful progression can result in fixations in libido within a specific stage. These fixations result in inappropriate levels of certain kinds of behavior which would be motivated by factors associated with the stage in which fixation occurs. For example, if a child is fixated on the oral stage of psychosexual development, psychodynamic theories suggest that individuals may experience compulsive oral behaviors, such as smoking, eating, or drinking in adulthood. Problems dealing with these and other behaviors can result in the formation of defense mechanisms to ensure that the troublesome impulses remain unmanifested in the person's unconscious. However, the need to maintain these powerful defenses can result in still more problems.

Directly relevant to OCD, Freud (1920) suggested that during the phallic stage of development, in which children are believed to seek genital stimulation, children experience the unconscious sexual desire for their mother and hatred for their father, commonly known as the Oedipus complex. Typical development, according to this theoretical perspective, involves resolving this complex by the child identifying with the adult rival to reject the child's desire. In individuals with OCD, it is believed that the Oedipus complex is not resolved appropriately, and unconscious feelings of sexuality and aggression become fused, symbolically at least, resulting in excessive expression of thoughts or actions (Brenner, 2002). It is believed that the repetitive thoughts and behaviors in those with OCD, such as the

need for orderliness and cleanliness, are behaviors that are a safe and acceptable expression of feelings of guilt, shame, and punishment associated with unresolved Oedipal desires (Rice, 2004).

Although psychodynamic theories have long been used to attempt to explain psychopathology in general, there is little objective evidence to suggest that these theories actually account for the acquisition and maintenance of OCD. The notion that psychopathology is the result of poor development through the psychosexual stages proposed by Freud (1920), and further discussed by Rice (2004) with regard to OCD, has been based primarily on case studies, rather than direct function or even correlational analyses of causes. The existence of the purported underlying intrapsychic conflicts are difficult, if not impossible, to demonstrate empirically and may be unfalsifiable in principle (Popper, 1963). Moreover, there is no research to support the effectiveness of psychodynamic therapies for OCD. Certainly other types of interventions, such as exposure and response prevention, have proven more effective (Richard & Lauterbach, 2007). Therefore, a review of other theories that have well-established research is warranted.

Neurobiological theories. Markarian and colleagues (2010) reviewed the literature on the neurological basis for the etiology of OCD, specifically focusing on the compulsive behaviors. They reported that the supplementary motor area and premotor cortex are involved in initiating and organizing movements via connections with the primary motor cortex. Roth and colleagues (1996) used MRI technology to detect motor sequences imagined by six participants and found that the premotor cortex and supplementary motor area were activated bilaterally in four of the six participants. Therefore, Roth and colleagues (1996) suggest that these brain regions are activated not only in observable motor behavior

but also when these behaviors are imagined. Therefore, it is likely that these brain regions are activated when engaging in both compulsive behavior and obsessions. This may be especially true if obsessions are conceptualized as repetitive private events, as suggested by Skinner's view that unobserved internal behaviors are controlled by the same principles that are responsible for those behaviors that are observable to others (Skinner, 1953). That is, obsessions are not a different kind of behavior but are unseen private compulsions involving thinking rather than motoric activity.

Maia, Cooney, and Peterson (2008) reported that several brain regions have been implicated in OCD, including the orbitofrontal cortex (OFC), anterior cingulate cortex (ACC), and the head of the caudate nucleus in adults. Research has found that these areas are hyperactive at rest in adults with OCD and become more active when OCD symptoms are observed (Whiteside, Port, & Abramowitz, 2004). The OFC and ACC are connected to the basal ganglia through loops that receive input from multiple areas in the cortex and then go back to the original area closing the loop (Alexander, DeLong, & Strick, 1986; Middleton & Strick, 2000). OCD is believed to be the result of an imbalance between direct and indirect pathways through the basal ganglia, with the net effect of the direct pathway being excitatory and the indirect pathway being inhibitory (Saxena et al., 2001; Saxena et al., 1998; Saxena & Rauch, 2000). Abnormalities within these brain regions are believed to be the cause of OCD (Saxena & Rauch). However, these studies are correlational, and the altered neurological functioning was examined only in individuals already exhibiting OCD symptoms.

Given the neurobiological correlates associated with compulsive behaviors, it is important to also consider the associated neurotransmitters, particularly serotonin and dopamine. It has been hypothesized that serotonin and dopamine play an important role in

the compulsive behavior observed in both OCD and Tourette's Disorder (Insel, Zohar, Benkelfat, & Murphy, 1990). Many studies implicate the serotonergic system in OCD because of the symptom-reducing effects of pharmacological treatment through selective serotonin reuptake inhibitors (March et al., 1998; Rapoport, Leonard, Swedo, & Lenane, 1993). Husted, Shapira, and Goodman (2006) reported that individuals with OCD have excessive excitatory glutamatergic neurons in the orbitofrontal cortex, and serotonin inhibits these neurons. This leads to increased release of serotonin and a subsequent decline in OCD symptoms. In addition, Stein and colleagues (1999) found that serotonin agonists resulted in an increase of OCD symptoms. Research has also found that there are elevated levels of glutamine and glutamate in left caudate in children who engage in compulsive behaviors (Rosenberg et al., 2000).

Other neurophysiological research has focused on the effects of excess dopamine on generating compulsive behaviors (Denys, Zohar, & Westenberg, 2004). For example, Taylor, Rajbhandari, Berridge, and Aldridge (2010) found that dopamine agonists increased repetitive and rigid sequences of grooming behavior and simple forms of motor behavior (e.g., scratching and biting) in rats. In addition, schizophrenic patients treated with levodopa, which increases dopamine in relevant brain structures, began to exhibit OCD symptoms (Neale & Oltmanns, 1988). This research suggests that there is a relationship between serotonin and dopamine neurotransmitters. McDougle, Goodman, and Price (1994) found that medications that block dopamine receptors led to changes in patients' responses to SSRIs, thereby making treatment of OCD symptoms more effective. Further, it has been suggested that serotonin receptor antagonism increases OCD symptoms by increasing the firing rate of dopamine neurons, lending more support to this theory (Ramasubbu, 2002).

Cognitive theories. A variety of "cognitive" theories have also been used to explain compulsions. These generally suggest that some kind of private thought processes or anxiety state mediates the observed aspects of OCD. Thus, the term "cognitive" in this context refers to the intervening verbal behavior and anxiety response associated with OCD, and not the unobservable or metaphorical rule-implementing mechanisms that are invoked in traditional cognitive psychology (e.g., Baars, 1986; Neisser, 1965; Piaget & Inhelder, 1969). That is, these cognitive events are thought of as a kind of private behavior: internal versions of externally applied explicit rules and intervening affective states that serve as instructions and motivating conditions, respectively. These often include obsessions related to health, death, others' welfare, sex, and religion. For example, one cognitive theory suggests that compulsive behavior in OCD develops from a tendency to exaggerate typical concerns of ordinary people combined with a tendency to have unusually high expectations for negative outcomes (Beck, 1976; Carr, 1974). Another theory that has developed from the cognitive literature is that obsessions provoke certain types of negative automatic thoughts. As a result, anxiety develops only if the automatic thoughts affect the individual's belief system (Salkovskis, 1985). It is believed that thinking about unacceptable actions is the same as engaging in these acts. Salkovskis (1985) believed that individuals with OCD had dysfunctional assumptions regarding their responsibility and self-blame and that compulsive behavior was a way to reduce the sense of responsibility and minimize blame.

Foa, Amir, Bogert, Molnar, and Przeworski (2001) examined the role of responsibility in people with OCD, social phobia, and non-anxious controls. The researchers provided OCD-related high-risk and low-risk scenarios for each of the three groups to estimate (1) the degree to which they wanted to remedy these scenarios, (2) their own

personal discomfort if the situations were not remedied, and (3) their responsibility for harm if the scenarios resulted in harm to others. Participants with OCD reported more distress, discomfort, and responsibility in low-risk situations than members of the other two groups, while in the high-risk situations there were no group differences. These results further support the role of responsibility as a motivator for compulsive behaviors specifically aimed at minimizing harm.

Foa and Kozak (1985) suggested that anxiety disorders are the result of impairments in emotional networks and that fear networks that reside in memory are related to erroneous estimations of threat, excessive physiological activities, and resistance to modification. They further contend that OCD is different from other anxiety disorders in that clients with OCD have impairments in interpretive rules for making inferences about harm; they come to a conclusion that a situation is dangerous based on the absence of evidence of safety and fail to induce that a situation is safe based on the absence of danger. It is believed that OCD-related fears are resistant to treatment in part because the fear networks are difficult to access and continue because of impairments in extinction and failure to habituate to physiological reactions, further discussed below. Sher, Frost, and Otto (1983) reported that individuals with OCD have impairments in organization and integration of personal experiences and may be related to compulsive checking behavior, which is consistent with the cognitive explanation regarding impairments in emotional processing.

Learning theories. Theories that invoke basic learning processes may provide the best avenue for understanding and treating OCD, especially because they tend to be more objective and point to variables that can be directly manipulated in experimental evaluations. This is already the case with treatment of OCD and other anxiety disorders, with the most

successful treatments being based on extinction, habituation, or counter-conditioning models traceable to Watson's original formulations (Thyer, Baum, & Reid, 1988; Todd & Pietrowski, 2007; Watson & Morgan, 1916). Furthermore, to the extent that they treat public and private verbal behavior and anxiety responses as private events, they can account for the development and maintenance of intervening and self-instructional responses, such as those invoked by the various cognitive theories. However, because most research and clinical work is concerned with the reduction of OCD behaviors, it is important to note that there are relatively fewer applied examples demonstrating the acquisition of obsessive and compulsive behaviors (Sturmey, Ward-Horner, Marroquin, & Doran, 2007). That is, anxiety disorders are engendered in the laboratory primarily to study methods of elimination, with signaled avoidance situations being primary among the techniques used to create them (Todd & Pietrowski, 2007). Even so, the experimental literature offers some examples of research into etiology, which will be presented here in terms of the main learning processes referenced.

Operant learning theory has developed from the work of Thorndike (1898) and Skinner (1938; 1953) and is concerned substantially with the consequences of an organism's behavior on its future behavior. When considering compulsive behaviors, operant learning theories suggest that obsessive and compulsive responses are the result of reinforcement contingencies maintaining those responses. Thus, these reinforcement contingencies might be capable of establishing persistent high rate public and private behavior through shaping and chaining, essentially by differentially reinforcing successive approximations toward a final behavior or set of behaviors (Skinner, 1938, 1951). It is certainly possible that the reinforcements that initially established the OCD were accidental but powerful, resulting in a highly persistent behavior that would be hard to explain if the initial reinforcement had not

been observed. Skinner (1938) demonstrated that a rat, when given a single reinforcer for lever pressing, would emit more than a hundred responses when placed on extinction with a subsequent decrease or complete elimination of the response. If the opportunities to engage in the behavior were rare, once a month in a human, for instance, the response engendered by a single reinforcement might persist for many years. OCD, however, is characterized by long-term persistence and frequency (Stampfl, 1987). Thus, once the behavior is acquired, OCD-related responses might be maintained on lean schedules of variable, intermittent, or periodic reinforcement (Ferster & Skinner, 1957; Perone, 1991) or maintained by combinations of behavioral processes (Stampfl, 1987). These factors could make compulsions both highly persistent and difficult to analyze. A subtle reinforcer that occurs only once every few days, weeks, or even months would probably not be seen often enough to be noticed. Thus, even though the compulsive response appears to be resistant to extinction or not associated with any particular consequence, it is actually being reinforced. The seeming paradox is that as the variable ratio reinforcement becomes less frequent, the responses maintained by it can become even more persistent (Ferster & Skinner, 1957). Such behavior would, however, be reducible by differential reinforcement for other behavior, especially if an alternative behavior produced the same reinforcer.

Response-independent reinforcers are another means through which compulsive behaviors may be acquired. For example, Skinner (1948) demonstrated what he referred to as "superstitious" behavior in pigeons on response-independent schedules. The consequence of a series of regularly timed reinforcement presentations was the development of unusual repetitive behavior in the pigeon, such as turning counter-clockwise between food deliveries and engaging in unusual head movements. Skinner (1948) attributed the acquisition and

maintenance of these apparently compulsive behaviors to accidental reinforcement of spontaneously occurring behavior. Skinner reported that prior to the initial delivery of food, the pigeon had emitted some type of response when food happened to be delivered. If the interval of food deliveries is brief enough that extinction of the response did not occur and the response occurred frequently enough, the response is strengthened because subsequent reinforcement is more likely to occur with limited opportunities for the organism to engage in alternative responses. Such behavior may be relatively unstable. The reinforcement relation would be unreliable by definition. While the existence of superstitious behavior is well established in the literature (Bloom, Venard, Harden, & Seetharaman, 2007; Mellon, 2009; Rudski, 2001), especially in situations in which multiple and concurrent schedules result in many opportunities for accidental reinforcer relationships, the relatively open model Skinner suggested in 1948 with his pigeons may not be a good model for explaining compulsive behaviors. According to Staddon and Simelhag (1971), Skinner did not adequately distinguish between behavior that might have been established through adventitious reinforcement and that which might be engendered by other processes, such as schedule-induction (Staddon & Simmelhag, 1971). However, in 1948, schedule-induction did not exist as a concept, and more recent analyses have cast doubt on the entire construct of "schedule-induction" as a distinct process and questions whether the range of induced behavior is as broad as the concept suggests (Todd et al., 1997; Todd & Pietrowski, 2005; Wetherington, 1981).

In contrast to operant conditioning, Pavlovian conditioning theories suggest that OCD may be the result of repeated pairing of stimuli, resulting in a response that becomes reflexive despite being essentially nonfunctional. It is particularly possible that certain

stimuli pairing might establish conditioned anxiety to harmless stimuli, which then results in avoidance or escape from those stimuli by responses that are themselves nonfunctional, except to the extent that they serve to separate the organism psychologically or physically from the anxiety-producing stimuli. In other words, OCD might be an example of the two-process theory of avoidance (Mowrer, 1947). This theory suggests that a neutral stimulus becomes associated with fear when paired with a stimulus that is likely to cause harm. The stimulus, either real or imagined, leads to feelings of discomfort, and escape or avoidance responses are then used to minimize this discomfort, which may be related to operant conditioning. These responses then become conditioned and are maintained through their ability to reduce the discomfort that is experienced. This theory is supported by the fact that obsessions tend to increase anxiety (Hodgson & Rachman, 1972; Rabavilas & Boulougouris, 1974), while compulsions reduce it (Roper & Rachman, 1976).

Habituation and sensitization are considered the most fundamental conditioning processes (O'Donohue & Ferguson, 2004). Habituation is the process of a decrease in observed behavior following repeated elicitations, while sensitization occurs when an observed response increases after being elicited multiple times (Groves & Thompson, 1973). Since habituation results in a decrease in response magnitude, this process is probably not relevant in helping to demonstrate the etiology of compulsive behavior—although habituation might explain some aspects of the organism's apparently reduced sensitivity to the negative consequences of the behavior.

Sensitization, however, offers distinct possibilities of a mechanism engendering OCD. Groves and Thompson (1973) have argued that both habituation and sensitization are the result of underlying neural processes and compete for control of behavior. For the present

purposes, however, the possible underlying processes are less important than the manifestation. A large body of research has shown that highly stereotyped excessive behavior can be easily induced in rats and pigeons by the repeated presentation of relevant stimuli, so called "adjunctive" behavior (Falk, 1971), which is now seen by some as sensitized by repeated eliciting events (Todd et al., 1997; Todd & Pietrowski, 2005; Wetherington, 1981). This excessive stereotyped behavior, particularly drinking in rats and attack in pigeons, would be weakened when elicited by individual stimulus presentations but would be strengthened by repeated and frequent stimulus presentations to the point at which they resemble compulsions. And it appears that they are compulsions; they are reduced by SSRIs, highly resistant to environmental modification, and highly stereotyped in topography and temporal features (Pietrowski, 2005; Todd et al., 1997). Thus, adjunctive behavior has been suggested as an animal model of OCD, with limitations in explaining the highly restricted range of compulsive behaviors exhibited.

Animal Models of OCD

Animal models have been used as a means of obtaining relevant information regarding the etiology of OCD, likely due to the ethical issues associated with conducting this research in the human population and the relative advantages of working with nonhumans. However, in the animal literature much attention has focused on the neurological contributions to OCD (Korff & Harvey, 2006; Stein, 2000; Szechtman, Sulis, & Eilam, 1998), with relatively few studies focusing on behavioral aspects (Odberg & Meers, 1987). For example, a series of studies have examined the role of the amygdale in the acquisition and retention of fear-potentiated startle responses in nonhuman primates (Antoniadis, Winslow, Davis, & Amaral, 2007, 2009). These studies found that nonhuman primates were

able to acquire a startle response to a visual cue through conditioning. They further found that primates with lesions to the amygdale were not able to acquire this startle response. Antoniadis and colleagues (2009), however, found that if primates learned the startle response through conditioning with an intact amygdale and then lesions were made to the amygdale, they maintained the memory of the conditioned response. This research clearly demonstrates the close interaction between environmental and neurological variables responsible for repetitive fear responses and learning; however, this specific startle response to a visual cue is an adaptive response to a perceived threat. When considering compulsive behaviors, key features are that the compulsion is a maladaptive response and occurs excessively. Studies conducted by Antoniadis and colleagues seem to be more related to anxious or phobic behaviors than the compulsions observed in OCD. It should be noted that cognitive theorists suggest that anxiety is the underlying mechanism through which compulsive behaviors occur; however, this issue is currently debated by behavioral theorists, as mentioned earlier (Beck, 1976; Carr, 1974; Foa & Kozak, 1985; Foa et al., 2001).

On the behavioral side, research on dogs and other animals under confinement may lead to the excessive expression of observable behaviors such as those found in OCD (Gluck & Pearce, 1977; McKinney, 1974). In dogs, these are often characterized as "separation anxiety" or "frustration" but consist of repetitive nonfunctional responses including barking, pacing, and chewing. In primates, Gluck and Pearce (1977) found that some primates engaged in increased perseveration on tasks without being rewarded when confined to a specific place for extended periods of time, thus having difficulty undergoing extinction. McKinney (1974) found that socially deprived rhesus monkeys engaged in repetitive behaviors, such as self-injury, self-mouthing, aggression, and inappropriate sexual behavior.

These studies suggest that these environmental factors may induce compulsive behaviors; however, this literature may not be relevant to the issue of OCD in humans, as long-term confinement is probably not a factor in the lives of most OCD sufferers. It may, however, play a role in the etiology of maladaptive repetitive behavior and other psychopathology in human prisoners and hostages (Lohner & Konrad, 2006; Meltzer, Jenkins, Singleton, Charlton, & Yar, 1999).

In contrast, schedule-induced polydipsia (SIP) in rats may be a credible animal model of compulsive behaviors observed in individuals with OCD (Moreno & Flores, 2012; Pietrowski, 2005; Toscano, Kameyama, Garcia-Mijares, Silva, & Santerem, 2008; Woods et al., 1993). Polydipsia is the excessive drinking that often occurs when an organism is exposed to food delivered on a response-independent schedule of reinforcement. This excessive drinking has been developed in food-deprived rats and was first reported by John Falk (1961). Although post-food drinking is typical in food-deprived rats, the excessive water consumption is not typical. Rats who have acquired polydipsia on a reinforcement schedule can consume as much water in 30 minutes as they would ordinarily drink in an entire day. SIP has been reported in both human and animal populations (Allen & Butler, 1990; Keehn, & Stoyanov, 1986; Lotter, Woods, & Vasselli, 1973; Wallace, Singer, Wayner, & Cook, 1975). In addition, this behavior is topographically and temporally stereotyped and is differentially sensitive to SSRIs. That is, giving SSRIs to polydipsic rats reduces the schedule-induced drinking but not ongoing operant responding (Moreno & Flores). Unpublished research by Pietrowski (2005) and other research by Pietrowski and Todd (2004) demonstrated that induced drinking in rats is highly stereotyped in terms of

topography and timing, maintaining the behavior even as the rats are made to work more to engage in it. Cumulative record data in Todd et al. (1997) illustrate the stereotypy of SIP.

According to an analysis conducted by Moreno and Flores (2012), polydipsia serves as a valid model of compulsive behavior based on four criteria that have been established to evaluate the use of animals in the laboratory as models of human behaviors (Geyer & Markou, 1995, 2002; Markou et al., 2009; Willner, 1984). These criteria focus on polydipsia in animals having face validity, construct validity, predictive validity, and reliability. Schedule-induced polydipsia (SIP) meets the criteria for face validity because it is excessive, persistent, and maladaptive and may be used to reduce the stress that goes along with not being able to have unlimited access to food as desired. In addition, it is not the result of a physiological need and not related to the direct effect of drugs. SIP also has construct validity in that the neurobiological processes underlying this behavior in rats involve the same processes in the human population. Pharmacotherapy used to treat compulsive disorders in humans has reduced SIP in rats without altering water or food regulatory intake. Last, Moreno and Flores (2012) reported that SIP meets reliability criteria, which is one of the most important criteria by which animal models are evaluated. It was reported that SIP can be demonstrated consistently within subjects and has been reproduced across laboratories.

The theories mentioned above can reasonably explain the etiology of repetitive behaviors. However, a notable feature of OCD is the stereotyped topography of the compulsion itself that cannot be adequately explained by these theories. For example, psychodynamic approaches are untestable, and processes are, by definition, unobservable. Neurobiological theories may predispose individuals to engage in compulsive behaviors, but it is unlikely that the stereotyped topography of these behaviors is directly related to the

neurobiological correlates associated with them. That is, the neurology cannot predict the specific form of the behavior, which had to come from some kind of learning process. Cognitive theories may better explain the acquisition of the specific topography of the compulsion given that the individual engages in the compulsive behavior to reduce the anxiety that arises from the obsession. However, the etiology of the specific obsession reaction remains unknown. Therefore, cognitive theories may account for the obsession at a motivational level but not inform us of how the specific reaction came to be.

Unlike the aforementioned theories, operant and respondent learning theories will potentially more fully explain both the etiology and topography of compulsive behavior. Acquisition and maintenance are the strengths of this perspective. And, because the relevant behaviors and presumed generating conditions can be simulated in the laboratory, behavioral theories are both more testable and more easily evaluated for validity. However, the etiology of compulsive behaviors in humans and other organisms may involve factors other than pairing and operant shaping, especially since these behaviors may develop independently of known reinforcers in a schedule. Moreover, reinforcement theory predicts that behaviors so maladaptive that they interfere with ordinary functions should be self-limiting, and other known behavioral processes should take over. For example, the individual should habituate to the anxiety associated with the behavior, or the compulsive behavior will eventually undergo extinction because of the harm that it produces and it reduces access to so many other reinforcers.

Social Facilitation and Adaptive Responding

Given the limitations in the theories mentioned above, alternatives should be considered to explain the etiology of compulsions in an attempt to demonstrate another potential mechanism through which compulsive behavior is engendered and modulated. For example, social or observational learning theories may be a simpler and more viable explanation for the development of these compulsive behaviors, because in some cases individuals tend to engage in the same behaviors as their parents or friends without receiving reinforcement for these acts. Bandura and Walters (1963) reported that these variables are often not addressed when determining the etiology of some behaviors.

Some recent experimental studies have demonstrated the acquisition of behavior in animals through social learning by observing other animals engage in adaptive behaviors in response to fear (Kavaliers, Choleris, & Colwell, 2001; Kavaliers, Colwell & Choleris, 2003) or novel food (Rymer, Schradin, & Pillay, 2008). For example, Kavaliers, Choleris, and Colwell (2001) conducted a study in which they examined the acquisition of fear-related behaviors in mice individually and in a social context. Some mice were exposed to biting flies that are naturally aversive, while other mice were observing this exposure but were not being exposed to the flies themselves. The researchers found that self-burying is elicited when mice are exposed to these biting flies. The researchers further examined the behavioral effects of the observer mice and found that when exposed to biting flies altered to be nonthreatening, the observer mice engaged in the self-burying behavior, even though the flies that they were directly exposed to were not a threat. This study demonstrated that mice learn to engage in behaviors that they have observed, especially if there is a possibility that they are in danger. Although this study demonstrates the acquisition of behavior through social

facilitation, the behavior that the mice are engaging in is adaptive. It is not clear, however, if this is the case with maladaptive compulsive behavior.

In addition to the use of mice, several researchers have examined the effects of observational learning in monkeys in response to feared stimuli (e.g., Mineka, 1987; Mineka, Davidson, Cook, & Keir, 1984; Mineka & Cook, 1986, 1989; Plimpton, Swartz, & Rosenblum, 1981). Mineka and Cook (1986) examined the effects of snake fear in rhesus monkeys by assigning monkeys to either observe other monkeys respond nonfearfully to snakes, be alone not responding fearfully to snakes, or watching another monkey responding nonfearfully with a neutral object. Then the researchers allowed the monkeys in each group to observe another monkey respond fearfully to a snake, and the results were that the monkeys who were exposed to a nonfearful model did not acquire fear when later observing another monkey being fearful of snakes. Plimpton, Swartz, and Rosenblum (1981) found that juvenile macaques differentially responded in a socially appropriate manner to color videotapes of unknown adult male and female conspecifics that were threatening or passive. These studies suggest that socially facilitated learning can occur across species and is not limited to in-vivo situations. However, these studies are limited in that they are more relevant to the etiology of other anxiety disorders related to fear, such as phobias, and are not specific to compulsive behaviors.

Even though some research is being conducted to explore these social learning theories, social variables are rarely discussed as they relate to compulsive behaviors, which is surprising given that much of adaptive human behavior occurs through imitation of others (Bandura & Walters, 1963). If not through direct imitation, acquisition of adaptive behavior certainly exists in social situations. In fact, it is believed that individuals with deficits in

imitation have psychological disorders and that imitation is a necessary part of social skill development (Bandura & Walters, 1963). Within the developmental literature, some researchers have conducted studies on imitation, most of which focus on enhancing skills in those with autism. For example, some studies report that individuals with autism have difficulty imitating others (Rogers & Williams, 2006; Williams, Whiten, & Singh, 2004) when compared with typically developing peers. However, with instruction, individuals with autism are able to imitate some behaviors, such as play skills (Ingersoll, 2010) and construction tasks (Tereshko, MacDonald, & Ahearn, 2010). Ingersoll (2010) conducted a randomized control trial to evaluate a naturalistic imitation training procedure to develop play skills in children with autism. They evaluated the children's elicited and spontaneous imitation and found that children who received the training intervention gained more and varied play skills than those who did not. It is important to note, however, that higher functioning children who engaged in more spontaneous play skills made more gains than those who did not begin with as varied of a repertoire.

Social Facilitation and Maladaptive Responding in OCD

Although these studies examined the social variables on adaptive behavior, it is possible that compulsive maladaptive behavior may also occur through social processes rather than through most of those discussed above. The role of social variables is an important one because many compulsive behaviors in those with OCD tend to occur at higher rates within families (Hollander et al., 2003; Nestadt et al., 2000). It is possible that genetic and neurobiological factors play a role in an individual's susceptibility to engage in these behaviors. However, it is also likely that some of these behaviors are socially facilitated, in that individuals learn to engage in these behaviors from observing family members or peers

engaging in them. At least, it is suspected that the actual type of compulsive behavior is not tightly controlled by physiology.

Even so, there is little research focusing on social variables to determine the etiology of compulsive behaviors observed in those with OCD. It would be ethically difficult to generate a true compulsion in humans. However, the few studies that have examined social variables, such as peer modeling as it relates to the drinking of alcoholic beverages by college students, found that participants would match their alcohol intake to peer models (or confederates) who consumed either high or low amounts of alcohol (Dericco & Niemann, 1980; Garlington & Dericco, 1977). This research suggests that the peer models' behavior directly influenced the behavior of the individual with whom they were paired, even if the behavior was not strictly imitative.

Given the limited research in explaining the mechanisms related to maladaptive compulsive behavior, this study will evaluate the potential of social facilitation to engender the development of compulsive-like behaviors in an animal model of OCD. The target behavior will be SIP in rats. As noted earlier, SIP appears to meet the criteria of an animal model of compulsive behavior.

Since there is sufficient research on response acquisition through reinforcement schedules and small but experimentally-sound research demonstrating the acquisition of polydipsia (Flores & Pellon, 1997; Lopez-Crespo, Rodriguez, Pellon, & Flores, 2004; Todd, Cunningham, Janes, Mendelson, & Morris, 1997), this study will create this highly stereotyped compulsive behavior using a response-independent schedule of reinforcement in normal rats. Polydipsia can be engendered in rats without weight reduction (Todd et al., 1997), thereby further adding to the clinical relevance of polydipsia as animal model of

compulsive behavior. Compulsive behaviors observed in the human population do not always appear to be the result of environmental deprivation. In addition, polydipsic responding can be measured with a high degree of precision, giving researchers the ability to detect very subtle changes in the behavior, which might not be observable in human OCD even under clinical conditions. Once rats have developed polydipsia, the experimenter will attempt to determine whether an experimentally naïve rat will learn to engage in the drinking behavior simply by observing the polydipsic rat.

Two main questions will be asked by this study. First, will rats exposed to a rat with elevated drinking be more likely to drink than rats exposed to a rat that did not drink? That is, one purpose of this study was to test whether preliminary observations of socially facilitated drinking in rats could be replicated. The second question is whether rats exposed to a drinking rat would be more likely to acquire SIP under standard generating conditions (e.g., fixed-time food delivery) than rats that had not been exposed to rats with elevated drinking. That is, the study would test whether a type of observational learning, social facilitation, would enhance the later acquisition of a compulsive response. If the answer to both questions—"Is social facilitation is a reasonably reliable phenomenon" and "Can a compulsion be primed by social facilitation?"— is "Yes," we will have expanded SIP as a potentially useful model of OCD and provided evidence that social facilitation is a factor in the etiology of specific compulsive topographies.

Chapter 2: Method

Subjects

Twenty-four experimentally-naïve male Sprague-Dawley rats that were 10 weeks old served as subjects for this study. The rats were obtained at approximately 4 weeks of age from Harlan/Sprague-Dawley, a commercial laboratory animal supplier. At the start of the study, each rat weighed between 170g and 255g. Each rat was housed separately in a home cage to permit individual feeding and to limit social interaction outside of the experimental setting. Prior to the beginning of the study, all rats were given free access to food and water.

Shortly before the study started, the rats were arbitrarily assigned to the various experimental groups. The "naïve" rats—the ones which would be given the opportunity to learn to drink through social facilitation—continued to have free access to food and water in their home cages. The model rats, which would be made polydipsic, underwent a weight reduction procedure to motivate eating in the experimental chambers. Weight reduction was accomplished by feeding five grams of rat chow per day until the target weight was reached. This was done rather than complete food elimination to avoid overstressing the rats. The target weights were maintained by adjusting daily post-session rations. These post-session rations were typically between 12 and 15 g. The model rats were reduced to 85% of their free-feeding weight. The naïve rats did not undergo the weight reduction procedure at any point throughout the study. Food and water were changed daily, or as needed, in each home cage. Bedding was changed weekly, or more often, as needed.

Apparatus

The experimental setup used in this study was the same as the setup used in Todd et al. (1997). All sessions were conducted in standard operant conditioning chambers for rats

with response levers removed (inside dimensions: 19 cm high, 20 cm wide, and 23 cm long). The sides and top of the chambers were made of clear plastic (0.5 mm thick), and the front and back walls were stainless steel (0.2 mm thick). The food cup was located just outside the front wall of the chamber in a container 5 cm in length and width, and 1 cm in depth. The food cup was 1.5 cm above the floor of the chamber and to the left of the drinking tube. Forty-five milligram food pellets were delivered into the food cup by mechanical food dispensers. These food dispensers made a clicking sound when each pellet was delivered, thereby providing rats with an immediate signal that food would be delivered. Steel bars extended across the floor of the chamber and were electrically connected together to form a ground-potential electrode for electrical contact drinkometers. These were used to measure number of drinking tube licks and duration of drinking. The drinking tube protruded into the chamber through a hole in the front wall and was located 8 cm above the cage floor and 4.5 cm to the right of the opening for the food cup. An 8-mm diameter drinking tube extended through this hole, 3 to 3.5 cm into the chamber at an angle of approximately 30 degrees from horizontal. A plastic cover was placed at the end of the drinking tube to help prevent the rat from shorting the drinkometers with their paws and producing erroneous readings. (Some rats rest a paw on or hold the tube itself while drinking.) These drinking tube guards are cup-shaped end caps for standard 1 inch (2.54 cm) PVC water pipe and are 2 cm long and 3 cm wide. The drinking tube was connected to a 100 ml graduated cylinder attached to the back of the front wall. It should be noted that only ball-end drinking tubes were used under the presumption that the clicking noise they make when a rat drinks would make drinking by a model rat more salient—essentially harder for the naïve rat to miss.

Data recording and experimental programming were performed by computers and other electronic interface equipment located in the same room as the experimental chambers. The electronic equipment operated silently, so no masking noise was used.

Response Measurement

The duration, frequency, probability, and post-food latency of drinking tube contacts are the main dependent measures in this study. In most studies of schedule-induced drinking, only overall quantity consumed during the entire session is reported (see Todd et al., 1997, for a discussion). This finer-grained analysis provides superior information about the development and relevant dimensions of any potential socially facilitated drinking. However, because two rats would sometimes be sipping from the same drinking tube, certain electric contact measures would be nonspecific to which rat is drinking, representing a combined measure of the responding of both rats. Therefore, during any session in which the two rats were in the cage together, each rat's drinking was measured by the experimenter scoring video records of drinking tube contacts. During sessions in which only one rat was in the chamber, electrical measures were used. In all instances of electronic and visual data collection, drinking was defined in terms of continuous contact of the rat with the drinking tube for at least one second. If a rat stopped drinking for at least two seconds, a new instance of drinking was counted.

Procedures

At the beginning of the study, the experimenter randomly assigned each rat to one of the following groups of three rats each (see Table 1): 1a) Drinking Model Rats – rats that will be trained to drink excessively on a FT-60 sec feeding schedule during the SIP Acquisition Training phase and serve as models for its matched drinking naïve rat during the Social

Facilitation phase; 1b) matched Drinking Naïve Rats – rats that will be paired with Drinking Model Rats during the Social Facilitation phase to determine if they will engage in excessive drinking during the SIP Acquisition Testing phase; 2a) Feeding Control Model Rats – rats that will be trained to eat, but not drink, during the SIP Acquisition phase and serve as models for their matched feeding control naïve rats during the Social Facilitation phase; 2b) matched Feeding Control Naïve Rats – rats that will be paired with the Feeding Control Model Rats during the Social Facilitation phase of the study; 3a) Social Contact Control Model Rats – rats that will be used to control for socialization as a factor for elevated drinking and will serve as models for their matched Social Contact Control Naïve Rats during the Social Facilitation phase; 3b) Social Contact Control Naïve Rats – rats that will be paired with the Social Contact Control Model Rats during the Social Facilitation phase of the study; and 4) Naïve Control Rats – rats used as controls for the Drinking Model Rats and the Drinking Naïve Rats during the final phase of the study SIP Acquisition Testing. Rat assignments to the various groups are summarized in Table 1.

Table 1

Rat Assignments after Randomization

	Drinking Modeling		**Feeding Controls**		**Social Contact Controls**		**Naïve Controls**
Rat Assignments	*Model*	*Naïve*	*Model*	*Naïve*	*Model*	*Naïve*	
	1	2	7	8	13	14	
	3	4	9	10	15	16	
	5	6	11	12	17	18	
							19, 20, 21, 22, 23, 24

Each experimental session was approximately 30 minutes long. Prior to each session, the rats were removed from their home cage, weighed, transported individually to the experimental room, and placed into an experimental chamber. Each rat spent the same amount of time in the experimental chamber during each session in all of the conditions to control for environmental exploration and acclimation effects. Each rat was returned to its home cage following each session and, if appropriate, given a measured ration designed to maintain a target weight.

This study included the following four phases: 1) Massed-Food Baseline, 2) Schedule-induced Polydipsia Training, 3) Social Facilitation, and 4) Schedule-induced Polydipsia Testing. Each phase is discussed in detail below.

Phase 1: Massed-Food Baseline. The Massed-Food Baseline consisted of five sessions in which each rat was placed in the chamber for 30 minutes, with 30, 45 mg food pellets (Bioserve # F0165 grain-based food) available in the feeding dish and water freely available from the drinking tube. The purpose of the sessions was to determine the amount of drinking associated with non-scheduled consumption of the same amount of food that would be delivered during the experimental sessions. It also assessed the general amount of drinking the rats would engage in during the sessions. As noted by Todd et al. (1997), these types of baselines have never been associated with excessive drinking and are predicated on a fallacious assumption that the only defining feature of SIP is excessive drinking, rather than it being a sensitization phenomenon consisting of a series of discrete drinking bouts which together result in more water consumption than if nothing had been done. However, the literature on SIP expects such measures, so it was included.

Phase 2: Schedule-induced Polydipsia Acquisition Training. During this phase of the study, the experimenter attempted to establish SIP in all of the drinking model rats by using the procedures outlined by Falk (1961) and adapted by Todd et al. (1997), so that they would drink reliably when a food pellet was delivered. Thus, they could later serve as drinking models for their matched drinking naïve rat pair. All other rats assigned to the other groups were placed in the experimental chambers for the same amount of time as the models. This equalized exposure time to the experimental environment across rats and allowed for environmental exploration and familiarity with the experimental chambers. Thus, there should not be an issue of the relative familiarity of the experimental environment interfering with the rats' behavior. The specific procedures for each group of rats were as follows:

***Drinking model rats*.** The experimenter placed rats that were assigned to be in the drinking model group individually in the experimental chamber. Since the purpose of this phase was to establish high-probability drinking, the goal was to get drinking following every pellet delivery; therefore, the experimenter reduced the weight of each of these rats to 85% of their free-feeding weights by limiting their post-session food rations. The experimenter placed the drinking model rats alone in the experimental chamber on a FT-60 sec feeding schedule with water freely available. From the rat's perspective, the session consisted of a brief waiting period after it had been put in the experimental chamber, then 30 minutes of food delivery, with one food pellet arriving each minute regardless of what the rat did.

***Drinking naïve rats*.** The experimenter placed rats that were assigned to be in the matched drinking naïve rat group individually in the experimental chamber. These rats were not reduced in weight. The food dispenser and drinking tube were operational, but food was not delivered. Thus, the rat heard a click when the food would have been delivered.

However, no food appeared. Essentially, the sessions mirrored exactly the treatment of the drinking model rats, but without food delivery. The purpose of this procedure was to control for familiarity with experimental sessions, ensuring that these control rats did not become spontaneously polydipsic (not a likely occurrence, but required by methodology) and making them insensitive to pellet-dispenser clicks to eliminate any emotional responses the noise might create, reducing the possibility that they would later learn the food-click association and compete for food at the pellet dispenser.

***Feeding control model rats*.** The experimenter placed rats that were assigned to be in the feeding control model rat group individually in the experimental chamber. These rats were reduced in weight to 85% of their free-feeding weight like the drinking model rats. The food dispenser and drinking tube were operational, but water was not available. The purpose of this procedure was to control for the possibility that eating, and eating-related behavior in a model, might elicit or otherwise engender drinking in the naïve rats. However, the fundamental purpose of this group was to show that the naïve rats needed to observe drinking in another rat to develop drinking themselves.

***Feeding control naïve rats*.** The experimenter placed rats that were assigned to be in the feeding control naïve rat group individually in the experimental chamber. These rats were not reduced in weight. No food or water was available during these sessions. Thus, the experimental experience for these rats consisted of 30 minutes of waiting in the experimental chamber. The purpose of this procedure was to control for environmental familiarity and prepare these rats to later serve as naïve rats for the feeding control model rats during Social Facilitation (Phase 3).

Social contact control model rats. The experimenter placed rats that were assigned to be in the social contact control model rat group individually in the experimental chamber. These rats were reduced in weight to 85% of their free-feeding weight. The food dispenser and drinking tube were operational, but food was not delivered. Essentially, these rats waited in the experimental chamber for 30 minutes but, unlike the Feeding Control Rats, had water available. The purpose of this group was to provide a control for the possibility that merely being in a chamber with another rat would engender drinking in the naïve rats. These rats would be expected to do a very small amount of drinking and therefore could also serve as controls for the relative rates of drinking in the naïve rats relative to the drinking in the model rats.

Social contact control naïve rats. The experimenter placed rats that were assigned to be in the social contact control naïve rat group individually in the experimental chamber. These rats were not reduced in weight. The food dispenser and drinking tube were operational during the session, but food was not delivered. Like the others, these rats became familiar with the experimental environment but would not become polydipsic. They would, however, be familiar with the location and purpose of the drinking tube. These rats would later serve as test naïve rats for the social contact control model rats during Social Facilitation (Phase 3). That is, they were matched to the non-polydipsic models to ensure that drinking would not be engendered by their eating or other behavior, or develop spontaneously.

Naïve control rats. The experimenter placed rats that were assigned to be in the naïve control rat group individually in the experimental chamber. These rats were not reduced in weight. During their sessions, the food dispenser and drinking tube were operational, but

water was not available. The purpose of this procedure was to prepare these rats to later serve as controls when the naïve rats in the drinking modeling group are completing Phase 4: Schedule-induced Polydipsia Testing. These rats will show, by comparison, whether the rate of drinking acquisition by the naïve rats exposed to drinking models is due to exposure to the models or simply to being acclimated to the experimental chamber. That is, it is expected that the naïve control rats will acquire SIP more slowly than those rats that have been exposed to drinking models.

Phase 3: Social Facilitation. During this phase of the study, the experimenter examined the effects of social facilitation (and other types of exposure to experimental conditions) on the acquisition of polydipsia for each of the rats. The model rat and its matched naïve rat pair were placed in the same chamber with one food dispenser and one drinking tube available. The sessions, like all the others, were 30 minutes long and consisted of the delivery of 30 food pellets, one per minute. The experiences of each group were as follows:

***Drinking modeling*.** A drinking model rat and its matched drinking naïve rat pair were placed in an experimental chamber together. The model rat continued at reduced body weight; the naïve rat continued to have free access to food in its home cage and therefore was not weight-reduced. Food was delivered on a FT-60 sec feeding schedule, and water was available from the drinking tube. The main question was whether the naïve rat would start drinking upon observing the model drinking, how much it would drink, and exactly when the drinking would occur. Preliminary work suggested that the naïve rat would tend to drink at the same time as the model. Eventually, the question would be whether these rats, due to this

exposure, would acquire SIP more rapidly than rats which had not acquired socially facilitated drinking.

Feeding control. A feeding model rat and its matched feeding naïve rat pair were placed in an experimental chamber together. The model rat continued at reduced weight, while the naïve rat was given free access to food and was not thereby weight-reduced. Food was delivered on a FT-60 sec feeding schedule, and water was available in the drinking tube. The purpose of this phase was to serve as a control. If the naïve rats drank a significant amount in this condition, even if the model was not polydipsic and, therefore, unlikely to drink very often, social facilitation could not be an explanation for the drinking by the rats which had a drinking model. However, since this experiment measured the duration, probability, and latency of drinking bouts, differences between the drinking of the rats with and without a drinking model could be assessed. The acquisition of SIP under typical generating conditions in Phase Four could also be compared to the acquisition by the rats with a drinking model. This would show whether either socially facilitated drinking, or having seen drinking, was enhanced the acquisition of drinking. It was not expected that these rats would drink much more than in baseline; rather, these rats would acquire SIP more slowly than the rats with a drinking model. It is also important to show that these naïve rats would become polydipsia drinkers to ensure that, for some reason, they simply would not have ever consumed significant amounts of water.

Social contact control. A social contact control model rat and its matched social contact control naïve rat pair were placed in an experimental chamber together for the standard 30-minute sessions. The model rat continued at its reduced weight, while the naïve rat continued to have free access to food. The food dispenser and drinking tube were

operational during these sessions, but food was not delivered. The purpose of this phase was to serve as a control for the drinking models and drinking naïve rats with regard to environmental exploration and to later be tested in Phase 4 for the acquisition of polydipsia. It was expected that the naïve rats in this group would not drink significantly; rather, they are expected to acquire SIP more slowly than the rats which had exhibited socially facilitated drinking. It is also important to show that these naïve rats would become polydipsic to ensure that, for some reason, they simply would not have ever consumed significant amounts of water.

Naïve controls. These rats were not included in this phase because they were to be used as controls later when testing for rate of acquisition of polydipsia in Phase 4. Procedures were conducted the same way as in Phase 2. The experimenter placed rats individually in the experimental chamber. These rats were not reduced in weight. The food dispenser and drinking tube were operational, but water was not delivered. These rats will show, by comparison, whether the rate of drinking acquisition by the naïve rats exposed to drinking models is due to exposure to the models or simply to being acclimated to the experimental chamber. Essentially, these are totally naïve rats against which other comparisons could be made as to the acquisition of SIP. Even though the Todd et al. (1997) study used identical procedures, and its results were also available for use as control, it is possible that differences could arise from incidental aspects of Todd's group's procedures relative to those used in this study.

Phase 4: Schedule-induced Polydipsia Acquisition Testing. The Schedule-induced Polydipsia Testing phase consisted of 26 sessions in which each rat was placed individually in the experimental chamber. The model rats continued at reduced weight, while the naïve

rats continued to have free access to food in their home cages. Each rat was placed on an FT-60 sec feeding schedule and had water freely available in the drinking tube during all sessions. Drinking was expected to be elevated for the drinking model rats and the drinking naïve rats, which should have exhibited some socially facilitated drinking, relative to all other rats (see Figure 1 for an outline of all study procedures).

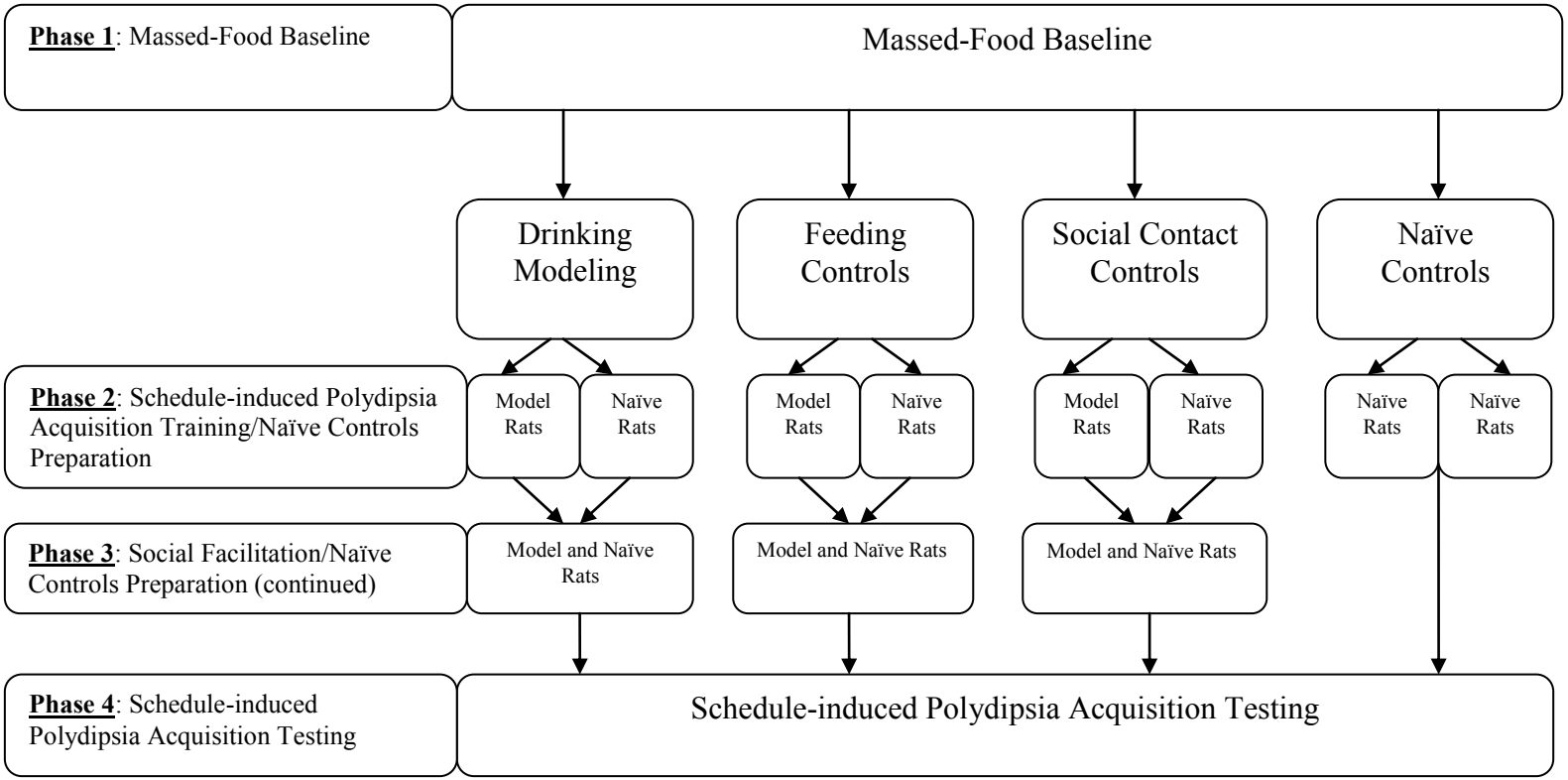

Figure 1. Outline of study procedures across groups.

Table 2

Summary of Rat Exposure to Food and Water in Each Phase

	Model vs. Naïve	**Phase 1: Baseline**		**Phase 2: SIP Acquisition Training**		**Phase 3: Social Facilitation**		**Phase 4: SIP Acquisition Testing**	
		Food	Water	Food	Water	Food	Water	Food	Water
Drinking Modeling (n=6)	Model (n=3)	+	+	+	+	+	+	+	+
	Naïve (n=3)	+	+	+	-	+	+	+	+
Feeding Modeling (n=6)	Model (n=3)	+	+	+	-	+	+	+	+
	Naïve (n=3)	+	+	-	-	+	+	+	+
Social Contact Control (n=6)	Model (n=3)	+	+	-	+	-	+	+	+
	Naïve (n=3)	+	+	-	+	-	+	+	+
Naïve Control (n=6)	Naïve (n=6)	+	+	+	-	X	X	+	+

Chapter 3: Results

The data of the rats in the Drinking Modeling Group represented in Tables 3 – 5 and in Figures 2 – 4 support the hypothesis that naïve rats exposed to a model rat with elevated drinking are more likely to drink than naïve rats exposed to a model rat that does not have elevated drinking. These data replicate the preliminary observations of socially facilitated drinking in rats. There was clearly a high level of variability across all drinking dimensions within and between the rats.

Table 3

Average Rates of Drinking for the Model Rat (1) and Naïve Rat (2) in the Drinking Modeling Group

	Phase 1: Massed-Food Baseline		**Phase 2: SIP Acquisition Training**		**Phase 3: Social Facilitation**		**Phase 4: SIP Acquisition Testing**	
	Model (1)	**Naïve (2)**	**Model (1)**	**Naïve (2)**	**Model (1)**	**Naïve (2)**	**Model (1)**	**Naïve (2)**
Drinking Tube Contacts	33.8	118	37.5	12.7	63.4	285.9	49.4	421.4
Water Intake (mL)	0.34	1.18	0.38	0.13	0.63	2.86	0.49	4.21
Intervals with Drinking	2.8	3.4	1.89	1.06	2.76	5.88	3.19	7.04
Drinking Probability	0.09034	0.10968	0.060939	0.034072	0.089182	0.189735	0.102756	0.227004
Total Duration (s)	9	21.8	7.61	3.28	15.18	59.12	11.70	71.67
Average Duration (s)	3.2	4.2	3.11	2.33	5.18	10.06	4.11	10.04
Total Latency (s)	95.2	107	47.89	23.67	67.88	128.88	48.44	90.70
Average Latency (s)	33.4	26.4	25.33	18.06	20.76	26.76	13.37	12.52

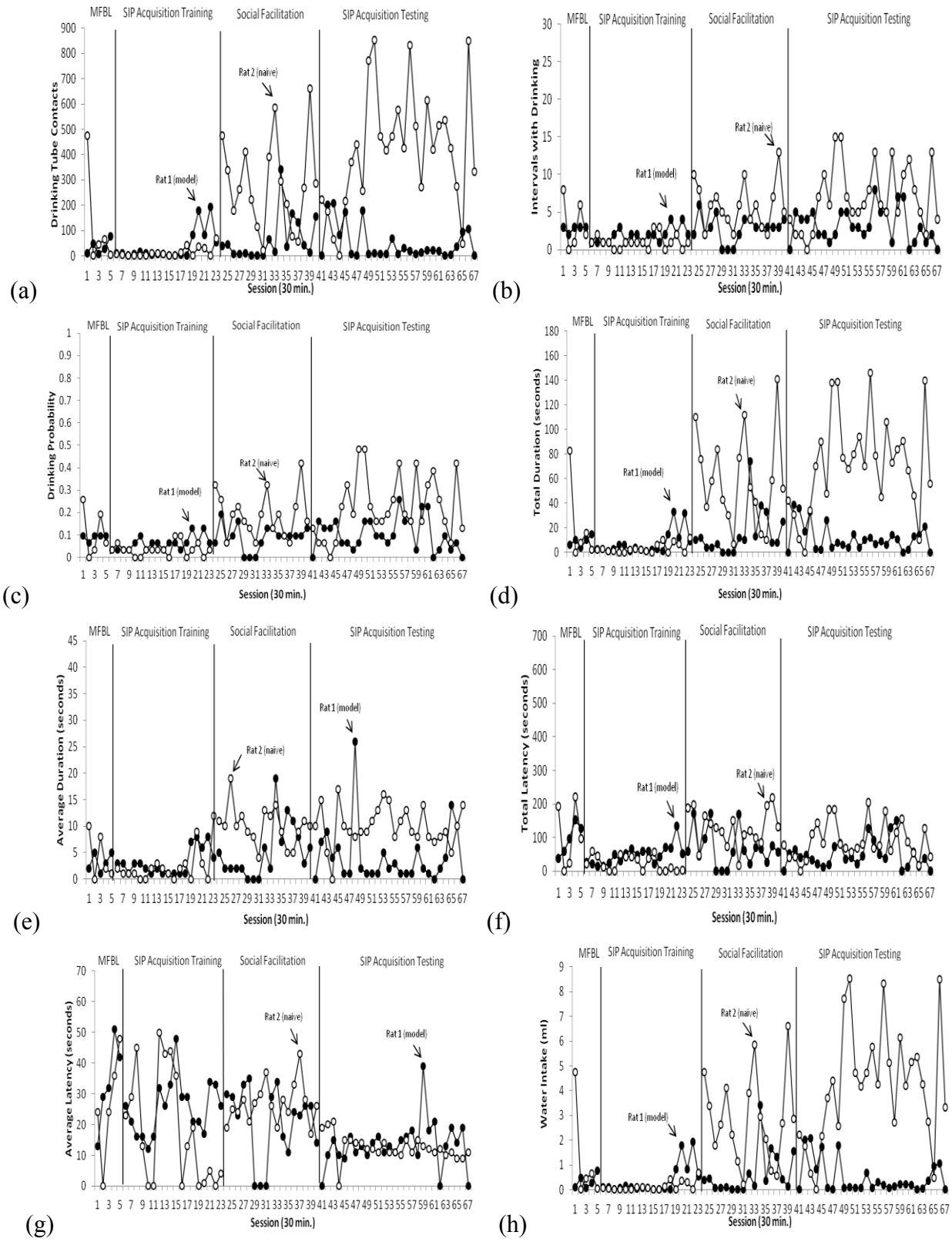

Figure 2 (a-h). Rates of drinking across sessions for the model rat (1) and naïve rat (2) in the Drinking Modeling Group.

Table 4

Average Rates of Drinking for the Model Rat (3) and Naïve Rat (4) in the Drinking Modeling Group.

	Phase 1: Massed-Food Baseline		**Phase 2: SIP Acquisition Training**		**Phase 3: Social Facilitation**		**Phase 4: SIP Acquisition Testing**	
	Model (3)	**Naïve (4)**	**Model (3)**	**Naïve (4)**	**Model (3)**	**Naïve (4)**	**Model (3)**	**Naïve (4)**
Drinking Tube Contacts	290.4	115.2	295	10.33	310.3	99.1	383.3	121.9
Water Intake (mL)	2.90	1.15	2.95	0.10	3.10	0.99	3.83	1.22
Intervals with Drinking	10.4	3.8	9.9	1.9	13.3	2.8	14.4	5.3
Drinking Probability	0.33548	0.12258	0.320783	0.060928	0.401213	0.090731	0.465941	0.169659
Total Duration (s)	58.6	20.6	58.9	4.4	72.2	21.9	80.8	24.3
Average Duration (s)	4.4	3.4	6.2	1.5	5.7	5.9	5.1	4.3
Total Latency (s)	258.8	112	228.6	46.2	268.4	59.7	191.2	142.4
Average Latency (s)	18.8	23.2	21.9	17.7	19	24.6	12.9	24.4

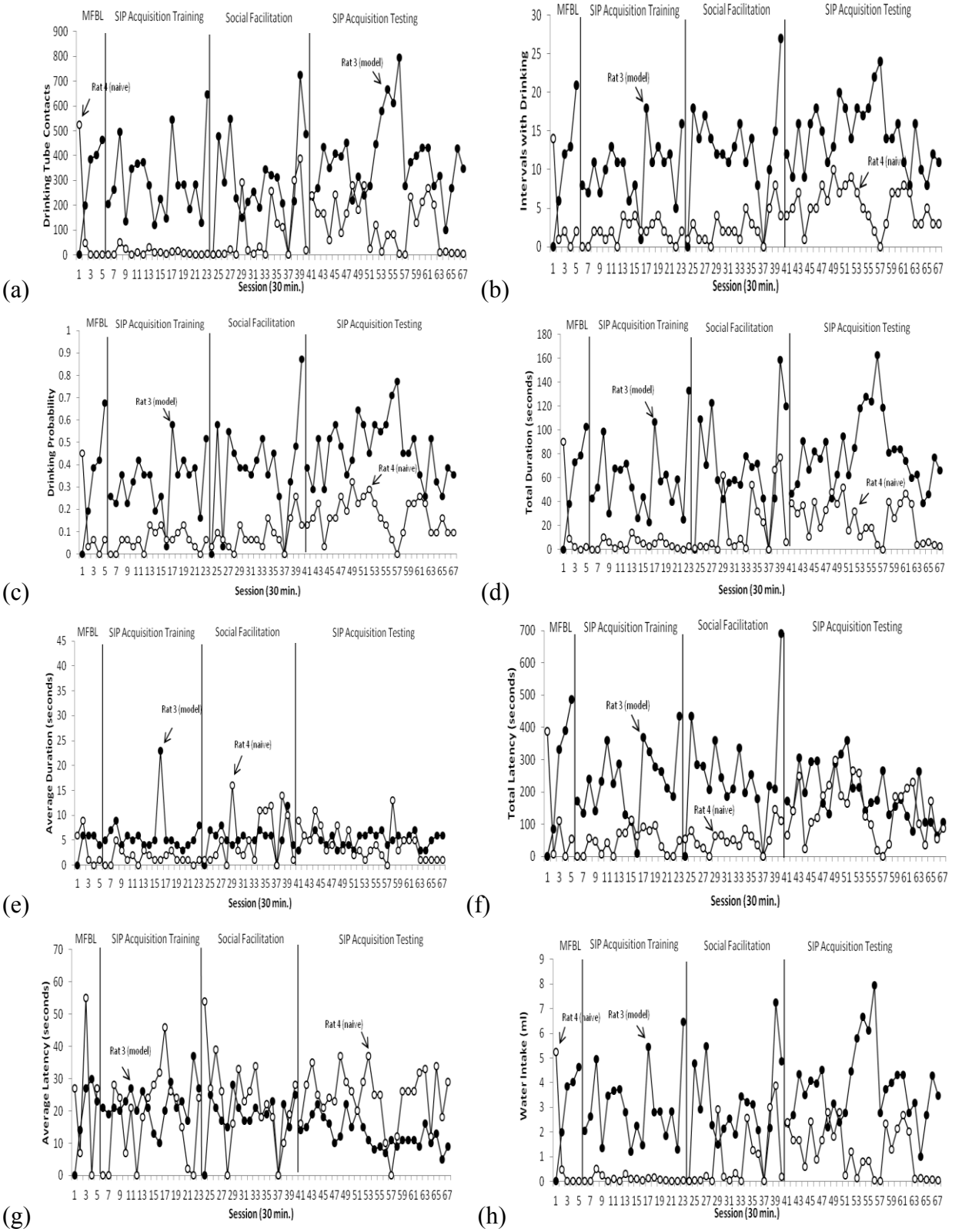

Figure 3 (a-h). Rates of drinking across sessions for the model rat (3) and naïve rat (4) in the Drinking Modeling Group.

Table 5

Average Rates of Drinking for the Model Rat (5) and Naïve Rat (6) in the Drinking Modeling Group

	Phase 1: Massed-Food Baseline		**Phase 2: SIP Acquisition Training**		**Phase 3: Social Facilitation**		**Phase 4: SIP Acquisition Testing**	
	Model (5)	**Naïve (6)**	**Model (5)**	**Naïve (6)**	**Model (5)**	**Naïve (6)**	**Model (5)**	**Naïve (6)**
Drinking Tube Contacts	157.2	15	313.3	23.3	59.2	16.1	349.7	402.7
Water Intake (mL)	1.57	0.15	3.13	0.23	0.59	0.16	3.49	4.03
Intervals with Drinking	2.6	2.0	6.1	5.1	1.6	3.3	8.0	11.5
Drinking Probability	0.08388	0.06452	0.197122	0.163072	0.051247	0.108159	0.258063	0.371578
Total Duration (s)	31.2	6.4	57.4	13.2	13.1	8.2	65.7	84.1
Average Duration (s)	12.4	3.0	8.9	1.6	4.2	2.4	8.0	7.1
Total Latency (s)	65.6	47.6	115.4	130.4	34.6	91.5	127.1	233.9
Average Latency (s)	23.4	25.0	20.8	21.3	15.8	21.9	16.1	18.7

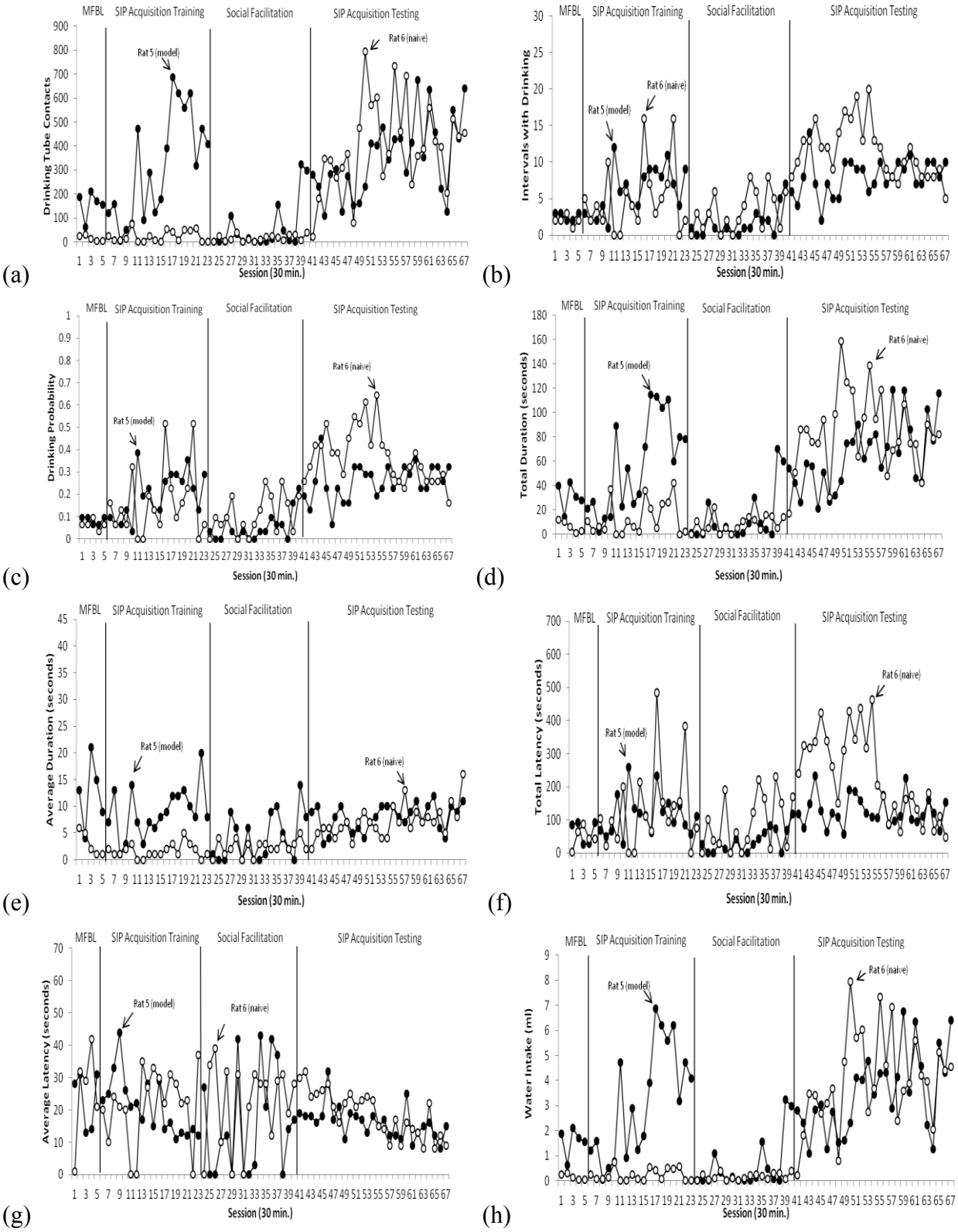

Figure 4 (a-h). Rates of drinking across sessions for the model rat (5) and naïve rat (6) in the Drinking Modeling Group.

Additionally, Tables 6 – 9 and Figures 5 – 8 demonstrate that the rats which did not have a drinking model did not acquire elevated drinking. This further supports the observation that socially facilitated drinking was exhibited in at least some of the naïve rats exposed to drinking rats. Furthermore, the rats that did not exhibit socially facilitated drinking also showed less rapid—if any—acquisition of schedule-induced polydipsia in the final phase of the experiment.

Table 6

Average Rates of Drinking for the Model Rat (9) and Naïve Rat (10) in the Feeding Control Group

	Phase 1: Massed-Food Baseline		**Phase 2: SIP Acquisition Training**		**Phase 3: Social Facilitation**		**Phase 4: SIP Acquisition Testing**	
	Model (9)	**Naïve (10)**	**Model (9)**	**Naïve (10)**	**Model (9)**	**Naïve (10)**	**Model (9)**	**Naïve (10)**
Drinking Tube Contacts	245.8	66.8	11.8	3.8	84.9	201.7	124.4	29.7
Water Intake (mL)	2.46	0.67	0.12	0.04	0.85	2.02	1.24	0.30
Intervals with Drinking	5.2	1.6	1.5	1.2	2.2	4.5	3.0	2.4
Drinking Probability	0.16774	0.05164	0.048394	0.039444	0.070563	0.145163	0.095585	0.07647
Total Duration (s)	45.8	12.0	3.6	1.9	16.9	38.5	23.7	8.6
Average Duration (s)	7.8	4.4	1.7	1.3	4.6	8.3	9.1	2.0
Total Latency (s)	136.2	44.2	38.4	29.6	54.9	110.7	51.9	56.8
Average Latency (s)	26.4	20.6	17.4	14.2	19.1	23.1	17.9	22.0

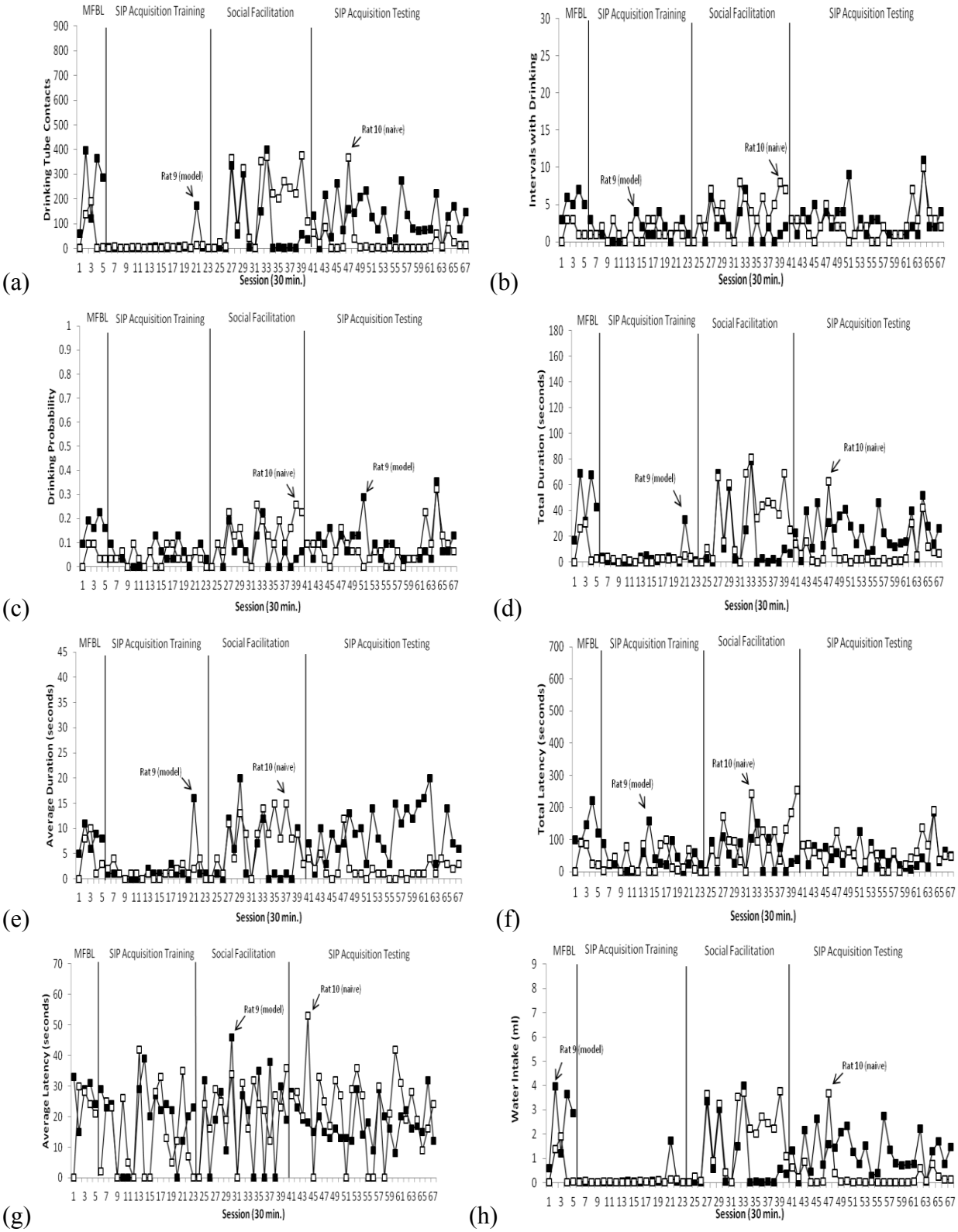

Figure 5 (a-h). Rates of drinking across sessions for the model rat (9) and naïve rat (10) in the Feeding Control Group.

Table 7

Average Rates of Drinking for the Model Rat (11) and Naïve Rat (12) in the Feeding Control Group

	Phase 1: Massed-Food Baseline		**Phase 2: SIP Acquisition Training**		**Phase 3: Social Facilitation**		**Phase 4: SIP Acquisition Testing**	
	Model (11)	**Naïve (12)**	**Model (11)**	**Naïve (12)**	**Model (11)**	**Naïve (12)**	**Model (11)**	**Naïve (12)**
Drinking Tube Contacts	17.8	60.8	41.6	181.1	47.6	17.5	32.4	15.4
Water Intake (mL)	0.18	0.61	0.42	1.81	0.48	0.18	0.32	0.15
Intervals with Drinking	1.8	2.6	0.9	5.6	1.5	1.7	3.8	0.6
Drinking Probability	0.05806	0.08386	0.028678	0.179211	0.04946	0.058067	0.123056	0.020322
Total Duration (s)	6.2	11.4	8.3	40.8	10.5	5.2	10.1	3.5
Average Duration (s)	2.2	3.4	3.4	6.6	4.3	1.5	2.8	2.4
Total Latency (s)	60.0	98.4	16.2	132.8	40.0	47.6	71.4	18.0
Average Latency (s)	26.6	38.2	7.2	22.4	17.5	20.2	16.4	12.7

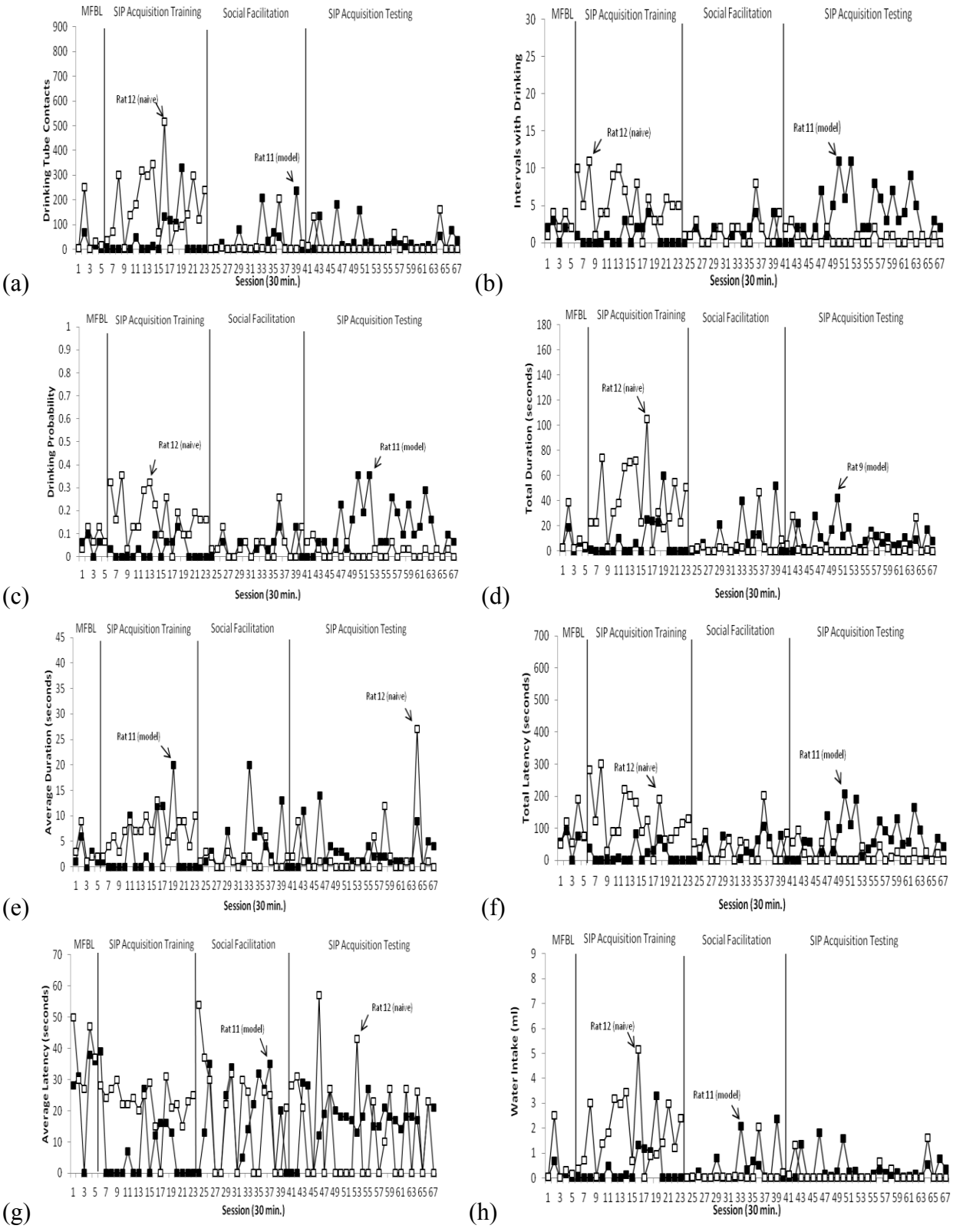

Figure 6 (a-h). Rates of drinking across sessions for the model rat (11) and naïve rat (12) in the Feeding Control Group.

Table 8

Average Rates of Drinking for the Model Rat (13) and Naïve Rat (14) in the Social Contact Control Group

	Phase 1: Massed-Food Baseline		**Phase 2: SIP Acquisition Training**		**Phase 3: Social Facilitation**		**Phase 4: SIP Acquisition Testing**	
	Model (13)	**Naïve (14)**	**Model (13)**	**Naïve (14)**	**Model (13)**	**Naïve (14)**	**Model (13)**	**Naïve (14)**
Drinking Tube Contacts	90.6	115.0	5.9	65.9	47.9	25.6	2.4	5.3
Water Intake (mL)	0.91	1.15	0.06	0.66	0.48	0.26	0.02	0.05
Intervals with Drinking	2.8	2.0	0.4	1.8	2.2	2.4	0.9	1.4
Drinking Probability	0.0903	0.06454	0.012561	0.057356	0.075912	0.064529	0.028685	0.046611
Total Duration (s)	19.2	18.0	1.5	12.2	11.4	7.7	1.7	2.6
Average Duration (s)	5.6	8.2	1.5	4.7	3.1	3.8	0.7	1.4
Total Latency (s)	96.4	56.2	8.8	38.1	56.1	63.0	18.3	45.4
Average Latency (s)	35.4	28.6	8.8	14.6	21.9	21.1	9.8	27.0

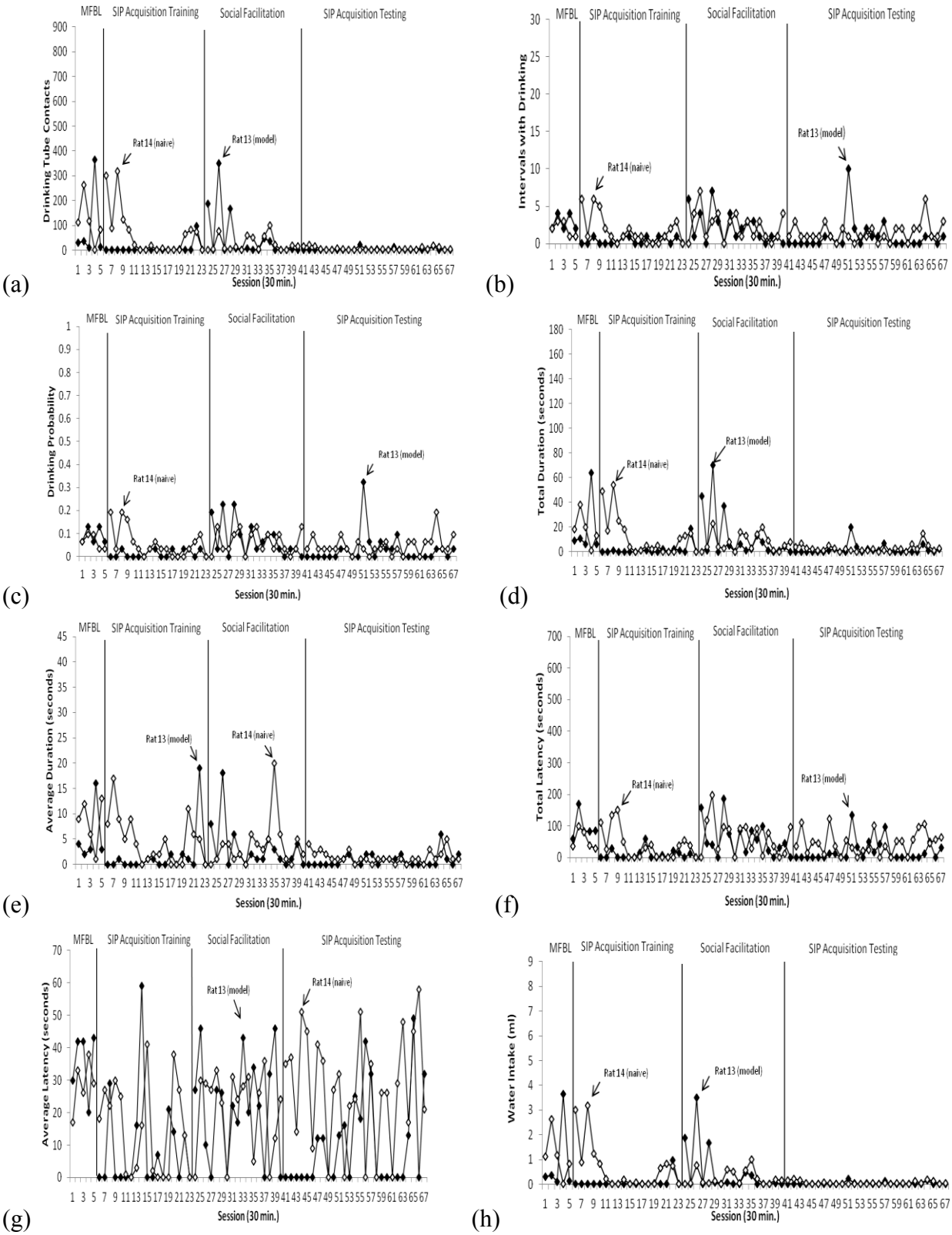

Figure 7 (a-h). Rates of drinking across sessions for the model rat (13) and naïve rat (14) in the Social Contact Control Group.

Table 9

Average Rates of Drinking for the Model Rat (15) and Naïve Rat (16) in the Social Contact Control Group

	Phase 1: Massed-Food Baseline		**Phase 2: SIP Acquisition Training**		**Phase 3: Social Facilitation**		**Phase 4: SIP Acquisition Testing**	
	Model (15)	**Naïve (16)**	**Model (15)**	**Naïve (16)**	**Model (15)**	**Naïve (16)**	**Model (15)**	**Naïve (16)**
Drinking Tube Contacts	398.8	96.8	43.9	123.4	154.4	194.4	480.5	32.7
Water Intake (mL)	3.99	0.97	0.44	1.23	1.54	1.94	4.81	0.33
Intervals with Drinking	5.4	3.4	3.9	2.7	2.9	4.3	7.3	4.6
Drinking Probability	0.17418	0.10966	0.127239	0.086022	0.092738	0.1391	0.234178	0.146959
Total Duration (s)	73.4	21.0	17.3	26.8	31.8	41.1	91.2	12.7
Average Duration (s)	14.8	4.4	2.4	7.8	8.8	9.8	13.2	2.5
Total Latency (s)	129.8	83.2	83.7	60.8	55.2	104.5	132.0	111.9
Average Latency (s)	22.2	16.2	16.3	20.4	17.3	24.6	17.7	23.8

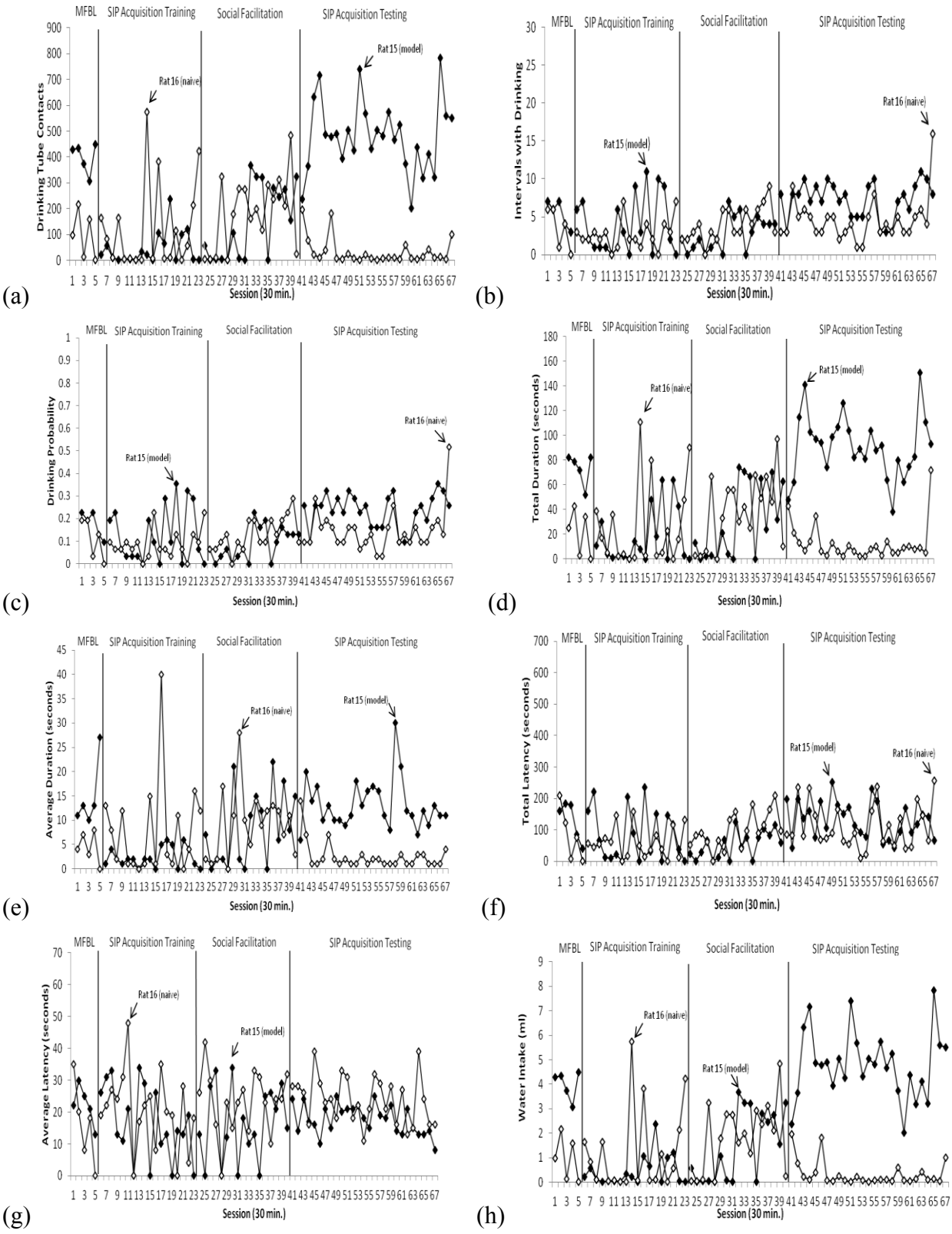

Figure 8 (a-h). Rates of drinking across sessions for the model rat (15) and naïve rat (16) in the Social Contact Control Group.

Average data presented in Table 10 and session trends depicted in Figure 9 demonstrate that the rats assigned to the Naïve Controls Group did not acquire elevated levels of drinking during the final phase. These data further support the hypothesis that social facilitation may enhance the rate of acquisition of SIP and that rats that were not exposed to a drinking model were slow to acquire elevated drinking rates, if they acquired elevated drinking at all.

Table 10

Average Rates of Drinking for Rats Assigned to the Naïve Controls Group

	Phase 1:Massed-Food Baseline	**Phase 2 and 3: Naïve Controls Preparation**	**Phase 4: SIP Acquisition Testing**
	Rats 19-24	**Rats 19-24**	**Rats 19-24**
Drinking Tube Contacts	44.3	13.7	25.6
Water Intake (mL)	0.44	0.14	0.26
Intervals with Drinking	2.8	1.9	2.1
Drinking Probability	0.091403	0.06222	0.068304
Total Duration (s)	11.2	4.7	6.9
Average Duration (s)	2.5	1.4	1.8
Total Latency (s)	80.6	53.8	51.5
Average Latency (s)	24.5	21.2	16.4

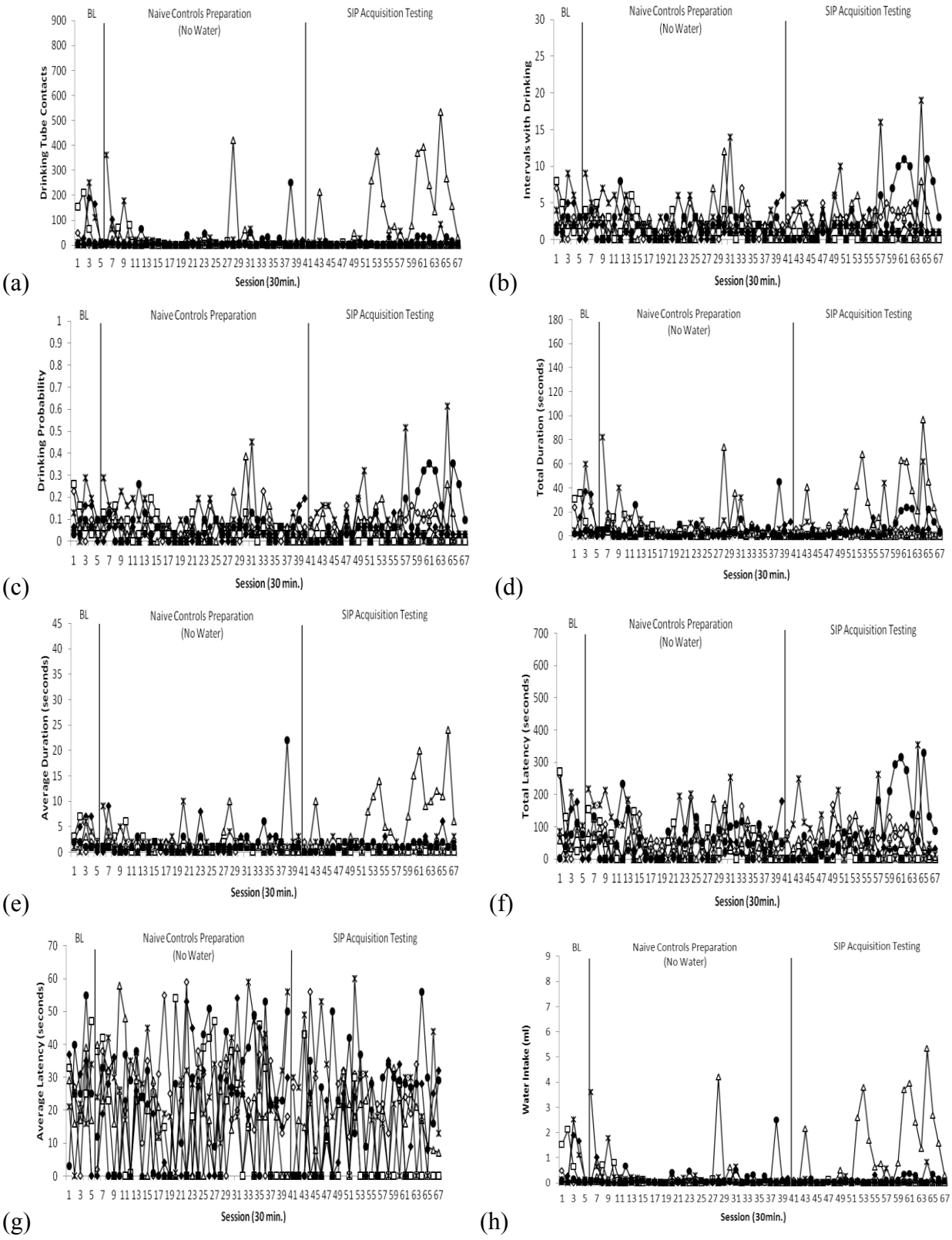

Figure 9 (a-h). Rates of drinking across sessions for naïve rats in the Naïve Controls Group.

To more closely examine acquisition rate between groups during the final phase of the study, data were compiled for each group and are presented in Table 11, while session data are presented in Figure 10. These data indicate that, on average, rats exposed to a drinking model engaged in more elevated drinking rates than rats in any other group.

Table 11

Average Rates of Drinking for Rats in Each Group during Phase 4: SIP Acquisition Testing

	Phase 4: SIP Acquisition Testing						
	Drinking Model	**Drinking Naïve**	**Feeding Controls Model**	**Feeding Controls Naïve**	**Social Contact Controls Model**	**Social Contact Controls Naïve**	**Naïve Controls**
Drinking Tube Contacts	260.8	315.3	54.6	88.5	163.9	54.9	25.6
Water Intake (mL)	2.61	3.15	0.55	0.88	1.64	0.55	0.26
Intervals with Drinking	8.5	7.9	3.3	2.1	3.2	3.3	2.1
Drinking Probability	0.275586	0.25608	0.106335	0.067707	0.10196	0.106333	0.068304
Total Duration (s)	52.7	60.0	13.0	16.2	32.2	13.3	6.9
Average Duration (s)	5.7	7.1	4.3	4.9	5.2	2.8	1.8
Total Latency (s)	122.2	155.6	61.6	44.2	61.1	74.2	51.5
Average Latency (s)	14.1	18.5	17.0	17.0	14.4	22.4	16.4

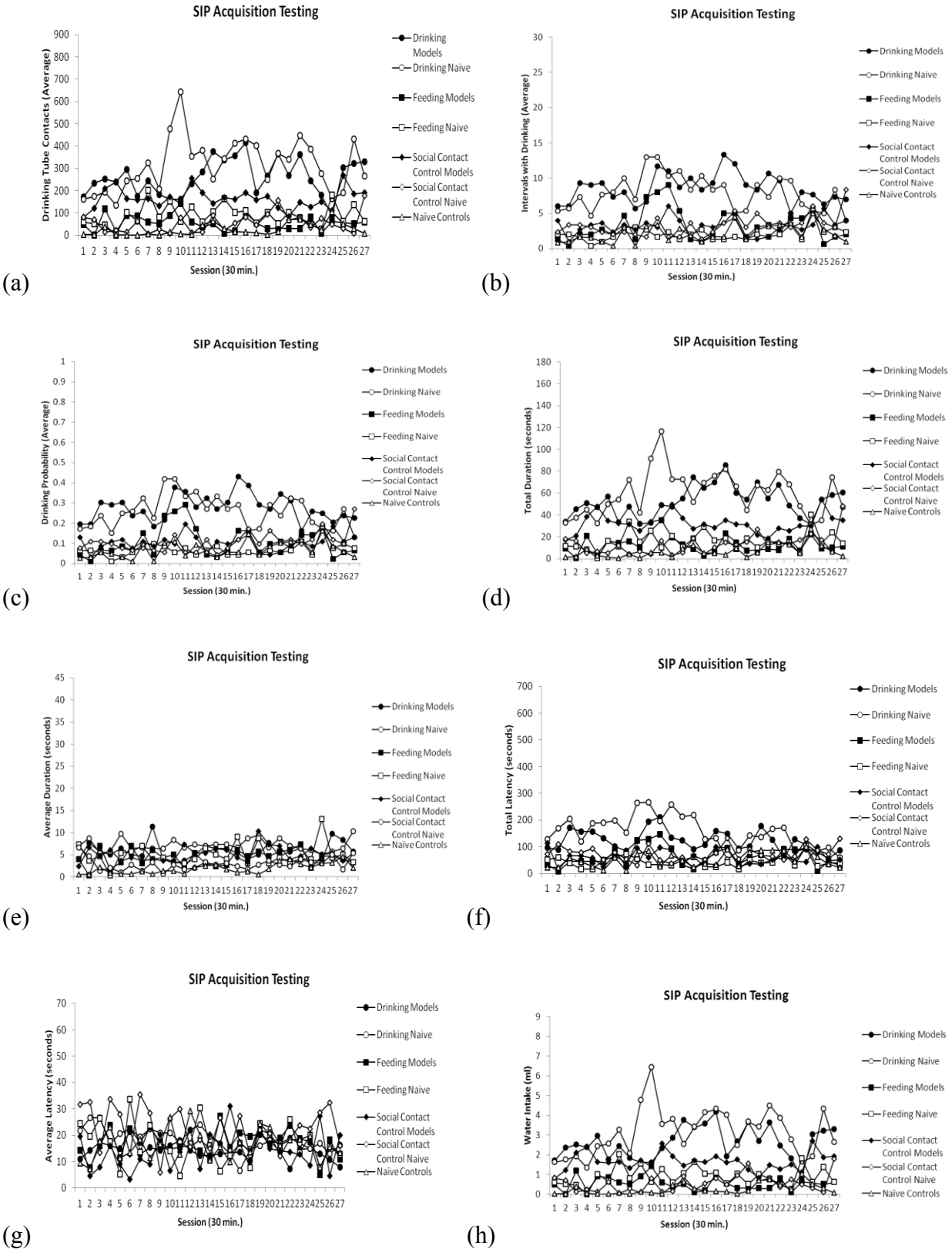

Figure 10 (a-h). Average rates of drinking for all rats during Phase 4: SIP Acquisition Testing.

Although the data mentioned above support the hypotheses proposed by the current study, there were some individual differences noted in particular with one pair of rats in the Feeding Control Group and one pair of rats in the Social Contact Control Group. These data patterns will be reviewed in detail because they do not follow the same data paths observed with other rats in their group.

Results for rats 7 and 8 in the Feeding Modeling Group indicate that the naïve rat engaged in higher rates of drinking tube contacts than the model rat in baseline; however, a decreasing trend was observed (refer to Figure 11a) skewing the overall averages reported in Table 12. The naïve rat also engaged in higher rates of total and average duration during baseline. Low rates were observed in both rats during the SIP Acquisition Training condition across all measurements with the exception of average latency. These data are to be expected, given that there was no water available during the SIP Acquisition Training condition and behaviors observed in this condition were likely the result of initial contact to determine if water was accessible and subsequent accidental contact with the drinkometer. When the model rat and naïve rat were placed together, water was made available through the drinking tube. Results indicate that the naïve rat started to engage in higher rates of drinking tube contacts across these sessions and into the SIP Acquisition Testing condition, while the model rat maintained its low rates in both conditions. This data pattern was also observed when measuring total duration and average duration.

When considering measurement of intervals with drinking, drinking probability, and total latency, low rates of drinking were observed in baseline and in SIP Acquisition Training. During conditions in which the rats were paired and water was made available, there appeared to be an increase in variability along these dimensions for both rats. This increase in variability continued

and appeared to become more evident into the SIP Acquisition Testing condition. It should be noted that rates were not differentiated along these dimensions.

The model and naïve rat's average latency seemed to be highly variable during baseline, SIP Acquisition Testing, and model and naïve pairing conditions. Both rats' varaibility along the dimension of average latency appeared to decrease during SIP Acquisition Testing.

Table 12

Average Rates of Drinking for the Model Rat (7) and Naïve Rat (8) in the Feeding Control Group

	Phase 1: Massed-Food Baseline		**Phase 2: SIP Acquisition Training**		**Phase 3: Social Facilitation**		**Phase 4: SIP Acquisition Testing**	
	Model (7)	**Naïve (8)**	**Model (7)**	**Naïve (8)**	**Model (7)**	**Naïve (8)**	**Model (7)**	**Naïve (8)**
Drinking Tube Contacts	10.8	103	17.9	9.2	12.4	118.8	7.1	220.4
Water Intake (mL)	0.11	1.03	0.18	0.09	0.12	1.19	0.71	2.20
Intervals with Drinking	1.8	1.6	1.8	1.8	1.0	2.9	3.1	3.3
Drinking Probability	0.05808	0.05162	0.059139	0.059144	0.032265	0.094876	0.100363	0.10633
Total Duration (s)	3.8	19.6	4.8	4.7	2.9	21.2	5.1	36.6
Average Duration (s)	1.6	11.2	2.1	1.9	1.6	10.6	0.9	10.4
Total Latency (s)	35.8	34.4	52.6	47.8	29.9	80.2	61.6	57.9
Average Latency (s)	23.8	16.8	21.8	21.9	18.1	24.2	16.6	16.6

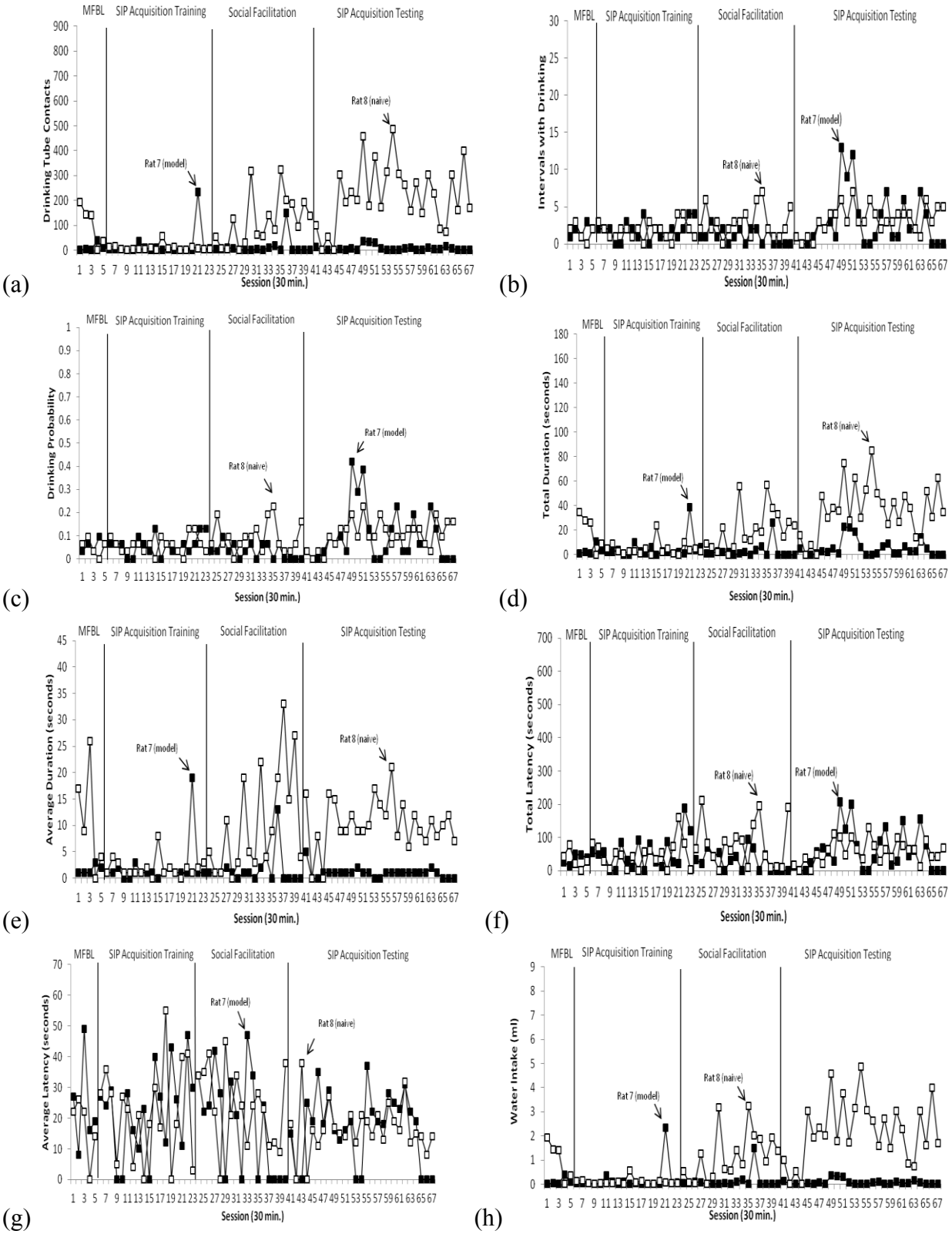

Figure 11 (a-h). Rates of drinking across sessions for the model rat (7) and naïve rat (8) in the Feeding Control Group.

Baseline data for Model Rat 17 and Naïve Rat 18 in the Social Contact Control Group indicate that the model rat demonstrated higher rates of responding along all measured dimensions, except average latency, than the naïve rat. Variability was also higher for the model rat along the dimensions of drinking tube contacts, total duration, average duration, and total latency. Low stable rates were observed with the naïve rat along all dimensions, with the exception of average latency where the naïve rat's average latency was higher than the model. Data continued to be differentiated during the SIP Acquisition Training condition along all dimensions, except for average latency. The model rat engaged in higher, more variable rates of responding along all dimensions, with the exception of average latency, than the naïve rat in the SIP Acquisition Training condition. The naïve rat was observed to have low stable rates, while the model rat demonstrated a slight downward trend in total latency. Trends during the SIP Acquisition Training condition along the dimension of average latency indicate that results were differentiated at the start of the condition with the model rat having higher latency, while the naïve rat demonstrated low stable rates. Towards the middle of this condition, data become undifferentiated, with the naïve rat engaging in more variable rates along this dimension (refer to Figure 12g).

When the rats were paired together, the model and naïve rats' results were undifferentiated, with both showing an increasing trend and increased variability along dimensions of drinking tube contacts, total duration, and average duration. Some differentiation was observed on the graph of average duration, with the naïve rat demonstrating higher rates than the model rat. Data on the intervals with drinking, drinking probability, and total latency show that both the model rat and naïve rat have increases along these dimensions, with the model rat showing more significant increases than the naïve rat. Variability seemed to increase for both

rats towards the end of the condition. During rat pairings, the data trends were highly diferentiated, with the model rat demonstrating higher rates than the naïve rat at the start of the condition. This data trend changed over a few sessions, with the naïve rat's rate of average latency increasing to match the model rat's data. Results from this point on appear to be more stable and undifferentiated.

Data show a clear differentiation between the model rat and the naïve rat along the dimensions of drinking tube contacts, intervals with drinking, drinking probability, total duration, and average duration. Both rats' data trends along these dimensions started low and stable. The naïve rat had an increasing trend, which became more variable over time. The model rat had increased variability along these dimensions towards the end of the condition. When tracking the data trends along the dimension for total latency, a reduction was observed in the total latency, and data stabilized and was undifferentiated for the model and naïve rat. An inverse trend was observed with average latency where variability increased for both rats, resulting in undifferentiated results during the first half of the condition and then a subsequent stabilization of the naïve rat's rates with continued variability for the model rat.

Table 13

Average Rates of Drinking for the Model Rat (17) and Naïve Rat (18) in the Social Contact Control Group

	Phase 1: Massed-Food Baseline		**Phase 2: SIP Acquisition Training**		**Phase 3: Social Facilitation**		**Phase 4: SIP Acquisition Testing**	
	Model (17)	**Naïve (18)**	**Model (17)**	**Naïve (18)**	**Model (17)**	**Naïve (18)**	**Model (17)**	**Naïve (18)**
Drinking Tube Contacts	209.0	15.8	191.8	6.2	159.9	154.5	8.9	126.8
Water Intake (mL)	2.09	0.16	1.92	0.06	1.60	1.55	0.09	1.27
Intervals with Drinking	4.8	1.6	3.8	0.5	8.3	3.2	1.3	3.9
Drinking Probability	0.15486	0.05162	0.121867	0.016144	0.264527	0.13334	0.043019	0.12543
Total Duration (s)	38.4	4.6	36.4	1.3	39.8	30.1	3.5	24.5
Average Duration (s)	7.0	2.6	8.9	1.2	4.5	8.3	1.6	4.6
Total Latency (s)	117.4	65.2	85.5	16.6	195.5	76.6	33.1	65.3
Average Latency (s)	20.8	42.4	23.0	14.1	26.9	20.7	15.7	16.3

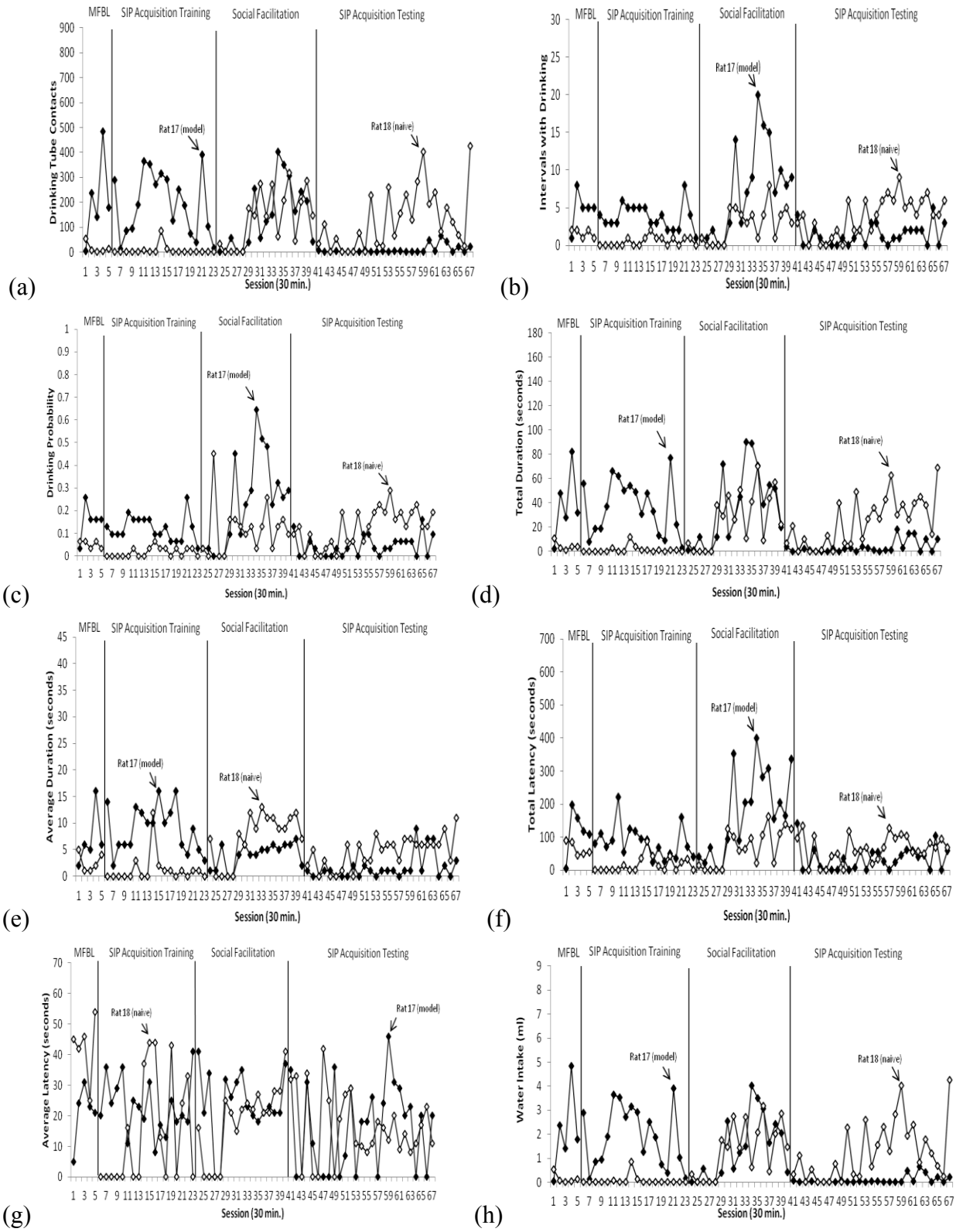

Figure 12 (a-h). Rates of drinking across sessions for the model rat (17) and naïve rat (18) in the Social Contact Control Group.

Chapter 4: Discussion

The purpose of the current study was to evaluate the hypothesis of social facilitation of compulsive behavior using an animal model. It was hypothesized that a naïve rat will begin to engage in elevated rates of drinking if placed with a model rat that itself engages in elevated rates of drinking, and do so primarily when the model drank. A second hypothesis held that rats with a history of socially facilitated drinking would subsequently acquire SIP, a type of compulsive behavior, more rapidly than rats which had not exhibited socially facilitated drinking. The former and latter together would show the fact of social facilitation of these forms of behavior and the enhanced acquisition of a compulsion through this mechanism. In general, the data from this study supported both hypotheses. The naïve rats that were paired with the drinking models drank more often and acquired polydipsia during the SIP Acquisition Testing condition more readily than the naïve rats that were paired with feeding models, social contact control models, and other naïve rats. These findings are consistent with preliminary work on the social facilitation of drinking by rats and suggest a mechanism by which the acquisition of a specific compulsive response topography might be engendered.

However, it is important to note that this general effect was not universally seen or strong across the subjects. That is, the results seen in the three model rats and their matched naïve rats that were placed in this drinking modeling group were highly variable. Two of the three drinking models increased their rates of drinking during SIP Acquisition Training, while the third maintained its baseline rates. All the naïve rats maintained their baseline rates during this condition, as expected. High variability within and between subjects also occurred during the Social Facilitation condition in which the drinking model rats were paired with the naïve rats. The drinking model in the first pair had a significant decline in drinking at the start of the

condition, with a steady increasing trend towards the end. Its matched naïve rat had a sudden increase in behavior that continued throughout the condition. The drinking model in the second pair maintained its baseline rates, while its matched naïve rat had a gradual increasing trend. The drinking model in the third pair had a sharp decrease in drinking, with a slight increasing trend towards the end of the condition, while its matched naïve rat maintained its low baseline rates.

Baseline rates of drinking were similar between the model and the naïve rat in the first pair. However, the model rats in the second and third pair engaged in consistently higher rates of drinking than their matched naïve rat. It is possible that these different baseline rates may have impacted the patterns of responding in future conditions. This high level of variability differs from previous research using these procedures (e.g., Todd et al., 1997), perhaps due to subtle effects of handling and other difficult-to-define procedural factors. Todd's results, using the same equipment in the same laboratory, were obtained through a long-term project run by researchers with years of experience relative to the months of the present researcher. Additional research might discover the reason for this difference and would be an important factor in evaluating these hypotheses. If the effect is dependent on particular expertise and can be strongly modulated by handling effects, the case for this being a useful animal model is weakened.

During SIP Acquisition Testing, there was a great deal of variability in the acquisition of drinking by the naïve rats and difficulties maintaining drinking for the model rats. Although the data averaged across all rats in the Drinking Modeling group demonstrate that the naïve rats' rate of drinking matched the drinking model rats' rates, this is not true for each individual pairing. The drinking model rat in the first pair had a significant decline in drinking during SIP Acquisition Testing, while its matched naïve rat engaged in high rates of drinking. The drinking model rat in the second pair had a slight increase in drinking rates, while its matched naïve rat

maintained its increased rates. The naïve rat's rate of drinking in the second pair never increased enough to match the model rat's rates. Last, the model rat in the third pair had an increasing trend in drinking rates, while the naïve rat matched its rate of drinking to the model rat's rate, even though they were not in the same chamber throughout this condition.

It should be noted that the pattern observed in the third pair of rats is what was expected of all rats in the Drinking Modeling group if social facilitation was the sole mechanism through which this repetitive behavior was acquired. However, there is empirical support for other processes, besides social facilitation, that can lead to the acquisition and maintenance of polydipsia, specifically, and other maladaptive repetitive behaviors in general. These processes include response acquisition through response-independent schedules (Falk, 1971; Ferster & Skinner, 1957; Flores & Pellon, 1997; Lopez-Crespo et al., 2004; Todd et al., 1997; Perone, 1991; Skinner, 1948), which were used to train the models in the current study, shaping and chaining (Skinner, 1938), sensitization (Groves & Thompson, 1973), confinement (Gluck & Pearce, 1977), and social deprivation (McKinney, 1974).

Although these theories have some empirical support, many of the factors were controlled for by the experimenter in the current study. For example, the experimenter controlled for age-related and sex-related variables by conducting the study with rats that were all the same age and sex. It is not known, at this time, that age is a relevant factor in the development of socially facilitated polydipsia. However, some studies in the applied literature report age-related influences on the development of compulsive behaviors, in general (Butwicka & Gmitrowicz, 2010; Geller et al., 2001). According to the DSM-IV-TR, age of onset of OCD is between six and 15 years in males and between 20 and 29 years for females. However, it is important to note that it is possible that there is a critical period in which compulsive behavior is more likely to

develop, and that might have been different than the rats used in the current study. This idea is, in part, supported by the fact that none of the naïve controls became polydipsic after being placed on the same schedule used to train the Drinking Model rats to drink earlier in the study.

The experimenter controlled for space by ensuring that all of the rats had the same-size home cages and experimental chambers. This was done because Gluck and Pearce found that primates had increased perseveration on tasks without being rewarded when they were confined for long periods of time. However, it should be noted that the amount of room for each rat to explore was minimized during the conditions in which model rats were paired with the naïve rats. That said, we must consider that by putting the rats in a very small space, next to one another, and using drinking tubes that are noisy, there is little chance that the naïve rat could not notice the model drinking. If social facilitation is to be a model of the acquisition of compulsive behavior, then we might have to look for situations in which the observer has a high probability of repeatedly observing the model. The nature of the behavior is probably less important. Here, it is a physical activity involving the competition of two organisms for access to one resource. Typical OCD behavior may involve competition between the OCD behavior and other demands, but probably not direct competition with another organism that could potentially countercontrol.

The experimenter also controlled for socialization factors, which was a critical aspect of the study, to determine the effects of social facilitation on drinking. Socialization was limited to only the times in which model rats were paired with naïve rats. Rats had no opportunities to socialize outside of experimental sessions because each rat was housed alone with no access to other rats. In addition to these variables, the study was designed in such a way that there was a control rat for each experimental condition that was used as a comparison for the experimental rats, and the schedule of food delivery was controlled in and out of experimental sessions.

Environmental Variables Impacting SIP Acquisition Leading to Within-group Differences

Based on the current data, it is likely that drinking by the naïve rats was socially facilitated in at least two of the three pairs of rats. However, there may be other variables that were not taken into account or controlled experimentally. The reasons for the individual differences in rate of drinking are unclear. It is possible there are alternative variables that were not accounted for throughout the course of the study that may have greatly impacted the pattern of responding. These variables and the effects that they may have had in the current study require some review.

Time of day. Experimental sessions were conducted at various times throughout the day. The majority of the sessions were conducted during the early evening hours. However, this was not the case on some days given the schedule of the experimenter. Experimental sessions were occasionally conducted in the afternoon or late evening hours. All of the rats completed experimental sessions within two hours of each other. This may be an important factor that may have affected drinking patterns, in that it is possible that rats are more likely to engage in higher rates of eating and drinking during the late evening hours than the early morning or afternoon hours. A study conducted by Kaya, Karakaş, and Coşkun (2011) experimentally demonstrate time of day being an important variable in the anxiety-like behavior of Wister rats travelling through an elevated maze along the dimensions of distance travelled, mobility, and velocity. This study suggests that, if time of day was not held constant, variability in responding within each subject and across subjects may have been a result of the this factor rather than something specific to the subject itself.

Temperature. Experimental sessions were conducted indoors. It is important to note that the experimenter did not have any control over the thermostat in the rooms that housed the

experimental chambers. It is possible that rats behave differently in certain temperatures than in others. It is also possible that if SIP is defined as a form of adjunctive behavior and the temperature during the experimental sessions was not optimal, then it could have minimized the rats' propensity to engage in the specific drinking behaviors that were being examined. It did appear as though there was a shift in the temperature in the building from the start of the study to the end of the study, given that the study was started in a winter month and continued through the summer months. In a study conducted by Baker (1980), he found that feeding may strongly depend on cues from the natural environment such as temperature, light, social cues, and prior feeding experiences. If these environmental variables remain constant, then feeding behavior in rats is considered to be aperiodic (i.e. feeding does not occur in a cyclical or time based pattern), if their pattern is put on extinction. This study suggests that the temperature in the room may have had an effect on feeding behaviors, which would in turn have an effect on drinking, especially since eating is an important component in SIP acquisition.

Light. The current study was conducted with the light on throughout all of the experimental sessions. It is not clear if the presence of light affected the behavior of the rats in the current study. In Todd and colleagues' earlier work, the lights were off during the experimental sessions. Thus, it is possible that rats are more likely to engage in higher rates of drinking in a darker environment than a lighter one. Baker (1980) did consider that, in addition to temperature, the presence or absence of light can serve as an environmental signal to eat. It is also believed that this may affect drinking patterns in the current study if the pattern of SIP usually begins with the rat eating a food pellet on the time-based schedule and then drinking.

Noise. The noise level varied throughout the course of the study. It seemed that the rats were more likely to drink in the experimental chambers when there was some noise compared to

times in which it was extremely quiet. However, no data were collected on noise level to determine if this affected the rats' behavior in any way. Noise level during experimental sessions was difficult to control for because construction was going on in the building, sometimes when the study was being conducted. In addition, other experiments were being conducted in the adjoining rooms and other experimenters and participants were frequently interacting in the hallways, while the current experimental sessions were being conducted. Moreseth, Dengerink, and Wright (1985) found that noise exposure that simulated an industrial setting significantly increased blood pressure and water consumption in female rats exposed to the noise for two weeks. In another study, Krebs, Macht, Weyers, and Weijers (1996) found that high decibel levels of white noise increased the duration of eating, exploring, grooming, and resting behaviors of rats. Although Krebs and colleagues (1996) did not examine the effects that noise can have on drinking rate, it is likely that that their results can be generalized to this behavior, as well. This is especially true if the researchers considered drinking an "adjunctive" behavior and classified drinking under the category of eating or exploring.

Individual Variables Impacting SIP Acquisition Leading to Within-group Differences

The current data indicate that polydipsia was socially facilitated with at least two of the three pairs of rats in the Drinking Modeling group. However, drinking significantly increased in one naïve rat in the Feeding Modeling group and in one model rat in the Social Contact Control group. Given that there were increases in drinking with other rats that were not expected to drink under the hypotheses presented, it is important to consider that there may have been individual variables affecting drinking rate in those rats that should be considered as a potential explanation for the acquisition of polydipsia or other compulsive behaviors.

Neurobiological variables. Some of the variation observed in the current study may be the result of differences in the rats' neurobiology that inhibited or promoted the acquisition of polydipsia. A study conducted by Pellon and colleagues (2011) found dopaminergic differences in rats that were classified as low drinkers and rats that were classified as high drinkers when exposed to a time-based schedule of food delivery for SIP. They found that low drinkers showed higher binding than high drinkers for D1 receptors in the nucleus accumbens, medial prefrontal cortex, amygdala, and the ventral tegmental area, while high drinkers showed higher binding than low drinkers for D2 receptors in the same areas. Given that SIP may be the best model for compulsive behavior observed humans (Moreno & Flores, 2012; Pietrowski, 2005; Pietrowski & Todd, 2004) these neurobiological variations may have been present or developed with the subjects over the course of the current study. This also may explain why many medications used to treat compulsive behaviors alter dopamine and serotonin levels. As mentioned previously, McDougle, Goodman, and Price (1994) demonstrated that using medications to block dopamine receptors led to changes in their patients' responses to SSRIs, increasing the efficacy of treatment for these patients. However, it is still unclear whether the acquisition of this behavior and other maladaptive behaviors in humans causes these neurobiological changes or if these neurobiological changes result in this compulsive behavior pattern.

Baseline rates of drinking. The rats that were under investigation in this study were expected to have baseline rates that were comparable. However, this was not the case. The data demonstrate that baseline rates of drinking varied significantly for each of the rats. This variability seemed to be related to environmental exploration of each rat. Observations of the rats during baseline in the experimental chambers involved a variety of behaviors that specifically involved sniffing the food dish and the water dispenser for some rats, while other rats seemed to

be less likely to explore the environment. Some rats were just observed to lie down in a corner of the experimental chamber and maintain that position for long periods of time. It appears as though rats that were more actively exploring their environment may have inflated baseline rates of drinking than the rats that remained stationary throughout most of the baseline sessions. This is likely due to the fact that they had more contact with the drinkometer than the rats that engaged in less environmental exploration and thus contacted the drinkometer less often.

Because there was so much variability in baseline rates of drinking, it may have been useful to continue with baseline sessions for longer than five days to ensure that each rat's rate of drinking stabilized or to determine if the variable patterns of drinking persisted. The experimenter also randomly paired rats together regardless of baseline rates of drinking. Water consumption and environmental exploration could have been controlled for if the experimenter paired together rats with similar baseline rates to ensure that changes in drinking were primarily related to the environmental contingencies in place, rather than individual differences in water consumption or environmental exploration.

Rate of SIP acquisition. In addition to the baseline rates of drinking for each rat, it seemed as though there were some individual differences in the rate at which the model rats became polydipsic. One of model rats in the Drinking Modeling group had significant increases in drinking during SIP Acquisition Training, while another model rat had low rates of drinking throughout the majority of the condition and then had some slight increases in drinking rate. The third model rat in this condition maintained its high and variable baseline rate of drinking. It is not clear what factors may have played a role in the rate at which the model rats acquired or, in one case, did not acquire polydipsia. It is possible that the individual differences in baseline rates, as mentioned previously, may have affected drinking behavior during the SIP Acquisition

Training condition. These differences may also be related to slight changes in weight; even though the same weight reduction procedures were used with all rats, slight variations that were not readily measured or observed may have affected drinking acquisition (Roper & Nieto, 1979).

Similar to baseline rates of drinking, it may have proven to be more useful for the experimenter to continue running out the SIP Acquisition Training condition for several more sessions, until stable patterns of responding were observed. However, there were concerns that some of the rats patterns would not stabilize and that the variability in drinking rate was related to some of the environmental variables mentioned above, specifically noise, temperature, and time of day.

Competition for food. In addition to the current study demonstrating variable rates of drinking and the rate at which the model rats acquired SIP, competition for food seems to play a bigger role than was expected. This is especially true in the conditions in which the rats were paired. The model rats in all groups were food deprived to enhance eating in the chamber. However, when each model rats' matched naïve pair was placed into the experimental chamber during session, the model rats seemed to spend the majority of their time with their heads close to or inside the food dispenser. It is assumed that the model rats wanted to remain near the food dispenser to ensure that they received the food pellet as soon as it was delivered and wanted to limit opportunities for its matched naïve rat to access the food. This behavior seemed to be the result of the model rat having decreased rates of drinking in the paired condition. However, it is not clear what the resulting effect would be on the naïve rat. On one hand, the model rat was not engaging in high rates of drinking behavior during this condition and, therefore, not modeling the behavior to the naïve rat, suggesting that the naïve rat would not adequately acquire high rates of drinking. However, it seems as though some of the naïve rats did engage in high rates of drinking

when paired with the model rat. This could be because the naïve rat had increased access to the water dispenser, since the model rat was allocating most of its time to positioning itself near the food dispenser.

Given that there were three models and three naïve rats in the Drinking Modeling group, it seems that there were individual differences in the model and naïve rats' response to the pairing condition. One of the model rats maintained its baseline rate of drinking, while the other two rats had an initial decline in drinking rate at the start of the condition and subsequent variability in drinking. This issue of competition for food is an important one and should not be underestimated. It could be controlled for if the rats had two separate food dispensers and two separate water dispensers, with each rat being able to access only on of them. However, conducting the study in this way would require a barrier of some sort to ensure that there was no way of accessing the other rat's food. If the study were conducted in this way, it is believed that the rats' socialization would be limited unnaturally because of limited physical contact.

Within-session timing. Individual rates of drinking may have been affected by the timing within sessions. This is specifically relevant to the condition in which the model rats were paired with their matched naïve rat. When considering the condition in which the rats were paired, it is important to note that there were multiple changes in the environment for both the model rat and the naïve rat that may have affected the rate of drinking. First, the model rat in each group was weight reduced, while the naïve rat was not. Observations of the model rat's behavior when paired with the naïve rat demonstrate that the model rat would limit its environmental exploration to being as close to the food dispenser as possible, as mentioned previously. However, there were times when the model rat would interact with the naïve rat. Depending upon the specific behavior of each rat at the time that food was delivered, it is

believed that it set the course for the pattern of responding that would occur throughout the session and possibly throughout the condition. For example, it is possible that at the first session in which the Drinking Model was placed with the Drinking Naïve rat, the naïve rat was closer to the food and ate it. This may have signaled to the model rat that was food deprived that it has more opportunities to get food if it stays closer to the food dispenser than if it interacts with the naïve rat or the drink. This may explain why, in some cases, the model rat regardless of group would have a reduction in drinking rate from baseline. However, if the Drinking Model was interacting with its matched Drinking Naïve rat and the first pellet was delivered and the model rat ate it because it just happened to be closer to the food dispenser, then it is likely that the model rat would be less concerned about the naïve rat eating the food pellet and become more likely to drink after the food pellet was delivered and complete its typical pattern. If within-session timing is an important variable that was not previously accounted for, then Skinner's (1938) hypothesis of accidental but powerful reinforcements that initially established the compulsive behavior may warrant more attention.

Limitations

One limitation of the current study is that baseline rates of drinking for all rats were not equal. In fact, there appeared to be significant variability in each rat's rate of drinking at the start of the study. It is often the case that an experimenter will conduct five sessions of baseline data to use as a comparison point and to allow the rats to explore the experimental chamber as their new environment. It was expected that each rat's rate of drinking would be similar to its matched pair. However, it was often the case that one rat engaged in higher baseline rates than the rat it was paired with. This variability made it difficult to define what was considered polydipsia, because for some rats high rates of drinking were observed in baseline, while for other rats, rates

of drinking were considerably lower or nonexistent when compared to their matched counterparts. Differentiated rates at baseline may lend more support to the notion that individual variables that were not accounted for may have had a stronger effect than initially expected. In addition, these baseline rates may have skewed the results during other conditions such that rats that engaged in high rates of drinking may have different amounts of dopamine or serotonin in their system, which may have made them more or less susceptible to acquiring the behavior under the different environmental contingencies that they were placed under.

Another limitation of the current study is that criterion to move to the next condition was based on the rat's behavior or the number of sessions that were conducted. However, more robust rates of drinking may have been observed if we extended the conditions with some rats, which would have led to a stronger effect. It is also possible that rats that were not believed to have excessive rates of drinking may have acquired the behavior with more exposure to the contingency or with more exposure to the model rat if criterion were more focused on the data that were being produced, rather than the number of sessions completed. In addition, extending the conditions may have allowed for more time for the variable results to stabilize, theoretically resulting in more stable and differentiatated results.

Order effects may be have been a limitation in the current study. The experimenter controlled for these effects by having naïve rats serve as controls. However, as mentioned previously, the amount of variability in rate of drinking that was observed between rats at baseline may have limited their ability to serve as appropriate controls over these variables. It seemed as though there were some carry-over effects from previous conditions, particularly from baseline to SIP Acquisition Training conditions. This could be related to the fact that the environments in both of these conditions were not discrepant enough when compared to being

placed alone in a chamber to being placed with a model rat. This issue should be explored in more detail, especially when considering the limited number of sessions that were allotted for the rats to learn the contingency and to acquire schedule-induced polydipsia or socially facilitated drinking.

Last, there were some methodological limitations associated with measurement technqiues that are worth noting. Data for all sessions were being recorded using electric contact drinkometers that were connected to computers that recorded drinking tube contacts, intervals with drinking, drinking probability, total and average duration, and total and average latency. Because there was only one drinkometer in each chamber, the computerized data from sessions in which the rats were paired were nonspecific as to which rat was drinking. The experimenter, therefore, took video records of each rat's behavior in the chamber and coded the video records for both the model rat and the naïve rat. At times, it was difficult to determine which rat was drinking by only reviewing the video records because it was often the case that the model and the naïve rat were drinking at the same time or one rat would block the viewers' access to the drinkometer, and it would be difficult to determine which rat was drinking at which point. Using different methods to record data may introduce another source of error in the data collection process that was not acocunted for in this study and may have increased some of the variability noted in the current study (Sidman, 1960).

Future Directions

Research is beginning to demonstrate that polydipsia in rats is a viable animal model of compulsive behavior often observed in those diagnosed with OCD and other psychiatric diagnoses (Moreno & Flores, 2012; Pietrowski, 2005; Pietrowski & Todd, 2004). Future research should replicate the current study. Sidman (1960) reported that replication of a study to

demonstrate its reliability is critical in the evaluation of experimental findings. A systematic replication of the experiment is recommended by specifically extending the baseline, SIP Acquisition Training, Paired, and SIP Acquisition Testing conditions to allow more time for rats to explore their environment, learn about the environmental contingencies that are occurring, and acquire the behavior. Extending the conditions will also allow some of the variability in responding that was observed in the present study to stabilize, providing clearer responses patterns.

Further research should also be conducted to try to identify the necessary and sufficient conditions that elicit this ongoing pattern of responding. The current study demonstrated that social factors may play a role in eliciting compulsive behavior with naïve rats. However, it is also apparent that other variables may play a significant role in a rat's acquisition of these compulsive behaviors or lack thereof. It is likely that compulsive behaviors are acquired by a combination of several factors, with social facilitation being one of them. Compulsive behaviors may be more readily acquired and/or occur at higher rates through a combination of social facilitation and environmental factors (time of day, temperature, light, noise, etc.) or social facilitation and individual factors (neurobiological differences, neuroanatomical differences, baseline rates, etc.). Future research should be conducted to specifically control for and manipulate these variables to determine the effects that they may have on the acquisition of compulsive behavior. In addition, research should also be conducted to determine the degree to which each of these variables increases or decreases the probability of acquiring compulsive behaviors.

Last, Sidman (1960) mentioned that generality of findings is of principal concern when considering the importance of experimental data. Given this issue of generality, translational

research should be conducted to determine if the findings associated with polydipsia can be readily generalized to the human population who have polydipsia or engage in other forms of behaviors that appear to be compulsive in nature. This research would be especially useful in helping researchers and clinicians to better understand and treat these behaviors. In addition, translational research in this field may also allow clinicians to better understand the process through which maladaptive behaviors are acquired and to develop more preventative strategies for individuals who may be at risk for developing these behaviors, rather than focusing on treating these behaviors after they have already developed, become maladaptive, caused distress, and become resistant to extinction.

Clinical Applications

The current study demonstrates that social facilitation may play a role in the acquisition of maladaptive repetitive behaviors, more commonly referred to as compulsions. When considering the results of this study and how it applies to individuals diagnosed with OCD, it appears as though some behaviors are likely to develop or are acquired through the process of social facilitation by others in the environment engaging in the behavior. Given that the study demonstrates that some behaviors may be socially facilitated, it may be useful for clinicians working with individuals engaging in early signs of compulsive behaviors to thoroughly assess social factors that may play a role in the acquisition of the specific behavior. This assessment may include issues related to a number of individuals who engage in this behavior, frequency of social contact with these individuals, and potential reinforcers that the individual may obtain from engaging in the behavior, among other things. This thorough assessment may help clinicians treat early signs of compulsive behaviors in the acquisition phase rather than when the behavior is well established.

In addition to the application of social facilitation on individuals demonstrating early signs of compulsive behavior, it is possible that social facilitation may play a role in other disorders that involve an individual engaging in repetitive maladaptive behavior (e.g. substance use, gambling, etc.). Because social facilitation is not the only mechanism for the acquisition of these behaviors, it may be beneficial for future research to consider the degree to which social facilitation impacts acquisition of maladaptive repetitive behavior and what other processes are necessary for an individual to acquire these behaviors. This information can help guide clinicians to more effective treatment options if they have a better understanding of the mechanisms through which these repetitive maladaptive behaviors are acquired.

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Appendix

APPROVAL NOTIFICATION

EASTERN MICHIGAN UNIVERSITY
Office of Research Development
Institutional Animal Care and Use Committee
Starkweather Hall, 2nd Floor
487-3090

Date: **March 8, 2011**

To: **James Todd**

From: Susan Campbell
ex officio

Eastern Michigan University's Institutional Animal Care and Use Committee (IACUC) has reviewed your *Application To Use Animals In Research or Instruction* referenced below. This project has been approved. The proposed animal use procedures are in compliance with University guidelines, State and Federal regulations and the standards of the "Guide for the Care and Use of Laboratory Animals."

When communicating with the IACUC office, please refer to the Approval Number referenced below. The appropriate Approval Number must accompany all requisitions for animals and pharmaceuticals. No research, testing or instructional use of vertebrate animals may be initiated without an Approval Number.

The Approval Period for your Approval Number is also indicated below. However, the United States Department of Agriculture (USDA) requires an annual review of applications to use animals. Therefore, each year of this application, prior to the anniversary of its approval date, you will receive a short Annual Review Form. Your continued animal use approval is contingent upon the completion and return of this form. You will also be notified prior to the expiration of the Approval Period so that any renewal application can be prepared, submitted and reviewed in a timely manner and an interruption in the approval status of this project avoided.

Committee approval must be obtained prior to changes in procedures that could affect the humane use of animals. If changes are contemplated, a revised Animal Use Form (with the changes highlighted) must be submitted and approved prior to initiation of the modified procedures. Contact this office for more information.

Title: **Social Facilitation of Polydipsia as an Animal Model of Compulsive Behavior**

Approval Period: **03/08/2011 to 03/07/2014**

IACUC Approval No.: **2011-050**

cc: Committee