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IS ETHANOL CONSUMPTION BY RATS REINFORCED BY TENSION REDUCTION OR EVOKED BY RELIEF FROM FEAR?

by

Tara Lynne Fidler

Submitted in partial fulfillment of the requirements

for the degree of Doctor of Philosophy

at

Dalhousie University

Halifax, Nova Scotia

April 14, 1999

DALHOUSIE UNIVERSITY

FACULTY OF GRADUATE STUDIES

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by Tara Lynne Fidler

in partial fulfillment of the requirements for the degree of Doctor of Philosophy.

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Dedicated to the memory of my great uncle,

John George Church

1922-1996

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ABSTRACT

Two hypotheses, tension reduction and relief, have been proposed to explain the mechanism by which ethanol consumption is reinforcing. According to tension reduction, ethanol reduces the tension, fear or anxiety associated with the expected occurrence of an unpleasant event and because this is negatively reinforcing organisms learn to consume ethanol to obtain its tension reducing effects. By contrast, the relief hypothesis suggests that ethanol is reinforcing because it can counteract deficiencies in receptor stimulation which occur after the cessation of aversive events, during periods of relief. In these experiments adult male Sprague Dawley rats had restricted access to ethanol in order to maximize its consumption in temporal proximity to the shock session. When access was restricted to a brief period away from the home cage immediately after (Experiment 1) or before shock sessions (Experiments 2-6) or during extinction (Experiments 7 and 8) consumption data failed to support either hypothesis whether comparisons were made within or between groups. Ethanol consumption did not differ between groups that had access to ethanol contiguous with shock or at the same time on no-shock days (Experiments 1 and 2). Consumption by Ethanol groups was lower than consumption by Sucrose and Water groups before shock sessions (Experiments 3 and 4). Thirst partially accounted for ethanol consumption in Experiment 5. In Experiment 6 there was no evidence for differential consumption of ethanol between periods with and without signals for aversive events. In Experiments 7 and 8, there were no differences between the ethanol and sucrose consumption of Shock and No Shock groups. Because ethanol could have had tension reducing effects (even if these effects were not eliciting consumption) freezing and bolus production were recorded as measures of conditioned fear (in Experiments 5, 7 and 8). Non-specific effects of ethanol could adequately account for differences between groups in these measures.

LIST OF ABBREVIATIONS AND SYMBOLS

ACTH	adrenocorticotropic hormone
ANOVA	Analysis of Variance
cm	centimeter
dB	decibel
FFW	free feeding weight
FI	Fixed Interval
FT	Fixed Time
g	gram
g/kg	grams per kilogram
h	hour
i.p.	intraperitoneal
l	liter
mA	milliampere
mm	millimeter
min	minute
ml	milliliter
ml/kg	milliliters per kilogram
s	second
s.c.	subcutaneous
SEM	Standard Error of the Mean
TRH	Tension Reduction Hypothesis
v/v	volume per volume
VI	Variable Interval
VT	Variable Time
W	watt
w/v	weight per volume

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I have been concentrating on the past but just for a moment I want to make a nod to the future and thank Chris Cunningham. I am looking forward to joining his lab at OHSU in the very near future.

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T.L.F. (April 18, 1999)

INTRODUCTION

Clinicians and researchers have long been interested in determining what factors motivate ethanol (ethyl alcohol, alcohol) consumption. Many of these people have concluded that drinking is a learned response. However this leaves open the question of why? Hullian reinforcement theory (Hull, 1943) assumed that an association between a stimulus and response could only be learned in the presence of some sort of reward or reinforcement. Reinforcement was defined in terms of drive reduction and a drive was a state of tension resulting from an unsatisfied need. Hull's view was that if a response leads to reduction in the strength of a drive then the response should recur the next time a similar situation is encountered (Conger, 1956; Vogel-Sprott, 1972). On the basis of clinical observation, Conger (1956) suggested that anxiety is a drive that would be reduced by ethanol consumption.

Tension Reduction Theory

Tension reduction theory is a direct outgrowth of the application of drive reduction theory to an understanding of how ethanol is reinforcing. An aversive state such as anxiety is conceived of as a drive and reduction of this drive is the reinforcer (Cappell & Greeley, 1987). The theory that ethanol consumption could be reinforced by

tension reduction consists of two (deceptively simple) premises: first, that ethanol reduces tension and second, that organisms drink ethanol to obtain its tension-reducing effects (Cappell & Herman, 1972). The first premise is referred to as the Tension Reduction Hypothesis. According to the second premise the relief from tension provided by alcohol reinforces the drinking response. From an operant or instrumental perspective, organisms will repeat behaviors that are followed by some reinforcment and tension reduction is an example of negative reinforcement (Volpicelli, 1987). So, if consumption of ethanol were followed by tension reduction then organisms would learn to consume ethanol in order to obtain this tension reduction.

The complexity inherent in tension reduction theory only becomes apparent when one tries to design experiments to test it. First of all, it is necessary to create an operational definition of tension reduction. This is difficult because the term "tension" is a generic construct which refers to a number of constructs including fear, anxiety, stress, frustration and arousal, all of which have in common that they denote hypothesized aversive states which could control behavior (Cappell, 1975; Cappell & Herman, 1972; Hodgson, Stockwell & Rankin, 1979). Tension has also been referred to as something that can interfere with performance or well being (Cappell & Greeley, 1987). Much of the difficulty that has been encountered in evaluating tension reduction theory arose from the

fact that there was no universally accepted definition of general concepts like tension and stress (Cappell & Greeley, 1987).

For the purpose of this thesis the terms tension, anxiety, and fear are considered equivalent and are representative of the state that arises from the expected occurrence of an unpleasant event (Hodgson et al., 1979; Wilson, 1988) such as electric shock or restraint. If ethanol reduces tension (as I have just defined it) and organisms can learn to drink ethanol to obtain this effect then it should be possible to show experimentally that: (1) alcohol administration reduces subjective, behavioral and physiological indices of tension and (2) that organisms will increase alcohol drinking when tense (Volpicelli, 1987).

Relief Hypothesis

Tension reduction is not the only mechanism that has been proposed to reinforce ethanol consumption. Relief is another factor that has received attention. The Relief hypothesis has its basis in compensatory opponent-processes. The affective and physiological response following an event is often opposite to the response during the event (Solomon & Corbit, 1974; Volpicelli, 1987). To see how opponent processes could motivate ethanol consumption in response to aversive stimuli it is important to consider separately the response of the organism during the environmental event (the aversive

stimulus) and the response following the event according to Volpicelli (1987). Uncontrollable events (such as electric shocks) increase the release of beta-endorphins so after the cessation of these events there is a decrease in endorphinergic activity and there may be a relative deficiency in opiate receptor stimulation. Ethanol can stimulate opiate receptors and compensate for this deficiency. Since this compensation is reinforcing, organisms can learn to counteract the post-event decrement in endorphinergic activity by consuming more ethanol (Volpicelli, 1987). The same mechanism has been proposed to explain morphine addiction (Volpicelli, 1987). According to this mechanism, ethanol is not reinforcing in anticipation of or during the aversive event but rather is reinforcing after the aversive event has been discontinued, when the organism is in a state of relief. The hypothesized mechanism described here by which a deficiency in endorphinergic stimulation following the cessation of an uncontrollable event motivates the consumption of ethanol might apply equally well to any of a number of other systems (such as norepinephrine, corticosterone, dopamine (Patel & Pohorecky, 1988), or ACTH (Sprague & Maickel, 1994)), that are affected by both uncontrollable events and ethanol. In any case, according to the relief hypothesis ethanol does not reduce tension, but rather tension reduction (relief) elicits ethanol consumption (Volpicelli, 1987).

According to the relief hypothesis, the chain of events by which ethanol can become reinforcing goes as follows. First, uncontrollable aversive events stimulate receptor activity (in some pathway). Following the termination of uncontrollable events, when relief is experienced, a relative deficiency in receptor activity exists. Next, ethanol can stimulate receptor activity. Finally organisms learn to compensate for deficiencies in receptor activity by increasing ethanol consumption (Volpicelli, 1987). The relief hypothesis would be supported if increased ethanol consumption occurred only after the cessation of the aversive event.

Non-specific effects of ethanol

Before invoking tension reduction or relief to explain patterns of ethanol self-administration, it would be necessary to eliminate other non-specific drug effects. For instance, if ethanol had locomotor or analgesic effects these would have to be ruled out before changes in responding were attributed to tension reduction. Similarly, the possibility that ethanol was being consumed for its caloric value would have to be eliminated before attributing increased ethanol consumption to its value as a reinforcer. Effects of ethanol on gastric motility in non-fearful animals would have to be assessed before reduced defecation by fearful animals following ethanol administration could be attributed to tension reduction.

Three non-specific effects of ethanol on locomotor behaviour have been identified. First, ethanol impairs motor behaviour at high doses. Second, at low to moderate doses ethanol enhances motor performance and this enhancement may be more evident after exposure to shock. Third, strong fear can inhibit the ethanol-induced arousal (Cunningham & Brown, 1983). Ethanol's effects on locomotor activity have been investigated in a number of different ways. Results have shown these effects to be strain and species-specific as well as dose-dependent. For example, a 2.0 g/kg i.p. dose of ethanol stimulated locomotor activity in DBA/2J mice beginning within the first few minutes after injection and lasting for at least 30 minutes. This elevation in locomotor activity increased across trials (Cunningham & Prather, 1992). In another study, the same 2.0 g/kg i.p. dose of ethanol increased locomotor activity by DBA/2J and CD-1 mice but not by C57BL/6J mice (Frye & Breese, 1981). It took a larger dose of ethanol (3.0 g/kg) via the oral route to produce the same locomotor stimulation (Frye & Breese, 1981). An even higher dose (4.0 g/kg i.p.) suppressed activity in all three mouse strains that were tested (Frye & Breese, 1981). In unstressed mice locomotor activity was generally stimulated by low doses of ethanol and suppressed by high doses of ethanol.

The effect of ethanol on the locomotor behaviour of rats varied widely between studies and different activity measures were differentially affected by ethanol

(Dalrymple-Alford & Benton, 1981). Low doses of ethanol (0.25-1.0 g/kg i.p.) had no effect on the locomotor activity of alcohol-preferring or alcohol-nonpreferring rats (Stewart, Gatto, Lumeng, Li & Murphy, 1993). A wider range of doses (0.1 to 3.0 g/kg i.p.) either had no effect or decreased the locomotor activity (during a 1 hour session immediately following injection) of adult male Sprague Dawley rats in a circular runway (Frye & Breese, 1981). In another report, doses from 1.0 to 2.0 g/kg i.p. ethanol had no effect on the locomotor activity of Sprague Dawley rats (Erickson & Kochhar, 1985). Motor activity of Sprague Dawley rats was depressed by a 1.5 g/kg i.p. dose of ethanol (Brick, Pohorecky, Faulkner & Adams, 1984). A 0.5 g/kg i.p. dose of ethanol had no effect on motor activity (relative to saline-injected controls) except in the middle of the night (dark phase of the light/dark cycle) when ethanol enhanced motor activity (Brick et al., 1984). Long Evans rats injected with 2.0 g/kg but not 1.0 g/kg i.p. ethanol 10 minutes prior to an open-field test were less active than water injected controls during both the first and second five minute blocks of the test (Aragon, Spivak & Amit, 1989). A 1.5 g/kg i.p. injection of ethanol suppressed activity in Long Evans rats (Trudeau, Aragon & Amit, 1990). Long Evans rats that consumed 0.7 g/kg ethanol during the 10 minutes prior to testing showed enhanced locomotor and exploratory behaviour (Gill, France & Amit, 1986). In some cases, effects of ethanol on locomotor behaviour emerged only following

repeated administrations of ethanol. Beginning on day 3 spontaneous activity was suppressed relative to baseline by repeated daily doses of 4.0 g/kg ethanol administered by stomach gauge in Long Evans rats (Alvarez, Prunell & Boada, 1998). By contrast, the same dose of ethanol increased activity counts in the open field beginning on day 3 (Alvarez et al., 1998). A daily 2.0 g/kg dose of ethanol by stomach gauge suppressed spontaneous activity beginning on day 9 relative to day 3 (Alvarez et al., 1998). Ethanol's effects on the locomotor activity of unstressed rats appeared to vary not only across strains but also across experiments within strains.

The analgesic effects of ethanol have not been widely investigated. However, there is a suggestion that ethanol can reduce sensitivity to shock in a dose-dependent manner (Cunningham & Brown, 1983). A 2.0 g/kg i.p. dose of ethanol had an analgesic effect in Sprague Dawley rats when tail-lifting response, escape behaviour, and vocalization were used as measures of aversive threshold to footshock and tailshock. All three measures were elevated 30 minutes after the ethanol injection (Brick, Sun, Davis & Pohorecky, 1976). The analgesic properties of ethanol are important to keep in mind since many of the stressors, such as electric shock, that have been used in the investigation of the tension reduction and relief hypotheses are painful.

Ethanol is high in calories so in some situations it may be consumed for its food value rather than for any of its other effects (Cunningham & Niehus, 1997). Arguing against this possibility are the findings from a microstructural analysis of consummatory behaviours: food, ethanol and water (Gill, Amit & Smith, 1996). This analysis revealed no association between total food intake and total ethanol intake in 70 male Long Evans rats that consumed between 1-5 g/kg/day of ethanol (Gill et al., 1996). Ingestion of food was not reduced to compensate for the extra calories ingested as ethanol. Meal size did not change whether the meal was accompanied by a bout of water or of ethanol consumption. Postprandial bouts of ethanol consumption were larger than all types of water drinking bouts. The absence of trade-offs between calories from food and ethanol in this study should not be interpreted as evidence that the rats could not shift their caloric intake to food or ethanol in the absence of the other.

The relationship between ethanol consumption and food deprivation has been examined (Stiglick & Woodworth, 1984). Although all groups preferred water to ethanol, ethanol preference and absolute consumption of ethanol were higher in the food-restricted than in the non-restricted groups at all concentrations of ethanol. For the food-restricted groups consumption of absolute ethanol increased sharply as concentration increased (from 8 to 16 to 32%). The greater ethanol consumption by food-restricted than

non-restricted groups is consistent with the claim that the food-restricted rats used ethanol as a source of calories. The different volumes of absolute ethanol consumption at the three concentrations of ethanol might have represented limitations due to fluid volume. In order to maintain the caloric intake, four times more 8% ethanol than 32% ethanol would have to have been consumed (Stiglick & Woodworth, 1984). There may be other differences between food-restricted and non-restricted groups besides differences in body weight and level of hunger that contribute to the different patterns of ethanol consumption observed in the two groups. Food restriction could have resulted in endocrine or neurophysiological changes that affected ethanol preference by some mechanism related to the pharmacological effects of ethanol.

The combination of food deprivation and access to a running wheel results in activity-maintained self-starvation or activity anorexia—running activity increases as weight decreases. In this experiment the survival rate of Sprague Dawley rats with access to a running wheel was examined under different levels of food deprivation and fluid access (Spigelman, McLeod & Rockman, 1991). Rats had free access to either 7% ethanol, 7% propylene glycol, or just water. All of the animals in the ad lib food groups survived to the end of the experiment (that is, they never dropped below 70% of their FFW). Food-deprived rats did more wheel running than the ad lib food groups. Among

the food-deprived groups the propylene glycol group showed a better survival rate and lower activity level than either the ethanol or water groups. Ethanol consumption was unaffected by level of food deprivation. By contrast, consumption of propylene glycol was significantly greater by rats that were food deprived than by rats that were non-deprived. It would appear that the rats were able to use propylene glycol for its nutritive value but that ethanol was not utilized in that way.

With regard to studies which have investigated the ethanol consumption of stressed animals it has been suggested that stress may alter the responsivity to the taste of ethanol (Cappell, 1975) so that increases in consumption could be a function of diminished sensitivity to taste and smell (Dole & Gentry, 1984). Supporting the idea that palatability of ethanol solutions is a factor in ethanol consumption is the finding that intake of ethanol solutions was increased by the addition of sweetening agents such as sucrose and saccharin. These increases could be obtained even when the baseline consumption of ethanol was already high (Meisch, 1984; Samson & Falk, 1974).

Defecation rate or rate of bolus production in certain situations has been used as a measure of conditioned fear (Inoue, Koyama & Yamashita, 1993). Before using this measure in studies involving ethanol it was necessary to examine whether ethanol had non-specific effects on gastrointestinal motility. No significant correlation between

ethanol consumption and defecation was observed for Sprague Dawley rats (Tobach, 1957) or across Alcohol-Preferring, Alcohol-Accepting, Fawn-Hooded, Alcohol-Nonpreferring, Alcohol-Nonaccepting, Flinders Resistant Line, Maudsley-Reactive and Maudsley-Nonreactive strains (Badishtov et al., 1995) although there was some suggestion that rats with high defecation rates consumed less ethanol. In chronic alcoholics or healthy nonalcoholics no correlation has been reported between the rate of gastric emptying and the amount of ethanol intake, age, or gastrointestinal symptoms prior to or at the time of the test (Keshavarzian, Iber, Greer & Wobbleton, 1986).

In spite of the absence of observed correlations between ethanol consumption and defecation there is some evidence that ethanol administration interfered with gastric motility and emptying in dogs and rats (Keshavarzian et al., 1986). Both acute and chronic ethanol consumption interfered with stomach functioning. Even small doses of ethanol have been shown to interfere with gastric and intestinal motility. Ethanol interfered with the activity of the muscles surrounding the stomach and the small intestine and thus altered the transit time of food through these organs (Bode & Bode, 1997). In humans, beverages with high ethanol concentrations (> 15%) appeared to inhibit gastric motility and delayed the emptying of the stomach (increased gastric transit time) (Bode & Bode, 1997). In the large intestine (of dogs) ethanol reduced the transit

time and compaction of the intestinal contents and caused diarrhea (Bode & Bode, 1997). Unfortunately, none of these experiments refer to the exact timing of effects (either increased or decreased gastric transit time) relative to when ethanol was administered.

Ethanol has been shown to affect locomotor behaviour, sensitivity to painful stimuli, caloric intake, and gastric motility. It is important to be aware of these non-specific effects so that they will not be misinterpreted and/or misattributed as specific treatment effects. The interference between non-specific and specific effects can be minimized by the inclusion of appropriate control groups. For example, inclusion of unstressed groups consuming ethanol and/or stressed and unstressed control groups consuming an isocaloric solution would allow the effects of ethanol and stress to be untangled. The inclusion of multiple behavioural measures, which might be differentially affected either by the stressor or by ethanol, is another way to prevent misinterpreting non-specific effects of ethanol as being the result of tension reduction or relief.

Does ethanol reduce tension?

The first premise of the tension reduction hypothesis has been assessed by administering ethanol to animals that were experiencing or anticipating an aversive situation and observing their behaviour. If ethanol reduces tension then behaviours associated with fear or anxiety should be reduced under the influence of (the

experimenter-administered) ethanol. The question of whether ethanol reduces tension has been addressed using a number of different paradigms including escape and avoidance, conditioned suppression, extinction and the partial reinforcement extinction effect. All of these paradigms have in common the fact that they are indirect measures of fear or anxiety. Performance of an operant response (such as a bar press) is used to infer a Pavlovian state (such as fear). The evidence from these paradigms has been reviewed elsewhere and the conclusion drawn from these reviews is generally that in some situations ethanol can reduce fear or tension (even after non-specific drug effects have been ruled out) (Cappell, 1975; Cappell, 1987; Cappell & Greeley, 1987; Cappell & Herman, 1972; Hodgson et al., 1979) and may even block biochemical responses to stress at some doses (Cappell, 1987; Cappell & Greeley, 1987). Experiments employing passive avoidance or approach-avoidance conflict have provided the most consistent support for the tension reduction hypothesis.

Avoidance is one example of a behaviour believed to be controlled by fear or anxiety (Cappell, 1975; Cappell & Herman, 1972; Mowrer, 1960). Disruption of avoidance performance following administration of ethanol would be consistent with tension reduction, although the influence of motor impairment or enhancement due to non-specific drug effects would have to be considered. The rate of extinction of an

avoidance response is considered to be an inverse function of the anxiety that was generated during avoidance training. Therefore, any drug that reduced anxiety would be expected to hasten extinction (Amit & Brown, 1982). Alternatively, if ethanol inhibits fear and fear must be evoked in order for it to extinguish, then ethanol might be expected to increase resistance to extinction of a fear-based response (Cappell & Herman, 1972). In fact, there have been reports of disrupted avoidance and enhanced avoidance with ethanol. Many of these effects could be accounted for in terms of nonspecific sensory or motor effects of ethanol (Cappell & Herman, 1972). It is not the case that ethanol should be expected to have effects in all experiments which involve noxious stimuli. For example, escape responses are elicited by unconditioned stimuli (like electric shocks) rather than by conditioned stimuli (like tones) and therefore should not be affected by any tension-reducing effect of ethanol (Hodgson et al., 1979). Where controversy exists about the tension reducing properties of ethanol it exists because the factors eliciting a response (conditioned versus unconditioned stimuli) were not adequately examined (Hodgson et al., 1979).

Temporal pattern of ethanol consumption and relation to blood and brain ethanol concentration

Before changes in ethanol self-administration (via oral consumption) could be assessed the pattern of ethanol consumption in the absence of stressors had to be identified. In this section I have documented the pattern of ethanol consumption when light cycle, ethanol concentration, and the availability of fluid choices were varied. Further, where possible I have linked the temporal distribution of drinking to the resulting blood and brain ethanol concentrations.

Alko Alcohol (Alcohol-Accepting) rats with continuous access to 10% ethanol and water consumed 5.33 g/kg/day of absolute ethanol (Aalto, 1986). Peak ethanol consumption occurred at the onset, mid-point and offset of the dark phase of the 12-h/12-h light/dark cycle. Water consumption was roughly parallel to ethanol consumption. Ethanol consumption by Alko Non-Alcohol rats was uniformly low (0.95 g/kg/day) (Aalto, 1986).

When Long Evans rats had free access to both ethanol (2-10 % v/v) and water they consumed most of their daily food and fluid during the dark phase of a 12-h/12-h light/dark cycle (Boyle, Smith & Amit, 1997; Gill et al., 1986). One bout of ethanol consumption occurred immediately after lights out and there were six discrete bouts of

ethanol consumption per day. Each drinking bout was characterized by a rapid rate of ingestion (0.62 g/kg/bout during 1.8 minutes). Regardless of ethanol concentration, bout duration remained very short in all animals (Gill et al., 1986). Detectable brain levels of ethanol were reported approximately 4 minutes following gastric intubation in Wistar rats with maximal levels occurring at 30 minutes (Nurmi, Kiianmaa & Sinclair, 1994).

Group housed (24 rats per complex enclosure) and food deprived Long Evans rats showed two peaks in ethanol consumption. One peak occurred just before access to food (one hour in the middle of the dark phase) and another occurred during the hours just before the onset of the light phase (Ellison, 1987). By contrast, water consumption increased gradually from the onset of the dark phase until the hour of feeding and peaked during the next six hours before dropping off rapidly with the onset of the light phase.

Male Wistar rats with continuous access to water, 5%, 10% and 20% ethanol showed two to four peaks of ethanol intake during the night and one weaker peak during the daytime that was correlated with the time when the rats were disturbed by weighing and replacing food and bottles. Total fluid intake showed a very similar time course (Wolffgramm, 1990). Wise (1973) showed that preference for a 20% ethanol solution was highest in Wistar rats that had access to ethanol on alternate days. Rats that had

continuous access to ethanol consumed little ethanol and maintained a strong preference for water (Wise, 1973).

Holtzman rats with continuous access to water and 7.5% ethanol showed a sharp increase in both ethanol and water consumption with the onset of the dark phase (Hatton & Vieth, 1974). This heightened consumption persisted throughout the 10 hours of darkness. Fluid consumption was negligible during the light phase. When stressed groups drank during the light phase this consumption did not diminish intake in the dark.

Male mice of the C57BL/6J strain showed a pronounced preference for ethanol, with ethanol preference ratios (between 10% ethanol and water) of 0.60-0.90. Mean daily intake for these mice was approximately 5 g/kg/day with 4 times more consumption occurring during the dark phase of the light/dark cycle (Dole & Gentry, 1984). During the dark phase, the blood ethanol concentration reached pharmacologically significant levels for transient periods. In another experiment with C57BL/6J mice, the mice reportedly consumed 6.0 g/kg/day (Millard & Dole, 1983).

The pattern of fluid consumption was consistent across all of the animals tested. When the time course of fluid consumption was monitored in rats that had fluid continuously available, consumption was highest during the dark phase of the light/dark cycle (Aalto, 1986; Boyle et al., 1997; Ellison, 1987; Gill et al., 1986; Hatton & Vieth,

1974; Wolffgramm, 1990). A weak peak in ethanol consumption was observed during the light phase in response to some disturbance (handling or a stressor) (Hatton & Vieth, 1974; Wolffgramm, 1990). Although the animals achieved intoxication at certain times it was not sustained (Samson & Grant, 1990).

When access to ethanol was restricted to 10-min/day (with food and water available the rest of the time) Long Evans rats consumed a volume of ethanol comparable to that consumed during a single bout in animals given free access. Under the limited access paradigm, the rats began to drink immediately and continued to drink for 2-4 minutes, achieving consumption of 0.69-1.6 g/kg of absolute ethanol. Rapid oral ingestion of small quantities of ethanol resulted in detectable levels of blood and brain ethanol (Gill et al., 1986). Thus, non-deprived animals with limited access to ethanol consumed considerable quantities, even during the day, a time when they would not normally feed or drink. There was a significant positive correlation between ethanol consumption during 24-h access and during 10-min access when the same animals were tested under both conditions (Boyle, Smith, Spivak & Amit, 1994).

When access to ethanol was extended from 10 minutes to a period between 30 and 180 minutes (with ad lib access to food and water at other times) most consumption occurred during the first 10-15 minutes (Samson & Grant, 1990). The animals drank

rapidly and steadily at the onset of the session and stopped drinking after blood ethanol levels reached a predictable level, regardless of the concentration of ethanol.

Animals displayed persistent and stable intake of ethanol over long periods of time, both in 24-h free choice paradigms and during limited access schedules. This persistence and the similarity of bout size between the two schedules are indicative that there is some mechanism that limited the size and duration of individual drinking bouts (Gill et al., 1986). Wolffgramm (1991) reported that after six months of relatively stable ethanol intake Wistar rats gradually increased their ethanol consumption (in g/kg) from week to week even though environmental conditions remained the same. Even when ethanol was reintroduced after a nine-month abstinence period the rats showed high levels of consumption (approximately 4 g/kg/day). Age-matched controls with no ethanol experience consumed approximately half as much ethanol (Wolffgramm, 1991).

Rouhani et al. (1990) cited evidence from Marfaing-Jallat (1978) and Segovia-Riquelme, Vitale, Hegsted &Mardones (1956) that the maximum rate of ethanol metabolism for rats is approximately 0.3-0.4 g/kg/h or 8.0 g/kg/day. Eckardt (1975) placed the elimination rate at 7.0 g/kg/day. Consumption (or administration) of ethanol in excess of these amounts would produce chronic intoxication. Long Evans rats with continuous access to a 12% ethanol solution as the sole fluid choice consumed between 4

and 7 g/kg/day (Daoust et al., 1987). This amount appeared to be constrained by the ethanol elimination rate since total fluid consumption increased when water was available simultaneously. For rats that preferred ethanol as 60% or more of their total fluid intake during a two-week screening, ethanol consumption remained high (approximately 5 g/kg/day) when water was also available. Rats that chose ethanol as less than 20% of their total fluid intake during a two-week screening dramatically decreased their ethanol consumption (to less than 1 g/kg/day) when another fluid choice (water) was available (Daoust et al., 1987). Rate of ethanol elimination from the blood did not differ between the two groups of rats.

The rate of ethanol absorption from the gastrointestinal tract depends on the rate of ethanol consumption and on the stomach contents. The time to reach peak blood ethanol level may be shorter and the peak level higher if ethanol is consumed quickly as opposed to being distributed over a period of hours. When food was restricted prior to ethanol consumption, peak blood ethanol concentration occurred sooner (Pohorecky & Brick, 1988). When gastric emptying was slow (such as when the stomach was full) the absorption of ethanol was delayed and peak blood ethanol concentrations were reduced (Holt, 1981) since ethanol is absorbed slowly from the stomach and quickly from the small intestine. Pohorecky and Brick reviewed evidence that suggested that if ethanol

was consumed at low concentrations it would be absorbed more slowly due to the effect of the greater volume on gastric emptying time.

Impact of stressors on alcohol consumption

Research linking experimentally induced stress with changes in ethanol consumption and/or preference has employed a number of different experimental paradigms. Stressors have included social variables (such as isolation or crowding), psychological variables (such as conflict), and painful stimuli (such as signaled and unsignaled inescapable electric shocks). In addition to using different stressors, studies have varied in terms of when and where ethanol was available, what ethanol solution was available, and whether other fluid choices were available. The patterns of ethanol consumption that have emerged from these experiments varied, as did the psychobiological mechanisms invoked to explain the patterns. For the purposes of this review, I have sorted the literature according to whether there were discriminable safety periods in which the animals could drink and whether these safety periods were temporally or contextually discriminable. This sorting scheme should allow for distinctions between tension reduction and relief. In experiments that did not include discriminable safety periods, there could have been no relief. Animals would have

experienced chronic fear. By contrast, inclusion of discriminable safety periods would have established periods of relief and periods of acute fear.

Experiments including no discriminable safety period

Experiments including no discriminable safety period are ones in which the animals were housed in the same environment in which stress was administered. This includes experiments in which the housing conditions constituted the stressor, as was the case in experiments involving crowding. Also included in this group are experiments in which a discrete stressor such as electric shock was presented around the clock. In either of these situations, the animals were consuming ethanol in the presence of the stressor. Although the stressor may not have been administered continuously (as was the case with electric shock), the schedule (for example non-contingent variable time or response-contingent variable interval) was such that the offset of one stimulus did not signal a predictable duration of safety before the next stimulus.

In conflict experiments, approach responses produce some positive outcomes along with a negative outcome, while not performing the approach response allows some negative outcome to be avoided. In one such series of experiments (Anisman & Waller, 1974, Experiments 3-5) half of the floor of the operant chamber could be electrified. The floor was arranged so that conflict groups had to cross to the electrified half of the floor

in order to drink. This floor arrangement was reversed for non-conflict groups. Non-conflict groups could avoid shock simply by remaining near the drinking spouts. Water and ethanol were continuously available. In experiments using an increasing ethanol concentration (from 2-10%) and with the floor electrified during a random 20% of every hour neither Holtzman nor Sprague Dawley rats changed their ethanol consumption systematically. Stronger conflict was elicited by presenting shock during 80% of each hour. The conflict group consumed less of both ethanol and water than non-conflict and no-shock groups. Ethanol consumption by the conflict groups was similar whether conflict was weak or strong. Water consumption was suppressed when conflict was strong. In another strong conflict experiment a 3% ethanol solution was available throughout (instead of an increasing ethanol concentration). The non-conflict group consumed more ethanol than the conflict or no-shock groups. Conflict did not increase ethanol consumption in any of these experiments although the relative preference for ethanol was much higher when ethanol concentration was higher and conflict was strong. In each of these experiments, the rats had control over the shock in the sense that they had the choice between crossing and not crossing into the shocked side of the box. This choice or control might have served to reduce tension so that the rats did not need to use ethanol for this purpose. Data indicating the relative activity levels and the relative

success at avoiding shocks would have helped to identify what coping strategies the conflict and no-conflict groups used. The conflict groups, especially the strong conflict group, would have been punished for drinking at least some of the time. That is, shock would have occurred at the instant when they were drinking. No data were presented about the number or duration of drinking bouts so it is impossible to determine if the rats ever consumed enough ethanol at one time to experience any post-ingestive effects.

Several experiments have investigated whether ethanol consumption varied with social rank. Dominant animals control access to food, water, mates, and territory. The lack of control experienced by the subordinate animals has been associated with high levels of anxiety (Chorpita & Barlow, 1998). In order to reduce this anxiety, subordinate animals were expected to consume more ethanol than dominant ones. Within colonies of five male and three female Long Evans rats, food, water and ethanol were available continuously and ethanol consumption by individual animals was monitored (Blanchard, Hori, Tom & Blanchard, 1987). Bout frequency and lick frequency were both higher for subordinate rats than for dominant rats. It is possible that subordinate rats felt more anxiety than dominant rats and consumed ethanol to mitigate this anxiety. Ethanol consumption might have been more rewarding or less aversive to the subordinate rats than to the dominant rats. Food and water consumption were not monitored but it is

possible that the dominant males controlled access to these commodities (which were located in a different place than the ethanol bottle) and that subordinates consumed more ethanol to compensate for relative deficiencies in food or water. At low (4%) but not at high (8%) ethanol concentrations females consumed more ethanol than males. After the death of the dominant male and two other males (in one colony) the females increased their ethanol consumption. This increase, at least, could not be explained in terms of the dominant rat preventing access to food and water.

In another experiment investigating the interaction between social rank and ethanol consumption (Ellison, 1987), two colonies were raised for 6 months with continuous access to water and 10% ethanol flavoured with 0.05% anise. Food access was restricted to one hour per day. The 10 highest and 10 lowest ethanol consumers were removed from these colonies and placed into a new colony. In the three hours before the onset of the dark phase the high ethanol consumers spent 2.75 times longer at the ethanol drinking spout than the low ethanol consumers. The high ethanol consumers ate less food, were less active, and were lower on all measures of dominance than the low ethanol consumers. However, it was not clear whether the high ethanol consumers consumed less food because they were prevented from eating more (by the dominant, low ethanol consumers) or because they did not need calories from food since they were getting them

from ethanol. Ellison concluded that the high ethanol consumers had subordinate status but it was not clear whether the low dominance rank caused the high ethanol consumption or vice versa. These extremes of ethanol consumption observed in colonized or previously colonized rats were not observed in animals that had always been isolated (Ellison, 1987).

There was a consistent negative correlation between ethanol consumption and social rank (Blanchard et al., 1987; Ellison, 1987) in experiments using different methodologies and different ethanol solutions. Subordinate rats were perceived to be more anxious than dominant ones since the dominant rats were in control of resources within the colony. Subordinate rats consumed more ethanol than the dominant animals. This pattern of consumption was consistent with the subordinate animals consuming ethanol for tension reduction but these experiments do not constitute a strong test of the hypothesis since anxiety was only inferred. Isolation and overcrowding are additional stressors relating to housing condition and their role in ethanol consumption has also been investigated.

Heminway and Furomoto (1972) investigated whether population density would influence consumption of ethanol by albino rats. Population density was varied with respect to the amount of floor space per animal with the number of animals per cage held

constant across three population densities: undercrowded, moderately crowded, and overcrowded. Rats in the moderately crowded cage consumed more ethanol than rats in either of the other crowding conditions (which did not differ from each other). Rats in the overcrowded and moderately crowded cages in which ethanol was available consumed less total fluid than did rats in the corresponding cages with only water available. Among the cages with only water available, rats in the overcrowded cage consumed the most water, perhaps because of the higher temperature within this cage (due to the crowding) (Heminway & Furomoto, 1972). There are several methodological limitations of this study. There were only two cages (n = 12 per cage) for each housing condition, one in which water was the only fluid available and one in which both water and ethanol were available. This amounts to n = 1 per group so the generality of this result must be viewed very cautiously. Consumption by individual animals was not measured so it was not possible to determine whether social rank affected ethanol consumption or whether this effect was equivalent across crowding conditions. The rats were exposed to the same housing conditions throughout the experiment so there was neither a pre-stress nor a post-stress phase for comparison purposes.

Rodgers and Thiessen (1964) varied population density by holding the floor space per mouse constant but varying the number of mice per cage. Mice of the C3H/Crgl/2

strain were housed individually, or in groups of five or ten mice. For six weeks the mice had ad lib access to food and water. Mice in the various housing conditions were compared as though there were ten independent individuals in each group (in spite of the fact that individual consumption data was not available for grouped animals). Consumption of 10% ethanol did not differ between groups when it was the only fluid available or when there was a choice between ethanol and water. Under choice conditions, fluid intake per individual decreased as population density increased. This difference was accounted for by differences in water consumption. The absence of differences in ethanol consumption by animals in different housing conditions could indicate that none of these housing conditions constituted an effective stressor. Differences in consumption patterns between this experiment (Rodgers & Thiessen, 1964) and the previous one (Heminway & Furomoto, 1972) might have reflected differences in experimental design (floor space held constant versus number of animals per cage held constant) or differences between species (mice versus rats).

There is some evidence that isolation is a more potent stressor for rats than for other rodents like gerbils or guinea pigs (Einon, Humphreys, Chivers, Field & Naylor, 1980). Given the opportunity 30-day-old rats spent about 17% of their time engaged in "play". Given that rats of this age slept more than 30% of the time, play time constituted

a significant portion of the rats' waking hours. Gerbils spent a comparable percentage of their time sleeping but only 1% of their time playing. Guinea pigs were never observed playing. Since guinea pigs and gerbils engaged in less play and less social contact than rats, they would have been deprived of less social contact when they were isolated (Einon et al., 1980). Unfortunately mice were not included in this study.

Another experiment compared the ethanol consumption by Long Evans rats that were housed six to a cage to that of rats that were housed individually (Deatherage, 1972). The individually housed rats had almost twice as much floor-space per rat as the group housed rats. Housing condition did not affect consumption when water was the only fluid available or when 10% ethanol was the only fluid available. However, when 20% ethanol was the only fluid available the individually housed rats consumed more ethanol than the group housed rats. The individually housed rats may have consumed more of the 20% ethanol in order to reduce anxiety associated with social isolation. However, if this were the case, it is not clear why there was no similar difference between the 10% ethanol consumption of individually housed and group housed rats (Deatherage, 1972). Wistar rats housed individually for 17 weeks showed a higher preference for 10% ethanol than group housed rats. Total drinking volume was also higher in isolated rats compared with group housed rats (Roske, Baeger, Frenzel & Oehme, 1994).

The ethanol consumption of Long Evans rats in a naturalistic colony, a group cage, or individual cages was compared in order to determine whether deprivation and isolation were stressors that would influence ethanol consumption (Kulkosky, Zellner, Hyson & Riley, 1980). The rats housed in the naturalistic colony consumed less 10% ethanol (whether it was sweetened or not) than the other groups. Kulkosky et al. concluded that both crowding (in the group cage) and isolation were effective stressors and that this stress was responsible for the increased ethanol consumption by these groups.

The roles of social behaviour, dominance and social deprivation in determining ethanol consumption have been examined simultaneously (Wolffgramm & Heyne, 1991). Male Wistar rats housed individually, in contact cages, or in groups of four had simultaneous access to water, 5%, 10%, and 20% (v/v) ethanol. Contact cages were group cages that had been divided into four compartments with vertical bars thereby allowing the four rats to communicate with each other. Both short-term and long-term social isolation enhanced the consumption of ethanol, especially at their high concentration (20%). Group housed rats maintained a preference for 5% ethanol. The group housed rats drank small quantities of ethanol but distributed their drinking over the whole period. When these animals were isolated they shifted their preference to the 20%

ethanol solution and increased their consumption by 38%. Individually housed rats shifted their preference from 5-20% over the first week and a half. These animals had a long single bout of consumption followed by several hours with no ethanol consumption. Rather than inducing the motor stimulation displayed by the group housed rats, ethanol had a sedative effect on the individually housed rats (likely due to higher peak blood ethanol levels resulting from the large versus small bouts of consumption (Pohorecky & Brick, 1988)). When switched from individual to group housing, consumption decreased by 23%. The level of ethanol consumption by rats housed in contact cages was similar to that of group housed rats but the preferred concentration and temporal pattern of intake resembled that of the individually housed rats (Wolffgramm, 1990; Wolffgramm, 1991). The increased ethanol consumption by isolated rats could indicate that ethanol was an effective reinforcer in this situation, perhaps because ethanol reduced tension.

In addition to effects of housing condition, there were also effects of social rank on ethanol consumption. When short term isolation was imposed on subordinate rats, they maintained their high levels of ethanol consumption. By comparison, dominant rats increased their ethanol consumption only during social isolation (Wolffgramm, 1991). The negative correlation between social dominance and ethanol consumption has been observed in a number of experiments (Blanchard et al., 1987; Ellison, 1987;

Wolffgramm, 1991; Wolffgramm & Heyne, 1991). Since this relationship held for experiments in which social rank was measured prior to the introduction of ethanol (Blanchard et al., 1987; Wolffgramm & Heyne, 1991) there is evidence that subordinate status preceded high ethanol consumption rather than high ethanol consumption causing subordinate status.

Behavioural changes have been observed in isolated animals that are consistent with the belief that isolation is a stressor. After 13 weeks of isolation rats were reported to be nervous and aggressive (Hatch, Wiberg, Balazs & Grice, 1963). However, effects on ethanol consumption varied with the age at which isolation was imposed. Long Evans rats that were isolated at weaning consumed more ethanol than rats that were housed in groups of four. Rats that were isolated beginning at 65 days of age did not consume more ethanol than group housed rats (Schenk, Gorman & Amit, 1990). Social deprivation may have been more stressful for immature rats. Individually housed Long Evans rats consumed more ethanol over 80 days than did each of 36 rats housed in one colony (Ellison, Daniel & Zoraster, 1979).

The experiments which varied housing condition seemed to be successful at inducing chronic anxiety (at least in the subordinate animals). Chronic anxiety could also be induced by housing animals in an environment in which inescapable, and unavoidable

aversive stimuli such as electric shocks were presented at unpredictable intervals. Such experiments enjoy an analytic advantage over those relying on social factors in that the exact nature, duration, frequency, and intensity of the anxiety-inducing stimulus can be defined and controlled. Those experiments, which presented experimenter-controlled anxiety-inducing stimuli at unpredictable intervals with no discriminable safety periods, are described next.

Male Sprague Dawley rats were given continuous access to both 16% (v/v) ethanol and water throughout baseline, shock and post-shock phases. No shocks were administered during the 16-day baseline or 32-day post-shock phases. During the 16-day shock phase, unsignaled footshocks were administered on a variable time (VT) 11.3 minute schedule 24 hours per day (Casey, 1960). During the baseline phase, ethanol consumption accounted for 50% of fluid consumption. Ethanol consumption increased over the course of the experiment and peaked at 70% of consumption on the sixteenth post-shock day. After this, ethanol consumption decreased, returning to baseline levels by 32 days post-shock (Casey, 1960). The initial increase in ethanol consumption following the cessation of shock was consistent with the relief hypothesis—it was not shock per se that led to ethanol consumption, but rather the relief elicited by the cessation of shock that led to ethanol consumption (Volpicelli, 1987). The subsequent decrease in ethanol

consumption might have been due to diminishing effectiveness of relief as a reinforcer over time. Ethanol consumption decreased because it was no longer reinforcing. There are other possible explanations. First, the rats lost weight during the shock phase and it is possible that they used ethanol as a supplemental source of calories during their recovery from stress (Champagne & Kirouac, 1989). Comparing the consumption of ethanol to that of an isocaloric solution would have helped in assessing the extent to which ethanol's caloric value was responsible for the increase in consumption. Increased ethanol consumption during the post-shock phase could have been the result of the normal tendency for animals to increase their ethanol intake over time (Choca, Wilson & Garcia, 1977) although if this were the case ethanol consumption should have remained high. This was not what happened. Without the inclusion of a no-shock group it is impossible to determine whether the shock and post-shock phases altered ethanol consumption. In any case, the result was not consistent with a tension reduction interpretation, since consumption continued to increase for 16 days after the shocks were discontinued.

Myers and Holman (1967) included a shock group similar to that of Casey (1960). Wistar rats were acclimated to increasing concentrations of ethanol (from 2-20%). During the shock phase, water and 12% (v/v) ethanol were continuously available and shocks were administered on a VT 3-19 minute schedule (mean interval not reported). As in

Casey's experiment the period of relative safety between shocks was both unpredictable and short. Ethanol consumption did not differ between this shock group and a similarly acclimated no-shock group. Neither group showed any change in ethanol consumption during the shock phase and no post-shock phase was included in this experiment. In spite of similarities between the shock schedules in the two experiments (Casey, 1960; Myers & Holman, 1967) the observed patterns of ethanol consumption were different. The different patterns of consumption might have reflected differences between Sprague Dawley and Wistar rats, between the ethanol solutions (16% versus 12%), or between acclimation (Myers & Holman, 1967) and no-acclimation (Casey, 1960) procedures.

The results from experiments employing various stressors with no discriminable period of safety were varied. Two levels of conflict failed to increase ethanol consumption (although ethanol preference did increase for the strong conflict group since they suppressed their water consumption) (Anisman & Waller, 1974). Subordinate animals consumed more ethanol than dominant ones (Blanchard et al., 1987; Ellison, 1987; Wolffgramm & Heyne, 1991). Moderate crowding led to increased ethanol consumption (Heminway & Furomoto, 1972). Group housing did not affect ethanol consumption when the floor space per mouse was held constant (Rodgers & Thiessen, 1964). Inescapable electric shock administered at irregular intervals did not affect ethanol

consumption (Myers & Holman, 1967) unless there was a long post-shock period (Casey, 1960). The variable pattern of results revealed by these experiments could reflect differential effectiveness of the stressors although this possibility is difficult to assess since behavioural and/or physiological impact of the stressors was not measured. The experiments also varied whether ethanol and stress were introduced simultaneously and if not, which was introduced first. Different strains and species of mice and rats were used in these experiments. These variables, in combination with whether there was an opportunity for post-stress consumption of ethanol may account for the observed differences between experiments.

Experiments including temporally discriminable safety periods

In contrast to those experiments discussed above, the next set of experiments all included temporally discriminable safety periods so one can look for control of consumption by relief as well as by tension. That is, although the animals were still housed in the same environment in which stressors were presented, there were predictable stressor-free or safety periods as well. These predictable periods of safety were achieved by using signaled stressors or by having consistent durations between presentations of the stressful stimuli so that the end of one stressor actually signaled a

period of relative safety (Moscovitch & LoLordo, 1968). Stressors included signaled loud noise, activity stress, avoidable electric shock, and inescapable electric shocks.

Loud noise was used as the stressor in one experiment investigating whether stress influences ethanol consumption (Mollenauer, Bryson, Robison, Sardo & Coleman, 1993). C57BL/6J mice were exposed to five daily pairings of a 5-minute 75 dB pulsed noise and a 20-minute 90 dB pulsed noise or just to the 5-minute 75 dB pulsed noise. There were no groups that were exposed to both noises in an unpaired arrangement or to just the loud noise. Five weeks of noise conditioning preceded the introduction of ethanol. When the signal was paired with the loud noise consumption of 10% ethanol increased during the signal relative to other 5-min periods. This result was consistent with tension reduction theory; the mice drank more ethanol during a period when they were anticipating a stressor. Alternatively, the loud noise may have been painful and the mice might have consumed ethanol during the signal in order to obtain its analgesic properties. A 90 dB noise would fall somewhere between loud music from a radio or heavy traffic (80 dB) and the subway (100 dB) (Goldstein, 1984) and this noise was sustained for 20 minutes. In spite of the fact that Mollenauer et al. concluded that their paradigm did not involve pain stimulation I think it is worth considering. No data are provided about the volume of consumption during the signal so it is impossible to determine whether the

mice did in fact consume a pharmacologically significant dose during this period (or ever). So, although the data are suggestive, as far as tension reduction is concerned; they are not conclusive.

The relationship between avoidable and unavoidable shock stress and ethanol preference was assessed. Hooded rats with access to ethanol and water were trained to bar press to avoid shocks signaled by an 8-second warning light during three two-hour sessions per day (Cicero, Myers & Black, 1968). This arrangement of shock sessions allowed for three 6-hour shock free periods per day although the rats were housed in the shock boxes continuously. Once avoidance behaviour had stabilized, a schedule of unavoidable shocks was superimposed on the avoidance schedule. Ethanol preference increased dramatically and avoidance behaviour was markedly depressed when the unavoidable shocks were signaled (by the same signal as the avoidable shocks). Neither the increase in ethanol preference nor the depression of avoidance was apparent when the unavoidable shocks were unsignaled. These results were replicated in an experiment by Opsahl and Hatton (1972) in which fluid consumption occurred away from the shock environment. Responses made during the signal light reliably prevented the shock prior to the introduction of the signaled-unavoidable shocks. With the introduction of signaled-unavoidable shocks responding during the light was suddenly less effective in preventing

the shocks from occurring. The change in avoidance responding could have resulted from extinction of the avoidance response since bar presses no longer correlated perfectly with the ability to avoid shock (Opsahl & Hatton, 1972). When the unavoidable shocks were not signaled the contingency between responses made during the tone and prevention of shocks remained intact. Ethanol consumption may have increased in the presence of signaled unavoidable shocks in order to reduce the tension and frustration induced by loss of control over the occurrence of shock. The difference in response to signaled and unsignaled unavoidable shocks could have reflected the difference between a physical stressor and a psychological stressor (Myers, 1970).

In another experiment using avoidable shocks as a stressor rhesus monkeys consumed more ethanol when they had to bar press to avoid shock than they had consumed during a pre-avoidance phase (Clark & Polish, 1960). Increased ethanol consumption persisted during the first three post-avoidance weeks but had diminished somewhat by the final three post-avoidance weeks. Increased consumption was specific to ethanol and did not reflect a general elevation in fluid intake by the monkeys. Although it was possible that ethanol consumption increased during the avoidance phase because of an increased caloric demand there are at least two lines of evidence that refute this possibility. First, ethanol intake remained high during the post-avoidance phase

during which time the monkeys rarely pressed the lever. Second, intake of food did not change during the avoidance phase.

In both avoidance experiments changes to the shock schedule which affected the avoidance contingency, whether it was the initial commencement of shock or the termination of the shock schedule (Clark & Polish, 1960) or the introduction of signaled unavoidable shocks (Cicero et al., 1968) triggered increases in ethanol consumption. A change to the shock schedule that did not affect the avoidance contingency, the introduction of unsignaled unavoidable shocks (Cicero et al., 1968) did not affect ethanol consumption.

A number of experiments investigated whether inescapable, unavoidable footshocks would affect ethanol consumption. Myers and Holman (1967) included one group that received footshocks on a FT 1-h schedule so that each shock signaled a 59 minute 47.5 second shock-free period. Not only was this a longer period of safety between shocks than in the experiments already discussed but it was also of predictable duration. For this group and for a corresponding no-shock control group, daily ethanol intake did not increase during the period of shock. A post-shock phase was not included in this experiment so it was not possible to assess the effects of long-term relief on ethanol consumption. From a tension reduction view it would be tempting to conclude

that the shock schedule used in this experiment was not an effective stressor and therefore there were no group differences in ethanol consumption. If this shock schedule was an insufficient stressor then behavioural data (for example, level of freezing or bolus production) should not differentiate between the groups either. Unfortunately, no behavioural data were reported.

A series of experiments (Mills & Bean, 1978; Mills, Bean & Hutcheson, 1977) were conducted in which shocks were administered during the first twelve minutes of every hour so that there was a predictable 48 minute shock-free period between shock bouts. Fluid consumption was monitored continuously so that short term changes in consumption could be detected. When the fluid choices were ethanol (5% or 10%) and water (Mills et al., 1977), ethanol consumption increased immediately following shock bouts as compared to a similar period during baseline and post-shock phases. In additional experiments (Mills & Bean, 1978; Mills et al., 1977) rats had four fluid choices (water, saccharin or propylene glycol, 5% ethanol, and 10% ethanol). Again, ethanol consumption was higher during the shock phase and showed the same peak 4-12 minutes after shock bouts. Over successive shock days, rats shifted their fluid choice from 5% to 10% ethanol. This switch could indicate that palatability became less important over time. Alternatively, calories may have become more important as the

experiment progressed. However if this were the case then consumption of propylene glycol should have increased as well, since its caloric value was midway between the two ethanol solutions. A third possibility is that the rats became tolerant to the effects of 5% ethanol and switched to 10% in order to maintain the same post-ingestive effects. Mills and his colleagues (Mills & Bean, 1978; Mills et al., 1977) concluded that since the increased ethanol consumption was restricted to the shock phase and occurred in a very regular fashion that the selection of ethanol was a function of shock-induced stress. It is open to speculation whether the rats consumed ethanol while relieved that one shock bout was over or while anticipating the next shock bout. If the increase in ethanol consumption still followed the shock bout by 4-12 minutes when the interval between shock bouts was variable (VT schedule) this would have indicated that ethanol consumption was reinforced by relief. Unfortunately, such a group was not included in these experiments.

Even though ethanol consumption was greater during the shock phase than during baseline or post-shock phases, the rats did not consume enough ethanol to sustain intoxication (blood ethanol level of 50 mg/100 ml) over the entire 24 hour period (Mills et al., 1977). Blood ethanol levels during the shock phase averaged 35 mg/100 ml with a range from 5-160 mg/100 ml. In order to sustain intoxication during a 24 hour period, the rats would have had to consume at least 8.4 g/kg of ethanol per day, roughly equivalent to

212 ml/kg of 5% ethanol or 106 ml/kg of 10% ethanol. This would have been impossible; total fluid consumption only averaged 80 ml/kg (Mills et al., 1977).

In the experiments using inescapable shock as the stressor that have been discussed so far the safety period between shocks or bouts of shock was just under one hour (Myers & Holman, 1967) or approximately 48 minutes (Mills & Bean, 1978; Mills et al., 1977). In the next experiment, the maximum duration of safety was extended to 6 or 12 hours between shocks. Shock groups preferred ethanol to water and consumed more ethanol than a no-shock group regardless of whether the maximum duration of safety was 6 or 12 hours (Anisman & Waller, 1974). The difference between shock and no-shock groups disappeared in subsequent sessions during which the shocks were no longer presented. This result is consistent with the tension reduction hypothesis although it is also possible that the shock groups consumed ethanol for its caloric value since rats in these groups lost weight during the shock phase. The latter alternative is cast into doubt by the results of another experiment. Ethanol consumption of food deprived and non-deprived shock groups did not differ during the shock phase. Both shock groups consumed more ethanol than a no-shock food deprived group. A post-shock phase was not included (Anisman & Waller, 1974). Since the time course of daily ethanol

consumption was not reported it is impossible to completely eliminate relief as the factor eliciting ethanol consumption during the shock phase.

Another experiment employing a 12 hour maximum duration of safety manipulated initial preference for ethanol (prior to the introduction of shock) and then investigated the effect of inescapable electric footshock (FT 30 minute schedule during alternate 12 hour periods) on this preference (Bond, 1978). Shock groups consumed more ethanol and less water during the shock phase than a no-shock group when initial ethanol preference was low. A post-shock phase was not included in the experiment. In subsequent experiments, a higher initial preference for ethanol was achieved by sweetening the ethanol solution with saccharin or by making ethanol the only fluid available for 25-days. During the shock phase shocked rats consumed less ethanol than no-shock rats in both experiments. Bond concluded that changes in ethanol consumption could reflect an adventitious punishment contingency since the rats drank in the same environment where they were shocked. The initially preferred solution was being consumed the most and therefore it was more likely that a rat would be shocked while drinking the preferred fluid and therefore to decrease its consumption of that fluid.

The maximum duration of safety was increased to 23 hours per day in another experiment (Volpicelli, Tiven & Kimmel, 1982). Each phase of the experiment consisted

of five shock days (with a one-hour shock session) and two no-shock days. A no-shock control group was not included in this experiment so comparisons could only be made within subjects or relative to a safety cage group (which is discussed in a later section). When there was a choice between 6% ethanol and water the shock box rats increased their ethanol preference during the two post-shock days relative to their ethanol preference on the preceding shock days. This result was inconsistent with tension reduction theory but consistent with the relief hypothesis since ethanol preference increased after shocks were discontinued.

A similar experiment (Caplan & Puglisi, 1986) included a pre-shock baseline phase during which rats had a choice between 5% ethanol and water. Animals housed in the shock boxes significantly increased their ethanol consumption during a shock phase (with a 1-hour shock session per day) relative to their own baseline consumption. Consumption data for the five shock days was not separated from data for the two post-shock days. However, during a final seven-day phase in which no shocks were administered ethanol consumption decreased. In a subsequent experiment the fluid choices were switched from water and ethanol to sucrose and sucrose/ethanol. Sucrose/ethanol consumption was negligible and consumption of the sucrose solution was lower during shock phases than during the pre-shock or post-shock phases.

When rats had access to ethanol in the shock boxes during a long duration of safety between shock bouts (6, 12, or 23 hours) the results were varied. Shock groups consumed more ethanol than no-shock groups during a shock phase but not a post-shock phase (Anisman & Waller, 1974; Caplan & Puglisi, 1986) regardless of food deprivation (Anisman & Waller, 1974). Other shock groups (for which there were no baseline data) increased their ethanol preference during post-shock days relative to shock days (Volpicelli et al., 1982). In a final set of experiments ethanol preference increased during a shock phase when initial preference was low but decreased during a shock phase when initial preference was high (Bond, 1978). Differences in the results of these experiments result in part from whether or not a baseline (pre-shock) or post-shock phase was included.

Rats in two final experiments (Kinney & Schmidt, 1979; Merrimen, 1996) were not housed in the shock boxes, but their only opportunity to drink ethanol occurred in the shock boxes so these experiments have been included here. One experiment was designed to assess the influence of a signaled safety period (during a shock session) on ethanol consumption (Kinney & Schmidt, 1979). Unsignaled inescapable shocks (1 mA, 1-s, on a VT 2-minute schedule) were delivered to food and fluid deprived albino rats during a one hour session in the shock boxes. During one 15 minute period of the session no shocks

were delivered. When the 15 minute shock-free period was unsignaled, ethanol consumption did not differ between the shock and no-shock groups. Ethanol consumption was higher during the shock phase than during baseline or post-shock phases only for the group that experienced the signaled 15 minute shock-free period. The group that received the signaled shock-free period had an ethanol intake of 0.33 g/kg/h when ethanol and water were both available and this increased to greater than 2.0 g/kg/h when ethanol was the only fluid available. When there was a choice between ethanol/sucrose and plain sucrose, sucrose was consumed during the shock-free period and ethanol/sucrose consumption occurred in bursts following each shock. Total fluid consumption increased during the post-shock phase but this increase was accounted for by the intake of the sucrose solution.

Merrimen (1996) compared the consumption of ethanol and sucrose by groups of Sprague Dawley rats that had access to fluid in the shock box during the 30 minutes before or after a 20 minute shock bout. Consumption did not differ among the four groups although there was a tendency for the ethanol groups to consume more fluid than the sucrose groups. Fluid consumption increased across days for all groups. Bolus production, a measure of conditioned fear (Inoue et al., 1993), was not significantly correlated with consumption for any group. These results provided no support for either

the tension reduction or relief hypotheses. Rather, the increased consumption across days and the absence of differences between groups on any measure could suggest that consumption was reinforced by thirst or hunger reduction. Including control groups that had access to solutions with no calories or with different caloric density would have helped to discriminate between the thirst and hunger reduction explanations.

The issue of adventitious punishment is an important one because if it were affecting the relationship between ethanol consumption and stress then its influence should be considered in all of the studies in which animals were exposed to shock in the same place that they were drinking. The frequency of punished responses should have varied with the shock schedule. When shocks were more frequent, the number of instances of a rat being shocked while drinking should have increased. Information about the time course of fluid consumption (as was provided in Mills & Bean, 1978; Mills et al., 1977) should help to evaluate adventitious punishment by allowing researchers to determine if consumption of the preferred fluid was in fact punished with shock more often than consumption of the less-preferred fluid(s). Experiments by (Mills & Bean, 1978; Mills et al., 1977) included hourly consumption data (and showed that consumption of all fluids was suppressed during shock bouts. There was little support for the adventitious punishment idea. Ethanol was initially preferred to water (Mills et al.,

1977) and this remained true throughout the shock and post-shock phases although absolute volumes of ethanol consumed decreased in the post-shock phase. In the experiments where there were four fluid choices, there were no reliable differences in baseline preferences so adventitious punishment could make no prediction about the results. Deviation from results predicted by the adventitious punishment hypothesis could have reflected differences in shock schedules between experiments conducted by Mills and his colleagues (Mills & Bean, 1978; Mills et al., 1977) and experiments conducted by Bond (1978). The former administered shocks on a variable time schedule during the first 12 minutes of every hour while the latter researcher administered a single shock every 30 minutes during alternate 12 hour periods. The role of adventitious punishment in fluid selection could not be assessed in studies that did not include a baseline phase (Anisman & Waller, 1974; Myers & Holman, 1967).

Experiments including contextually discriminable safety periods

In these experiments, animals had access to ethanol in an environment other than the one in which they were exposed to stress. Thus the animals were able to drink during periods when they might have experienced anticipatory fear or post-stress relief but not during periods when the stressor was being administered. Since consumption did not occur in the same environment as the aversive event, adventitious punishment should not

have been an issue in any of these experiments. Stressors used in these experiments included: social dominance, housing conditions, avoidable shock, and inescapable shock.

The impact of social factors on ethanol consumption was investigated in an experiment in which consumption occurred in a different context from the stress (Blanchard, Flores, Magee, Weiss & Blanchard, 1992). Long Evans rats were housed in colonies composed of 5 males and 2 females and in six of the eight colonies a dominance hierarchy formed. Rats were removed from the colony for 7 hours per day for individual two bottle tests between water and ethanol. Prior to grouping, rats which became dominant drank more water and had lower ethanol preferences than rats that became subordinate. After grouping this pattern persisted; dominant males consumed less ethanol than subordinates did. The subordinate rats increased their consumption of both fluids relative to the dominant rats.

The effect of anxiety induced by crowding on ethanol consumption also has been investigated (Hannon & Danlon-Bantz, 1976; Thiessen & Rodgers, 1965). In these experiments, unlike previous ones that examined the effects of various housing conditions on ethanol consumption, fluid was available during periods of relative safety away from the group situation. C57BL mice that had never been grouped consumed more ethanol than did mice that had been grouped for five days and then separated (Thiessen &

Rodgers, 1965). This difference could not be accounted for in terms of caloric necessity since the mice that had been housed in groups lost weight, not the isolated mice. Individually housed Wistar rats consumed more 25% ethanol than rats that were housed in pairs for eight hours per day from day 25 until the end of the experiment (day 60) (Parker & Radow, 1974). In one experiment, individually housed Sprague Dawley rats consumed less of a 10% ethanol/milk solution than group housed rats. Fluid consumption in this experiment was restricted to two daily 10 minute sessions (Hannon & Danlon-Bantz, 1976). Individually housed mice and rats consumed more ethanol than group housed mice and rats. This difference does not appear to be an artifact of ethanol's caloric value but there is no definitive evidence (for example behavioural or physiological) to indicate that the individually housed animals consumed ethanol in order to reduce tension.

The effect of control over food delivery or over the termination of electric footshocks on ethanol consumption (and preference) by rats has been investigated (Volpicelli & Ulm, 1990). Rats unable to control the delivery of food pellets preferred ethanol more than rats that had control over food deliveries. Similarly, rats unable to control the termination of electric footshock preferred alcohol more than rats that could escape shock. These results provide additional evidence that the psychological dimension

of control over environmental events (whether appetitive or aversive) influences ethanol preference by rats. Rats with no control over aspects of their environment would have been expected to feel more anxiety than rats with control (Mineka, Cook & Miller, 1984). Increased ethanol consumption by the rats without control is consistent with tension reduction.

A feeding and fasting schedule was combined with a shock schedule and the combined effect of these stressors on consumption of three fluids (water, 10% ethanol, and a food solution) examined (von Wright, Pekanmaki & Malin, 1971). Weak unavoidable shocks (0.2 mA, 5-s, VT 55 minute) had little effect on ethanol consumption but the effects of strong unavoidable shocks (1.2 mA) were similar to those found by Casey (1960). During the stress period there was no increase in ethanol intake but there was an abrupt rise in consumption when the shocks were terminated and the rats remained in their home cages (von Wright et al., 1971). Other groups barpressed to gain access to food and each bar press was punished with a shock (0.2 or 1.2 mA). Weak contingent shock had no effect on ethanol consumption whereas animals that received strong contingent shocks increased their ethanol consumption on conflict days and decreased their ethanol consumption to pre-experimental levels when the shocks were terminated.

A second experiment (von Wright et al., 1971) showed no consistent increase in ethanol intake during non-contingent shock periods although there was a uniform and persistent increase in intake following the termination of shocks. The failure to see increased ethanol consumption during the shock period was explained in terms of response incompatibility. When strong shocks were given at unpredictable intervals, the animals learned various freezing and jumping responses that were incompatible with drinking. After shock was terminated, animals were removed to their home cages, an environment not associated with shock and which, therefore, did not elicit responses incompatible with drinking. The increased consumption of ethanol during the post-shock period in an environment removed from the shocks is consistent with the relief hypothesis although it is also possible that ethanol consumption would have increased over the course of the experiment even if no shocks had been administered (Choca et al., 1977).

Avoidable shocks were presented to Holtzman rats during 1-hour sessions five days per week (Hatton & Vieth, 1974). The rats had continuous access to 7.5% ethanol and water and consumption was monitored continuously. Ethanol preference (and consumption) during the two hours after the shock session was higher on shock days than during the comparable period on days when no shocks were delivered. This pattern of

ethanol consumption is consistent with relief. Ethanol consumption that occurred during the light phase (after the shock session) did not diminish ethanol consumption during the dark phase, which could indicate that consumption that occurred during the light phase served a different purpose than ethanol consumption during the dark phase. No control groups were included in this experiment.

Several experiments (Nash & Maickel, 1985; Sprague & Maickel, 1994; Ton, Brown, Michalakeas & Amit, 1983) compared the effects of acute stressors on ethanol consumption by rats. Both shock and restraint suppressed the gradual increase in ethanol intake that was apparent in a no-stress control group of Wistar rats (Ton et al., 1983). Group housed Sprague Dawley rats with a choice between saccharin and ethanol/saccharin decreased their home cage fluid consumption during weeks in which there were sessions of immobilization or isolation (Nash & Maickel, 1985). Both groups increased their ethanol/saccharin consumption and preference during post-stress weeks. When rats were subjected to immobilization or isolation stress on an irregular, unpredictable schedule they decreased their ethanol or ethanol/saccharin consumption (Roske et al., 1994; Sprague & Maickel, 1994). Stress did not affect saccharin consumption. Ethanol consumption returned to baseline levels during the post-stress phase. Krishnan, Nash and Maickel (1991) alternated 4-day periods of restraint (1-3

hours per day) with 4-day stress-free periods. Male Sprague Dawley rats suppressed their ethanol consumption on stress days and increased their consumption during the stress-free periods. Rats with an initially high preference for ethanol did not show as big a rebound on stress-free days as rats that had a lower initial preference for ethanol.

A number of acute inescapable, unavoidable stressors had similar effects on ethanol consumption. Rats subjected to shock, restraint, immobilization, or isolation either suppressed or did not change their ethanol consumption on days when the stressor was administered (Krishnan et al., 1991; Nash & Maickel, 1985; Roske et al., 1994; Sprague & Maickel, 1994; Ton et al., 1983). After the stressor was discontinued, ethanol consumption increased (Krishnan et al., 1991; Nash & Maickel, 1985; Roske et al., 1994; Sprague & Maickel, 1994).

The consumption of safety cage groups (groups housed away from the shock boxes except for a one hour shock session) was compared to the consumption of shock box groups described previously (Caplan & Puglisi, 1986; Volpicelli et al., 1982). All of the groups were exposed to the same 1 hour shock session on all shock days. On shock days rats in the safety cage group drank reliably more ethanol and less water than rats in the shock box group (Caplan & Puglisi, 1986; Volpicelli et al., 1982). Both groups increased their ethanol preference during two post-shock days relative to the five shock

days (Volpicelli et al., 1982). A no-shock control group was not included in either experiment.

The basic schedule from the previous experiments, that is, rats housed in safety cages away from the shock boxes except for the one hour daily shock session (Caplan & Puglisi, 1986; Volpicelli et al., 1982) was retained in several other experiments which varied the control groups (Fidler & LoLordo, 1996; Volpicelli, Davis & Olgin, 1986; Volpicelli, Ulm & Hopson, 1990). In one of these experiments rats were injected with either naltrexone (an opiate antagonist) or placebo on each of six post-shock days to assess the possibility of an interaction between ethanol and the endorphin system (Volpicelli et al., 1986). As did the safety cage group in the earlier experiment (Volpicelli et al., 1982) rats that received placebo injections increased their ethanol consumption and decreased their water consumption during the post-shock phase relative to the shock phase (Volpicelli et al., 1986). The group that received naltrexone injections did not show this ethanol increase. Naltrexone injections blocked the post-shock increase in ethanol consumption, which suggests that endogenous opiates are involved in the voluntary drinking of ethanol by rats.

Another experiment used a similar shock regimen but included both shock and no-shock groups (neither of which was injected with anything) (Volpicelli et al., 1990).

Total fluid consumption by the shock and no-shock groups did not differ during the baseline and post-shock phases. During the shock phase, shocked rats consumed significantly less fluid than control rats. Ethanol preference increased across phases for both groups. During the post-shock phase ethanol preference was higher for the shock group than for the no-shock group. This result paralleled those of Volpicelli et al. (1982, 1986) and Caplan and Puglisi (1986). Volpicelli and Ulm (1990) also reported that inescapable shock decreased ethanol consumption on shock days but increased it on the day after shock days. The presence of contextually discriminable relief periods, over and above the 23 hours of safety during the shock phase, appeared to be an important condition for observing increased ethanol preference.

The role of initial preference on subsequent ethanol preference was assessed by performing a median split on baseline preference data (Volpicelli et al., 1990). Rats in the shock group with low initial ethanol preference increased their ethanol preference during the shock phase relative to rats in the no-shock group with low initial ethanol preference. Rats in the shock group with higher initial ethanol preference decreased their ethanol preference during the shock phase relative to their no-shock counterparts. The reason for the different effect of shock on animals with initially lower or higher ethanol preference was not clear. Previously (Bond, 1978), the role of initial preference was explained in

terms of adventitious punishment contingencies but this explanation cannot apply when shock presentations occurred in a different environment from fluid consumption.

Fidler and LoLordo (1996) conducted a series of experiments similar in form to that of Volpicelli et al. (1990). Each experiment included baseline, shock, and post-shock phases. In the first experiment the consumption and preference for 5% ethanol and an isocaloric sucrose solution by a 60-shock group (2-s, 0.8 mA shocks on a FT 60-s schedule for one hour, like the shock parameters used by Volpicelli et al., 1986) were compared to that of a 2-shock group. There was a small but significant interaction between group and phase such that relative to the 60-shock group, the 2-shock group decreased its ethanol preference across phases. However, ethanol preference did not differ significantly between groups during any phase of the experiment. This result was inconsistent with both the tension reduction and relief hypotheses.

In four additional experiments (Fidler & LoLordo, 1996), the ethanol consumption and preference of a 60-shock group (0.8 mA, 2-s shocks on a FT 60-s schedule for one hour) were compared to that of a group that remained in their home cages, and to a group that was moved to the shock boxes and not shocked. Regardless of whether treatments occurred during the light or dark phase there were no group differences in ethanol preferences in these experiments. The increased ethanol preference

by shocked animals relative to controls that had been observed by Volpicelli et al. (1990) was not replicated regardless of whether baseline ethanol preference was higher, lower, or the same as that reported in the earlier experiment (Volpicelli et al., 1990). The results from this series of experiments offer little support for a relationship between regularly scheduled 1-hour shock sessions and ethanol consumption, either anticipatory or soon after the shock session. The absence of group differences in these studies is problematic for both tension reduction and relief hypotheses of ethanol consumption, since both predicted an increase in ethanol preference for the shock group relative to controls. Mean consumption of absolute ethanol during baseline by rats in the shock groups varied from 1.10 to 3.02 g/kg/day (Fidler & LoLordo, 1996), amounts that are comparable to the 2.31 g/kg/day observed by Volpicelli et al. (1990). Therefore, differences in pre-shock exposure to ethanol were not responsible for the discrepancy in the results. The rats in these experiments consumed significant quantities of ethanol but they did not consume enough to maintain continuous intoxication (Mills et al., 1977). It is possible that they did not consume enough ethanol within temporal proximity of the shock session (either before or after) to learn about any effects that ethanol might have. However, if this is the case it is not clear why there were significant effects in other experiments of similar design with comparable levels of consumption.

A similar design using a 1 hour restraint session instead of a 1 hour shock session compared the ethanol consumption in the post-restraint phase for groups with or without access to ethanol during the restraint phase (Bowers, Sabongui & Amit, 1997). Repeated sessions of restraint increased ethanol consumption during the post-restraint phase only by Long Evans rats that had not had access to ethanol during the restraint phase. This result is explained as follows (Bowers et al., 1997). Groups that had ethanol available during the restraint phase could use ethanol to reduce the impact of the stressor. If the impact of restraint was attenuated by ethanol consumption then the post-restraint increase in ethanol intake would also be attenuated. For groups that did not have access to ethanol during the restraint phase, the full impact of the stressor would have occurred in the post-restraint phase and ethanol consumption would have increased in order to attenuate this (larger) stress response. Unfortunately, consumption was only measured once per day in this experiment so there are no data to indicate just how the group that had access to ethanol during the restraint period actually used ethanol; for example, whether consumption occurred in anticipation of or during relief after daily restraint sessions.

Instead of assessing initial preference for a fixed concentration of ethanol, Champagne and Kirouac (1987) held initial preference constant and used individually determined ethanol concentrations for each rat. Ethanol preference did not change over

the course of the experiment (unlike in Volpicelli et al., 1982, 1990) although total consumption did decrease during the shock phase. Unfortunately, a no-shock group was not included in this experiment.

Powell, Kamano and Martin (1965) used a very different shock regimen from any of those described so far. On shock days, rats were subjected to five two-minute shock sessions with one (0.2 mA, 5-s) shock per session. Male Wistar rats increased their ethanol consumption over the course of the experiment regardless of whether they were shocked. During the post-shock phase, male rats in the no-shock group had a higher preference for ethanol than the male rats in the shock group. Females in the shock group increased their ethanol consumption but this increase was only apparent following a period of forced ethanol consumption. Rats with high initial ethanol preference (as defined by a median split) consumed increasing amounts of ethanol regardless of experimental treatment. By contrast, rats with low initial ethanol preference increased their ethanol consumption only after a period of forced intake. This pattern is contrary to that reported by Volpicelli et al. (1990).

In a number of experiments discussed so far (Fidler & LoLordo, 1996; Volpicelli et al., 1986; Volpicelli et al., 1982; Volpicelli et al., 1990) rats had a long period of safety away from the shock boxes during which fluids were available. In the next two

experiments (Boyd, Callen & House, 1989; Cox & Stainbrook, 1977), rats also had a long period of safety away from the shock boxes but access to fluids was restricted to a brief session within this period. After shock sessions, which consisted of a 90-s placement in the shock box terminating in a 1-s shock, fluid deprived albino rats had access to 10% ethanol in a drinking environment for 10 minutes. Five hours later the rats had a choice between ethanol and water in their home cages (Boyd et al., 1989). Ethanol consumption in the drinking environment did not differ between shock and no-shock groups. In the home cage, rats in the shock group consumed more ethanol than rats in the no-shock group. Boyd et al. concluded that ethanol had become reinforcing for the shock group because of its association with relief from fear.

Cox and Stainbrook (1977) restricted fluid access to a 15 minute period in a distinctive drinking environment immediately before or after the 15 minute shock session. Shock sessions occurred every other day for the duration of the experiment. All of the rats had access to ethanol on one quarter of the single bottle days, sucrose on another quarter of the single bottle days and water on half of the single bottle days. For one group of animals water was available on shock days and the other fluids on alternate no-shock days. For the other group water was available on no-shock days and the other fluids on alternate shock days. Data for the days on which only one fluid was available

was not reported. Two bottle tests between ethanol and sucrose were interspersed among the single bottle days. The group that had access to ethanol and sucrose before shock had a higher ethanol preference than any of the other three groups (which did not differ from each other).

The results of experiments in which animals had the opportunity to consume fluid in an environment other than the one in which they were stressed produced varying results. Experiments varied on a number of dimensions including the paradigm used to induce stress, the duration of stress and safety periods, and duration of the drinking period. Subordinate rats consumed more ethanol than dominant rats (Blanchard et al., 1992). Isolated rats consumed more ethanol than group housed rats (Parker & Radow, 1974; Roske et al., 1994; Thiessen & Rodgers, 1965) except when fluid consumption was restricted to 10 minutes twice per day (Hannon & Danlon-Bantz, 1976). Rats with no control over the delivery of food pellets or the termination of footshock preferred ethanol more than rats that had control (Volpicelli & Ulm, 1990).

A number of experiments induced stress using a one-hour session of inescapable footshocks. In these experiments rats housed away from the shock boxes consumed more ethanol than rats housed in the shock boxes on shock and post-shock days (Caplan & Puglisi, 1986; Volpicelli et al., 1982). Rats in a similar safety cage group increased their

ethanol consumption in the post-shock phase unless they were injected with naltrexone (Volpicelli et al., 1986). Ethanol preference during the post-shock phase was higher for a shock group than for a no-shock group according to Volpicelli et al. (1990) but not according to Fidler and LoLordo (1996). Several studies examined the relationship between initial ethanol preference and ethanol preference during shock and post-shock phases. According to Volpicelli et al. (1990), the ethanol preference of rats with low initial ethanol preference increased across phases and the reverse was true for rats with high initial ethanol preference. When initial ethanol preference was held constant ethanol preference remained constant over the course of the experiment (Champagne & Kirouac, 1987). Fidler and LoLordo (1996) observed no consistent relationship between initial ethanol preference and subsequent pattern of consumption. Fidler and LoLordo saw no evidence that shock enhanced ethanol preference. Results varied between these experiments in spite of the fact that they employed similar shock schedules.

The final two experiments combined very short shock sessions with restricted access to fluid. Rats that were exposed to ethanol immediately after shock consumed more ethanol in the home cage five hours later than rats in the no-shock group (Boyd et al., 1989). Rats exposed to ethanol and sucrose before shock sessions showed a higher

ethanol preference than those rats with the same fluid pairing after shock or those rats that were exposed to ethanol and sucrose on no-shock days (Cox & Stainbrook, 1977).

Previous research in our laboratory (Fidler & LoLordo, 1996) which allowed rats continuous access to ethanol and water or sucrose except during a 1-h footshock session failed to support either the Tension Reduction Hypothesis or the Relief Hypothesis despite use of parameters identical to those employed in one published report (Volpicelli et al., 1986). Since the rats had 23-h access to ethanol, it is possible that they were not drinking enough ethanol in temporal proximity to the shock session (either before or after) to obtain any beneficial effects that it might have. Further, since the rats had simultaneous access to ethanol and sucrose or to ethanol and water, and would drink some of both, they could have had difficulty associating the taste of ethanol with any pharmacological effects that did occur.

In order to remedy these problems I adopted a procedure based on that of Cox and Stainbrook (1977), in which rats' access to fluid was restricted to a brief period immediately before or after a shock session. Further, all access to fluids occurred in a distinctive drinking environment and on most days only one fluid: ethanol, or sucrose, or water was available so that the animals would be better able to sort out the post-ingestive consequences of the different solutions. Two groups, Group E/S-Sh Before and Group

E/S-Sh After, had access to ethanol on some shock days and to sucrose on the remaining shock days. Water was available on no-shock days. A second pair of groups, Group E/S-NoSh Before and Group E/S-NoSh After, had access to water on all shock days. Ethanol was available on some no-shock days and sucrose was available on the remaining no-shock days.

Providing the animals with one solution at a time, instead of two, should result in an increase in consumption of a particular solution on a given day. In the case of ethanol, this should increase the likelihood that enough will be consumed to have an intoxicating effect. Moreover, if such an effect does occur, the rats should be able to learn that the taste of the ethanol solution precedes that effect (since it was the only taste available), while the different taste of the control solution (sucrose or water) precedes no effect. This should cause the positive reinforcing value of the ethanol to increase (at least for groups that receive ethanol on shock days, Group E/S-Sh Before or After), relative to that of sucrose or water. Occasional two-bottle tests (ethanol versus sucrose, ethanol versus water, and sucrose versus water) on shock and no-shock days were included in order to assess fluid preferences.

The first two experiments varied only in whether access to fluid occurred after the shock session (Experiment One) or before the shock session (Experiment Two). In the

first experiment, the Relief Hypothesis would be supported if ethanol consumption, or ethanol preference, increased across days relative to sucrose consumption in Group E/S-Sh After, which had access to ethanol or sucrose after shock sessions, and not in Group E/S-NoSh After, which had access to ethanol or sucrose on no-shock days. In the second experiment, the TRH would be supported if ethanol consumption, or ethanol preference, increased over successive days relative to sucrose consumption in Group E/S-Sh Before, which had access to ethanol or sucrose before shock sessions, and not in Group E/S-NoSh Before.

In Experiments 1 and 2 each rat had access to three different fluids in the drinking environment (on different days). Rats were placed in the drinking environment every day and in the shock boxes half of the time. The task for the rat was complicated. In Experiments 3 and 4, I simplified the task for the rats. Each rat consumed only one fluid in the drinking environment and placement in the drinking environment always preceded shock sessions. This arrangement should have allowed the rats to learn an association between the drinking environment and the shock session and therefore they should have experienced anticipatory fear in the drinking environment. If ethanol reduces tension then ethanol should have become more reinforcing and consumption should have increased. Further, rats in the ethanol group should have shown lower levels of fear when it was

measured. Beginning towards the end of Experiment 3 fecal bolus production in the drinking environment and shock boxes was recorded as an index of fear. This measure has been used previously (Inoue et al., 1993). Experiment 4 was essentially a replication of Experiment 3 except that fecal bolus production was measured from Day 1.

In Experiments 1-4 fluid deprived rats had access to fluids for only one hour per day. As a result, whatever consumption occurred must have been motivated at least partially by thirst. In Experiment 5, the role of thirst was assessed by allowing rats (from the Ethanol, Sucrose, and Water groups of Experiment 4) to consume fluid in the drinking environment under different levels of fluid deprivation. On some shock days the rats were able to consume water in their home cages prior to fluid access in the drinking environment. To the extent that different factors motivated the consumption of different fluids (for example thirst, caloric value, tension reduction) their consumption should have been differentially effected by the preloading manipulation. Fecal bolus production was measured throughout the experiment. If ethanol reduced tension (regardless of whether that was why it was consumed) bolus production should have been higher on days when consumption was lower.

In Experiments 1 and 2 the consumption of ethanol before or after shock could be compared between subjects to the consumption of ethanol on a no-shock day. In

Experiments 3-5 the consumption of ethanol before shock could be compared to the consumption of other fluids before shock. In Experiment 6, the same animals had access to ethanol during a signal for shock and in the absence of a signal for shock. This was accomplished by giving the rats their fluid access in different drinking environments on different days. Placement in one drinking environment always preceded a shock session and placement in the other always preceded return to the home cage. According to tension reduction theory, ethanol should have been more reinforcing during signals for a noxious event so ethanol consumption would have been expected to increase more rapidly in the shock-associated drinking environment.

Experiments 7 and 8 differed from the earlier experiments in several important respects. First of all, rats in these experiments had access to ethanol or sucrose in the shock boxes. Fecal bolus production in the drinking environments was very low in Experiments 4-6 which could indicate that the rats were not experiencing anticipatory fear. If the rats were not experiencing anticipatory fear then they could not have been expected to consume ethanol to reduce their fear. The number of shocks was reduced (from Experiments 1-6) to increase the likelihood that the rats would drink in the shock boxes. In Experiments 2-6 access to ethanol occurred prior to shock sessions. In these experiments ethanol was not introduced until after shock had been discontinued. This

manipulation ensured that fear was established prior to the introduction of ethanol and it eliminated the possibility that any changes in behaviour could be attributed to analgesic properties of ethanol. Rats in Experiment 7 were non-deprived to reduce the influence of thirst on consumption in the shock boxes. Rats in Experiment 8 were fluid deprived because consumption in Experiment 7 was too low to be meaningful.

EXPERIMENT 1

One group of thirsty rats, Group E/S-Sh, had the opportunity to drink ethanol in a distinctive environment after alternate shock sessions and to drink an isocaloric sucrose solution after others, with water available for one hour on intervening no-shock days. A second group of rats, Group E/S-NoSh, had access to ethanol or sucrose in a distinctive environment on no-shock days and to water after shock sessions. Since the distinctive drinking environment always followed shock for both groups of animals, placement in this environment was expected to act as a signal for safety, causing the rats to be less afraid or relieved in this environment. Only the group that had ethanol paired with shock, Group E/S-Sh, had the opportunity to drink ethanol during this period of relief. If relief is the primary motivator of ethanol consumption, as suggested by the Relief Hypothesis, then these rats should increase their ethanol consumption relative to those that had access to ethanol on no-shock days.

Method

Subjects

The subjects in this experiment were twenty-four adult male Sprague Dawley rats from Charles River Canada. At the start of the experiment the mean weight of the rats

was 287 g with a range between 255 and 310 g. The rats were individually housed in Wahmann hanging cages under a 16 h/8 h light/dark cycle with lights on at 7:00 am. All experimental manipulations occurred during the light part of the cycle. Rats had ad lib access to food in the home cage throughout the experiment. Fluid access was restricted as described in the procedure section.

Apparatus

Shock Environment. Shock sessions were conducted in four identical operant chambers, 24 x 29 x 20 cm. The top of each chamber was white Plexiglas and the walls were of Plexiglas in vertical stripes of black (2 cm) and white (2.5 cm). A lever was located in the center of one of the sidewalls and under it was a food cup. The door of each chamber was made of transparent Plexiglas with a 12 x 13 cm removable metal panel. The floor of each chamber was made of stainless steel bars, 1.5 mm in diameter, spaced 1.3 cm apart. Grason-Stadler shock generators and scramblers (Model E1064GS) could electrify the floors. Each chamber was enclosed in a 60 x 28 x 28 cm sound attenuating shell fitted with a fan that was turned on during the sessions to provide ventilation and to provide a masking noise. During sessions, a 7.5-W house light illuminated each chamber. Presentation of the shocks was controlled by an Apple IIe microcomputer. The room

containing the computer and relay rack separated the shock environment from the drinking environment.

Drinking Environment. The drinking environment consisted of four plastic tub cages, 43 x 27 x 15 cm, placed side by side on a table. The cages were fitted with wire lids that held the drinking bottle at an appropriate angle. Bottle position (right versus left) was varied from session to session. The room was dimly lit and white noise was used to mask noise from outside the room.

Fluids. The three fluids used in this experiment were tap water, a 5% (v/v) ethanol solution made from 95% ethyl alcohol and tap water and sweetened with 0.05% (w/v) saccharin, and a 7.01% (w/v) sucrose solution made with 0.5% (v/v) acetic acid. Pilot work indicated that these solutions were equally acceptable to the rats. Ethanol and sucrose solutions were equicaloric and were identical to those used by Fidler and LoLordo (1996, Experiment 1). Solutions were delivered in glass bottles fitted with rubber stoppers that held straight stainless steel drinking tubes.

Procedure

The ad lib supply of water was discontinued, and access to water was restricted to 1-h/day in the home cages for three days. Rats were randomly assigned to one of six squads of four rats each. The four rats in each squad had access to fluids at the same time

and the squads were run in the same order and at the same time (± 0.5 h) each day. For the next five days, the Baseline Period, animals were confined in the drinking boxes for their 1-h daily access to water (in accordance with the "training" procedure used by Cox and Stainbrook, 1977). The experimental procedure began the next day.

Shock days were alternated with no-shock days. On shock days, each animal was weighed and placed in the shock box for one hour prior to being placed in the drinking environment. In the shock boxes, rats received 0.8-mA shocks, each of 2-s duration, on a FT 60-s schedule under white light. Grids were wiped clean and pans emptied between rats. On no-shock days animals were weighed and taken to the drinking boxes for one hour and then returned to their home cages. Shock and no-shock days were counterbalanced such that on any given day half of the rats were shocked and half were not.

Each animal continued to have access to fluid in the drinking box for 1 h/day. Only one bottle was present on any given day, and its position was counterbalanced across days. For half of the animals (Group E/S-Sh), the availability of either ethanol or sucrose was contiguous with shock, while on the alternate days when no shock was given, these animals had plain tap water to drink. For the other half of the animals (Group E/S-NoSh), tap water was available on shock days, and either ethanol or sucrose was

available on no-shock days. On days when ethanol or sucrose was available, half of the animals received ethanol and half sucrose. Table 1 shows a representative four-day cycle for Experiment 1.

Rats were assigned to groups so that within each squad there were two rats from Group E/S-Sh and two rats from Group E/S-NoSh. Further, one of the rats from each group had a shock day and the other a no-shock day. Of the four animals in each squad, on any given day, two had a water day, one had an ethanol day, and the fourth had a sucrose day. Order of fluid presentation, whether ethanol or sucrose came first, and order of days, whether shock or no-shock came first, were counterbalanced across animals.

Two-bottle tests were included at various intervals during the experiment and this schedule is outlined in Table 2. There were always two days of two-bottle tests so that all of the animals had a two-bottle test on a shock day and on a no-shock day. The first set of two-bottle tests, between ethanol and sucrose (Eth/Suc #1), occurred after the fourth 4-day cycle. During the 1-h drinking session the rats had two bottles available, one containing ethanol and the other containing sucrose. The position of the two fluid bottles was counterbalanced across days. The shock schedule was continued for these test days so that there was a 2-bottle test after shock and no-shock for each of the animals. A second pair of ethanol versus sucrose two bottle tests (Eth/Suc #2) occurred after the

Table 1: Representative four day cycle for the experiment in which fluid was available in the hour after the shock session

Group	Day 1	Day 2	Day 3	Day 4
E/S-Sh	Home Cage Water	Shock Ethanol	Home Cage Water	Shock Sucrose
E/S-NoSh	Home Cage Ethanol	Shock Water	Home Cage Sucrose	Shock Water

Note: Within each group the starting day (shock or no-shock) was counterbalanced so that half the animals in each group started with each type of day. Order of fluid presentation (ethanol or sucrose) was also counterbalanced. Therefore, on any given day, half of the animals in each group had access to water, one quarter had access to ethanol and the remaining quarter had access to sucrose.

Table 2: Schedule showing when (and which) two-bottle tests were conducted in Experiment 1.

Day	Four Day Cycle	Two-Bottle Tests
1-16	1-4	
17-18		Ethanol/Sucrose #1
19-34	5-8	
35-36		Ethanol/Sucrose #2
37-44	9-10	
45-46		Ethanol/Water
47-54	11-12	
55-56		Sucrose/Water

eighth 4-day cycle. After two more 4-day cycles (the ninth and tenth) there were two-bottle tests between ethanol and water (Eth/Wat). Alternating shock and no-shock days continued through the eleventh and twelfth 4-day cycles, and the experiment finished with a pair of two-bottle tests between sucrose and water (Suc/Wat).

Measures. The rats were weighed daily throughout the experiment. Consumption was measured in ml and in ml/kg for both single and two-bottle tests. On two-bottle tests, in addition to recording consumption, fluid preference was calculated. For tests between ethanol and sucrose (Eth/Suc #1 and Eth/Suc #2) or between ethanol and water (Eth/Wat) the ethanol preference ratio was calculated as consumption of ethanol divided by total fluid consumption. A preference ratio of 0 would represent an absolute aversion for ethanol such that no ethanol was consumed. A preference ratio of 1.0 would represent an absolute preference for ethanol such that only ethanol was consumed. A preference ratio of 0.5 would represent neutrality; half of the fluid consumed was ethanol. For tests between sucrose and water (Suc/Wat) the sucrose preference ratio was calculated as sucrose consumption divided by total fluid consumption. All analyses were performed using SuperANOVA by Abacus Concepts. All reported F-values are significant at $p < .01$, unless otherwise mentioned.

Results

Figure 1 shows the mean (± SEM) consumption of water in ml/kg by groups E/S-Sh and E/S-NoSh during the five baseline sessions in the drinking environment. A one factor ANOVA with consumption on day 5 as the dependent measure confirmed that the groups did not differ from each other prior to the first 4-day cycle.

Mean (± SEM) body weight for each group on the 24 days when water was the only fluid available in the drinking environment (2 days per 4-day cycle) is shown in Figure 2. Mean body weight increased over the course of the experiment for both groups. A 2 x 24 group by water day mixed ANOVA revealed only a significant main effect of cycle, $F(23,506) = 153.103$.

The mean (± SEM) consumption of ethanol and sucrose during single bottle tests is shown in Figure 3. Consumption is consistently higher on the days when sucrose is the only fluid available than on the days when ethanol is the only fluid available regardless of whether these fluids are available on shock or no-shock days. A 2 x 2 x 12 group by fluid by cycle mixed ANOVA revealed significant main effects of fluid and cycle and a significant fluid by cycle interaction, $F(1,22) = 138.392$, $F(11,242) = 10.313$, $F(11,242) = 3.269$, respectively. The simple main effect of cycle was significant for both ethanol, $F(11,253) = 5.518$, and sucrose consumption, $F(11,253) = 6.986$. The simple main effect

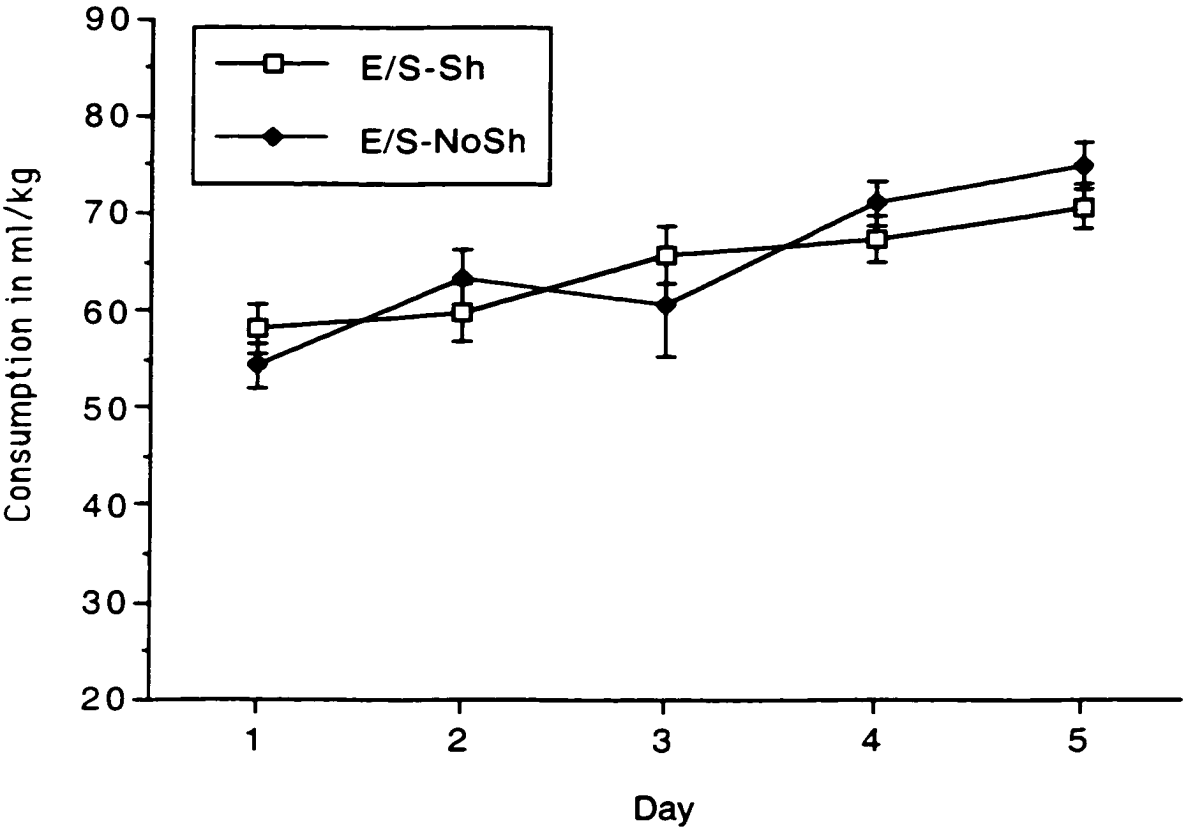

Figure 1: Mean (± SEM) water consumption in the drinking environment during the five-day baseline phase for Groups E/S-Sh and E/S-NoSh in Experiment 1.

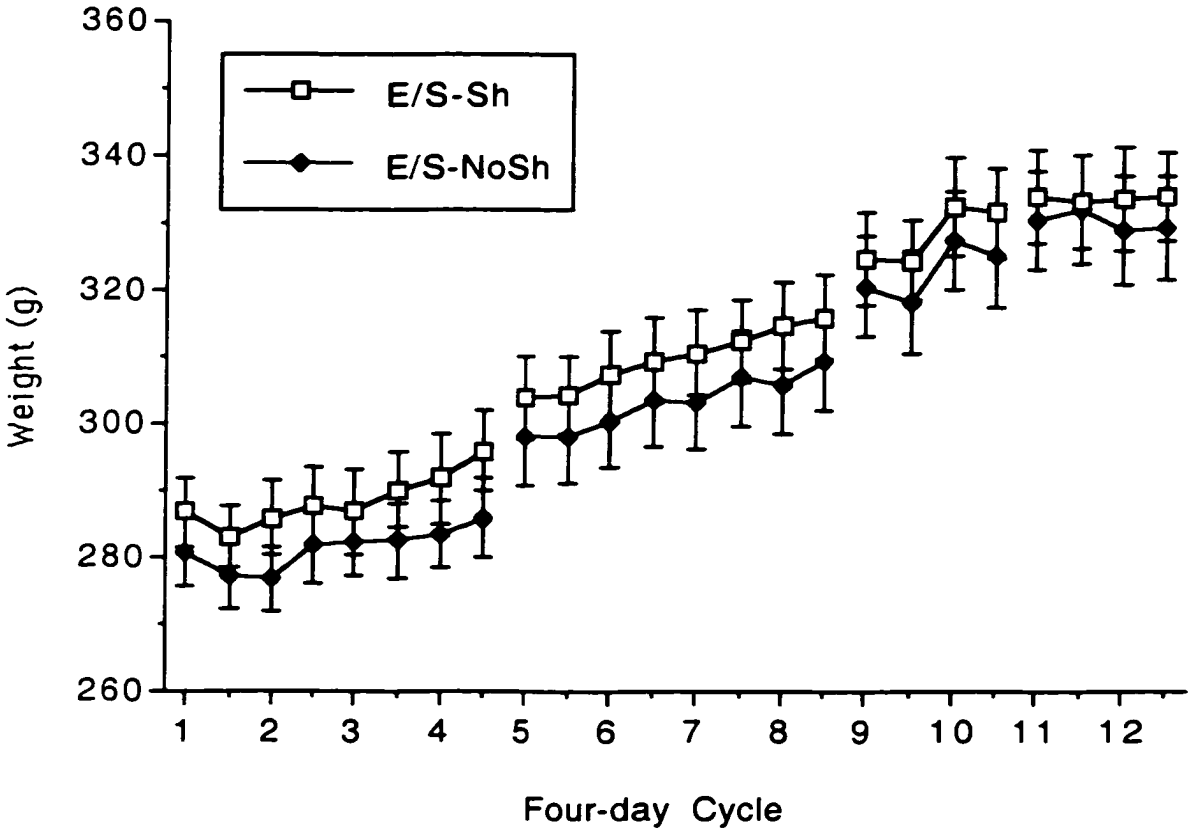

Figure 2: Mean (± SEM) body weight for the days when water was available in the drinking environment (2 days per cycle) during the 12 4-day cycles of the experiment for Groups E/S-Sh and E/S-NoSh in Experiment 1. Gaps in each curve indicate when each pair of two-bottle tests took place.

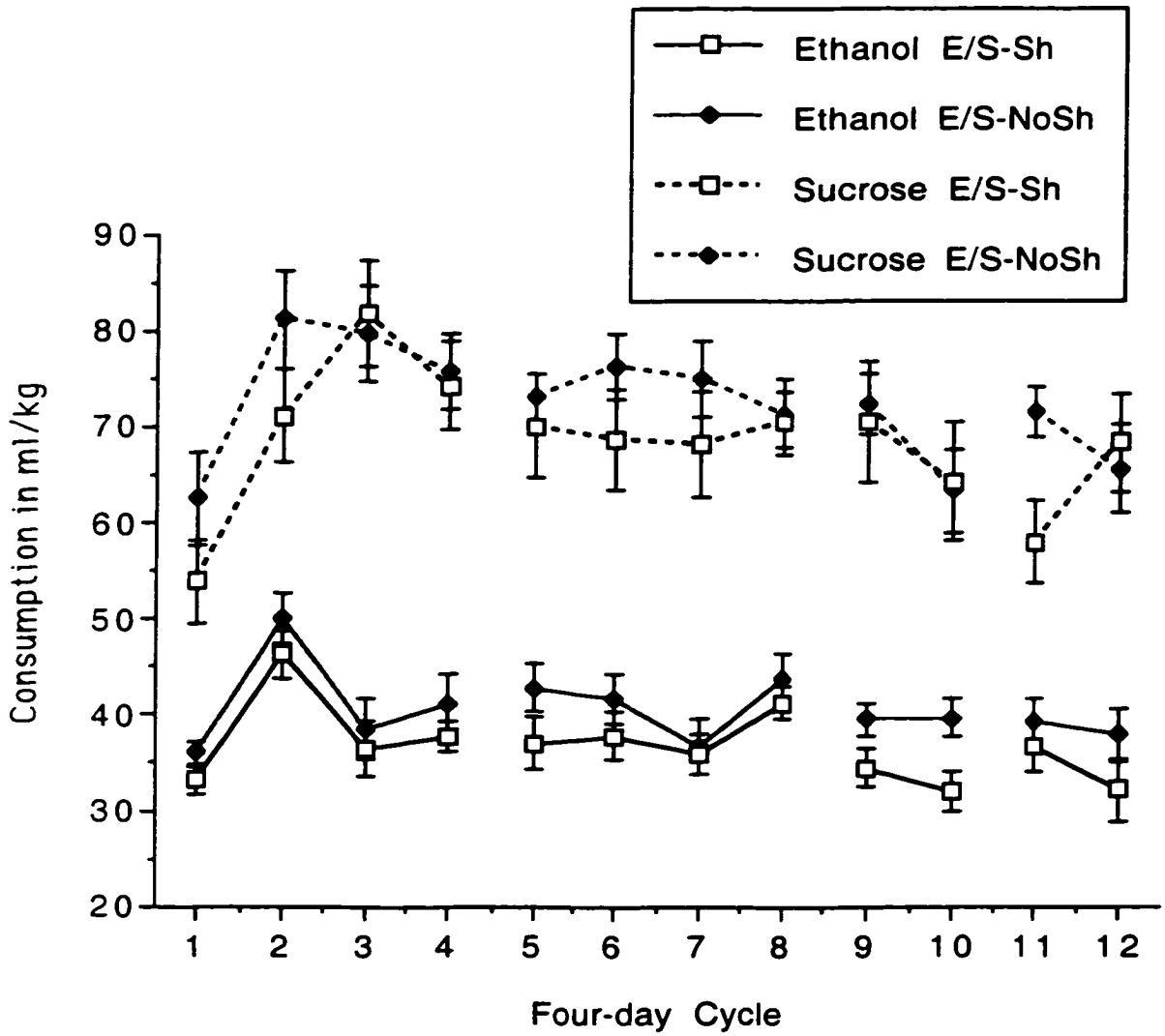

Figure 3: Mean (± SEM) consumption of ethanol and sucrose by groups E/S-Sh and E/S-NoSh during 1-bottle tests of cycles 1-12 in Experiment 1. Gaps in the curves represent the occurrence of 2-bottle tests.

of fluid was significant during every cycle, F(1,23) at least 37.962. There were no significant effects or interactions involving group, i.e. of whether access to the ethanol and sucrose solutions followed shock or occurred on no-shock days.

Since the biggest changes in consumption of ethanol and sucrose occurred during the first three cycles, these cycles were analysed separately. A 2 x 2 x 3 group by fluid by cycle mixed ANOVA identified no effect of group, but significant effects of fluid and cycle, $F(1,22) = 127.458$, $F(2,44) = 35.133$, and a significant fluid by cycle interaction, $F(2,44) = 11.601$. The simple main effect of cycle was significant for both ethanol, $F(2,46) = 20.184$, and sucrose consumption, $F(2,46) = 23.071$. Ethanol consumption during cycle 2 was significantly higher than the average of cycles 1 and 3, $F(1,46) = 38.734$. Ethanol consumption during cycle 1 did not differ from ethanol consumption during cycle 3. Sucrose consumption during cycle 1 was lower than the average of cycles 2 and 3, $F(1,46) = 44.471$. Sucrose consumption during cycles 2 and 3 did not differ. The simple main effect of fluid was significant during cycle 1, cycle 2, and cycle 3, $F(1,23) = 57.877$, 56.569, and 107.299.

Consumption on days when water was the only fluid available in the drinking environment (2 days per 4-day cycle) is shown in Figure 4. Water consumption appeared to decrease across cycles but did not appear to differ between groups. A 2 x 24 group by

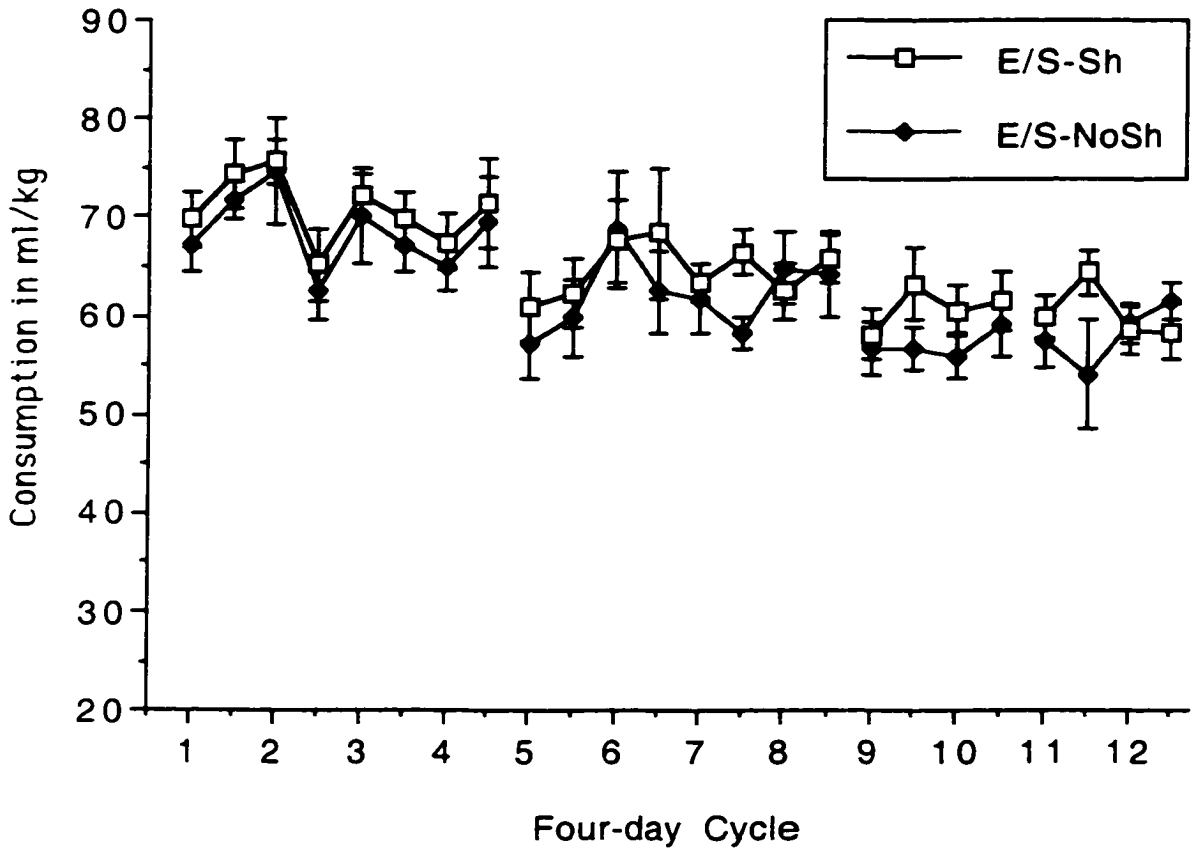

Figure 4: Mean (± SEM) water consumption by groups E/S-Sh and E/S-NoSh during 1-bottle tests of cycles 1-12 in Experiment 1. Gaps in the curves represent the occurrence of 2-bottle tests.

water day mixed ANOVA confirmed this observation. Only the main effect of water day was significant, $F(23,506) = 5.623$.

Mean (± SEM) preference during the shock and no-shock day of each pair of 2-bottle tests for Groups E/S-Sh and E/S-NoSh is shown in Figure 5. Each pair of 2-bottle tests was analysed separately using 2 x 2 group by test condition (shock day versus no-shock day) mixed ANOVAs. There was a significant effect of test condition for Eth/Suc #2, $F(1,22) = 12.292$. Ethanol preference was lower on the shock day than on the no-shock day for this pair of tests. There was a significant effect of group during the Eth/Wat preference tests, $F(1,22) = 4.968$, $p < .05$. Ethanol preference was higher for group E/S-Sh than for E/S-NoSh. Analyses revealed no significant effects or interactions for Eth/Suc #1 or for Suc/Wat preference tests.

Total consumption during the shock and no-shock day of each pair of preference tests is shown in Figure 6. Total consumption, like preference, was examined using 2 x 2 group by test condition (shock day versus no-shock day) mixed ANOVAs. There was a significant effect of test condition for Eth/Suc #1, $F(1,22) = 11.109$. Total consumption was greater during the no-shock day than during the shock day. For the second pair of preference tests, Eth/Suc #2, there was a significant effect of test condition, $F(1,22) = 4.465$, $p < .05$, and a significant interaction between group and test condition, $F(1,22) =$

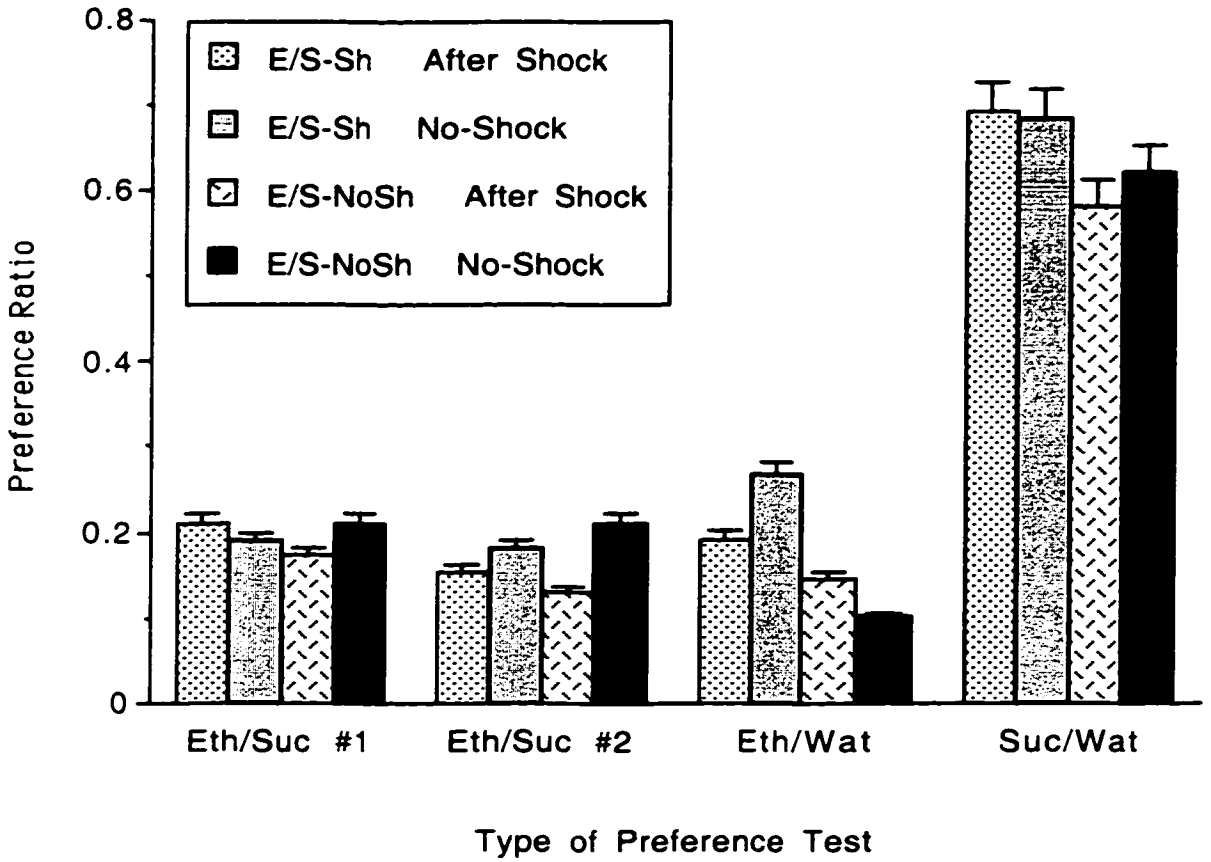

Figure 5: Mean (± SEM) preference ratios for Groups E/S-Sh and E/S-NoSh during shock and no-shock days of 2-bottle tests between ethanol and sucrose (Eth/Suc #1 and Eth/Suc #2), ethanol and water (Eth/Wat), and between sucrose and water (Suc/Wat) in Experiment 1. For the first three pairs of 2-bottle tests the preference ratio was calculated as ethanol consumption divided by total fluid consumption. For the final pair of 2-bottle tests the preference ratio was calculated as sucrose consumption divided by total fluid consumption.

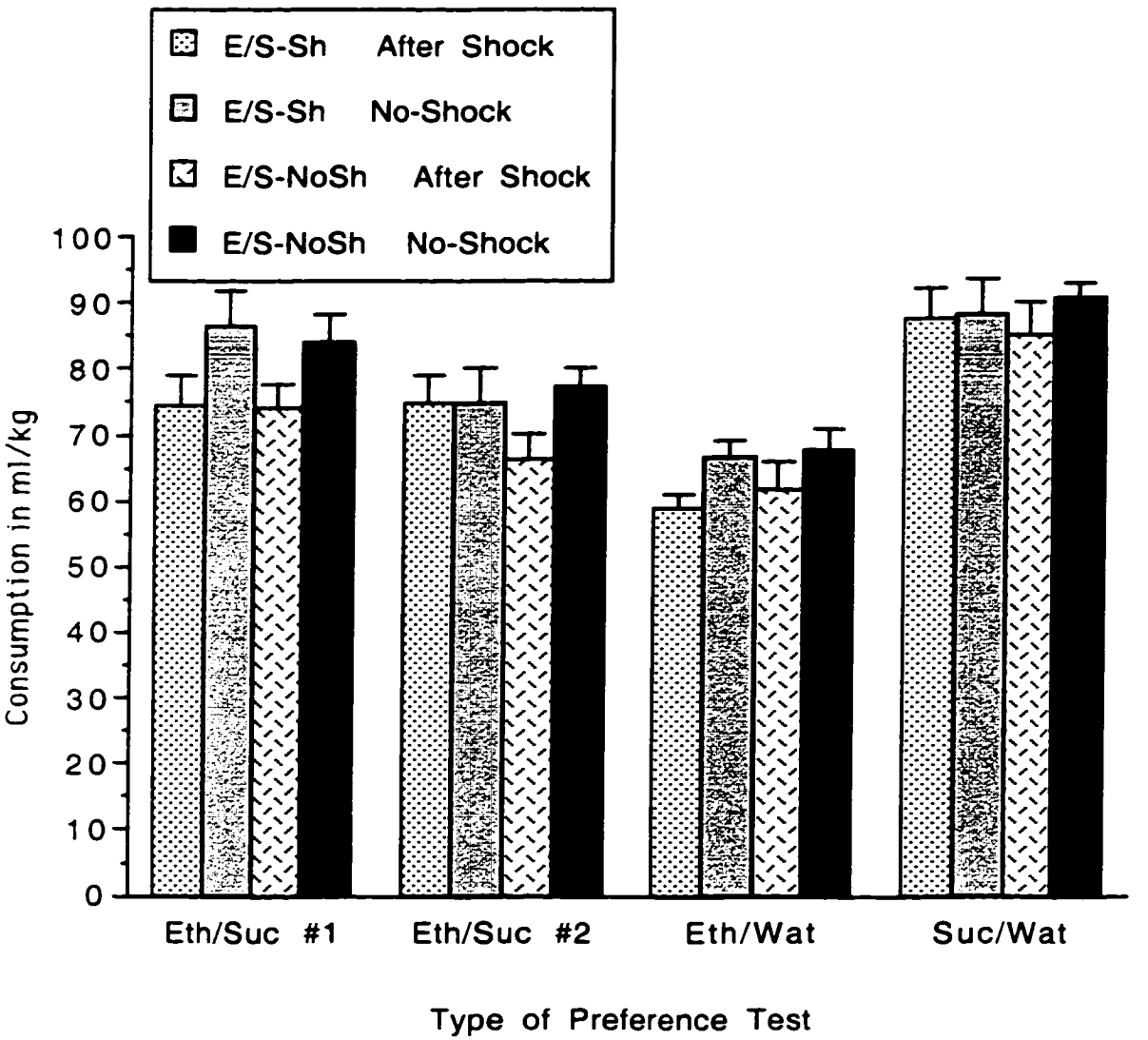

Figure 6: Mean (± SEM) total consumption for Groups E/S-Sh and E/S-NoSh during shock and no-shock days of 2-bottle tests between ethanol and sucrose (Eth/Suc #1 and Eth/Suc #2), ethanol and water (Eth/Wat), and between sucrose and water (Suc/Wat) in Experiment 1. For the first two pairs of 2-bottle tests total consumption was calculated as ethanol consumption plus sucrose consumption. For the third pair of 2-bottle tests, total consumption was calculated as ethanol consumption plus water consumption. For the final pair of 2-bottle tests total consumption was calculated as sucrose consumption plus water consumption.

4.532, $p < .05$. Total consumption was greater during the no-shock day than during the shock day. Simple effects analysis revealed only a significant simple main effect of test condition for Group E/S-NoSh, $F(1,11) = 7.586$, $p < .05$. Total consumption was greater on the no-shock day than on the shock day of the test. The effect of test condition was also significant for the Eth/Wat preference tests, $F(1,22) = 10.366$. Again, total consumption was greater on the no-shock day than on the shock day. There were no significant effects or interactions for the Suc/Wat preference tests.

Discussion

Contrary to predictions from the Relief Hypothesis, animals that had access to ethanol following shock, Group E/S-Sh, did not increase their ethanol consumption across cycles in absolute terms, or relative to their sucrose consumption, or relative to the ethanol consumption of Group E/S-NoSh. In fact, the pattern of fluid consumption by Groups E/S-Sh and E/S-NoSh was very similar. During cycle 1, consumption of ethanol and sucrose was low for both groups as compared to their baseline consumption of water. This suppressed consumption of ethanol and sucrose solutions during cycle 1 could be the result of a generalized post-shock deficit, finickiness, or neophobia. These explanations can be assessed by examining consumption during the first exposure to a flavoured solution. If the observed pattern of consumption was the result of a generalized

post-shock deficit, then consumption of all fluids should have been suppressed immediately after shock as compared to baseline. This is not what happened. Rats that had access to ethanol immediately following their first shock session (n = 6) consumed an average of 33.20 ml/kg as compared to the 47.77 ml/kg consumed by rats that had access to sucrose following their first shock session (n = 6). Rats that had access to water immediately after their first shock session (n = 12) consumed an average of 67.17 ml/kg. So, consumption was suppressed (relative to an average of 71.42 ml/kg for all animals on the fifth day of baseline) following the first shock session only for those animals that had access to one of the flavoured solutions. Therefore, the observed pattern of consumption cannot be explained by a generalized post-shock deficit.

Finickiness is the suppression of consumption of bitter-tasting substances such as quinine- or saccharin-adulterated water, but not unadulterated water, following exposure to uncontrollable shock (Ferguson & Job, 1997). In order to say that finickiness has occurred it would be necessary to show that consumption of ethanol and sucrose was significantly different from baseline in shocked but not in unshocked animals and that water consumption was unaffected by the shock manipulation. Rats that had access to ethanol, sucrose, or water immediately following their first shock session consumed only 46.3, 69.67, or 94.85% of the volume that they had consumed on the last day of baseline.

Rats that were not shocked and had access to ethanol, sucrose or water on the day after baseline ended (before they had ever been shocked) consumed 47.16, 79.53, or 101.44% of the volume of fluid that they had consumed on the last day of baseline. Ethanol and sucrose consumption were each substantially different from baseline regardless of whether the animals were shocked. Water consumption was unaffected by the shock manipulation. Since the suppressed consumption of the unfamiliar ethanol and sucrose solutions was not specific to animals that had been shocked, finickiness cannot be said to have occurred.

Neophobia, the tendency to be cautious about new substances, accounts for the suppressed consumption of the novel ethanol and sucrose solutions (regardless of shock treatment) during cycle 1. The increased consumption of ethanol and sucrose during cycle 2 can be described as recovery from neophobia. As a result of the higher ethanol consumption in cycle 2, the rats may have encountered the negative consequences of ethanol ingestion. Cappell (1987) suggested that an orally consumed dose of 1.5 g/kg absolute ethanol may be tension reducing but that higher doses may be aversive since there is a relatively small "'margin of safety' between anxiolytic activity and impairment" (Cappell, 1987; p. 244) and drug-induced impairment may be stressful in itself. During the first cycle, the rats consumed a mean of 34.63 ml/kg of the ethanol

solution or 1.73 g/kg absolute ethanol in one hour. During cycle 2 this increased to 48.17 ml/kg of the solution or 2.41 g/kg absolute ethanol, beyond Cappell's postulated tension reducing range. The reduced consumption observed during cycle 3 (and subsequent cycles) could reflect negative associations with high levels of consumption, regulation of ethanol intake at positively reinforcing levels, or minimum fluid requirements by the (thirsty) rats.

All of the rats in this experiment were fluid deprived, so thirst has to be considered as a factor motivating consumption in the drinking environment (regardless of what solution is available). However, this being said, there is no reason to expect that thirst alone should motivate greater consumption of sucrose than ethanol as was observed here. If thirst were the only factor motivating consumption, then consumption of the two solutions should have been approximately equivalent.

The various two-bottle tests were included in order to assess whether consumption of some fluids was altered more or less than other fluids following shock when there was more than one fluid choice. Both groups showed an aversion for ethanol (ethanol preference ratios less than 0.5) on all two-bottle tests where ethanol was one of the fluid alternatives. During the Eth/Wat two-bottle tests, there was a significant difference between groups, such that Group E/S-Sh displayed less of an ethanol aversion

than Group E/S-NoSh. Although this difference was in the direction predicted by the Relief Hypothesis, over the three pairs of 2-bottle tests Group E/S-Sh did not show a consistently higher ethanol preference than Group E/S-NoSh nor did Group E/S-Sh show a consistently higher ethanol preference on shock days than on no-shock tests. According to the hypothesis, ethanol should have been more reinforcing (and consequently more preferred) following shock than on no-shock days. During Eth/Suc #2 ethanol aversion was less during the no-shock day than during the shock day. There was no indication that access to ethanol immediately following shock was more reinforcing than access to ethanol at other times.

EXPERIMENT 2

In this experiment, instead of having access to fluid after shock rats had access to fluid in a distinctive drinking environment before shock. One group of thirsty rats, Group E/S-Sh, had access to ethanol before alternate shock sessions and to an isocaloric sucrose solution before the others. Water was available for an hour on intervening no-shock days. A second group of animals, Group E/S-NoSh, had access to ethanol or sucrose in a distinctive environment on no-shock days and to water before shock sessions. Since the distinctive drinking environment always preceded shock for both groups of animals, placement in this environment was expected to act as a signal for shock, causing the rats to become anxious or fearful in this environment. Only rats in Group E/S-Sh had the opportunity to consume ethanol during this signal for shock and therefore only animals in this group were expected to learn that ethanol was tension reducing and thus to increase their consumption. Therefore, according to the TRH, rats that received ethanol and sucrose on shock days were expected to increase their ethanol consumption relative to the other group, for which access to ethanol in the distinctive environment was negatively correlated with shock.

Method

Subjects

The rats in this experiment were 24 adult male Sprague Dawley rats from Charles River Canada which were housed as in the previous experiment. On the first day of the experiment, these rats had a mean weight of 360 g, with a range between 324 and 410 g.

Apparatus

Drinking and shock environments, ethanol and sucrose solutions were all as in Experiment 1.

Procedure

The procedure for this experiment was similar to that of the first experiment. The main difference was that this time fluid was available for 1 h before the shock session instead of after. The 4-day cycles and 2-bottle tests followed the same sequence as in the first experiment (see Tables 1 and 2, respectively). In addition, this experiment also included two additional 4-day cycles, Cycles 13 and 14, following the sucrose and water 2-bottle tests. During these two cycles access to fluid continued as outlined in Table 1 but no shocks were administered. Rats were returned to their home cages following all sessions in the drinking environment.

Measures. Body weight, consumption, and preference ratios were recorded and/or calculated as in Experiment 1. As before, all reported F-values are significant at $p < .01$, unless otherwise mentioned.

Results

Mean (± SEM) consumption of water in the drinking environment during the five days of baseline is shown in Figure 7. Groups do not appear to differ on any of these days. A one factor ANOVA on the consumption data from Day 5 confirmed that consumption by the two groups, E/S-Sh and E/S-NoSh, did not differ at the end of baseline.

Figure 8 shows the mean (± SEM) body weights for Groups E/S-Sh and E/S-NoSh on the two water days of each 4-day cycle. Body weight appeared to increase across both the shock cycles (1-12) and no-shock cycles (13 and 14). Separate analyses were conducted on body weight for the 12 cycles during which shock was delivered and for the two post-shock cycles (Cycles 13 and 14). A 2 x 24 group by water day mixed ANOVA revealed a significant effect of cycle, $F(23,506) = 177.627$, for the 12 shock cycles. A 2 x 4 group by water day mixed ANOVA on body weight during the two post shock cycles also revealed a significant effect of cycle, $F(3,66) = 8.678$. There were no significant effects or interactions involving group.

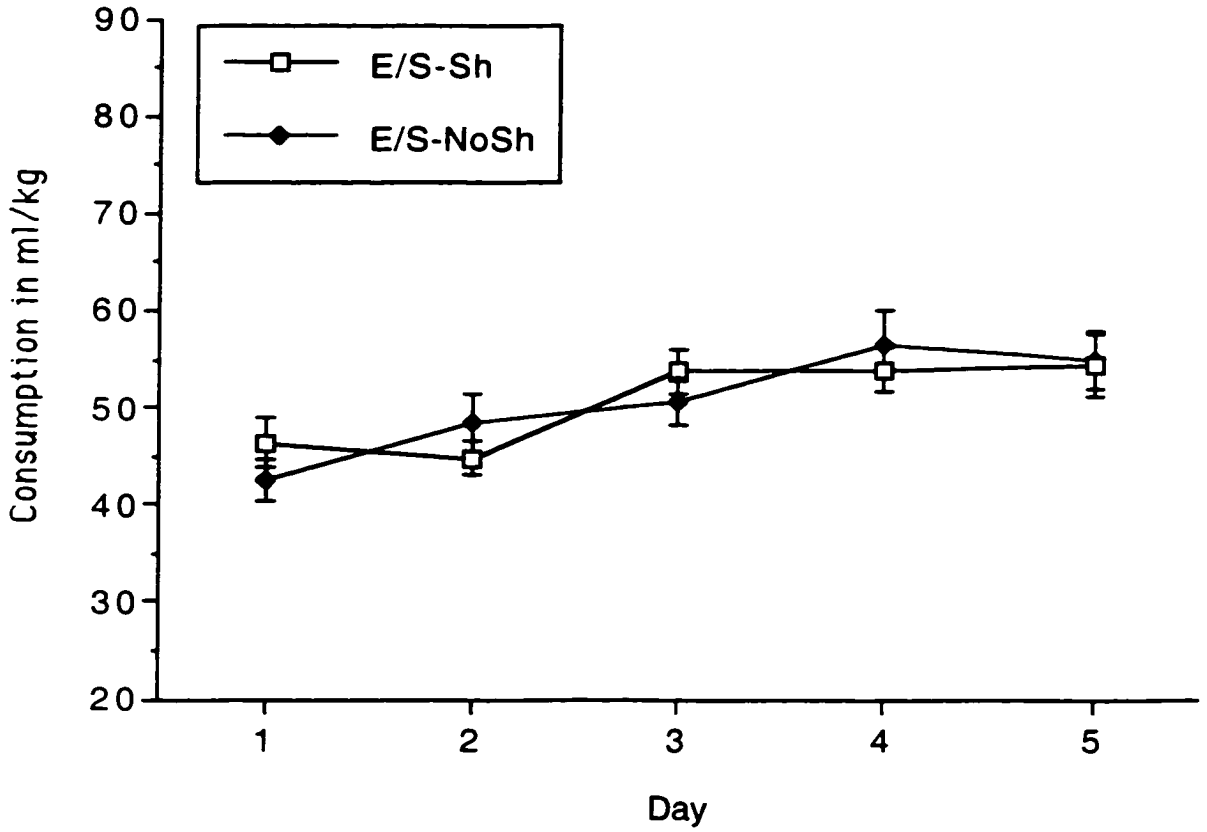

Figure 7: Mean (± SEM) consumption of water by Groups E/S-Sh and E/S-NoSh in the drinking environment during the five baseline days of Experiment 2.

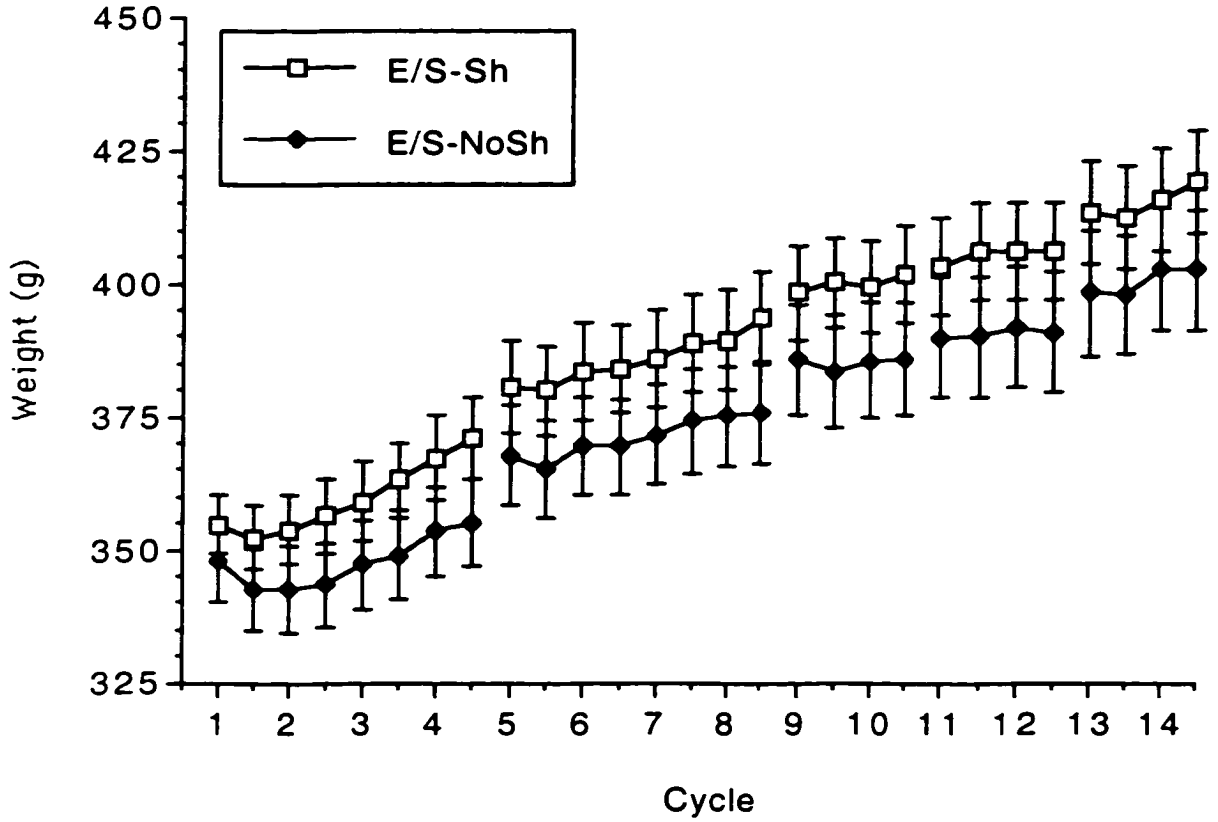

Figure 8: Mean (± SEM) body weight on the 2 water days of each 4-day cycle in Experiment 2. During cycles 1-12, this represents body weight on no-shock days for Group E/S-Sh and on shock days for Group E/S-NoSh. No shocks were delivered to either group during Cycles 13 and 14.

Consumption of ethanol and sucrose in ml/kg during single bottle tests for Group E/S-Sh and Group E/S-NoSh is shown in Figure 9. This represents consumption on 1-bottle, shock days for Group E/S-Sh and on no-shock days for Group E/S-NoSh during cycles 1-12. No shocks were administered to either group during cycles 13 and 14. For both groups, consumption of both ethanol and sucrose increased from cycle 1 to cycle 2. Ethanol consumption then decreased during cycle 3 and then remained relatively stable at this level. Sucrose consumption remained at the high level observed in cycle 2. Thus, sucrose consumption was substantially higher than ethanol consumption for both groups. Consumption during the 12 cycles when shocks were administered was analysed by a 2 x 2 x 12 group by fluid by cycle mixed ANOVA. This analysis revealed significant main effects of fluid and cycle and a significant interaction between fluid and cycle, $F(1,22) = 73.880$, $F(11,242) = 10.634$, and $F(11,242) = 6.666$, respectively. There were no significant effects involving group on 1-bottle tests. Simple effects analyses revealed significant effects of cycle for both ethanol, $F(11,253) = 4.922$, and sucrose, $F(11,253) = 12.353$. The simple effect of fluid was significant for each cycle from 2 to 12, $F(1,23)$ at least 25.319. The first cycle was the only one in which ethanol and sucrose consumption were not significantly different.

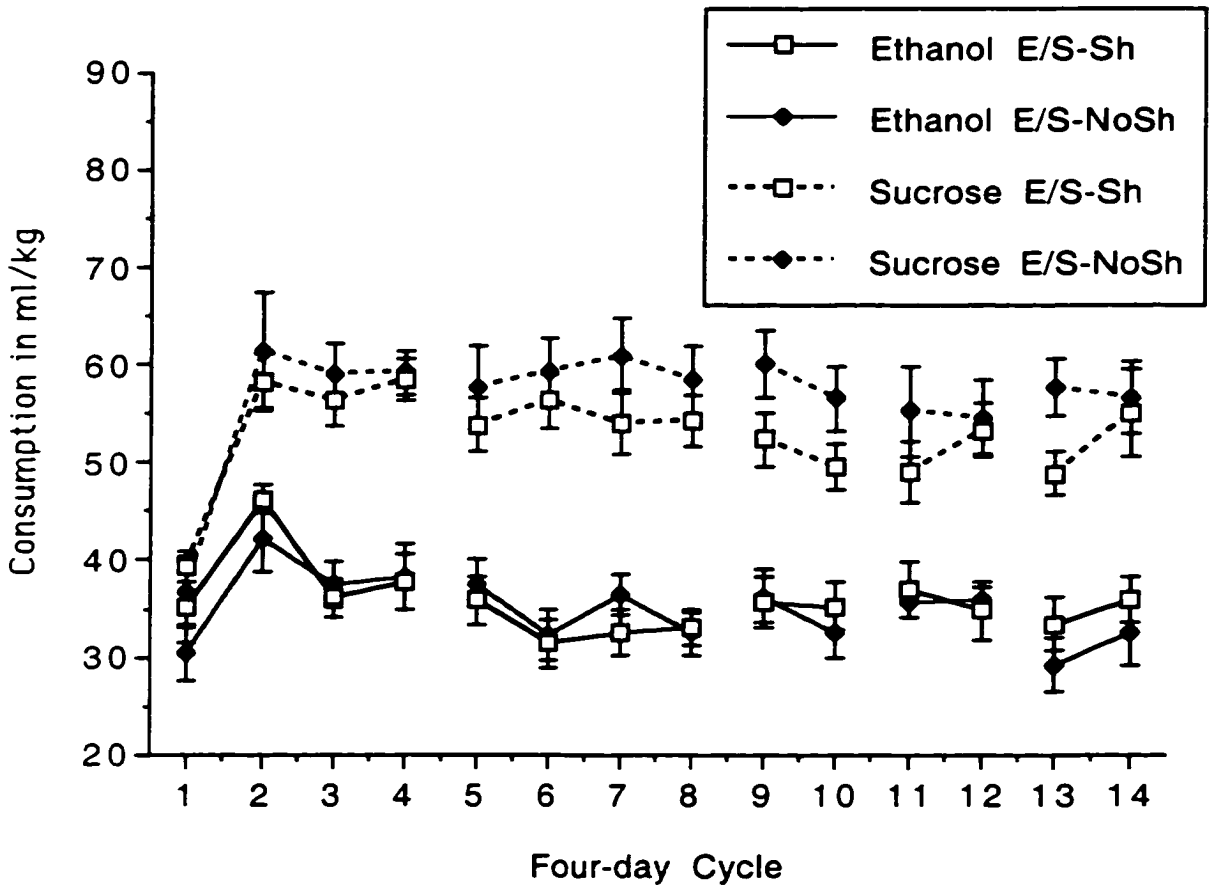

Figure 9: Mean (± SEM) consumption of ethanol and sucrose in ml/kg during 1-bottle tests of Experiment 2. Group E/S-Sh had access to ethanol or sucrose before shock sessions (except during Cycles 13 and 14 when no shocks were delivered). Group E/S-NoSh had access to ethanol or sucrose on alternate no-shock days.

Since it appeared that most changes in consumption occurred during the first three cycles (see Figure 9), these cycles were examined in a 2 x 2 x 3 group by fluid by cycle mixed ANOVA. This analysis revealed significant effects of fluid and cycle, $F(1,22) = 48.345$ and $F(2,44) = 39.334$. The interaction between fluid and cycle was also significant, $F(2,44) = 11.117$. Simple effects analysis showed that the effect of cycle was significant for both ethanol, $F(2,46) = 12.247$, and sucrose consumption, $F(2,46) = 37.991$. Subsequent contrast analysis showed that ethanol consumption during cycle 2 was higher than the average of cycles 1 and 3, $F(1,46) = 21.645$. Ethanol consumption during cycle 3 did not differ from ethanol consumption during cycle 1. Sucrose consumption during cycle 1 was lower than the average of cycles 2 and 3, $F(1,46) = 75.353$. Consumption during cycle 2 did not differ from consumption during cycle 3. The simple effect of fluid was non-significant during cycle 1. However, sucrose consumption was significantly greater than ethanol consumption during cycle 2, $F(1,23) = 25.319$, and cycle 3, $F(1,23) = 103.627$.

Consumption of ethanol and sucrose during the two post-shock cycles (Cycles 13 and 14) was examined in a 2 x 2 x 2 group by fluid by cycle mixed ANOVA. This analysis revealed only a significant effect of fluid, $F(1,22) = 86.166$. All of the rats consumed more sucrose than ethanol.

Water consumption by the two groups in ml/kg is shown in Figure 10. Group E/S-Sh had access to water on no-shock days while Group E/S-NoSh had access to water on shock days. There were no apparent differences in water consumption between groups during any cycle. This observation was confirmed by a 2 x 24 group by water day mixed ANOVA. This analysis revealed no significant effects or interactions involving group. However, there was a significant effect of water day, $F(23,506) = 8.607$.

Both groups showed an aversion for ethanol (a preference ratio less than 0.5) during two-bottle tests between ethanol and sucrose or between ethanol and water for both shock and no-shock days. Sucrose was highly preferred to ethanol (1 - ethanol preference ratio) and also preferred to water (see Figure 11). Preference ratios for each pair of preference tests were examined by 2 x 2 group by test condition (shock versus no-shock) mixed ANOVAs. These analyses revealed only a significant interaction between group and test condition, $F(1,22) = 4.327$, $p < .05$, for the 2-bottle tests between ethanol and water. None of the simple effects describing this interaction were significant.

Figure 12 shows mean (± SEM) total consumption on shock and no-shock days of each pair of 2-bottle tests for Groups E/S-Sh and E/S-NoSh. These data were examined using 2 x 2 group by test condition mixed ANOVAs for each pair of 2-bottle tests. There was a significant effect of group, $F(1,22) = 5.405$, $p < .05$, for the second pair of 2-bottle

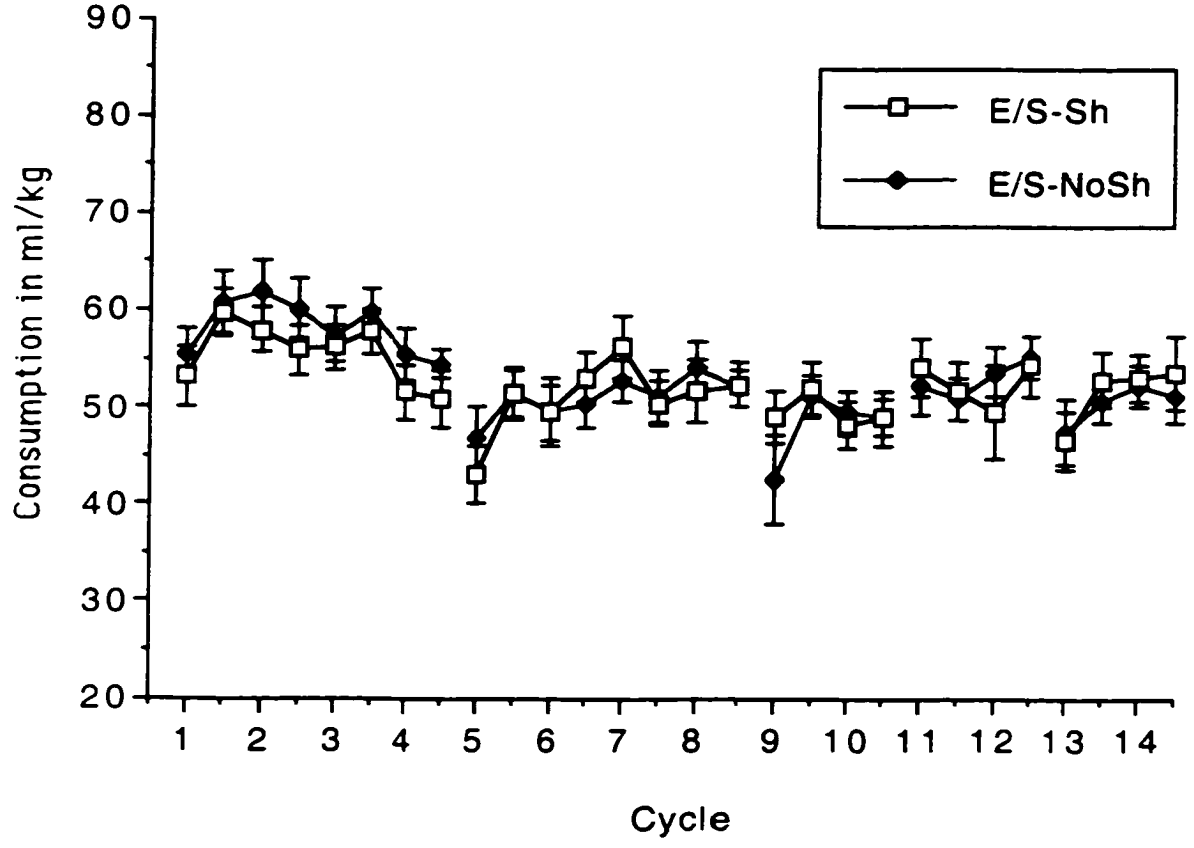

Figure 10: Mean (± SEM) consumption of water in ml/kg during 1-bottle tests of Experiment 2. Group E/S-Sh had access to water on no-shock days. Group E/S-NoSh had access to water before shock sessions except during Cycles 13 and 14 when no shocks were delivered.

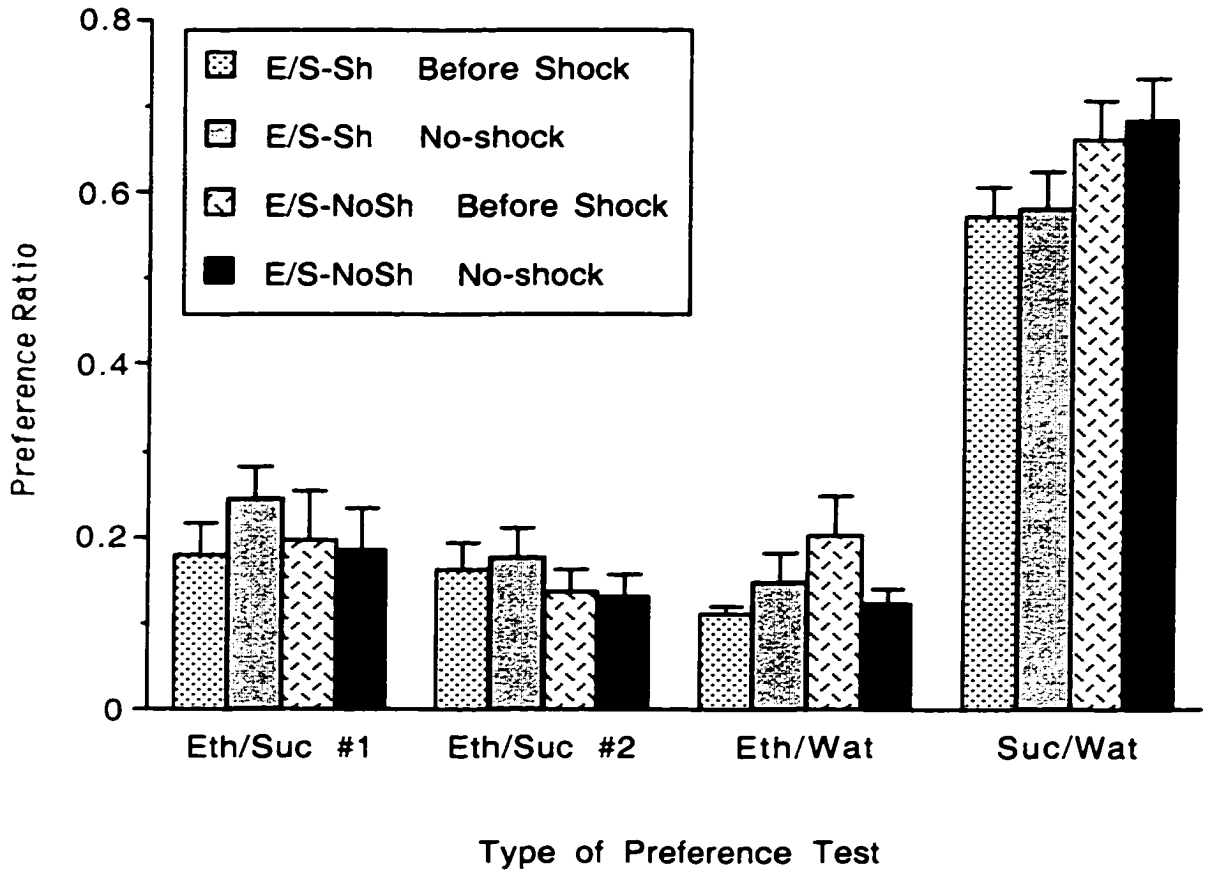

Figure 11: Mean (± SEM) preference ratios (consumption of first fluid/total fluid consumption) for Shock and No-Shock Days during each pair of 2-bottle tests for rats in Group E/S-Sh and Group E/S-NoSh in Experiment 2.

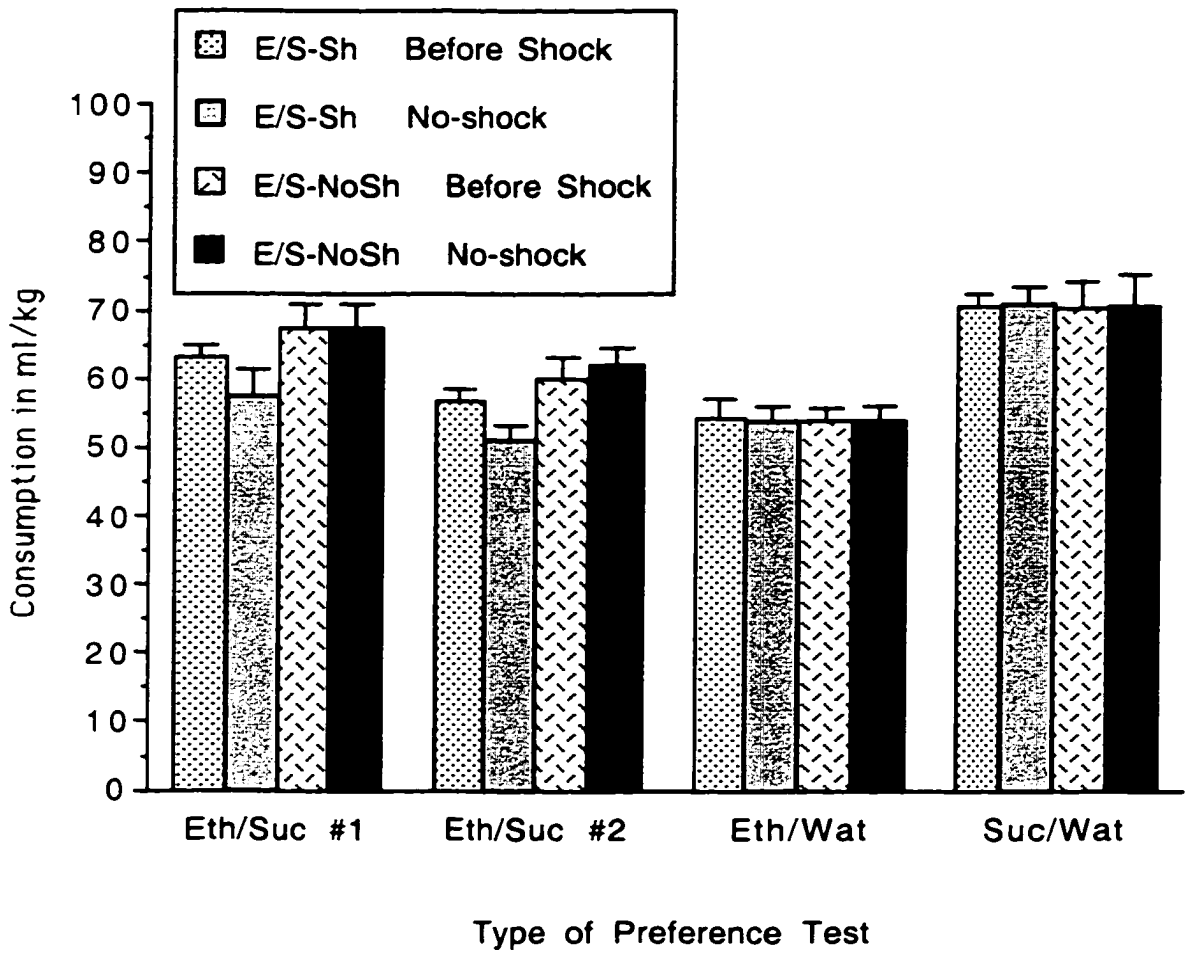

Figure 12: Mean (± SEM) total consumption (consumption of first fluid + consumption of second fluid) for Shock and No-Shock Days during each pair of 2-bottle tests for rats in Group E/S-Sh and Group E/S-NoSh in Experiment 2.

tests between ethanol and sucrose. Group E/S-NoSh drank more fluid than Group E/S-Sh. There were no other significant effects or interactions.

Discussion

Consumption did not differ between groups and was consistently higher on days when sucrose was available than on days when ethanol was available. Ethanol consumption did not increase across cycles. On all 2-bottle tests involving ethanol, both groups showed an ethanol aversion (preference ratios less than 0.5). Sucrose was strongly preferred to both ethanol and water. None of these observations are consistent with predictions from the Tension Reduction Hypothesis.

As in Experiment 1, the biggest changes in consumption occurred during the first three cycles. In Experiment 1, sucrose consumption was significantly greater than ethanol consumption during each cycle. In Experiment 2, sucrose consumption was not significantly greater than ethanol consumption until cycle 2. In both experiments, sucrose consumption started low and increased across cycles 2 and 3. Ethanol consumption was low during cycle 1 and increased during cycle 2 but then decreased during cycle 3. During the first cycle, the rats consumed a mean of 33.73 ml/kg of the ethanol solution or 1.69 g/kg absolute ethanol. During cycle 2 this increased to 47.23 ml/kg of the solution or 2.36 g/kg absolute ethanol, well beyond Cappell's (1987) tension reducing range. So, as

in Experiment 1, ethanol consumption beyond the second cycle could represent negative associations with high levels of consumption, regulation of ethanol intake at positively reinforcing levels, or minimum fluid requirements by the (thirsty) rats. Rats in these experiments were 23-h fluid deprived at the time of fluid access. Since rats consumed less ethanol than sucrose or water during drinking sessions, it is impossible to determine whether their consumption was motivated by biological necessity (and this amount coincidentally coincided with Cappell's tension reducing dose) or whether the animals were in fact regulating their ethanol intake in order to attain tension reduction. However, if the rats were regulating their ethanol consumption, then similar levels of ethanol consumption would have been expected even when other fluid choices were available as was the case during 2-bottle tests. This did not happen. On 2-bottle tests, in which ethanol was one of the fluid choices, not only were ethanol preferences very low (representing strong ethanol aversions) but ethanol consumption was also much lower than during 1-bottle tests.

These two experiments share many design elements with the Cox and Stainbrook (1977) experiment. For example, all of the experiments have three fluids: ethanol, sucrose, and water, which are available on different days. There are groups which have ethanol or sucrose contiguous with shock (Groups E/S-Sh Before and After) and groups

which have ethanol or sucrose contiguous with no-shock (Groups E/S-NoSh Before and After). Whether fluid is available immediately before or immediately after shock was manipulated in both studies.

In spite of these basic similarities between the studies, there are also differences which may have implications for interpreting the results. Although a 5% ethanol solution was used in my experiments and by Cox and Stainbrook (1977), the solution differed in that mine was an ethanol/saccharin solution and theirs was an ethanol/sucrose solution. In addition to this, the sucrose concentration used by Cox and Strainbrook was slightly higher than mine and theirs was not adulterated by acetic acid. While this means that the solutions used by Cox and Stainbrook were slightly more caloric than mine and probably more palatable, there is no reason to expect a greater change in preference with experience in their experiment than in mine although I would have expected relative differences if Cox and Stainbrook had included two-bottle tests of ethanol against water or sucrose against water.

There were differences in the number of 1-bottle days between studies, and these might be considered more important than the differences in solutions. Cox and Stainbrook (1977) included only eight 1-bottle days (2 ethanol, 2 sucrose, and 4 water) prior to the first 2-bottle tests. I included twice as many 1-bottle tests before the first 2-

bottle tests in order to allow consumption on 1-bottle days to stabilize before the introduction of the second bottle, yet I did not observe significant differences between groups on ethanol/sucrose 2-bottle tests. The absence of a group effect in my experiment leads me to question the conclusion put forward by Cox and Stainbrook that their finding of greater ethanol preference in their Group E/S-Sh Before than in Group E/S-NoSh Before "emerged because [their] new paradigm allowed sufficient opportunity for [their] animals to learn about the anxiety-reducing property of alcohol" (p. 29).

Although there are fewer 1-bottle days in Cox and Stainbrook's (1977) experiment, their animals experience more 2-bottle shock tests between ethanol and sucrose because these tests are conducted in pairs and because no other 2-bottle tests, e.g. ethanol and water, were included. Unfortunately, Cox and Stainbrook collapse the data across their six 2-bottle tests so that we are unable to see the emergence of the increased preference by animals in Group E/S-Sh Before.

Perhaps the most interesting interpretation of the discrepant results comes from a consideration of the duration of drinking and shock intervals incorporated into the studies. My experiments incorporated a 1-h shock session (consistent with previous work, Fidler & LoLordo, 1996) and a 1-h drinking session that occurred so that the animals would be drinking in temporal proximity to the shock session. For the Fluid Before

groups, this arrangement allowed some of ethanol's effects to take place prior to or during placement in the shock box. By contrast, Cox and Stainbrook (1977) used shock and drinking sessions each of 15 minutes in duration. These shorter durations for drinking and shock sessions might ensure that most of ethanol's intoxicating effects are not experienced until after the rat has been returned to its home cage even in Group E/S-Sh Before. So, Cox and Stainbrook's Group E/S-Sh Before might drink more ethanol (and show less of an aversion to it relative to the other groups) in order to obtain its effects during a period of relief, rather than during a period of tension. For Group E/S-Sh After in their experiment and mine, ethanol's effects may occur too long after the shock session to be reinforcing so that these groups do not increase their ethanol consumption or preference relative to Group E/S-NoSh After.

EXPERIMENT 3

When rats were given continuous and simultaneous access to two solutions (ethanol and sucrose or ethanol and water) (Fidler & LoLordo, 1996) it might have been difficult for them to determine which post-ingestive consequences were associated with each solution. In the two experiments just discussed, thirsty rats had access to three different solutions, ethanol, sucrose, and water, on different days with varying relations to shock. This arrangement should have made it easier for the rats to associate particular tastes with particular outcomes. However, with this procedure the rats had fewer exposures to individual solutions and their associated consequences. In order to increase the number of solution exposures and to keep the relationships between solutions and consequences for individual animals simple, in the next experiments I assigned different groups of rats to different solutions. That is, one group of rats always had access to ethanol in the drinking environment, a second to sucrose, and a third to water. Every time that the rats were placed in the drinking environment they received the same solution and placements in the drinking environment were always followed by shock sessions in another environment. Under these circumstances there should have been a large number of cues associated with the drinking environment that predicted that shock was to follow. Therefore, the rats were expected to become anxious in the drinking environment in

anticipation of the shock session to follow. If the Tension Reduction Hypothesis is correct then rats in the Ethanol group should learn to drink ethanol in order to relieve this anxiety. Further, animals in the Ethanol Group should display less fear/anxiety when it is measured via fecal bolus production (Inoue et al., 1993) than animals in the Sucrose and Water Groups.

Method

Subjects

The subjects in this experiment were 42 adult male Sprague Dawley rats from Charles River Canada. The rats were housed as described in Experiment 1. The average weight of the rats on the first day of the experiment was 357 g with a range from 325 to 396 g. Food was freely available in the home cages throughout the experiment. Fluid access was as described in the procedure section.

Apparatus

Environments. Drinking and shock environments were as specified in Experiment 1.

Fluids. The solutions in this experiment were changed from the 5% ethanol and isocaloric sucrose solutions used in the previous experiments to 10% ethanol and

corresponding new isocaloric sucrose. With the 10% ethanol solution, the rats had to consume less ethanol to achieve an intoxicating dose and since this involved a smaller fluid volume the ethanol the ethanol that was consumed should have been absorbed more rapidly. The 10% (v/v) ethanol solution was made from 95% ethyl alcohol and tap water and sweetened with 0.1% (w/v) saccharin. The 14.02% (w/v) sucrose solution was made with 1.0% (v/v) acetic acid (Fidler, 1992). Tap water was still the third fluid.

Procedure

Approximately 24 hours before the start of the experiment water bottles were removed from the rats' home cages. Subsequently, the rats had access to fluid for one hour per day in either the drinking environment or in the home cage.

For 36 rats the experiment began with a series of alternating shock and no-shock days. On shock days, rats were removed from their cages, weighed and transported to the drinking environment where they had access to one of ethanol, sucrose, or water for one hour. Rats were removed from the drinking environment and transported to shock boxes where they received 0.8 mA, 2-s footshocks on a FT 60-s schedule for one hour. At the end of the shock session rats were returned to their home cages. On no-shock days, rats remained in their home cages where they had access to water during the same hour that they had access to fluid on the shock day. After the twenty-eighth shock day, shocks were

discontinued but the alternating cycle of fluid access in the drinking environment one day and water in the home cage the next day continued for six more days. So, the rats had access to fluid in the drinking environment 31 times. The first 28 exposures were followed by a shock session and the final 3 were followed by return to the home cage.

Rats were randomly assigned to squads and groups. There were nine squads of four animals each and three of these squads were designated as the Ethanol Group, three as the Sucrose Group and three as the Water Group (so, for each group n = 12). Squads were run out of phase so that on one day five squads had a shock day and the remaining four squads had a no-shock day. On the next day, five squads had a no-shock day while the other four squads had their shock day. The squads were run in the following sequence: Ethanol 1, Sucrose 1, Water 1, Ethanol 2, Sucrose 2; and Water 2, Ethanol 3, Sucrose 3, Water 3. Animals in Ethanol 1 had access to fluids at the same time as Water 2; Sucrose 1 at the same time as Ethanol 3; Water 1 at the same time as Sucrose 3, and Ethanol 2 at the same time as Water.

Six additional animals were not moved to the drinking environment or to the shock boxes but rather had all of their fluid access in the home cage. Like the other rats, these rats, the Home Cage Ethanol group, had access to fluid for one hour per day. They

had access to water one day and to ethanol the next. These rats had access to fluid at the same time as the rats in Sucrose 2.

Measures. Body weights were recorded for each of the days that the rats were taken to the drinking environments. Consumption in the drinking environments and in the home cages was recorded in ml and ml/kg throughout the experiment. For drinking environment sessions 23 to 31, the number of fecal boli deposited in that environment by each animal was recorded. Similarly, fecal boli were counted in the shock boxes during sessions 23 to 28.

Results

Mean body weight (± SEM) for the Ethanol, Sucrose, and Water groups immediately prior to sessions in the drinking environment is shown in Figure 13. Body weight decreased over the first five cycles for all groups as the rats adjusted to the fluid deprivation schedule and then increased across the remaining cycles. Although the ethanol group appeared to be consistently heavier than the other groups, a 3 x 28 group by cycle mixed ANOVA did not reveal a significant effect of group. There was a significant effect of cycle, $F(27,891) = 144.846$, and a significant group by cycle interaction, $F(54,891) = 1.821$. The simple main effect of cycle was significant for each

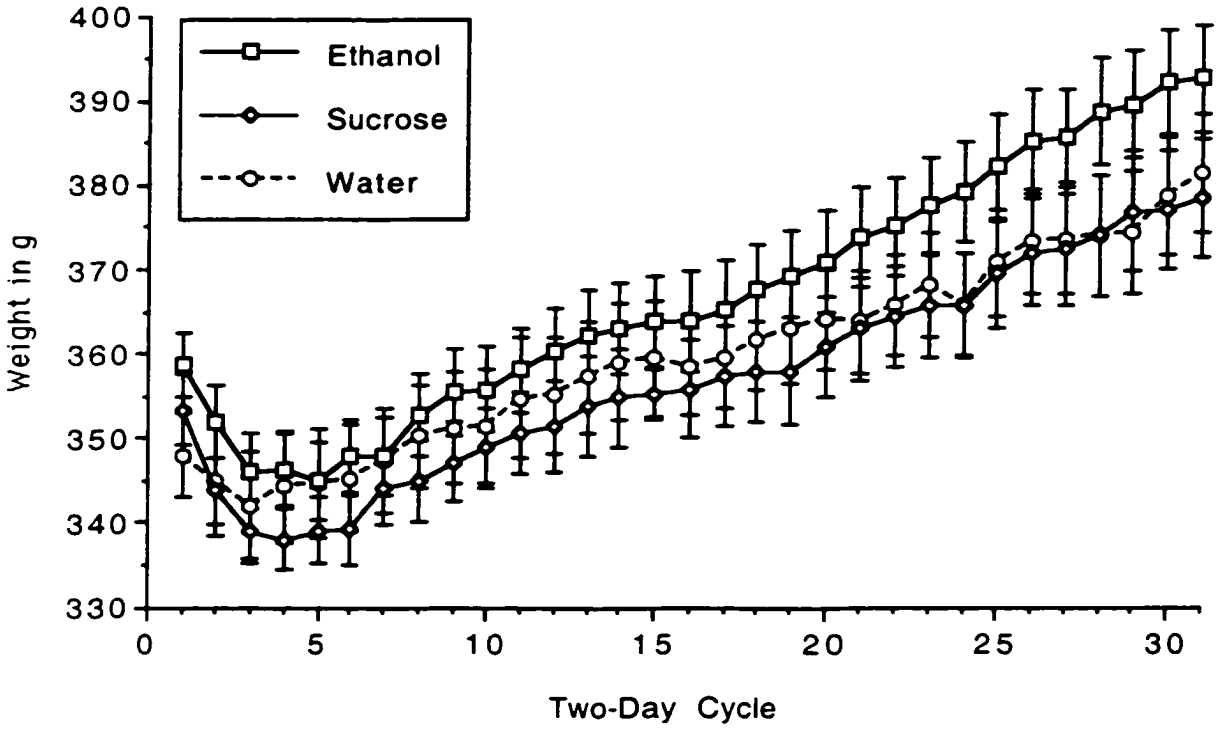

Figure 13: Mean (± SEM) body weight prior to sessions in the drinking environment for rats in the Ethanol, Sucrose, and Water groups in Experiment 3.

of the Ethanol, Sucrose, and Water groups, F(27,297) = 64.681, 39.635, and 44.330, respectively. The simple main effect of group was not significant during any cycle.

Consumption in the drinking environment, measured in ml/kg, during the twenty-eight shock cycles and the three no-shock cycles is shown in Figure 14. Consumption by the Water group appeared to be higher than consumption by the Sucrose group and both groups appeared to consume more than the Ethanol group throughout shock and no-shock cycles. This observation was confirmed for the shock cycles by a significant effect of group in a 3 x 28 group by (shock) cycle mixed ANOVA, F(2,33) = 49.188, on the first 28 cycles. Means comparisons showed that over the 28 shock cycles consumption by the Ethanol group was significantly lower than average consumption by the Sucrose and Water groups, F(1,33) = 65.553. Consumption by the Sucrose group was significantly less than by the Water group, F(1,33) = 32.823. The main effect of cycle, F(27,891) = 11.653, and the interaction between group and cycle, F(54, 891) = 2.219, were both significant for the twenty-eight shock cycles. Contrasts on the cycle main effect seem to indicate that most of the changes occurred within the first few cycles. Consumption during cycle 1 was significantly lower than during cycle 2, F(1,891) = 31.836. Consumption during cycle 2 was significantly lower than during cycle 3, F(1, 891) = 18.637. Consumption during cycle 3 was not different from consumption during

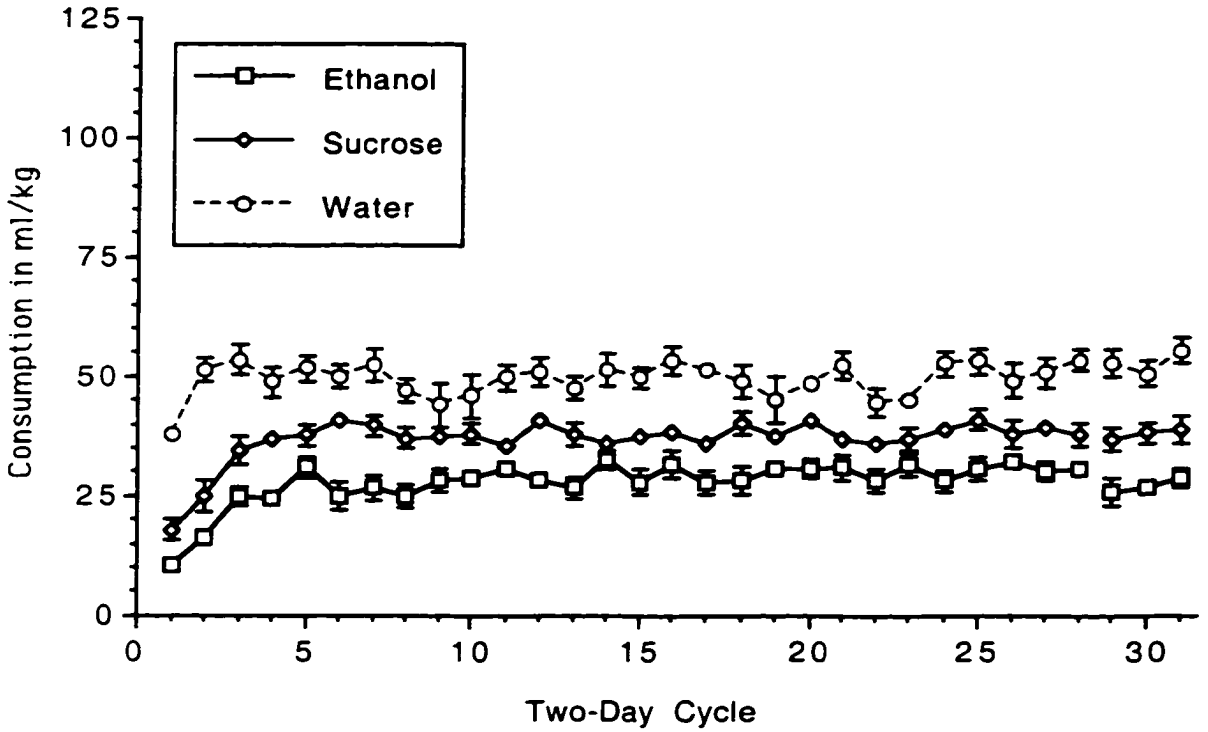

Figure 14: Mean (± SEM) consumption in the drinking environment in ml/kg for the Ethanol, Sucrose, and Water groups in Experiment 3. N = 12 per group. Rats in the Ethanol group consumed ethanol

cycle 4. The simple main effect of cycle was significant for each group, $F(27,297) = 6.862$, 9.508, and 2.498 for the Ethanol, Sucrose and Water groups. In each case, means comparisons showed that consumption increased significantly from cycle 1 to cycle 2, $F(1,297) = 5.087$, $p < .05$; 10.864; and 17.074. For the Ethanol and Sucrose groups, consumption during cycle 28 was significantly greater than during cycle 1, $F(1,297) = 62.398$ and 83.629. The simple main effect of group was significant during each cycle, $F(2,33)$ from 6.091 to 42.679. Means comparisons showed that consumption by the Ethanol group was lower than the average of the Sucrose and Water groups during each cycle, $F(1,33)$ from 8.651 to 72.156.

Consumption in the drinking environment during the no-shock cycles (29-31) was examined in a separate 3 x 3 group by cycle mixed ANOVA. This analysis revealed only a significant effect of group, $F(2,33) = 48.645$. As with consumption during the shock cycles, means comparisons showed that consumption by the Ethanol group was lower than the average of the Sucrose and Water groups, $F(1,33) = 64.796$. Consumption by the Sucrose group was lower than consumption by the water group, $F(1,33) = 32.494$.

In order to compare consumption during shock cycles to consumption during no-shock cycles a 3 x 2 x 3 group by treatment (shock versus no-shock) by cycle mixed ANOVA was performed on the consumption data for the last six cycles (26-31). This

analysis revealed a significant effect of group, $F(2,33) = 42.727$, and a significant treatment by group interaction, $F(2,33) = 4.644$, $p < .05$. Means comparisons showed that over the last six cycles consumption by the Ethanol group was significantly lower than the average by the Sucrose and Water groups, $F(1,33) = 54.832$. Examination of the interaction showed a significant simple main effect of group for both the shock and no-shock treatments, $F(2,33) = 27.987$ and 48.645, respectively. In both cases, means comparisons revealed that consumption by the Ethanol group was lower than the average for the Sucrose and Water groups, $F(1,33) = 34.147$, and 64.796 for the shock and no-shock cycles. Consumption by the Sucrose group was also lower than consumption by the Water group during both the last three shock and no-shock cycles, $F(1,33) = 21.827$ and 32.494. The simple main effect of treatment was significant only for the Ethanol group, $F(1,11) = 11.223$. Animals in this group consumed more during cycles in which drinking preceded shock than during cycles in which no shocks were administered.

Drinking environment consumption by the Ethanol group was compared to the ethanol consumption of the HCE group in order to see if being moved to other environments and receiving shocks altered ethanol consumption. Mean ethanol consumption for the Ethanol and HCE groups is shown in Figure 15. The data presented for the Ethanol group are identical to those in Figure 14. Data for the HCE group were

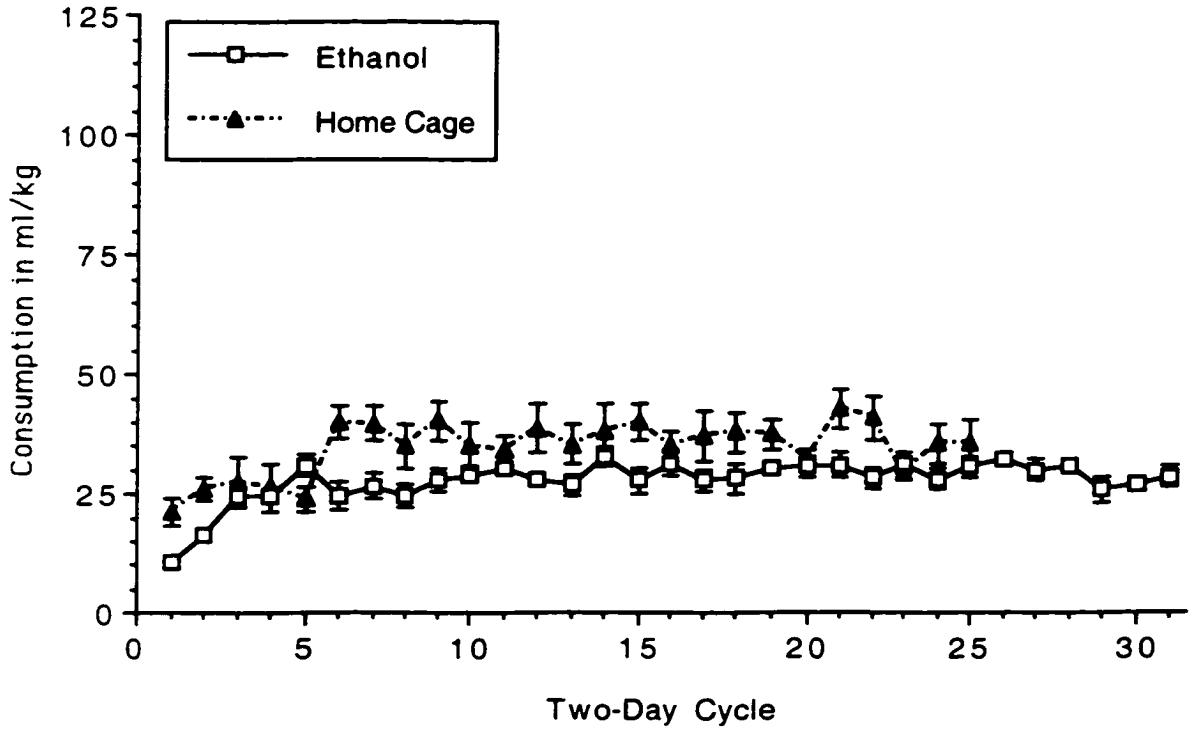

Figure 15: Mean (± SEM) ethanol consumption by the Ethanol group (n = 12) in the drinking environment and by the Home Cage Ethanol group (n = 6) in the home cage during Experiment 3.

only collected during cycles 1-25. A 2 x 25 group by cycle mixed ANOVA revealed a significant effect of group, $F(1,16) = 7.668$, $p < .05$. Consumption by the Ethanol group was lower than consumption by the HCE group.

Consumption of water in the home cage is shown in Figure 16. Consumption appeared to increase from cycle 1 to cycle 2 and then decline gradually for all three groups. A 3 x 28 group by cycle mixed ANOVA revealed only a significant effect of cycle, $F(27,864) = 5.082$. A 3 x 3 group by cycle mixed ANOVA on home cage consumption during the three no-shock cycles revealed no significant main effects or interactions.

Mean (± SEM) bolus production in the drinking environment during the last 6 cycles in which shock sessions followed sessions in the drinking environment (cycles 23-28) and during the three no-shock cycles (cycles 29-31) is shown in Figure 17. The six shock cycles were analysed in a 3 x 6 group by cycle mixed ANOVA. This analysis revealed a significant effect of group, $F(2,33) = 6.037$. Means comparisons showed that bolus production by the Ethanol group was less than the average of the Sucrose and Water groups, $F(1,33) = 9.642$. Bolus production by the Sucrose and Water groups was not significantly different. A 3 x 3 group by cycle mixed ANOVA on bolus production in the drinking environment during the no-shock cycles did not show any significant effects

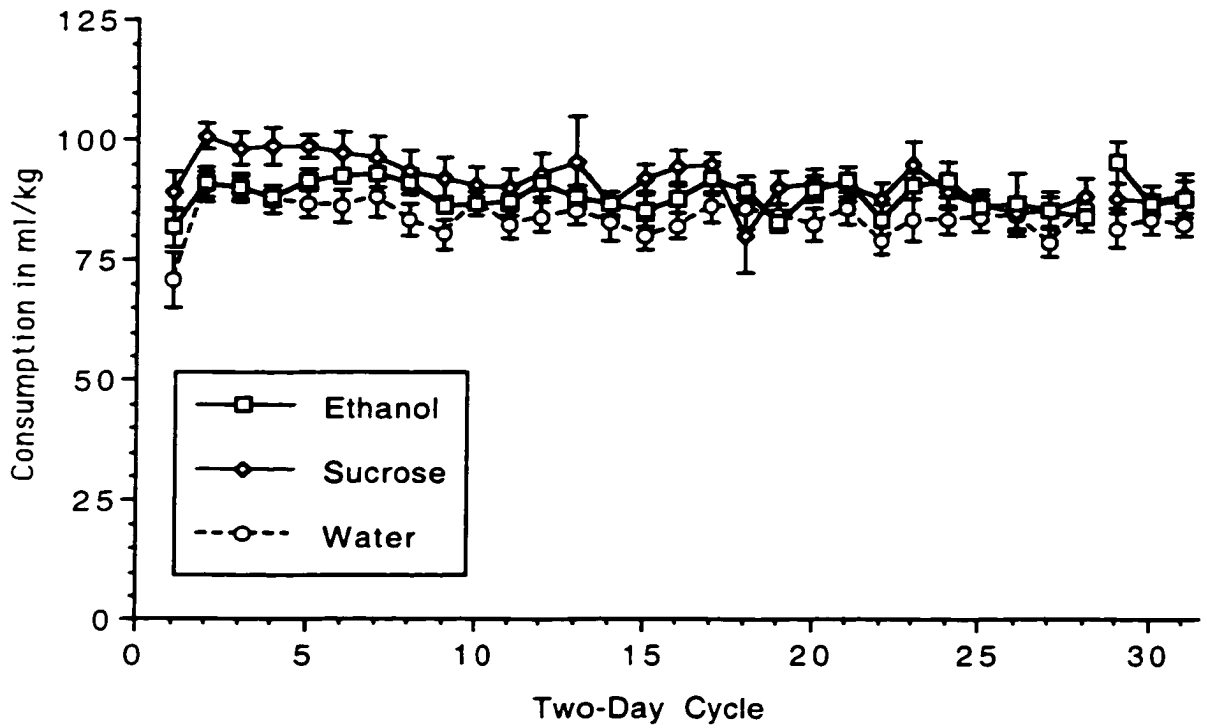

Figure 16: Mean (± SEM) consumption of water in the home cage by the Ethanol, Sucrose, and Water groups in Experiment 3. N = 12 per group.

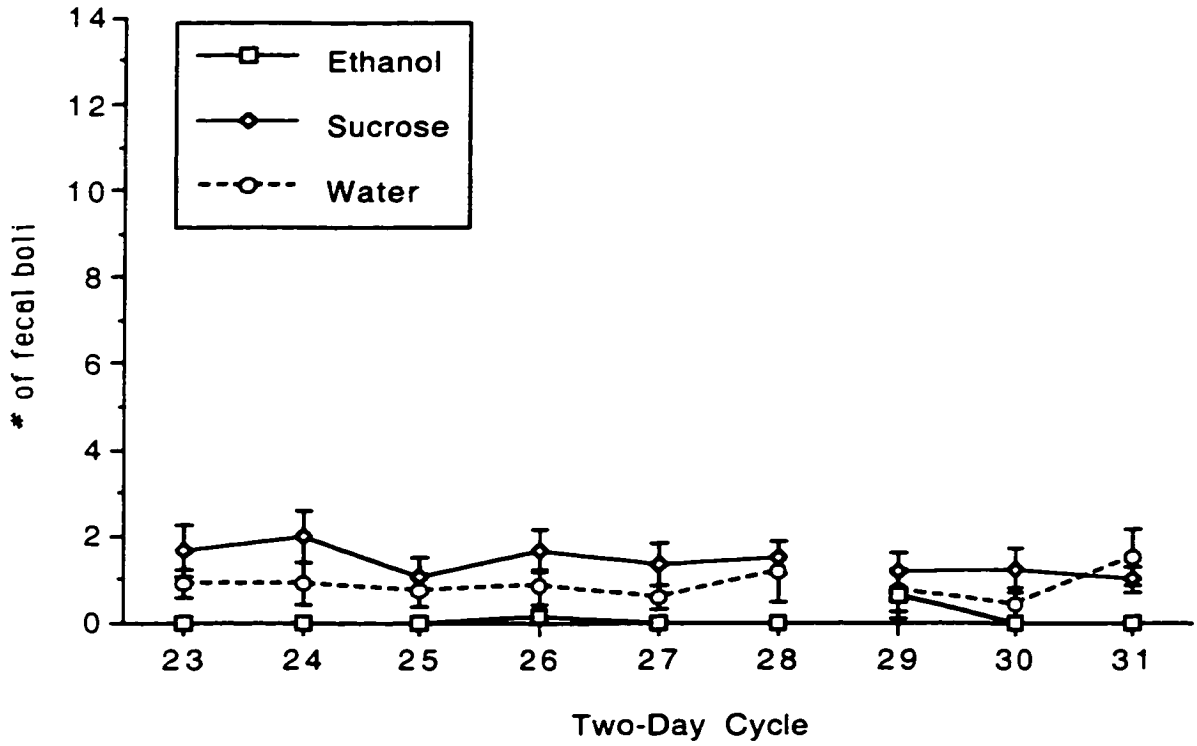

Figure 17: Mean (± SEM) fecal bolus production in the drinking environment during the last six shock cycles (23-28) and the three no-shock cycles (29-31) for the Ethanol, Sucrose, and Water groups in Experiment 3. N = 12 per group.

or interactions. The last three shock cycles were compared to the three no-shock cycles in a 3 x 2 x 3 group by treatment by cycle mixed ANOVA. Only a significant effect of group emerged from this analysis, $F(2,33) = 4.177$, $p < .05$. Means comparisons showed that bolus production by the Ethanol group was lower than the average of the Sucrose and Water groups, $F(1,33) = 6.911$, $p < .05$, which did not differ from each other.

Bolus production in the shock boxes during cycles 23 to 28 is shown in Figure 18. These data were analysed using a 3 x 6 group by cycle mixed ANOVA. As for bolus production in the drinking environment, only a significant effect of group emerged from the analysis, $F(2,33) = 5.162$, $p < .05$. Bolus production in the shock boxes by the Ethanol group was significantly lower than the average of the Sucrose and Water groups, $F(1,33) = 7.404$, $p < .05$. Bolus production by the Sucrose and Water groups was not significantly different.

Discussion

Examination of daily consumption in the drinking environment shows that the Water group consistently drank more than the Sucrose group and that both groups drank more than the Ethanol group. However, the Ethanol group did increase its consumption across cycles, which could indicate that the rats learned to drink ethanol to relieve their fear about the upcoming shock session. Comparison of the final three shock cycles and

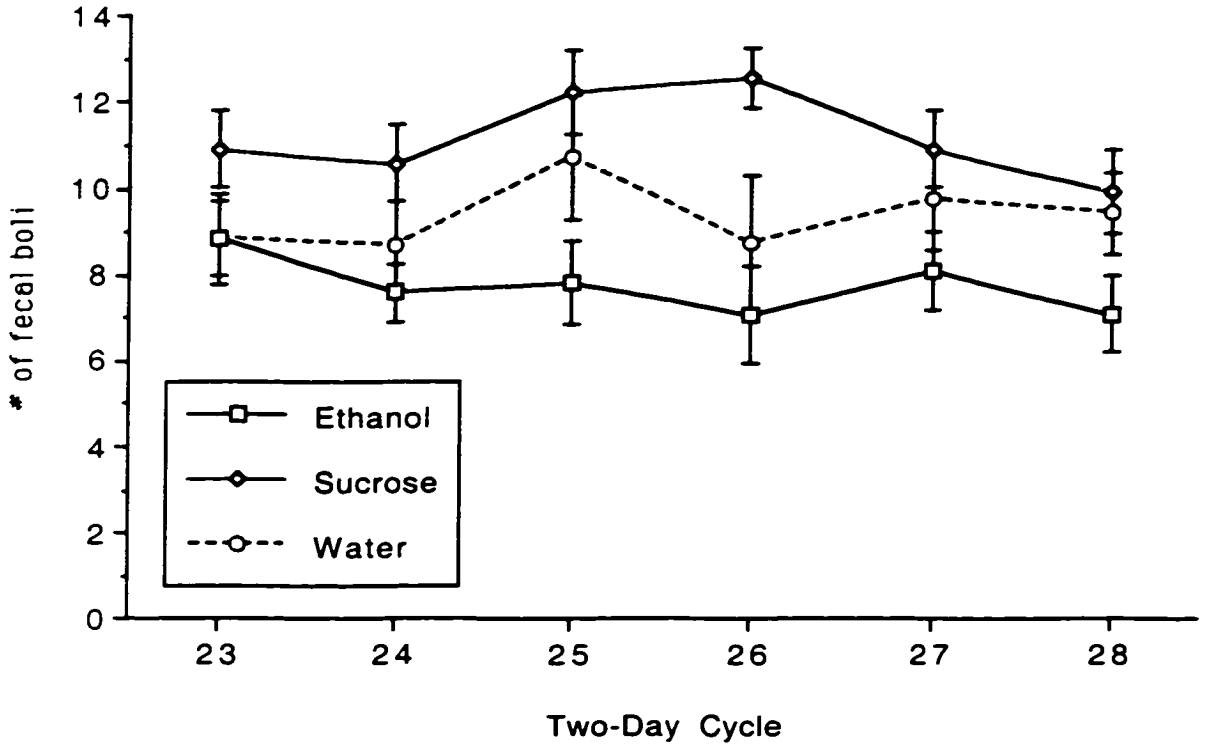

Figure 18: Mean (± SEM) number of fecal boli deposited in the shock environment by the Ethanol, Sucrose, and Water groups (n = 12 per group) during the last six shock cycles of Experiment 3.

the three no-shock cycles also provides some support for the Tension Reduction Hypothesis. Ethanol consumption was greater during the last three shock cycles than during the three cycles in which no shocks were administered. This pattern was not observed for either of the other two groups. However, the observation that the relatively unstressed HCE group consumed more ethanol than the Ethanol group is not what the Tension Reduction Hypothesis would have predicted.

If the Ethanol group consumed ethanol in order to reduce tension produced by cues associated with shock, then these animals should have experienced/expressed less negative affect than the other groups. Fecal bolus production in both the drinking and shock environments was recorded as a measure of this negative affect. Overall, there was substantially less defecation in the drinking environment than in the shock boxes, which was consistent with the animals being less fearful in the drinking environment than in the shock boxes. During the last six shock cycles, rats in the Ethanol group defecated significantly less than rats in the other groups in both the drinking environment and shock boxes. During the three no-shock cycles, defecation in the drinking environment was equivalent for all groups. (Rats were not taken to the shock boxes during no-shock cycles). Although the lower bolus production by the Ethanol group is consistent with this group experiencing less fear there are other possible explanations.

Animals in the Ethanol group consumed less fluid (on days when ethanol was their only fluid choice) and defecated less than did animals in the other groups. It is conceivable that there was a correlation between level of consumption and bolus production so that the Ethanol group defecated the least simply because they consumed the least. However, if consumption in the drinking environment fully accounted for defecation (in the drinking and shock environments) then defecation should have been highest for the Water group since its consumption was significantly higher than that of the Sucrose Group. This was not the case. Another alternative is that there is a correlation between caloric intake and bolus production. Such a correlation would account for the fact that the Sucrose group defecated the most but would suggest that the water group should have defecated less than the Ethanol group, which did not happen. A third explanation for the low level of defecation displayed by the Ethanol group is that ethanol had effects on gastric motility (Bode & Bode, 1997; Keshavarzian et al., 1986). That is, defecation may be altered by physiological effects of ethanol unrelated to effects of ethanol on tension.

Whatever the reason ethanol was consumed in this experiment (by the Ethanol group or by the HCE group), the rats did consume a pharmacologically significant volume of ethanol. Kinney and Schmidt (1979) indicated that rats can metabolize ethanol

at a rate of 0.30 g/kg/hr. Mean consumption of absolute ethanol by the Ethanol group was 1.06 g/kg during cycle 1, 1.63 g/kg during cycle 2, and in excess of 2.46 g/kg during all subsequent cycles. Cappell (1987) suggested that tension-reducing effects of ethanol can be apparent in animals following consumption of 1.5 g/kg absolute ethanol. Unfortunately, bolus data were not collected during the initial cycles so it cannot be determined whether group differences in defecation coincided with a particular level of ethanol consumption.

EXPERIMENT 4

In the previous experiment, we recorded fecal bolus counts beginning on the twenty-third shock day. The ethanol group defecated less than the other groups in both the drinking environment and the shock environment. Although this was consistent with the Ethanol group being less fearful than the other groups it was not possible to determine whether the difference in defecation emerged as a result of ethanol consumption. In this experiment, the basic methodology of Experiment 3 was replicated except that fecal bolus production was recorded from the very beginning of the experiment.

Once again, ethanol consumption was expected to increase across cycles and this increase was not expected for sucrose and water consumption. All of the animals were expected to be less fearful in the drinking environment than in the shock boxes and this was expected to be manifested as lower defecation in the drinking environment than in the shock boxes. Further, defecation by the Ethanol group was expected to be lower than defecation by the other groups.

Method

Subjects

The 36 rats in this experiment were obtained from Charles River Canada and were housed as described previously. Four rats were unable to complete the experiment due to ill health. All of the data for these animals were dropped from the analyses. On the first day of the experiment, the rats had a mean body weight of 345 g with a range from 316 to 378 g.

Apparatus

Environments and Fluids. The drinking environment and shock boxes were identical to those described in Experiment 1. Ethanol and sucrose solutions were identical to those described in Experiment 3.

Procedure

Like Experiment 3, this experiment consisted of a series of two-day cycles. On one day, the rats were removed from their home cages, weighed, and transported to the drinking environment. Following 1-h access to fluid, the rats were transported to the shock boxes for a 1-h shock session of 0.8 mA, 2-s shocks on a FT 60-s schedule. After the shock session, the rats were returned to their home cages. On the other day, the rats

remained in their home cages and had access to water for 1-h (at the same hour that they had had fluid access in the drinking environment).

Also as in Experiment 3, animals were assigned to one of nine squads which were assigned to groups in the same sequence as before. Again, the squads were run out of phase so that on one day five squads had a shock day and four had a no-shock day or vice-versa. Four animals were removed from the experiment due to ill health with the result that n = 10 for each of the Ethanol and Sucrose groups and n = 12 for the Water group.

Measures. Animals were weighed prior to their drinking session on all shock days. Fluid consumption was recorded for sessions in the drinking environment and in the home cage both in ml and in ml/kg. The number of fecal boli deposited by each rat in the drinking environment and in the shock boxes was recorded during each cycle.

Results

Mean (± SEM) body weight for each of the Ethanol, Sucrose, and Water groups on each shock day of the experiment is shown in Figure 19. There was a small decrease in body weight over the first few cycles followed by a gradual increase over the remaining cycles. A 3 x 20 group by cycle mixed ANOVA revealed only a significant effect of cycle, $F(19,551) = 26.495$.

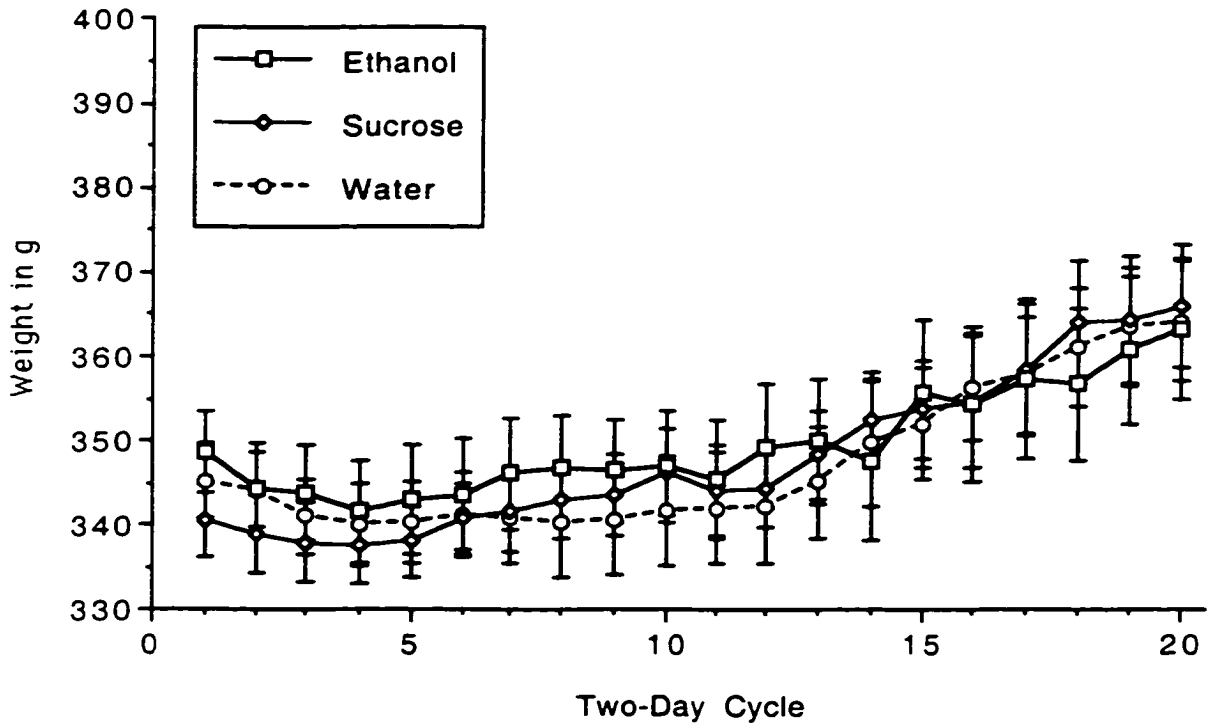

Figure 19: Mean (± SEM) body weight on the shock day of each cycle for the Ethanol (n = 10), Sucrose (n = 10), and Water (n = 12) groups in Experiment 4.

Figure 20 shows consumption in ml/kg in the drinking environment. Consumption by the Water group was consistently higher than consumption by the Sucrose group and both appeared to have higher consumption than the Ethanol group. For all three groups consumption appeared to increase across cycles. A 3 x 20 group by cycle mixed ANOVA revealed significant main effects of group, $F(2,29) = 23.178$, and cycle, $F(19,551) = 13.915$, and a marginally significant interaction between group and cycle, $F(38,551) = 1.486$, $p < .10$. Means comparisons on the group effect showed that consumption by the Ethanol group was less than the average consumption by the Sucrose and Water groups, $F(1,29) = 32.658$. Consumption by the Sucrose group was also significantly lower than consumption by the Water group, $F(1,29) = 11.594$. For the cycle effect, the biggest change occurred between cycles 1 and 2, $F(1,551) = 25.923$. Simple effect analyses were performed in order to further examine the interaction. The simple main effect of cycle was significant for each of the Ethanol, Sucrose and Water groups, $F(19,171) = 5.178$, 7.867, and $F(19,209) = 4.901$, respectively. Means comparisons showed a significant increase in consumption between cycles 1 and 2 only for the Ethanol and Sucrose groups, $F(1,171) = 8.363$ and 19.267. All three groups increased their consumption from cycle 1 to cycle 20, $F(1,171) = 22.630$, 72.348, and $F(1,209) = 22.474$ for the Ethanol, Sucrose and Water groups respectively. The simple main effect of group was significant for all

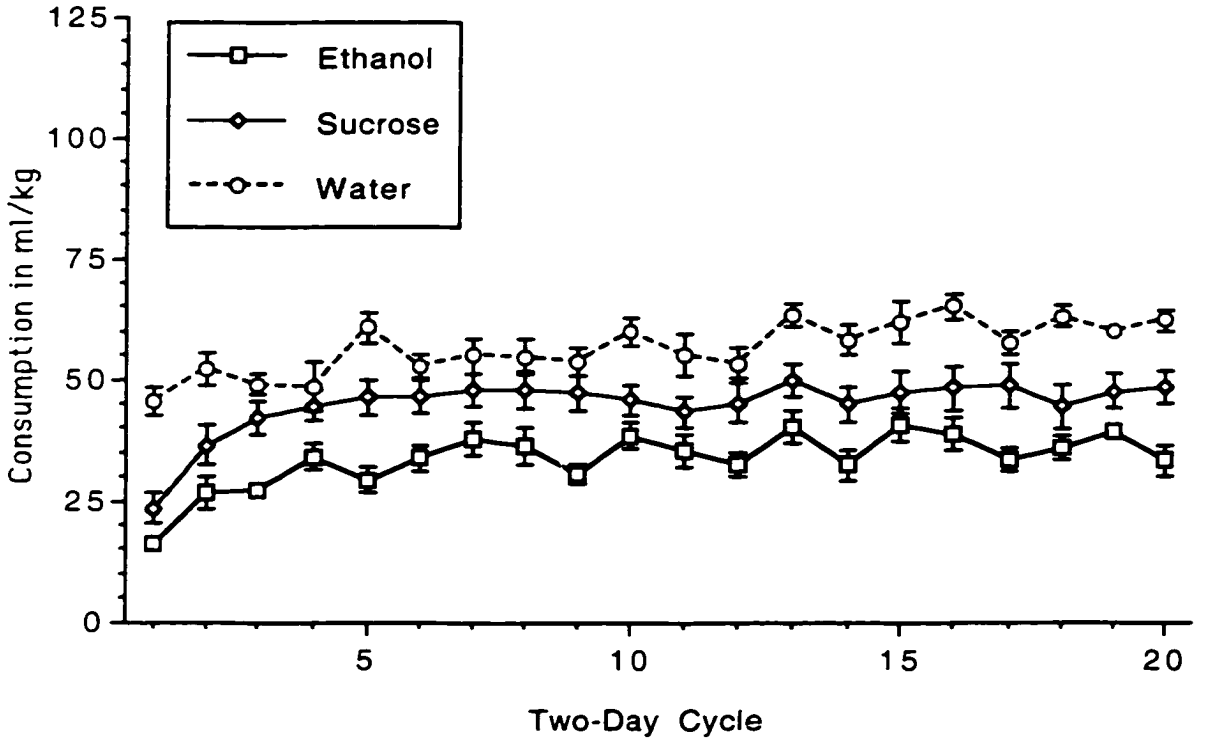

Figure 20: Mean (±SEM) fluid consumption in ml/kg in the drinking environment preceding shock sessions for Ethanol (n = 10), Sucrose (n = 10), and Water (n = 12) groups in Experiment 4.

but cycle 4. During all of the cycles in which the simple main effect of group was significant, the Ethanol group consumed less than the average of the Sucrose and Water groups.

Water consumption in the home cage during each cycle by each of the Ethanol, Sucrose, and Water groups is shown in Figure 21. One missing value during cycle 13 was replaced by the group mean, rather than dropping the animal from all analysis. A 3 x 20 mixed ANOVA revealed significant effects of group, $F(2,29) = 3.465$, $p < .05$, and cycle, $F(19,551) = 2.397$, but no significant interaction. Means comparisons for the group effect showed that home cage consumption by the Ethanol group was higher than the average home cage consumption of the Sucrose and Water groups, $F(1,29) = 6.668$, $p < .05$. The home cage consumption of the Sucrose and Water groups was not significantly different. Examination of the cycle effect by means comparisons showed a small but significant decrease in home cage water consumption from cycle 1 to cycle 20, $F(1,551) = 10.352$.

Bolus production in the drinking environment is shown in Figure 22. There was very little bolus production by any group during any cycle in the drinking environment. Defecation by the Ethanol group, in particular, was negligible. During several cycles there were no boli produced by any member of this group. A 3 x 20 group by cycle mixed ANOVA did not reveal any significant effects or interactions.

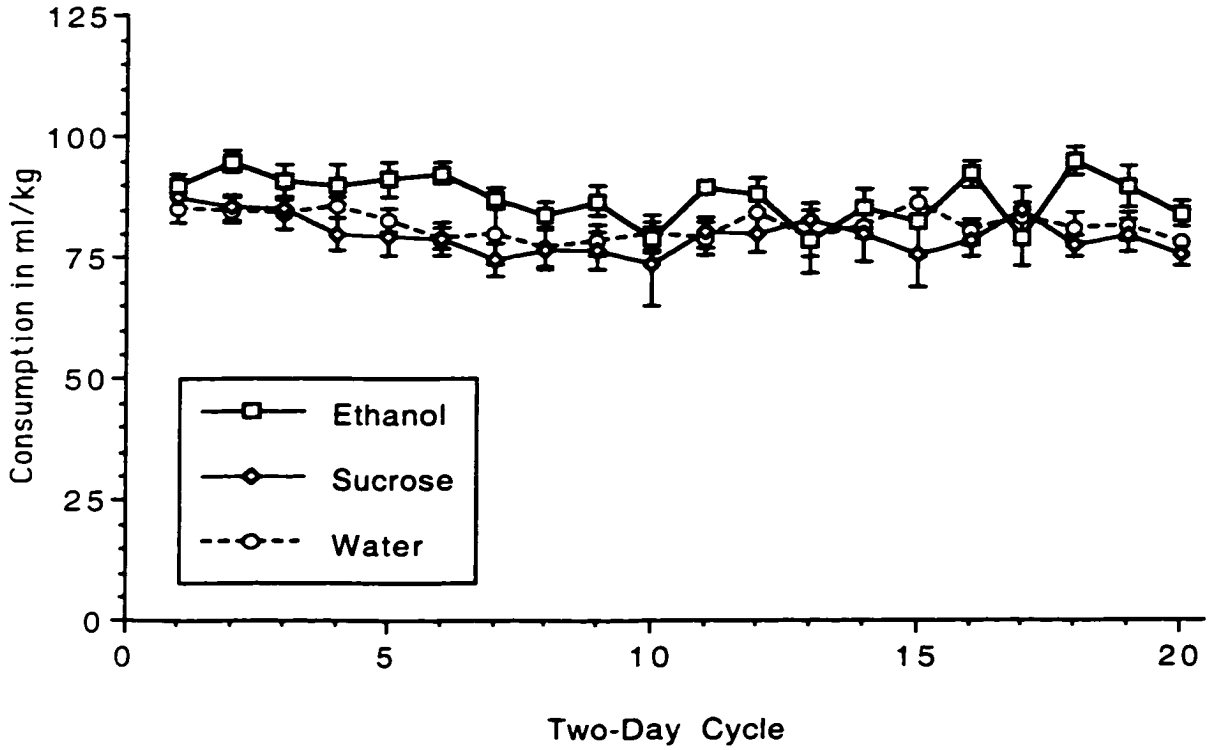

Figure 21: Mean (± SEM) home cage water consumption in ml/kg by the Ethanol (n = 10), Sucrose (n = 10), and Water (n = 12) groups in Experiment 4.

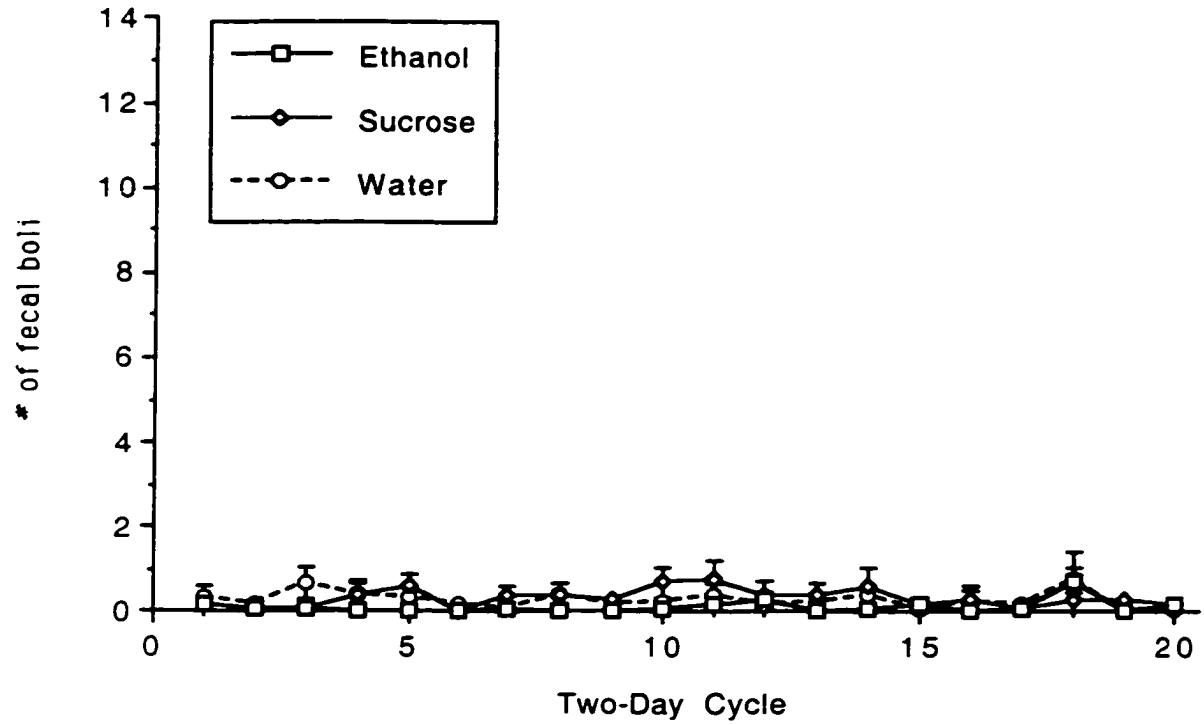

Figure 22: Mean (± SEM) fecal bolus production in the drinking environment preceding shock sessions by the Ethanol (n = 10), Sucrose (n = 10), and Water (n = 12) groups in Experiment 4.

Bolus production in the shock boxes is shown in Figure 23. Bolus production was much higher in the shock boxes than it was in the drinking environments. The Ethanol group appeared to defecate less than the other groups during many of the cycles. A 3 x 20 group by cycle mixed ANOVA revealed significant effects of group, $F(2,29) = 6.883$, and cycle, $F(19,551) = 2.374$, and a significant interaction between group and cycle, $F(38,551) = 1.951$. Means comparisons on the group effect showed that the Ethanol group defecated less than the average of the Sucrose and Water groups, $F(1,29) = 13.730$. There was no difference in fecal bolus production between the Sucrose and Water groups. Simple effect analyses were conducted to tease apart the interaction. The simple main effect of cycle was significant for the Ethanol group, $F(19,171) = 1.971$, and for the Water group, $F(19,209) = 3.093$, but not for the Sucrose group. For the Ethanol group, defecation appeared to decrease from cycles 1 to 8, increase during cycle 9, decrease during cycle 10 and then increase across the remaining cycles. As a result, means comparisons did not show a difference in defecation between cycle 1 and cycle 20. The simple main effect of cycle looks very different for the Water group. Defecation appeared to increase gradually across cycles. Consistent with this observation, means comparisons showed that bolus production was significantly lower during cycle 1 than during cycle 20, $F(1,209) = 19.272$. The simple main effect of group was significant during cycles 6-8,

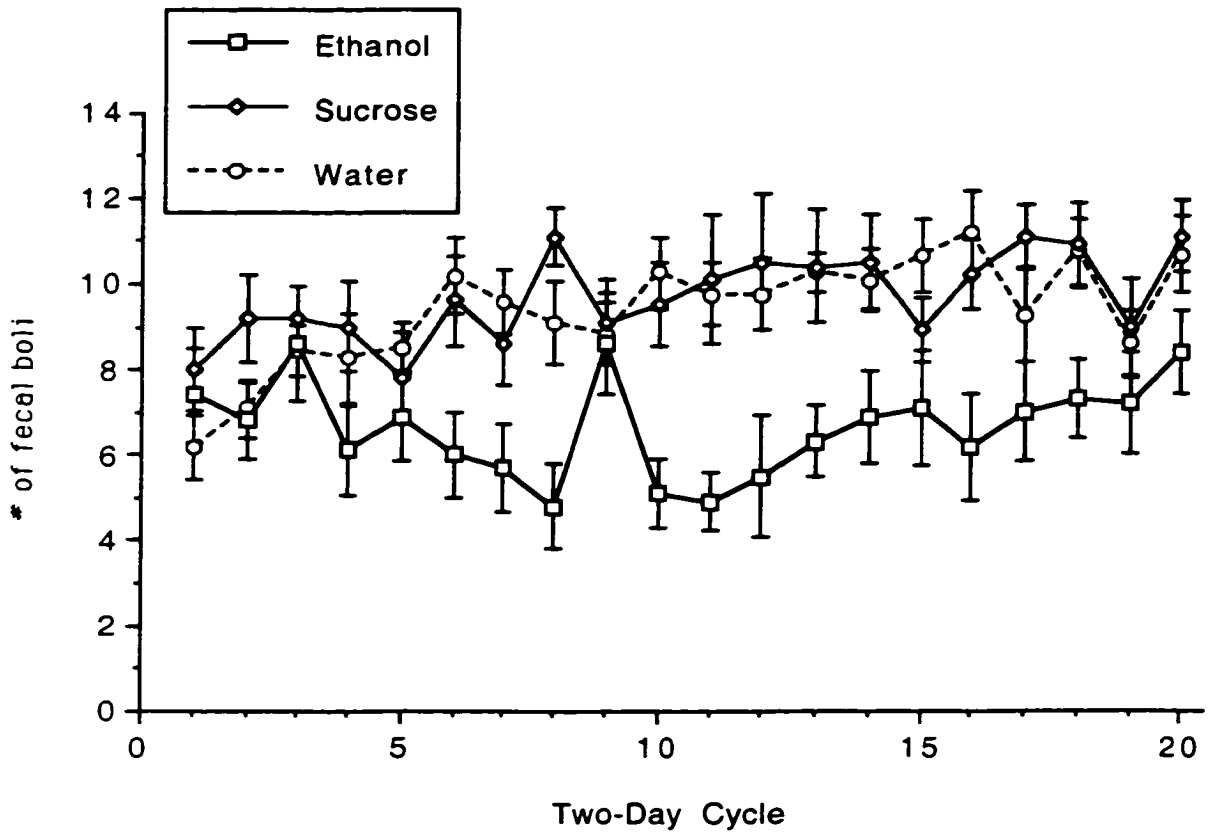

Figure 23: Mean (± SEM) fecal bolus production in the shock boxes during shock sessions by Ethanol (n = 10), Sucrose (n = 10), and Water (n = 12) groups in Experiment 4.

10-14, and 16-18, all F's(2,29) > 3.5. Every time there was a significant main effect of group, bolus production was significantly lower by the Ethanol group than by the average of the Sucrose and Water groups, all F's(1,29) > 5.75. The Sucrose and Water groups were never significantly different from each other.

Discussion

Rats in the Ethanol group consumed less fluid in the drinking environment than did rats in the Sucrose or Water groups. All three groups increased their consumption from cycle 1 to cycle 20. This pattern mirrors the consumption results from Experiment 3. Once again, the increasing consumption by the Ethanol group was consistent with predictions from the Tension Reduction Hypothesis. However, it is still unclear why the Ethanol group consumed less fluid than the other groups. One explanation that has to be considered is that the rats do not like the Ethanol solution and so only drink enough of it to satisfy their minimal fluid requirements. This possibility is addressed in Experiment 5.

During Experiment 3, bolus production data were not collected during the initial cycles and as a result there was no information available about the emergence of group differences for this measure. In this experiment, bolus production data were collected for the drinking environment and the shock boxes beginning in cycle 1. From the beginning, bolus production was very low in the drinking environment. The absence of both group

and cycle effects is consistent with the claim that the rats did not acquire fear in this environment. A very different picture emerged from examination of the bolus data for the shock boxes. Overall, the level of defecation was much higher in the shock boxes than it had been in the drinking environment and this level increased across cycles. This finding is consistent with the expectation that the animals were more fearful in the shock boxes than in the drinking environment and that the level of fear increased with exposure to shock. Group differences in defecation did not emerge until cycle 6, and during the next several cycles resulted from lowered defecation by the Ethanol group. During cycle 9, defecation was much higher by the Ethanol group with the result that the simple main effect of group was not significant during this cycle. Again during cycles 10-14 the simple main effect of group resulted from a decrease in defecation by the Ethanol group. Bolus production was consistently lower by the Ethanol group than by the other groups which is consistent with the Tension Reduction Hypothesis.

EXPERIMENT 5

In Experiments 3 and 4, thirsty rats that had the opportunity to consume ethanol in a distinctive drinking environment before shock consumed less fluid than did groups of rats that had access to sucrose or water before shock. Ethanol consumption increased across shock cycles in both experiments. The Ethanol group also defecated less than the other groups. Both the increased consumption across cycles and the low defecation by the Ethanol group are consistent with a Tension Reduction view. However, since the animals were fluid deprived and the Ethanol group consumed less than the other groups I wanted to know to what extent ethanol consumption was motivated by thirst rather than by other factors. The role of thirst was assessed in this experiment by allowing rats from the Ethanol, Sucrose and Water groups (of Experiment 4) to consume fluid in the drinking environment under different levels of fluid deprivation. On some shock days the animals had access to water for one hour in the home cage two hours prior to their fluid access in the drinking environment. If thirst were the primary motivator of ethanol consumption, then ethanol consumption should have been lower on the days when the rats had the opportunity to pre-load with water than on the days when ethanol was their only fluid source. If thirst were the primary motivator of consumption in the drinking environment,

then all of the groups should have consumed less fluid in the drinking environment on pre-loading days than on no pre-loading days. If consumption were motivated by the caloric content of the solution, then neither ethanol nor sucrose consumption should have been affected by the pre-loading manipulation. If the rats were motivated to consume ethanol mainly because of its pharmacological consequences then ethanol consumption alone should have been unaffected by the pre-loading manipulation.

In addition to recording bolus production, some sessions in the shock boxes (during cycles 31-34) were videotaped. Segments of these videotapes were examined and incidents of "freezing" (absence of visible movement other than what was required for respiration) were scored. Since freezing is a characteristic post-encounter fear response (Fanselow, 1994) reduced freezing following ingestion of ethanol could have been indicative of reduced fear. I expected that, to the extent that they were both indicators of fear, the freezing and bolus data would converge.

In order to see what effect ethanol would have on fear (as measured by bolus production) in animals that had prior experience with shock but not with ethanol, at the end of the experiment the fluid available in the drinking environment was switched to ethanol for the Sucrose and Water groups. Half of the animals in the Ethanol group continued to have access to ethanol (for comparison purposes) and the remaining animals

were switched to sucrose. If ethanol had been reducing the fear of the Ethanol group then those animals that started getting sucrose instead of ethanol should have increased their bolus production. If ethanol could reduce fear then the animals in the Sucrose and Water groups should have decreased their bolus production.

Method

Subjects

The rats from Experiment 4 were used in this experiment. At the start of this experiment, mean body weight was 426 g, with a range from 360-485 g. The rats were housed individually as described previously. Fluid was available as described in the procedure section.

Apparatus

Fluids and Environments. These were the same as in the last two experiments.

Bottles. In order to deliver the small quantities of fluid (15 or 25 ml) required during the pre-loading period in the home cage, it was necessary to use smaller bottles. One hundred-ml polyethylene bottles with rubber stoppers and curved drinking tubes were affixed to the front of the cages with springs for this purpose. At all other times,

fluids were delivered (as before) using large glass bottles with rubber stoppers and straight drinking tubes.

Video Equipment. Some sessions in the shock boxes were videotaped using a Panasonic WV-1550 camera with a SONY tv lens No. 309969 connected to a Panasonic AG-2500 videocassette recorder. A white noise pulse was presented every five seconds to facilitate scoring.

Procedure

This experiment began the day after Experiment 4 ended. The rats continued to have access to fluid for one hour per day either in the drinking environment or in the home cage. The schedule of alternating shock and no-shock days also continued. Shock sessions (0.8 mA, 2-s shocks on a FT 60-s schedule for 1 hour) followed fluid access in the drinking environment. A preloading manipulation was added to this experiment such that two hours prior to alternate drinking environment sessions, the rats had access to water in the home cage for one hour. The daily schedule for each day of a representative four-day cycle for this experiment is shown in Table 3. On the pre-loading day (Day 1 of the representative four-day cycle), a water bottle was attached to the animals' home cage for 1 hour. The animals remained in their home cages with no fluids available during the next hour. Two hours after the preloading period began; the rats were removed from their

Table 3: Daily schedule for each day of a representative four-day cycle for Experiment 5

Time	Day 1	Day 2	Day 3	Day 4
1st Hour	PL		No PL	
	Home		Home	
2nd Hour				
3rd Hour	E or S or W	Water	E or S or W	Water
	DE	Home	DE	Home
4th Hour	Shock		Shock	

Note: PL = Preloading period; No PL = No Preloading period; E = Ethanol; S = Sucrose; W = Water; DE = Drinking Environment.

home cages, weighed, and transported to the drinking environments. After 1 hour in the drinking environment with access to ethanol or sucrose or water, the rats were transported to the shock boxes for their shock session. After the shock session, rats were returned to their home cages where food was freely available. On no-shock days (Days 2 and 4 of the representative four-day cycle), rats remained in their home cages with food freely available. The rats had access to water for one hour at the same time that they had access to fluid in the drinking environment on shock days. The no-preloading day (Day 3) was identical to the preloading day except that there was no fluid access in the home cage prior to the drinking environment session.

Group membership continued from Experiment 4. Animals that had been in the Ethanol group in Experiment 4 continued to have access to ethanol in the drinking environment in this experiment. Similarly, rats in the Sucrose and Water groups in Experiment 4 continued to have access to sucrose and water, respectively, in the drinking environment in this experiment. The groups were treated identically except for which fluid was available in the drinking environment.

As before, squads of animals were run out of phase with each other. So, each day some animals had a no-shock day and others had a shock day. Of the animals being shocked half were preloaded and half were not. During the first 14 four-day cycles, 15 ml

of water was available during the preloading period. During cycles 15-28, 25 ml of water was available during the preloading period. After a 48-day break during which the animals were maintained on 23-h fluid deprivation, six more cycles (29-34) were run in which 25 ml of water were available during the preloading period. For the final three cycles of the experiment (35-37), the same preloading regimen continued and the fluid available to each group in the drinking environment was switched. Both the Sucrose and Water groups had access to ethanol in the drinking environment during these final cycles. Half of the animals in the Ethanol group continued to have access to ethanol in the drinking environment and the rest were given access to sucrose.

Over the course of the experiment (184 running days) there were several days during which the rats were not run (in addition to the intentional break between cycles 28 and 29). On days when the animals were not run, they received access to water in their home cages for at least one hour. The experiment resumed at the point in the cycle where it had been when the interruption occurred. The animals were not run for 1 day between cycles 13 and 14 when the fire alarms were being tested. During cycle 20, the animals were not run for 2 days and during cycle 24 the animals were not run for four days due to experimenter illness. During cycle 25, the cooling system for the colony room failed. As a result, the animals were not run for 1 day (but received access to extra water in the

home cage), and received 2 and 3 hours extra access to water after their session were over on two other days.

Between cycles 31 and 34, 16 rats were individually videotaped in a total of 45 shock box sessions. At any one time, one animal from one of the two boxes at which the camera could be pointed was videotaped. Each of the animals that was videotaped was taped during a shock session on at least one preloading and one no preloading day. For each of these 45 60-minute sessions, three 5-minute periods were scored: the first 5 minutes of the session, the middle 5 minutes of the session, and the last 5 minutes of the session. Freezing was scored using a time sampling procedure. The rat was scored as freezing or not freezing every 5 s. Scoring was signaled by a white noise pulse.

Measures. Body weights were recorded for all preloading and no-preloading days of the experiment. Consumption during the preloading period was recorded in ml in order to allow an easy comparison of the volume consumed relative to the volume available. Consumption during drinking environment sessions was recorded in ml and in ml/kg for preloading and no-preloading days. In addition, a difference score (drinking environment consumption on the no-preloading day minus drinking environment consumption on the preloading day) was calculated for each cycle. Positive values for this difference score would indicate higher consumption on the no-preloading day than on the preloading day

while negative values for this difference score would indicate that consumption was higher on the preloading day than on the no-preloading day. Larger absolute values for the difference score indicate greater impact of the preloading manipulation. Values near zero indicate little difference in drinking environment consumption as a result of preloading. Home cage water consumption on no-shock days was recorded in ml and in ml/kg for successive water days. The numbers of fecal boli deposited in the drinking environment and in the shock boxes were recorded for preloading and no-preloading days. As with drinking environment consumption, difference scores were also calculated for both of the bolus measures (bolus production on the no-preloading day minus bolus production on the preloading day for the drinking environment and for the shock boxes). Positive values for these difference scores represent more defecation on days with no preloading than on days with preloading and negative values represent the opposite. The larger the absolute value, the greater the change in defecation between days with preloading and days with no preloading. Freezing scores (# of intervals scored as freezing) were transformed to % freezing scores ((# of intervals scored as freezing divided by # of intervals) * 100) for early, middle and late 5-minute periods, and for total freezing over all three periods.

Results

Mean (± SEM) consumption during the preloading period is shown in Figure 24. During cycles 1 to 14, 15 mls of water were available to the animals. Mean consumption for the three groups is virtually identical and it is apparent that the rats were consuming virtually all of the available fluid. In cycles 15 to 28 and cycles 29 to 34, 25 mls of water were available during the preloading period. Again, all three groups consumed most of the available fluid. Three missing values were replaced by the group mean for that day. Group by cycle mixed ANOVAs revealed only significant effects of cycle for the first 14 cycles, $F(13,377) = 31.854$, and for cycles 15 to 28, $F(13,377) = 3.909$. There were no significant effects or interactions for cycles 29-34.

Mean body weight on each preloading and no-preloading day is shown in Figure 25. Standard errors are not shown on this figure as they tended to obscure the data points. A 3 x 2 x 14 group by treatment (preloading versus no preloading) by cycle mixed ANOVA revealed significant effects of cycle, $F(13,377) = 131.592$, and treatment, $F(1,29) = 1338.682$, and a significant interaction between cycle and group, $F(26,377) = 2.480$, during the first 14 cycles. In general, body weight increased across cycles and was higher on preloading days than on no-preloading days (animals were weighed after preloading). The same observations were supported for cycles 15-28. A 3 x 2 x 14 group

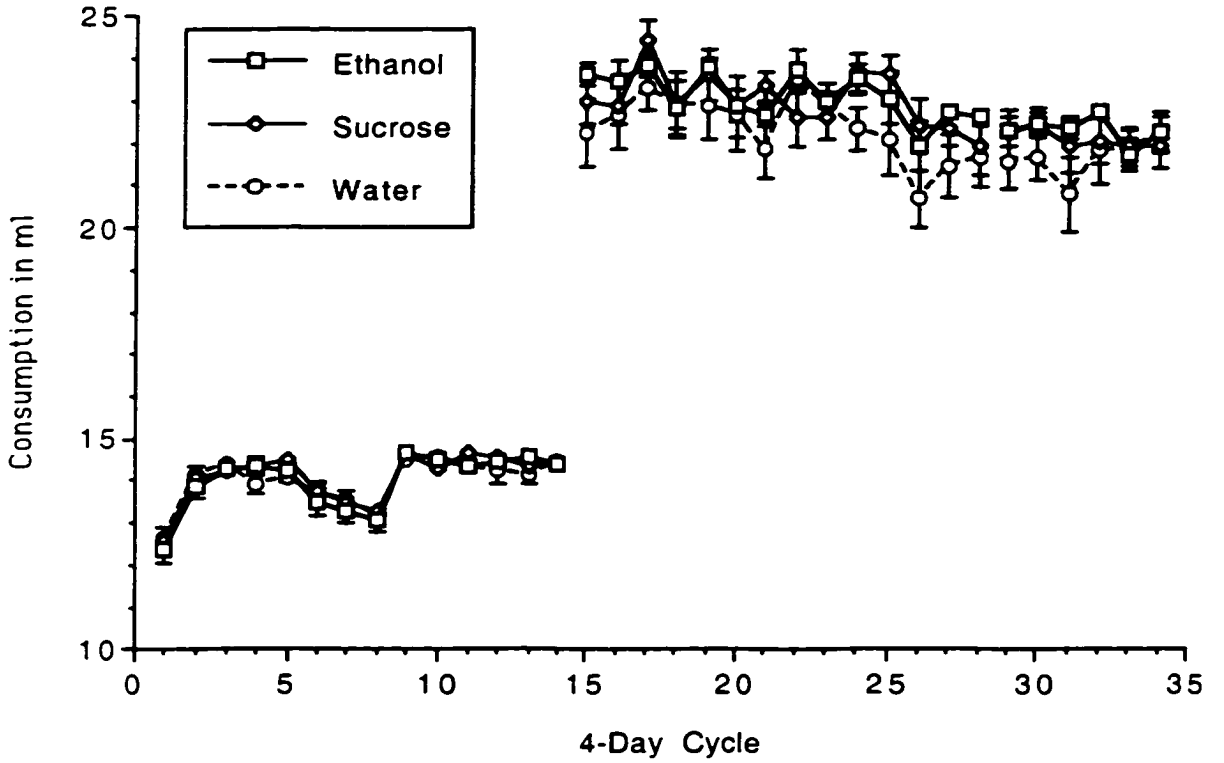

Figure 24: Mean (± SEM) consumption of water in ml during the preloading period for the Ethanol (n = 10), Sucrose (n = 10), and Water groups (n = 12) in Experiment 5. During cycles 1-14 15 mls of water was available. During cycles 15-28 and during cycles 29-34 (which began 48 days after cycle 28 ended) 25 mls of water was available.

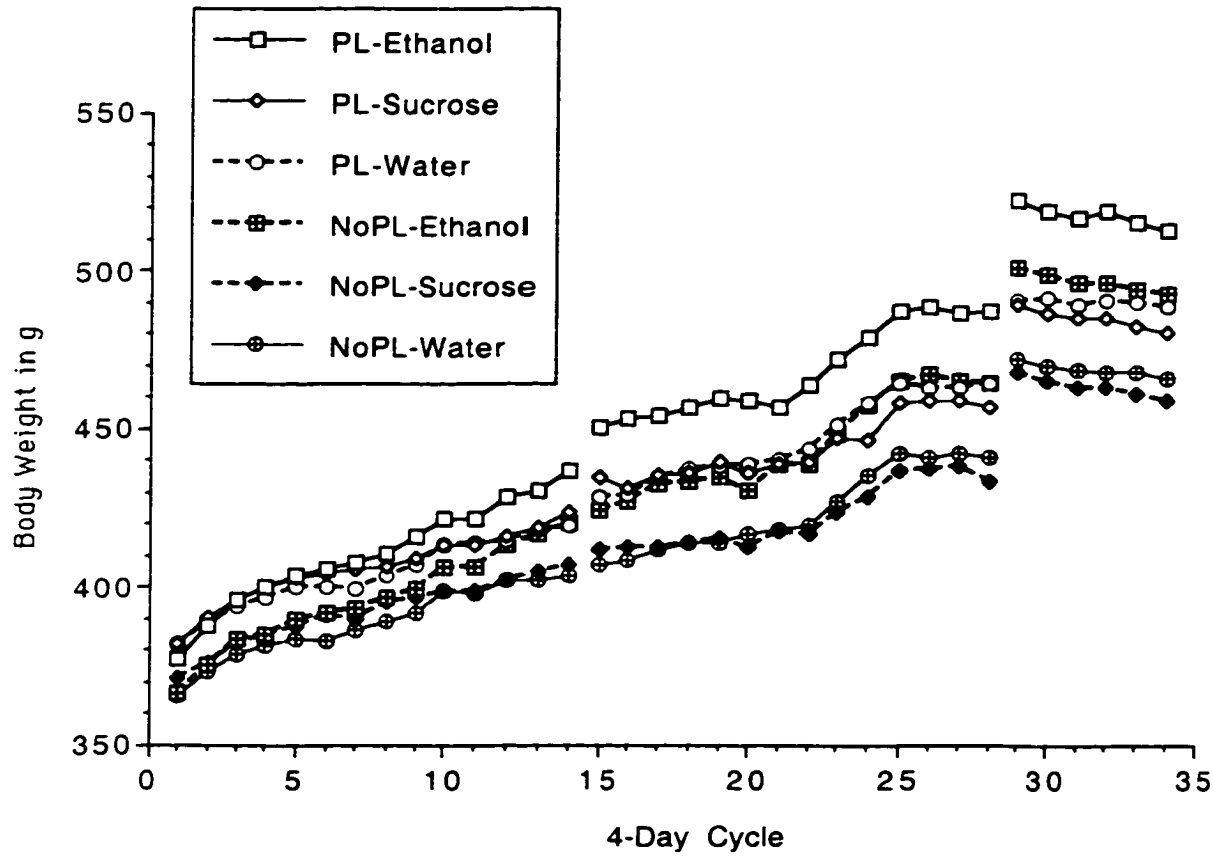

Figure 25: Mean body weight in grams on days with preloading and with no preloading for the Ethanol (n = 10), Sucrose (n = 10), and Water groups (n = 12) in Experiment 5. During cycles 1-14 15 mls of water was available during the preloading period (which ended an hour before the animals were weighed). During cycles 15-28 and 29-34 25 mls of water was available during the preloading period. Cycles 28 and 29 were separated by 46 days.

by treatment by cycle mixed ANOVA on these data also revealed significant effects of cycle, $F(13,377) = 181.580$, and treatment, $F(1,29) = 2355.279$, and a significant interaction between cycle and group, $F(26,377) = 2.894$. Body weights appear to be much higher during cycle 29 than during cycle 28, indicating that the rats grew during the intervening 48 days. A 3 x 2 x 6 group by treatment by cycle mixed ANOVA for the last six cycles revealed only significant effects of cycle, $F(5,145) = 9.898$, and treatment, $F(1,29) = 1728.350$. During these cycles, body weight was again higher on preloading than on no-preloading days. There was a general decrease in body weight across the last six cycles.

Mean consumption in ml/kg in the drinking environment on preloading and no-preloading days is shown in Figure 26. On eleven different occasions a bottle leaked so that the consumption value could not be obtained. These missing values were replaced by the group mean for the days in question. Generally, consumption on no-preloading days was higher than consumption on preloading days and this difference appeared to be much bigger for the Water and Ethanol groups than for the Sucrose group. The Water group consumed more than the Sucrose group, which consumed more than the Ethanol group. Parallel group by treatment (preloading and no-preloading) by cycle mixed ANOVAs were conducted on the drinking environment consumption data for cycles 1 to 14, 15 to

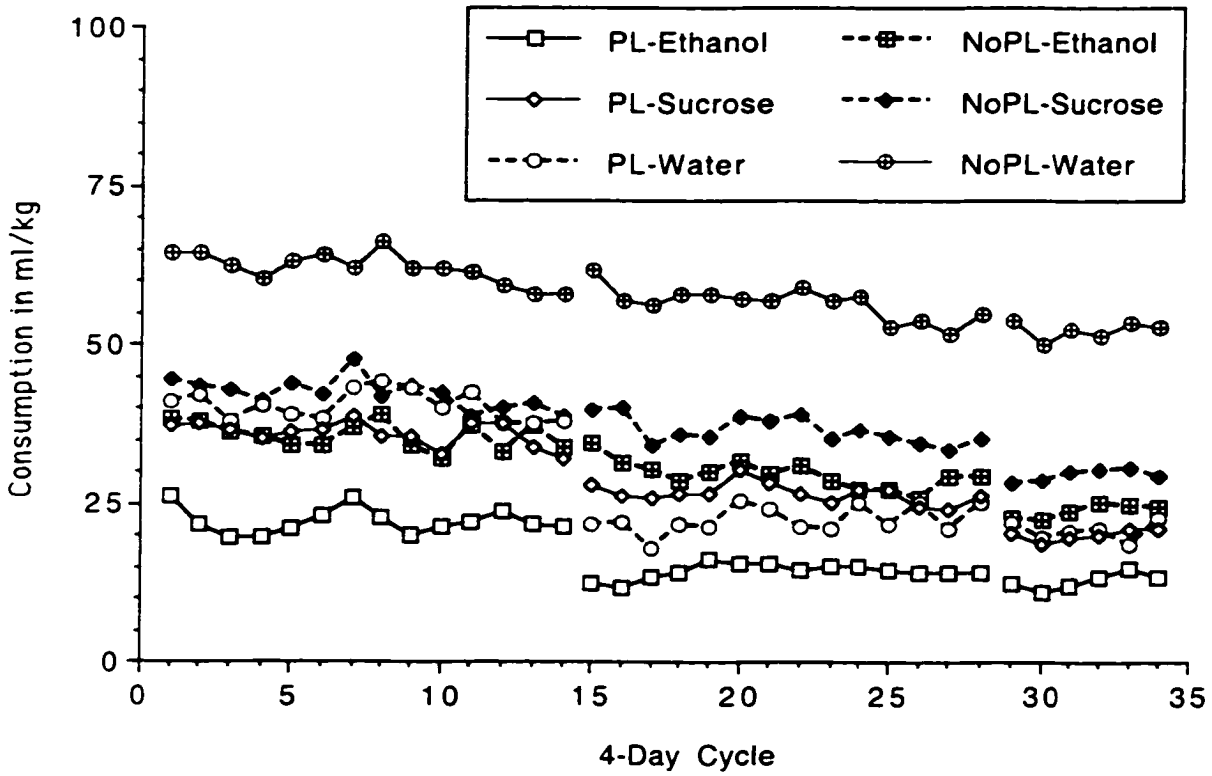

Figure 26: Mean (± SEM) consumption in the drinking environment in ml/kg on days with preloading and with no preloading for the Ethanol (n = 10), Sucrose (n = 10), and Water groups (n = 12) in Experiment 5. During cycles 1-14 15 mls of water was available during the preloading period (which ended an hour before the animals were weighed). During cycles 15-28 and 29-34 25 mls of water was available during the preloading period. Cycles 28 and 29 were separated by 46 days.

28, and 29 to 34. Each analysis revealed significant main effects of group, $F(2,29) = 32.551$, 24.306, 21.469, cycle, $F(13,337) = 3.771$, 4.869, $F(5,145) = 2.285$ ($p < .05$), and treatment, $F(1,29) = 304.119$, 437.689, 311.801. For each group of cycles, consumption was lower on preloading days than on no-preloading days. Means comparisons showed that average consumption by the Ethanol group was lower than the average of the Sucrose and Water groups, $F(1,29) = 43.010$, 36.534, and 23.697, for cycles 1-14, 15-28, and 29-34, respectively. Consumption by the Sucrose group was also lower than consumption by the Water group, $F(1,29) = 19.009$, 10.003, and 17.079. The interaction of group and treatment was significant for each group of cycles, $F(2,29) = 32.783$, 63.669, and 55.905. Simple effect analyses revealed the same pattern of results for each group of cycles. The simple main effect of treatment was significant for each group. Consumption was lower on preloading days than on no-preloading days for the Ethanol group during cycles 1-14, 15-28, and 29-34, $F(1,9) = 118.841$, 96.677, and 66.855. Consumption was also lower on preloading than on no-preloading days for the Sucrose group, $F(1,9) = 22.465$, 32.339, and 26.032. The same pattern held for the Water group, $F(1,11) = 219.320$, 462.704, and 314.521. The simple main effects of group for each treatment also revealed consistent effects across the three groups of cycles. The simple main effect of group was always significant for preloading days, $F(2,29) = 24.732$,

11.526, and 4.538 ($p < .05$), as it was for no-preloading days, $F(2,29) = 38.027$, 44.471, and 43.380. On preloading days, means comparisons revealed that consumption by the Ethanol group was lower than the average of the Sucrose and Water groups, $F(1,29) =$ 45.538, 21.059, and 8.92. Consumption by the Sucrose and Water groups did not differ. On no-preloading days, consumption by the Ethanol group was again lower than the average of the Sucrose and Water groups, $F(1,29) = 33.469$, 39.872, 33.999, and consumption by the Sucrose group was lower than consumption by the Water group, $F(1,29) = 38.718$, 44.543, and 48.400. During cycles 15 to 28, the interaction between cycle and treatment was also significant, $F(13,377) = 3.059$.

The mean difference in drinking environment consumption between days with no preloading and days with preloading for each cycle (no preloading minus preloading) is shown in Figure 27. All of the values are positive, indicating that drinking environment consumption on days with no-preloading was always higher than on days with preloading, an observation that is consistent with the treatment effect already discussed. Group by cycle mixed ANOVAs were conducted for the 14 cycles during which the rats were preloaded with 15 mls of water (cycles 1-14), the first 14 cycles during which the rats were preloaded with 25 mls of water (cycles 15-28), and the final six cycles during which the rats were preloaded with 25 mls of water (cycles 29-34) which started 48 days

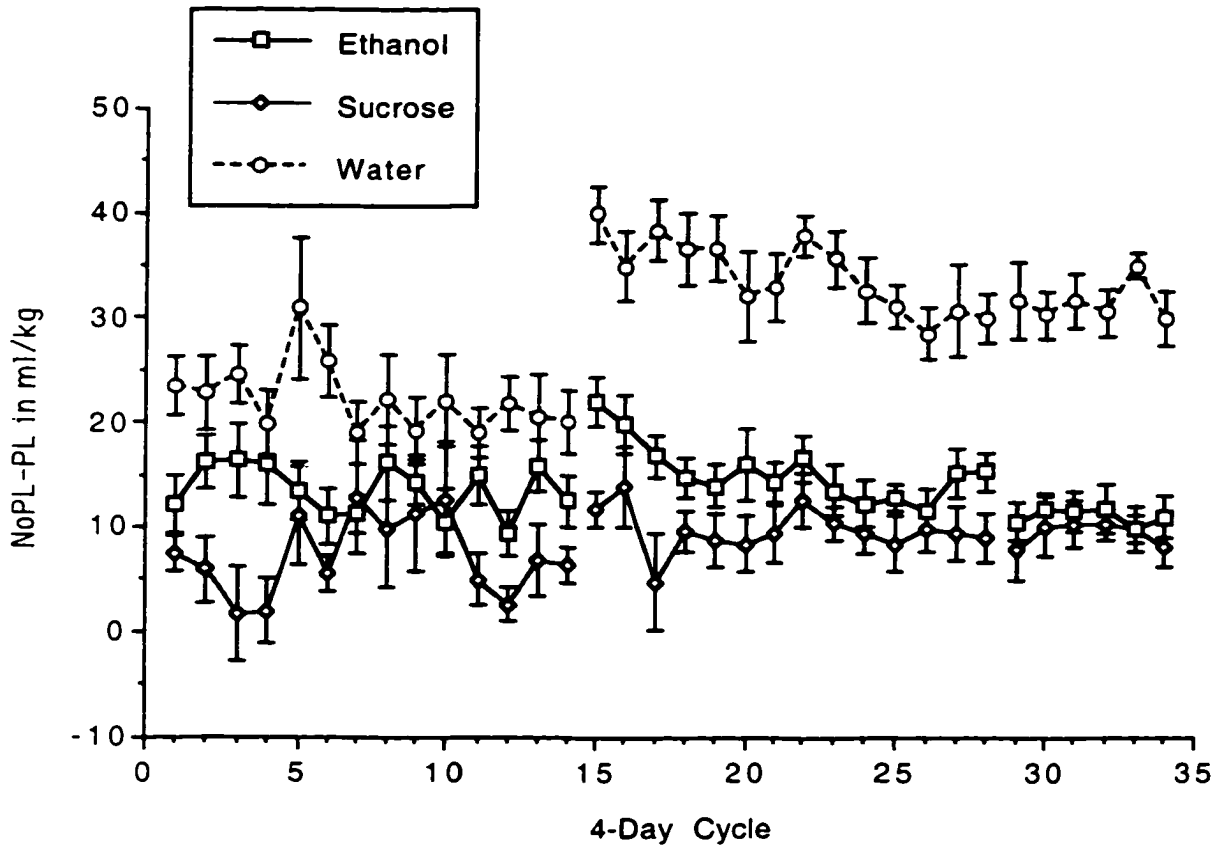

Figure 27: Mean (± SEM) difference in ml/kg between consumption in the drinking environment on days with and without preloading (NoPL-PL) for Ethanol (n = 10), Sucrose (n = 10), and Water groups (n = 12) in Experiment 5. Preloading was 15 mls of water during cycles 1-14 and 25 mls of water cycles 15-28 and 29-34.

after cycle 28. For each set of cycles, the main effect of group was significant, $F(2,29) = 26.811$, 66.906, and 55.908. In each case, means comparisons showed that the difference in drinking environment consumption between days with no-preloading and days with preloading was significantly greater for the Water group than for the average of the Sucrose and Ethanol groups, $F(1,29) = 44.975$, 127.976, and 111.372. The difference between days with no-preloading and days with preloading was significantly greater for the Ethanol group than for the Sucrose group only during cycles 1-14 and during cycles 15-28, $F(1,29) = 8.647$, and 5.835 ($p < .05$). The main effect of cycle was significant for cycles 15-28, $F(13,377) = 2.726$.

Home cage water consumption on successive days (two per cycle) of the experiment is shown in Figure 28. Standard error bars are not shown because they tended to obscure the data points. Eight values missing due to leaky water bottles were replaced by the group means. Water consumption by the three groups is strikingly similar across cycles. Group by cycle mixed ANOVAs were performed on the data from cycles 1-14, 15-28, and 29-34. These analyses revealed only significant effects of cycle, $F(27,783) = 7.675$, 7.815, and $F(11,319) = 3.939$.

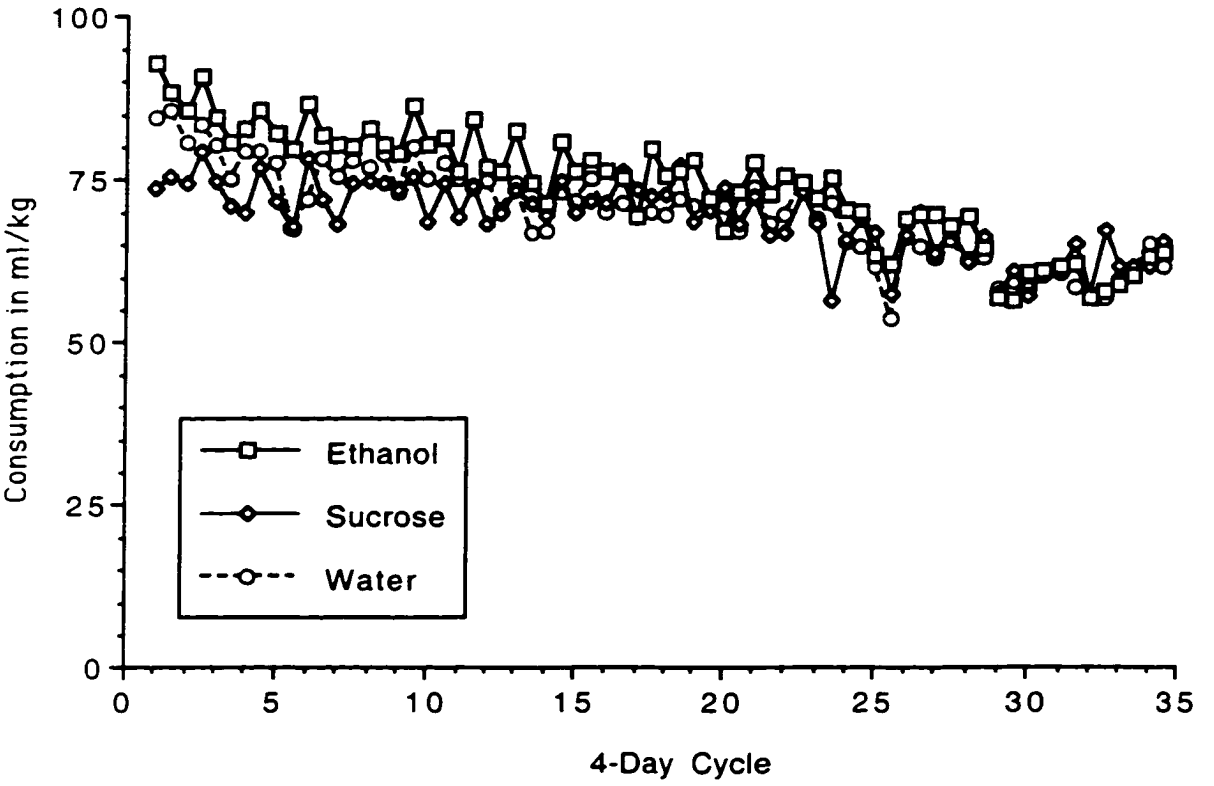

Figure 28: Mean home cage water consumption in ml/kg by Ethanol (n = 10), Sucrose (n = 10), and Water groups (n = 12) on successive water days (2 per 4-day cycle) during Experiment 5.

The mean number of fecal boli deposited in the drinking environment is shown in Figure 29. Bolus production in the drinking environment is very low across cycles and regardless of preloading.

Mean number of fecal boli deposited in the shock boxes on the preloading and no-preloading days of each cycle are shown in Figure 30. The Ethanol group appeared to produce fewer boli in the shock boxes on days with no preloading than any of the other groups on days with or without preloading. Separate group by treatment by cycle mixed ANOVAs were performed on the shock box defecation data for cycles 1-14, 15-28, and 29-34. These analyses revealed significant effects of treatment, $F(1,29) = 45.155$, 27.633, 11.344, and significant group by treatment interactions, $F(2,29) = 6.512$, 8.848, 7.452 for each set of cycles. During the first 14 cycles, the simple main effect of group was not significant for preloading or no-preloading days but the simple main effect of treatment was significant for each of the Ethanol, Sucrose and Water groups, $F(1,9) = 19.196$, 13.972, and $F(1,11) = 13.150$. In each case, defecation in the shock boxes was reduced on no-preloading days relative to preloading days. There were no other significant effects or interactions for cycles 1-14. Simple effect analysis on the group by treatment interaction for cycles 15 to 28 revealed simple main effects of treatment for the Ethanol and Sucrose groups, $F(1,9) = 14.124$, and 11.352 but not for the Water group. For the Ethanol and

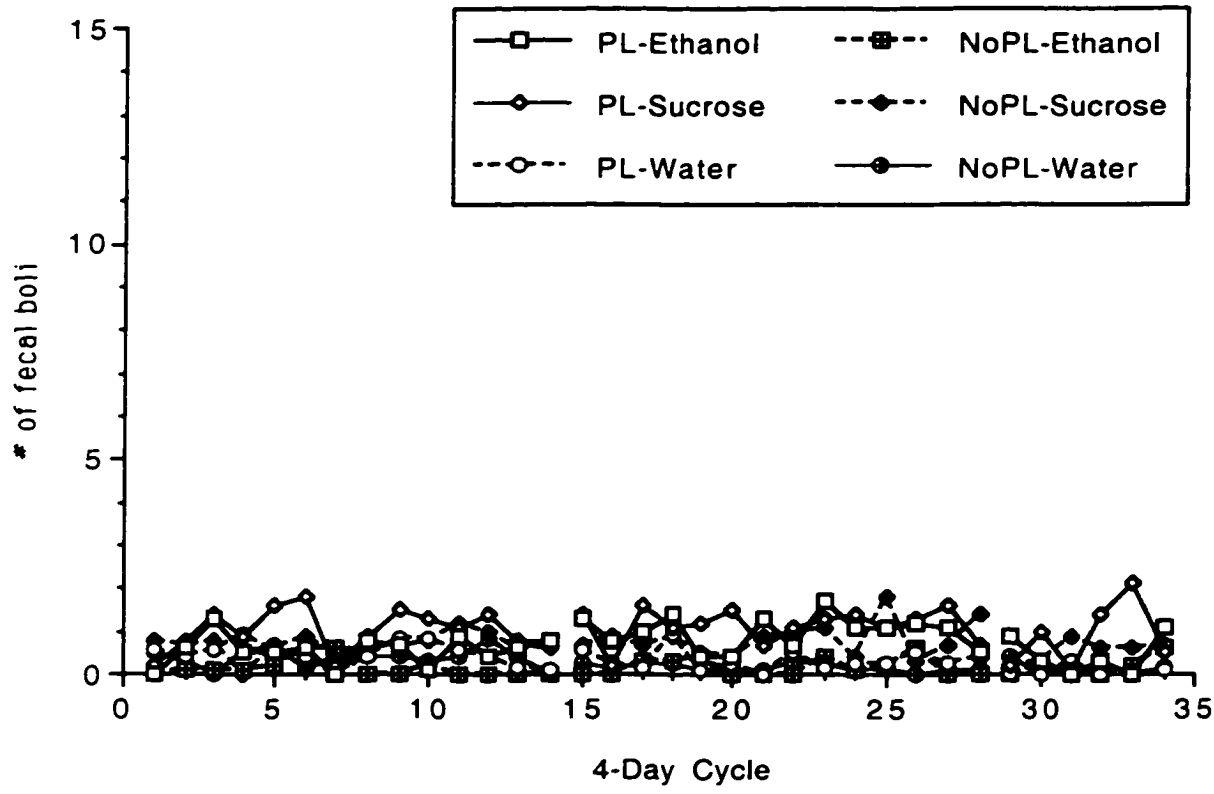

Figure 29: Mean # of fecal boli deposited in the drinking environment by Ethanol (n = 10), Sucrose (n = 10), and Water groups (n = 12) on days with and without preloading during Experiment 5.

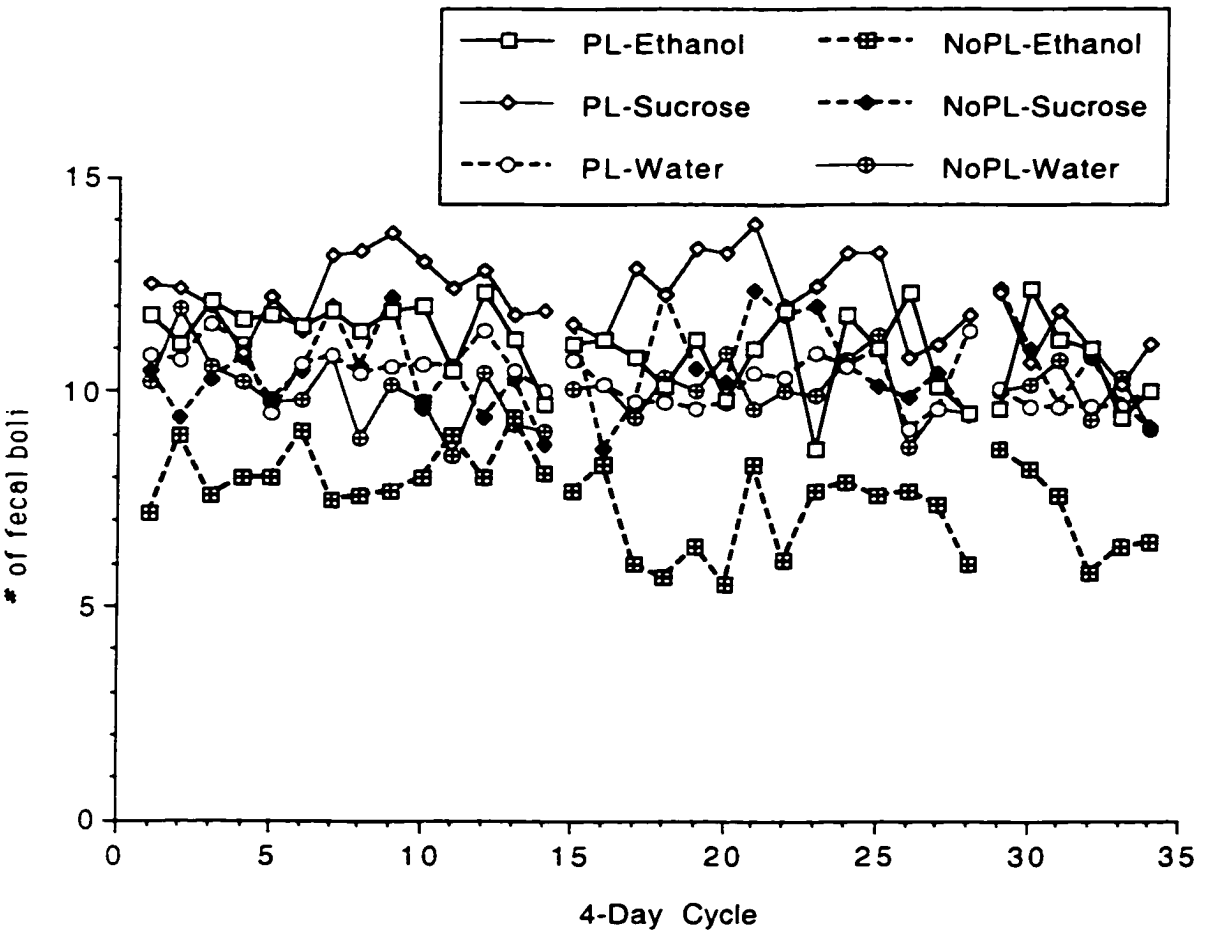

Figure 30: Mean # of fecal boli deposited in the shock boxes by Ethanol (n = 10), Sucrose (n = 10), and Water groups (n = 12) on days with preloading and days with no preloading in Experiment 5.

Sucrose groups, defecation in the shock boxes was higher on preloading days than on no-preloading days. The simple main effect of group was significant only on no-preloading days, $F(2,29) = 4.432$, $p < .05$. The Ethanol group deposited fewer boli in the shock boxes than the average of the Sucrose and Water groups, $F(1,29) = 8.392$. The Sucrose and Water groups did not differ from each other. Simple effect analysis on the group by treatment interaction for cycles 29-34 revealed a significant main effect of treatment only for the Ethanol group, $F(1,9) = 14.062$. For the Ethanol group, fewer boli were deposited in the shock boxes on no preloading days than on preloading days. The simple main effect of group was significant only on days with no-preloading, $F(2,29) = 3.503$, $p < .05$. Means comparisons showed that defecation in the shock boxes by the Ethanol group on days with no-preloading was lower than the average defecation of the Sucrose and Water groups, $F(1.29) = 6.937$, $p < .05$.

The difference between number of fecal boli deposited in the shock boxes on days with no preloading and days with preloading is shown in Figure 31. Most of the values are negative which indicates that more boli were deposited in the shock boxes on preloading days than on no-preloading days. The largest absolute values seem to be for the Ethanol group, which suggests that there was more of a difference in defecation between days with no-preloading and days with preloading for this group than for the

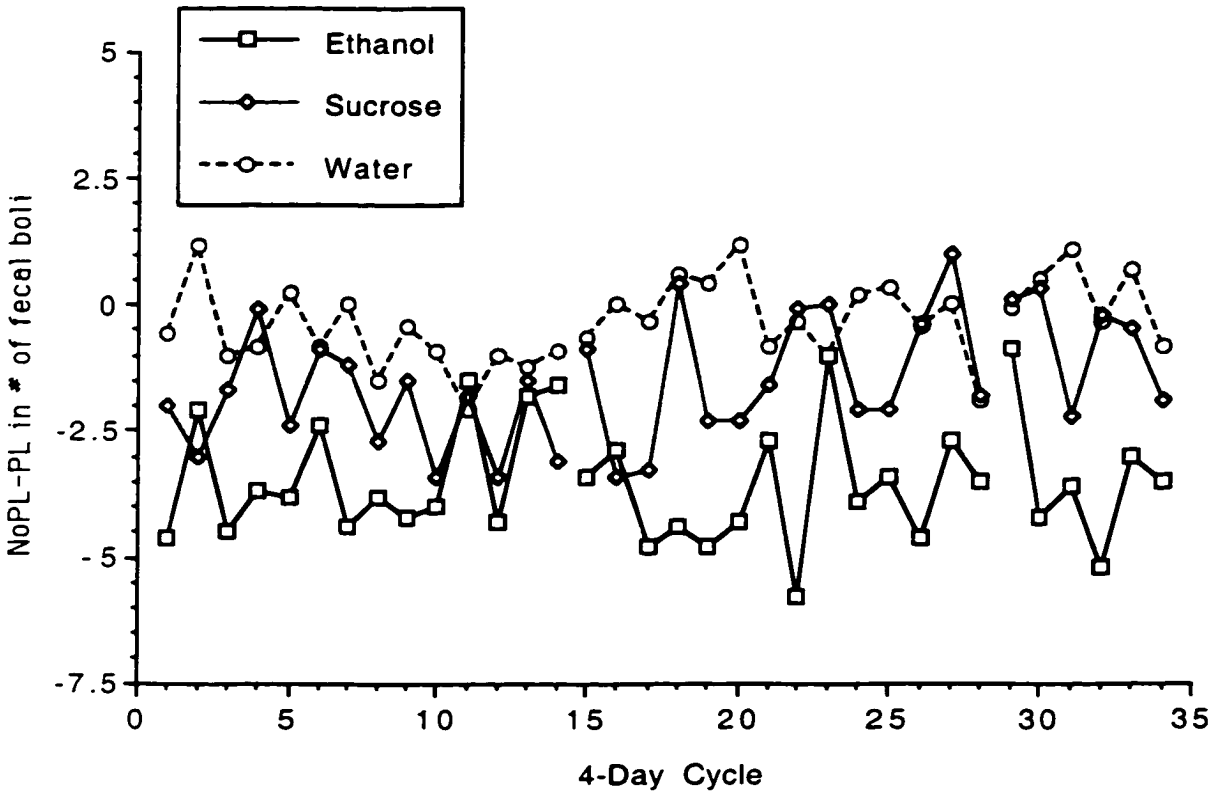

Figure 31: Mean (± SEM) difference between # of fecal boli deposited in the shock boxes on days with and without preloading (NoPL – PL) for Ethanol (n = 10), Sucrose (n = 10), and Water groups (n = 12) in Experiment 5.

Sucrose and Water groups. Separate group by cycle mixed ANOVAs were computed for the shock box difference scores from cycles 1-14, 15-28, and 29-34. In each case, only a significant effect of group emerged from the analysis, $F(2,29) = 6.512$, 8.648, and 7.452. Means comparisons showed that the difference score for the Ethanol group was significantly different than the average of the Sucrose and Water groups during each set of cycles, $F(1,29) = 9.043$, 14.929, and 13.606. The difference scores for the Sucrose and Water groups were never different.

Mean per cent freezing for the sessions that were videotaped during cycles 31-34 is summarized in Table 4. Freezing appeared to decrease across the three scoring intervals within a session. Animals in the Ethanol group generally froze less than animals in the Sucrose group and these animals froze less than animals in the Water group. No statistics were performed on these data since the number of animals (and sessions) for which I had data was so small. However, for none of the groups was there an appreciable difference between freezing on days with and without preloading.

During cycles 35-37, fluid access in the drinking environment was switched to sucrose for half of the Ethanol group and to ethanol for all of the animals in the Sucrose and Water groups. During the preloading period, 25 mls of water continued to be available. All four groups consumed most of the available fluid. A 4 x 2 x 3 group by

Table 4: Drinking environment consumption, shock box bolus production, and % freezing for some animals during cycles 31-34 of Experiment 5

	n*	DE Cons. ml/kg	SB boli	% Freezing			
				Early	Middle	Late	Total
ETHANOL							
Mean PL	7	13.62	11.3	52	46	29	42.29
Mean NoPL	8	27.73	7.1	52	47	27	42.00
SUCROSE							
Mean PL	7	21.23	8.3	69	47	43	52.95
Mean NoPL	8	27.47	7.8	71	48	37	51.92
WATER							
Mean PL	7	21.51	10.7	81	73	55	69.52
Mean NoPL	8	55.56	8.3	81	67	42	63.08

* n = number of videotaped shock sessions per group (data are from 5 different animals for each of the Ethanol and Sucrose groups; and from 6 different animals for the Water group)

treatment by cycle mixed ANOVA on the body weight data for cycles 35-37 revealed only significant effects of cycle, $F(2,56) = 11.228$, and treatment, $F(1,28) = 1701.259$. All of the groups were heavier on days with preloading than on days with no preloading.

Consumption in the drinking environment on preloading and no preloading days is shown in Figure 32 for the animals that have always had access to ethanol (E/E), animals that switched from ethanol to sucrose (E/S), animals that switched from sucrose to ethanol (S/E), and animals that switched from water to ethanol (W/E). A 4 x 2 x 3 group by treatment (preloading and no preloading) by cycle mixed ANOVA revealed significant effects of group, $F(3,28) = 8.321$, cycle, $F(2,56) = 4.315$, $p < .05$, and treatment, $F(1,28) = 220.831$, as well as a significant interaction between group and cycle, $F(6,56) = 2.773$. Fluid consumption was higher on days with no preloading than on days with preloading. Means comparisons on the group effect showed that consumption by E/E was greater than the average of S/E and W/E, $F(1,28) = 24.769$. Consumption by groups S/E and W/E did not differ. As before, a difference score was calculated for consumption on no preloading days minus preloading days. Analysis of these scores revealed no significant effects or interactions. Home cage water consumption was equivalent for the four groups. Bolus production in the drinking environment was low for all groups regardless of preloading. Bolus production in the shock boxes is shown in

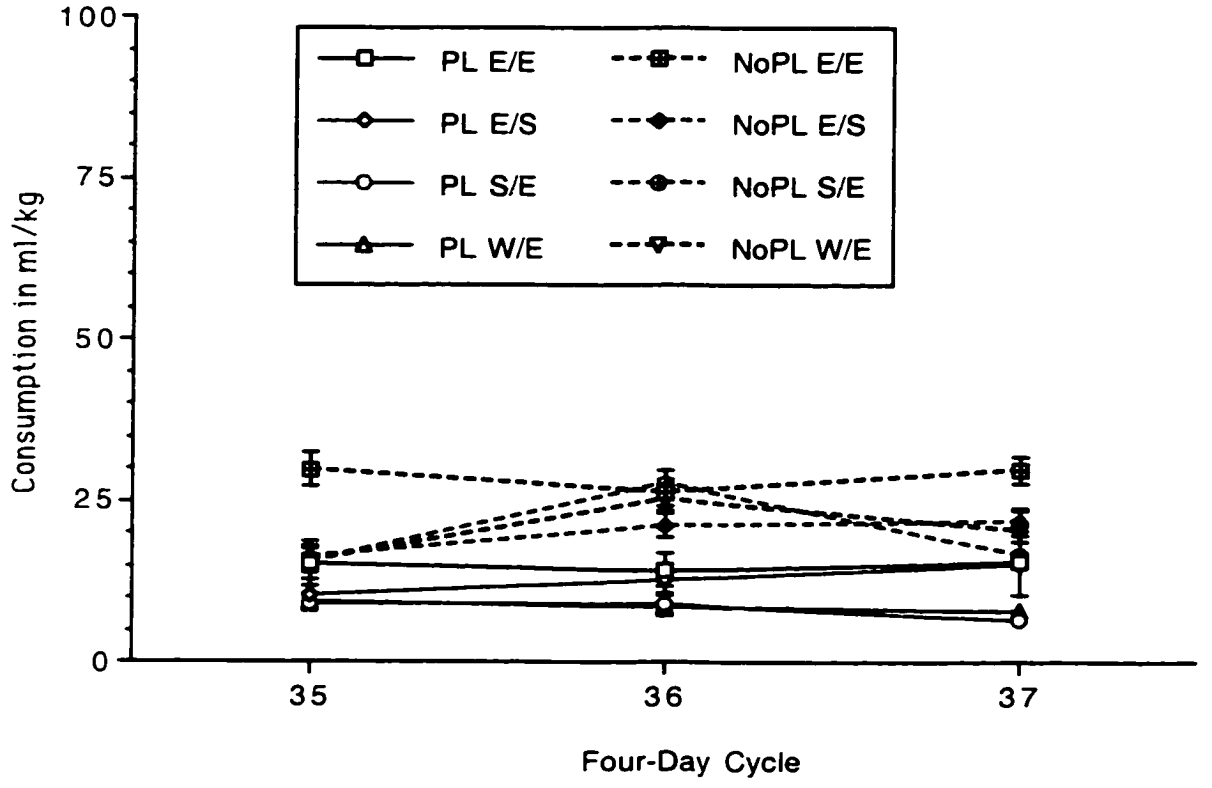

Figure 32: Mean (± SEM) consumption in ml/kg in the drinking environment on days with and without preloading during the three cycles (35-37) of Experiment 5 after fluids were switched. E/E (n = 5) is the group that had consumed and continued to consume ethanol in the drinking environment. E/S (n = 5) is the group that had previously consumed ethanol in the drinking environment and now consumed sucrose. S/E (n = 10) and W/E (n = 12) had previously consumed sucrose and water, respectively, and now consumed ethanol.

Figure 33. The number of fecal boli deposited in the shock boxes was higher than in the drinking environment. A 4 x 2 x 3 group by treatment by cycle mixed ANOVA on the shock box defecation data revealed only a significant effect of treatment, $F(1,28) = 24.443$. Bolus production was higher on days with preloading than on days with no preloading. Analysis on the difference score for shock box bolus data revealed no significant effects or interactions.

Discussion

When thirsty rats had the opportunity to consume 15 or 25 mls of water in their home cages prior to sessions in the drinking environment they consumed virtually all of the available fluid. Since there were no group differences in pre-loading consumption or in home cage water consumption each of the groups should have been equally thirsty (or non-thirsty) at the beginning of sessions in the drinking environment. All of the groups should have been more thirsty on days with no preloading than on days with preloading. So if thirst were the primary factor motivating consumption then consumption should have been equally suppressed on preloading days (relative to no preloading days) for all three groups. Ethanol, Sucrose, and Water groups did drink less in the drinking environment on preloading days than on no preloading days. However, the Ethanol group

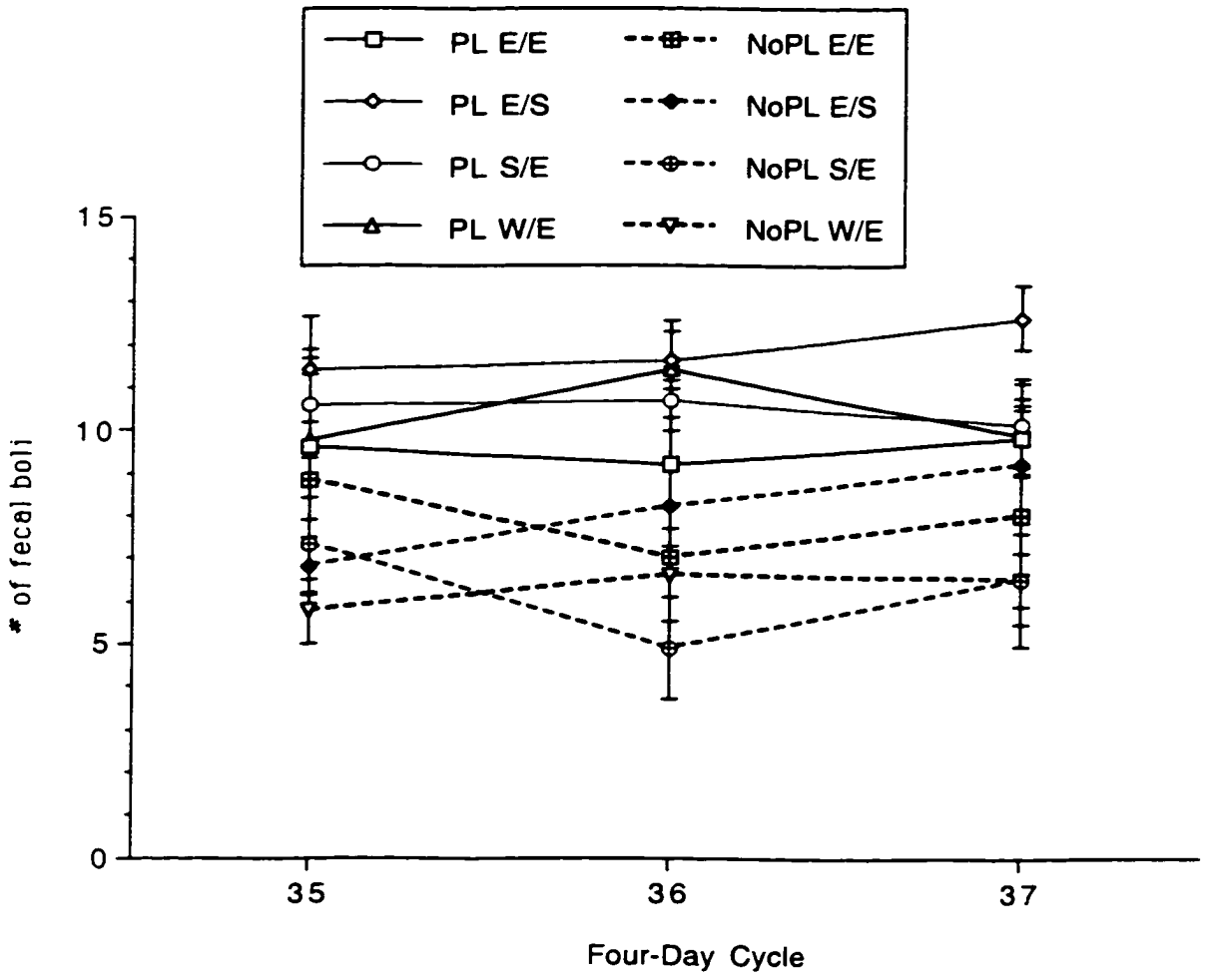

Figure 33: Mean (± SEM) number of fecal boli deposited in the shock boxes on days with and without preloading during cycles 35-37 of Experiment 5. Animals that had previously consumed ethanol had access to ethanol (E/E) or sucrose (E/S) during these cycles. Animals that had previously consumed sucrose and water all consumed ethanol during these cycles (S/E and W/E).

also always consumed less than the Sucrose and Water groups and the Sucrose group consumed less than the Water group. The question of whether consumption by individual groups was equally affected by the preloading manipulation is best addressed using the difference scores (consumption on days with no preloading minus consumption on days with preloading). The difference in drinking environment consumption between days with no preloading and days with preloading was greater for the Water group than for the average of the Sucrose and Ethanol groups. This difference suggests that thirst was a more important factor in motivating water consumption than in motivating ethanol or sucrose consumption. During the cycles with 15 mls of preloading and during the first set of cycles with 25 mls of preloading, the difference in drinking environment consumption between days with and without preloading was significantly greater for the Ethanol group than for the Sucrose group which could suggest that thirst was more important for ethanol than for sucrose. Sucrose consumption was less affected by the preloading manipulation than either ethanol or water consumption. This pattern of results suggests the involvement of some factor(s) other than thirst in motivating the consumption of sucrose and ethanol. Caloric regulation is one possible explanation. However, given that the ethanol and sucrose solutions were isocaloric, an explanation in terms of caloric

regulation cannot fully account for the results. So far, there is no evidence that the rats are drinking ethanol for its pharmacological effects.

The results of this experiment paralleled those of an experiment by Czirr, Hubbell and Reid (1987) who showed that providing water-deprived (but otherwise unstressed) Sprague Dawley rats with an opportunity to preload with water prior to an opportunity to consume ethanol or water markedly reduced consumption of water but not of ethanol. The authors concluded that ethanol was consumed for its nutrient value.

If the tension reduction hypothesis were correct then animals that had consumed ethanol should have shown less fear than animals that had consumed sucrose or water regardless of the reasons for that consumption. Bolus production measurements were taken in the drinking environment and in the shock boxes in order to assess fear. Bolus production was very low for all groups in the drinking environment (as was true in Experiment 4) which suggests that the rats were not afraid in this environment. Bolus production was higher in the shock boxes for all groups. The preloading manipulation had a greater influence on the defecation of the Ethanol group than the other groups (as evidenced by the larger absolute values of the difference scores shown in Figure 30). On days with no preloading, when ethanol consumption was higher, defecation was lower.

This could indicate that ethanol consumption on preloading days was not sufficient for the rats to obtain this fear reducing effect.

Examination of freezing in the shock box during the few sessions that were videotaped offered some suggestion that animals that had consumed ethanol (on days with or without preloading) froze less than animals that had consumed sucrose or water (see Table 4). However, these data must be viewed with caution since they represent only a small sample of the animals and sessions.

When solutions were switched so that Sucrose and Water groups had access to ethanol in the drinking environment (during cycles 35-37) there were overall group differences in ethanol consumption. Animals that had always had access to ethanol in the drinking environment consumed more than did animals that had previously had access to something else. All groups consumed more on days with no preloading than on days with preloading. For all of the groups, bolus production in the shock boxes was higher on days with preloading, when consumption was lower, than on days with no preloading. This result would seem to indicate that ethanol is having the same effect on defecation during initial exposures that it is having after many exposures.

EXPERIMENT 6

In the experiments described so far, thirsty rats had access to ethanol, sucrose or water in a distinctive drinking environment before shock. This design allowed me to compare the consumption of different groups of animals during a period when they were expected to anticipate shock. It did not allow me to compare an individual animal's consumption under different circumstances. This is an important comparison. If the tension reduction hypothesis were right, then during a signal for a noxious event, the animals should have been anxious and should have learned to drink ethanol to reduce or relieve this anxiety. Conversely, during a signal for safety or when there is no danger signal, the animals should not have become anxious and therefore should have consumed less ethanol. It is also possible that ethanol could become reinforcing because of tension reduction and then no longer depend on tension to set the occasion for drinking ethanol. If this were the case, I would expect to see increased consumption during the signal for a noxious event and followed by increased consumption in the absence of danger signals.

In this experiment, rats had the opportunity to drink an ethanol solution in two distinctive environments. Placement in one of these environments always preceded a shock session (0.8-mA, 2-s shocks delivered on a FT 60 s schedule for 1h, as in previous experiments) in another box. Placement in the other drinking environment always

preceded return to the home cage. Animals in a control group had access to an isocaloric sucrose solution in the two drinking environments. If the tension reduction hypothesis were correct, then animals in the ethanol group should have consumed more ethanol when they were anticipating shock than when they were anticipating no shocks. This pattern of consumption was not expected to hold for the sucrose group. In the drinking environment that preceded shock the sucrose and ethanol groups were expected to be initially equivalent on fecal bolus production but over successive days, the ethanol group was expected to defecate less than the sucrose group as their anxiety was relieved by ethanol. In the drinking environment preceding safety, defecation was expected to be very low and no difference was expected between groups. In the shock boxes, the ethanol group was expected to defecate less than the sucrose group because of its ethanol-reduced anxiety.

In all of the experiments discussed so far, thirsty rats have had access to fluid in a distinctive drinking environment with some relation to a shock session in another environment. Because the animals were thirsty when placed into the drinking environment and this was their only fluid access for 24 hours, I worried about the extent to which thirst could account for the consumption patterns observed in the drinking environment. The role of thirst was assessed in Experiment 5 by allowing the animals to

preload with water prior to some drinking environment sessions but not others. Ethanol and sucrose consumption were less affected by the preloading manipulation than was water consumption, which suggested that some factor other than thirst must have been involved. Since the animals were allowed to preload prior to sessions in the drinking environment it is possible that observed differences in drinking environment consumption did not reflect different factors motivating consumption but rather reflected satiation. The Water group in Experiment 5 may have been more affected by the preloading manipulation simply because they normally consumed more fluid in the drinking environment than the Sucrose and Water groups. In order to alleviate the satiation problem in this experiment supplementary water was made available two hours after the drinking environment session beginning with the sixteenth exposure to each drinking environment. The rats should have been able to learn that the drinking environment session was not their only opportunity to drink and therefore should have reduced their consumption in the drinking environment if thirst was the only factor motivating it. If consumption in the drinking environment were motivated by calories then neither sucrose nor ethanol consumption should have been affected by the supplementary water. If tension reduction motivated ethanol consumption then it alone should have been unaffected by the supplementary water.

Following the twentieth exposure to each drinking environment the fluids were switched. The Ethanol group was switched to sucrose in the drinking environment and the Sucrose group was switched to ethanol. This fluid switch was introduced in order to assess whether ethanol would have the same effects on animals that had been previously shocked without ethanol.

Method

Subjects

Subjects in this experiment were 32 experimentally naïve male Sprague Dawley rats from Charles River Canada, St. Constance, Quebec. The rats were housed as in all of the previous experiments. On the first day of the experiment, they had a mean weight of 359 g with a range from 282 to 399 g. The rats had ad lib access to food in their home cages throughout the experiment and fluid access was as described in the procedure section.

Apparatus

Environments. Four environments were used in this experiment: two drinking environments, Environment A and Environment B; a shock environment; and the rats' home cages. Home cages, shock boxes and drinking environment A were as described in

Experiment 1. Drinking environment B consisted of four operant chambers, 15.2 x 22.8 x 21.6 cm, with wire mesh floors in a dark room (with red lights) with a wintergreen odour. Background noise was provided by a white noise generator in Environment A and by a ventilation fan in Environment B. Thus Environment B differed from Environment A in terms of location, illumination, odour, sound, and cage type.

Fluids. Ethanol and sucrose solutions were identical to those used in Experiment 3 and Experiment 4.

Procedure

Access to water was reduced to 1 h/day beginning eight days before the start of the experiment. After this 8-day period, a singly alternating schedule was instituted such that the rats were weighed and then placed into a drinking environment on one day and remained in their home cages on the other day. During the first four 1-hour sessions in each drinking environment rats had access to water. Rats were returned to their home cages following the first exposure to each drinking environment (cycle 0). Shocks (0.8 mA, 2-s on a FT 60-s schedule) were delivered in another environment following the second exposure to one of the drinking environments (A or B) and this contingency was maintained for the rest of the experiment. The three cycles (cycles 1-3) in which water was available in both drinking environments and one was followed by shock served as a

baseline period. After this period, water was replaced by ethanol and sucrose solutions in the drinking environments for the Ethanol and Sucrose groups respectively. Water continued to be available in the home cage on intervening days. Table 5 shows a representative four day period for the Ethanol group. To restate: the rats remained in their home cages on alternate days. On days away from the home cage they were weighed and then placed in either drinking environment A or B on a pseudo-randomly alternating schedule. Drinking environment sessions lasted for one hour. Half of the rats were returned to their home cages after sessions in A and placed in the shock boxes after sessions in B. These contingencies were reversed for the remaining half of the animals. Shock sessions lasted for one hour and consisted of 0.8 mA, 2-s shocks delivered on a FT 60-s schedule. Fluid access occurred during the same hour each day. Ethanol and Sucrose groups differed only in terms of what fluid was available in the drinking environment. Drinking environments A and B were counterbalanced.

Water was the only fluid available during cycles 1-3. During cycles 4-15, rats had access to ethanol or sucrose for 1 hour in the drinking environments or to water for 1 hour in the home cages. Beginning with cycle 16 and continuing throughout the experiment, supplementary water was introduced so that two hours after the end of the initial daily drinking session (in the home cage or in a drinking environment) rats had

Table 5: Daily schedule for the Ethanol group during a representative four day period during Experiment 6

Hour	Day 1	Day 2	Day 3	Day 4
1	Home Water	**A** Ethanol	Home Water	**B** Ethanol
2	Home	Shock	Home	Home
3	Home	Home	Home	Home
4	Home Water	Home Water	Home Water	Home Water

Note: **A** = Drinking Environment A; **B** = Drinking Environment B; E = Ethanol; S = Sucrose; W = Water; DE = Drinking Environment. The 4th hour, supplementary water, was not included until after the 16th drinking environment exposures.

The Sucrose group received sucrose in A and B instead of ethanol. Both drinking environment (A and B) and sequence were counterbalanced. Home cage water days were always singly alternating. A and B occurred on a pseudo-random schedule with as many as three A's or B's in succession.

access to water for an additional hour. The shock schedule was switched from a FT 60-s schedule to a variable time (VT) 60-s schedule (with a range from 1-295 s) beginning with cycle 19. The inter-shock intervals were determined according to Fleshler and Hoffman's (1962) equation for constant-probability VI schedules and were randomized in three different sequences. These three sequences were used in the same order for all of the rats. Beginning with the twenty-first cycle, the solutions available in the drinking environment were switched so that the Ethanol group was given access to sucrose and the Sucrose group was given access to Ethanol. Finally, from cycles 27-32 drinking environment sessions were reduced to 30 minutes. Shock sessions and supplementary water were maintained at 1 hour.

The rats were assigned to eight squads of four animals each. As in previous experiments, the squads were run out of phase so that each day four of the squads were taken to drinking environments for their fluid access and the remaining four squads had a home cage water day. Within each squad, two animals were in the Ethanol group and two were in the Sucrose group. For one animal from each group, drinking environment A was paired with shock and drinking environment B was paired with return to the home cage. This contingency was reversed for the other squad member from the same group.

Measures. Rats were weighed prior to placement in each drinking environment. Fluid consumption was recorded in ml and in ml/kg for all drinking environment sessions, home cage water days, and supplementary water periods (on drinking environment and home cage days). The number of fecal boli deposited in the safe-paired and shock-paired drinking environments and in the shock boxes was also recorded.

The many days of the experiment were separated into 7 discrete sections for the purpose of analyses. The first section consisted only of the very first exposures to each drinking environment (cycle 0). The next three exposures (1-3), following the introduction of shock were grouped together as a baseline phase. The first 12 exposures to each drinking environment after the introduction of ethanol and sucrose solutions (exposures 4-15) were grouped together to form the third section. Drinking environment exposures 16-18 and 19-20 after the introduction of supplementary water and VT shocks formed the fourth and fifth sections. Drinking environment exposures 21-26, the first 6 exposures after availability of ethanol and sucrose solutions was reversed, were grouped together to form the sixth section. The seventh section was comprised of the final six exposures (27-32) to each environment.

Results

Mean (± SEM) body weight in grams for each drinking environment day for animals in the Ethanol and Sucrose groups is shown in Figure 34. Body weight increased across days for both groups. Group by day mixed ANOVAs on the body weight data for each section revealed no significant effects or interactions involving group. The effect of day was significant during sections 2-7.

Fluid consumption in the safe- and shock-paired drinking environments is shown in Figure 35. The safe-paired drinking environment is the one followed by return to the home cage and the shock-paired drinking environment is the one followed by a shock session in another environment. A 2 x 2 group by environment mixed ANOVA revealed no significant effects or interactions on consumption during the first exposure to each environment. Analyses performed on the data for consumption during the baseline period (section 2) showed no significant effects or interactions.

For section 3, a 2 x 12 x 2 group by exposure by environment mixed ANOVA revealed significant effects of group, $F(1,30) = 11.365$, and exposure, $F(11,330) = 13.354$. Consumption by the Sucrose group was higher than consumption by the Ethanol group. The exposure by group and group by exposure by environment interactions were both significant, $F(11,330) = 3.026$ and 1.835. Simple effect analysis showed that the

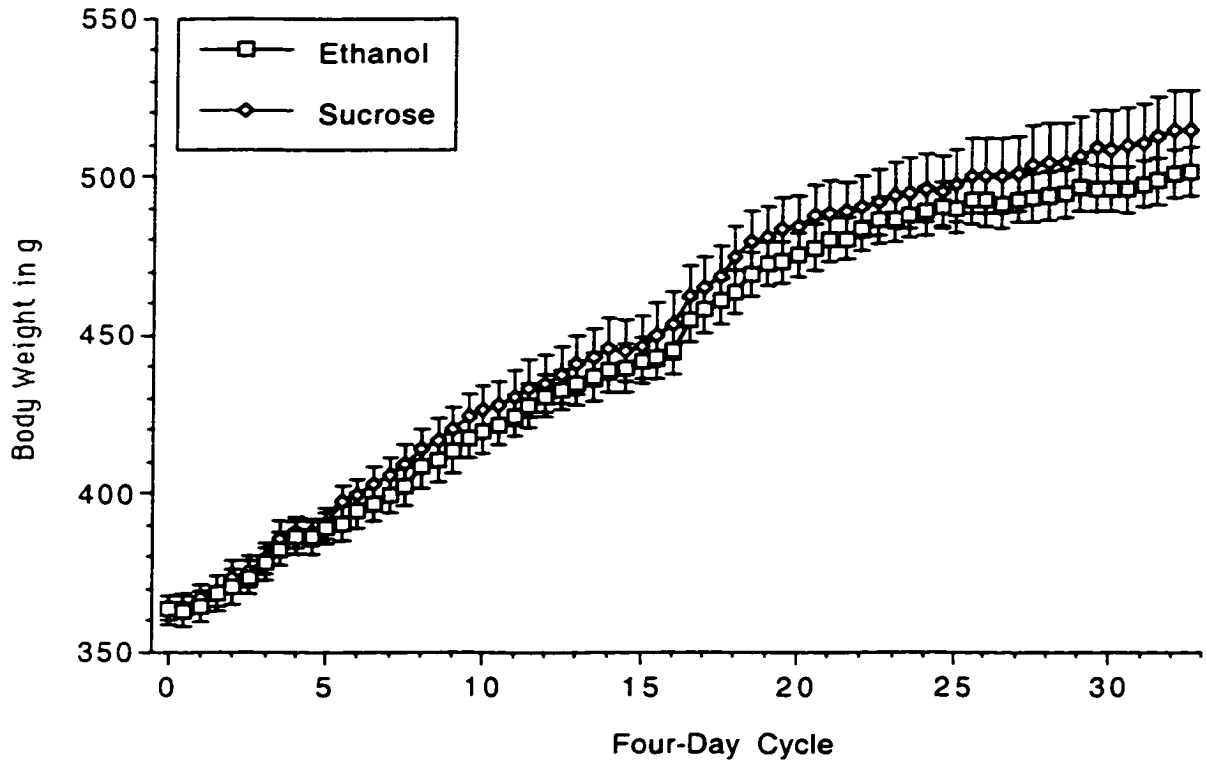

Figure 34: Mean (±SEM) body weight in grams for the Ethanol and Sucrose groups on successive drinking environment days (2 per 4-days) in Experiment 6.

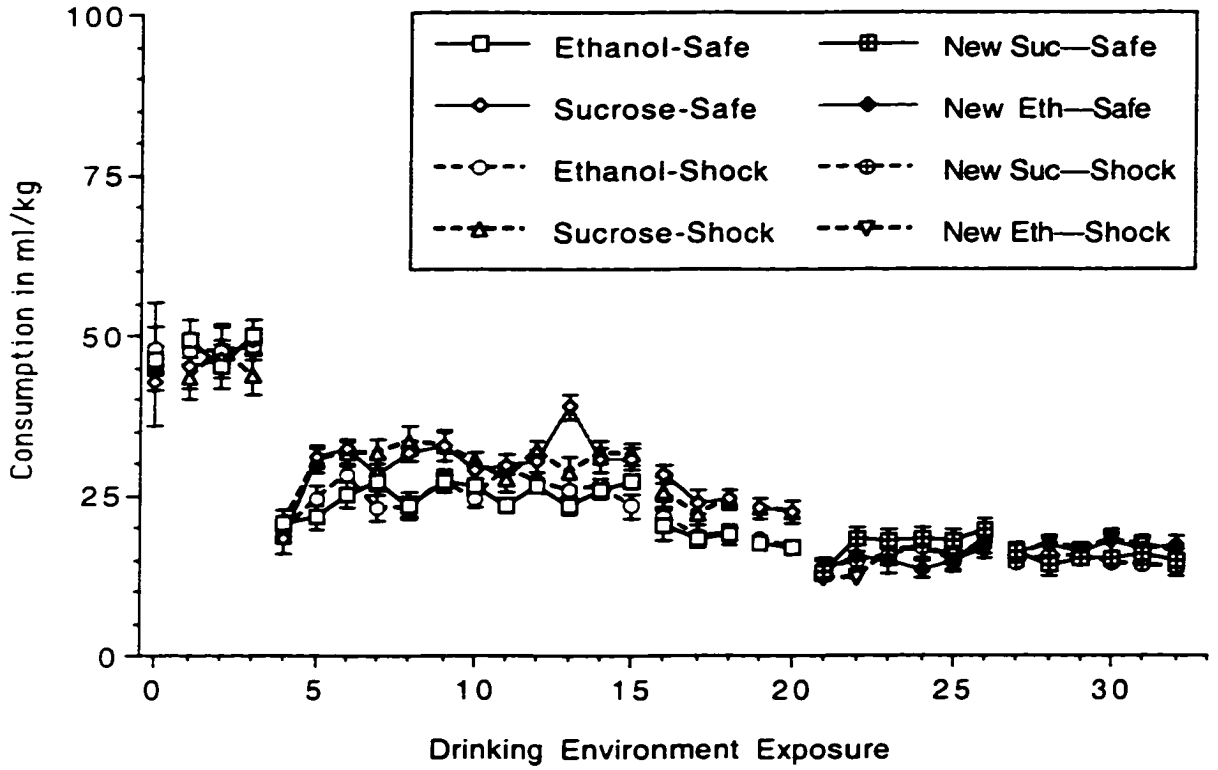

Figure 35: Mean (± SEM) consumption in the safe- and shock-paired drinking environments by the Ethanol and Sucrose groups in Experiment 6. Water was the only fluid available in the drinking environments during exposures 0-3. From cycles 4-20, the Ethanol group consumed ethanol and the Sucrose group consumed sucrose in each drinking environment. From exposure 21-32, the New Suc group (formerly the Ethanol group), consumed sucrose in the drinking environments and the New Eth group (formerly the Sucrose group) consumed ethanol in the drinking environments.

exposure by environment interaction was not significant for either group. The group by environment interaction was significant only for the eleventh drinking environment exposure, $F(1,30) = 9.425$. For this exposure, the simple main effect of environment was significant for the Ethanol group, $F(1,15) = 14.524$, and the simple main effect of group was significant only for the safe-paired drinking environment, $F(1,30) = 10.090$. Ethanol consumption was higher in the shock-paired than in the safe-paired drinking environment. In the safe-paired drinking environment sucrose consumption was greater than ethanol consumption.

The group by exposure interaction was significant for both the safe- and shock-paired drinking environments, $F(11,330) = 2.707$ and 2.157 ($p < .05$). The simple main effect of exposure was significant for the Ethanol group in both the safe- and shock-paired environments, $F(11,165) = 2.549$ and 2.895 and for the Sucrose group in both environments, $F(11,165) = 8.415$ and 4.784. For the safe-paired environment, the simple main effect of group was significant during the fifth, sixth, eighth, and eleventh and thirteenth drinking environment exposures. For the shock-paired environment, the simple main effect of group was significant during the fifth, seventh, eighth, tenth, twelfth, fourteenth and fifteenth drinking environment exposures. For both the safe- and shock-

paired environments, every time the simple main effect of group was significant, consumption by the Sucrose group was greater than by the Ethanol group.

A group by exposure by environment mixed ANOVA on the data for section 4 (drinking environment exposures 16-18) revealed significant effects of group, $F(1,30) = 9.960$, and cycle, $F(2,60) = 6.155$. Sucrose consumption was higher than ethanol consumption. During exposures 19 and 20 (section 5), when shocks were switched from a FT to a VT schedule, sucrose consumption continued to be higher than ethanol consumption, $F(1,30) = 13.639$, as shown by a 2 x 2 x 2 group by exposure by environment mixed ANOVA. Analysis of the data from sections 6 and 7, the first 6 exposures after the solutions were switched and the final 6 exposures, revealed no significant effects or interactions involving group.

Consumption in the home cage on water days is shown in Figure 36. The most striking feature of this figure is the precipitous decline in consumption coincident with the introduction of a supplementary water period (in cycle 16). Group by day mixed ANOVAs revealed no significant effects or interactions involving group during any section except section 3 (4-15). There was a significant effect of group during section 3, $F(1,30) = 4.233$, $p < .05$. The Ethanol group consumed more water in the home cage than the Sucrose group.

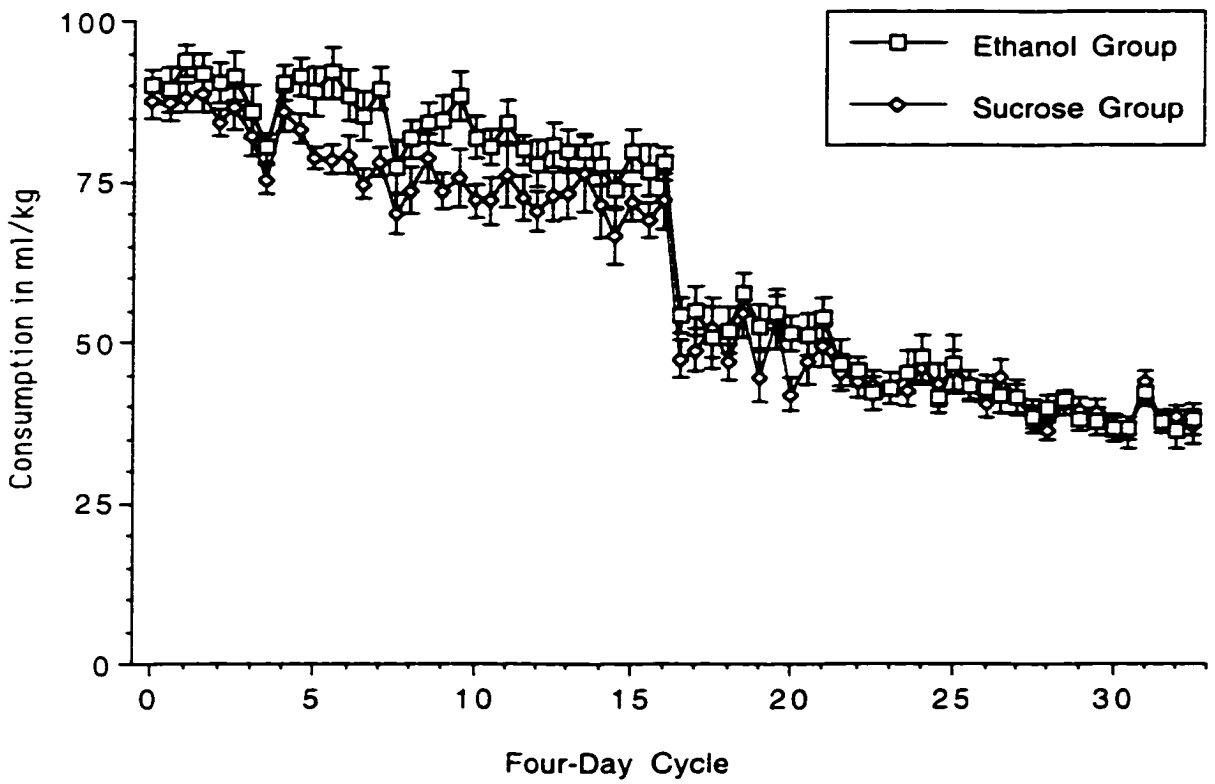

Figure 36: Mean (± SEM) home cage water consumption by the Ethanol and Sucrose groups in Experiment 6.

Consumption during the supplementary water periods on days with drinking environment exposures and on days spent in the home cage is shown in Figures 37 and 38 respectively. Group by exposure by drinking environment mixed ANOVAs carried out on the supplementary water consumption on drinking environment days revealed a consistent effect of environment, $F(1,30) = 24.45$, 9.254, 8.44, and 20.129 for sections 4-7. Consumption was higher during the supplementary water period following exposure to the safe-paired drinking environment than the shock-paired drinking environment. There was a significant effect of group only during section 7, $F(1,30) = 11.161$. The original Ethanol group (which had access to sucrose in the drinking environment during this section) consumed more supplementary water than the original Sucrose group (which had access to ethanol in the drinking environment during this section). There was also a significant group by environment interaction during section 7, $F(1,30) = 8.161$. Simple effect analysis of the interaction revealed a significant main effect of environment for the Ethanol group, $F(1,15) = 34.804$, but not for the Sucrose group. Consumption by the Ethanol group during the supplementary water period was higher on no-shock days than on shock days. The simple main effect of group was significant following both safe- and shock-paired drinking environment exposures, $F(1,30) = 13.912$ and 7.622. In both cases, supplementary water consumption was higher by the Ethanol group (which consumed

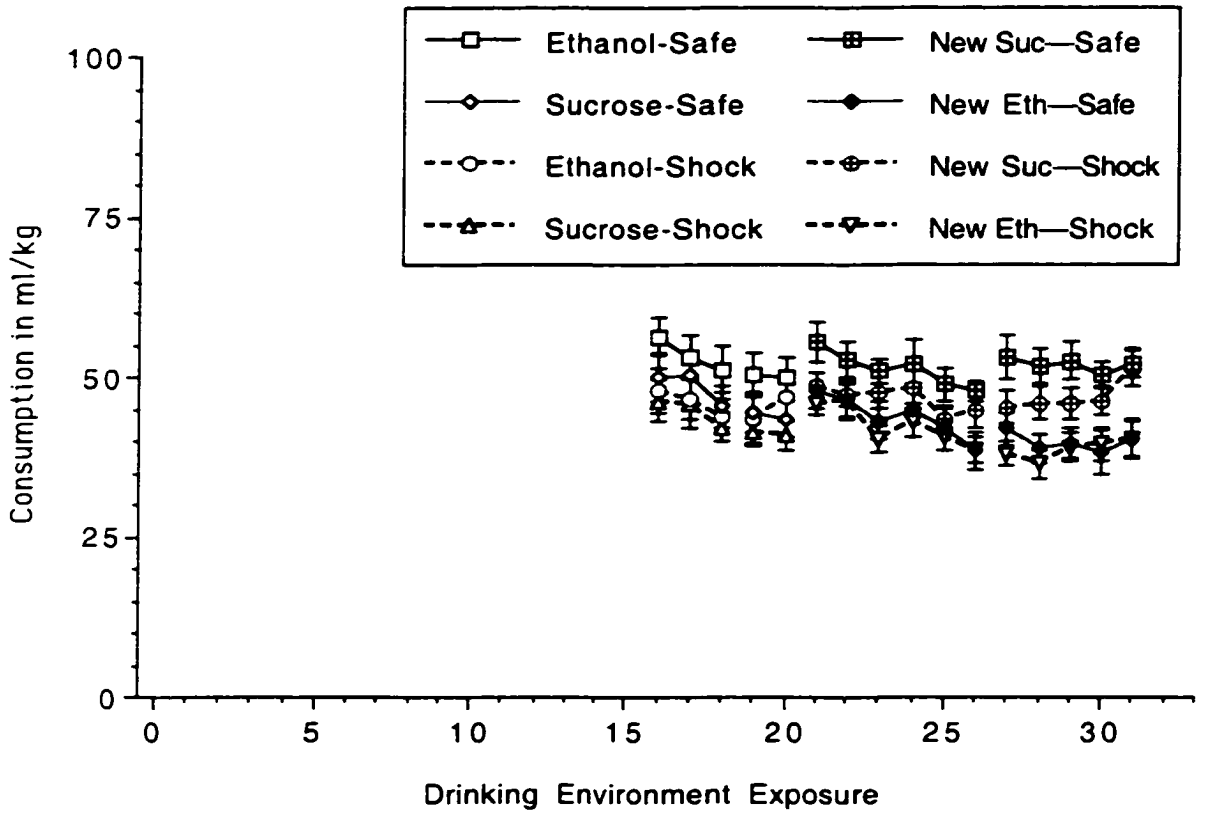

Figure 37: Mean (± SEM) supplementary water consumption in ml/kg in the home cage by the Ethanol, Sucrose, New Suc (previously Ethanol) and New Eth (previously Sucrose) groups following shock sessions or following exposure to the safe-paired drinking environment in Experiment 6.

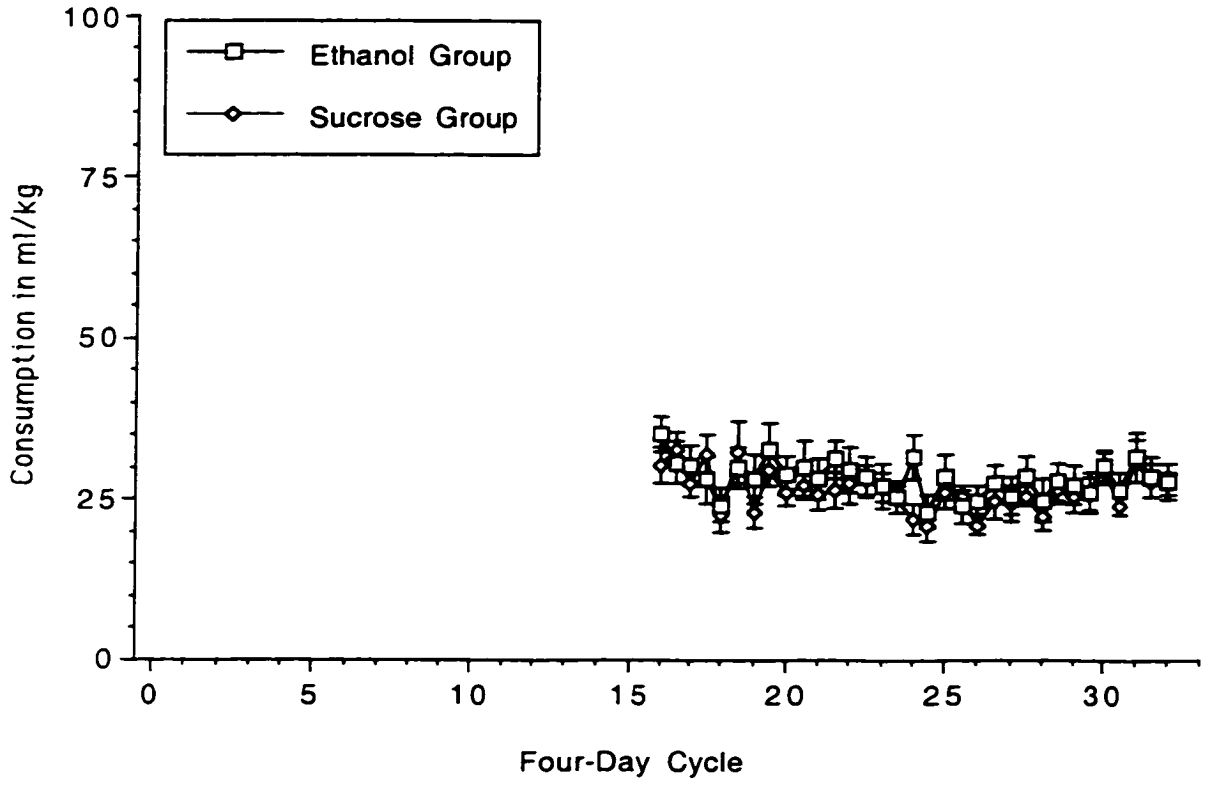

Figure 38: Mean (± SEM) supplementary water consumption in ml/kg by the Ethanol and Sucrose groups on home cage water days in Experiment 6.

sucrose in the drinking environment) than by the Sucrose group (which consumed ethanol in the drinking environment). Examination of supplementary water consumption on home cage water days (via group by day mixed ANOVAs) revealed no significant effects or interactions involving group.

Figure 39 shows the mean number of fecal boli deposited in the safe- and shock-paired drinking environments. Defecation was negligible in both drinking environments by both groups before and after the fluids were switched.

The number of fecal boli deposited in the shock boxes is shown in Figure 40. A group by shock day mixed ANOVA revealed no significant effects or interactions involving group during the baseline period although there was an increase in bolus production across shock days, $F(2,60) = 5.238$. Similar analyses for Section 3 (4-15) revealed a significant interaction between group and cycle, $F(11,330) = 2.088$, $p < .05$. The simple main effect of cycle was significant only for the Sucrose group, $F(11,165) = 1.883$, $p < .05$. The simple main effect of group was significant only for the eleventh and fourteenth shock days, $F(1,30) = 10.806$ and 6.039 ($p < .05$). For each of these shock days, the Sucrose group produced more boli than the Ethanol group. Group by shock day mixed ANOVAs revealed no significant effects or interactions for days 16-18 or 19-20.

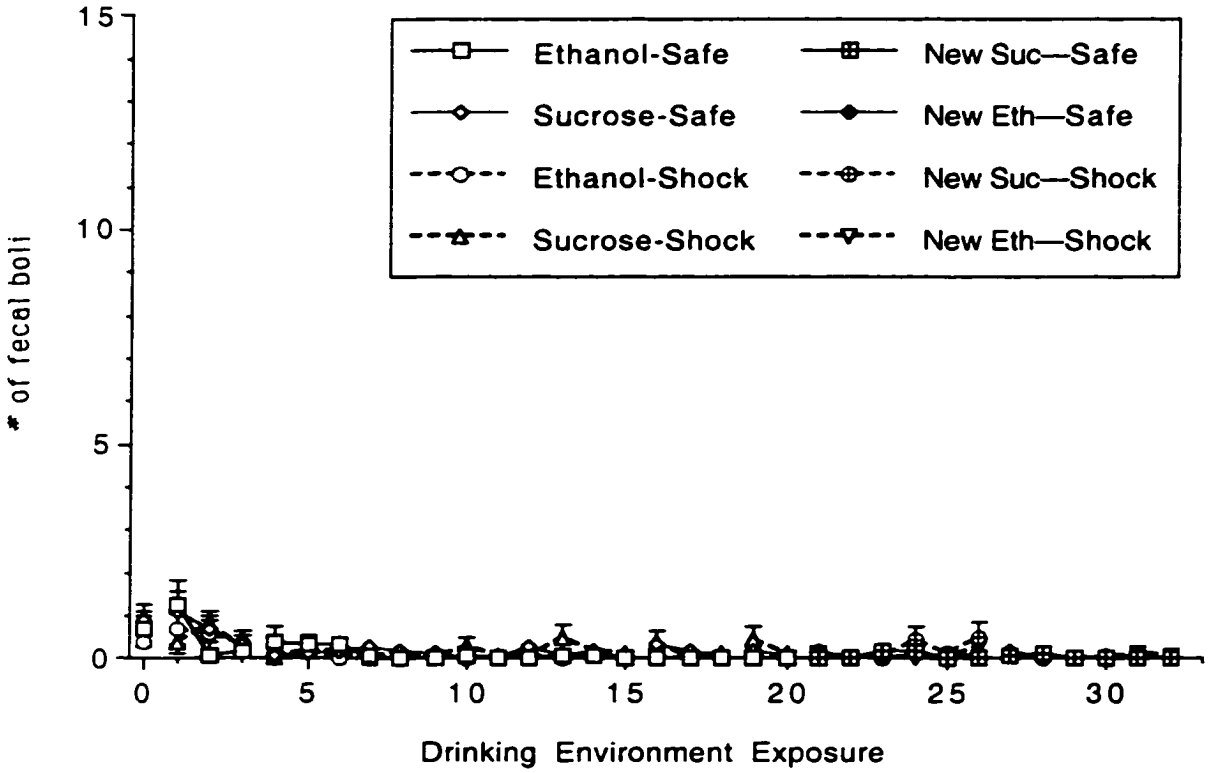

Figure 39: Mean (± SEM) number of fecal boli deposited in the safe- and shock-paired drinking environments by the Ethanol, Sucrose, New Suc (formerly Ethanol), and New Eth (formerly Sucrose) groups in Experiment 6.

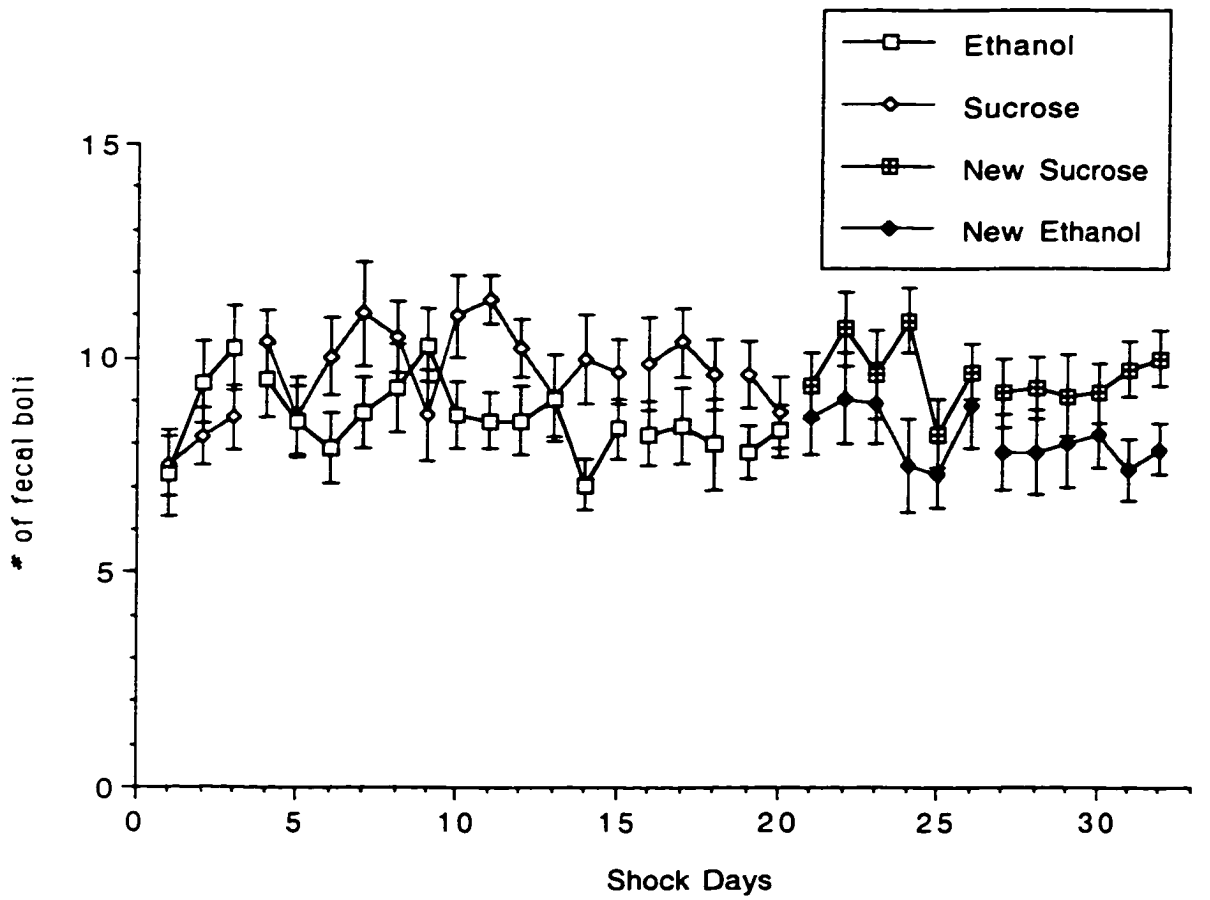

Figure 40: Mean (± SEM) number of fecal boli deposited in the shock boxes by the Ethanol, Sucrose, New Sucrose (formerly Ethanol), and New Ethanol (formerly Sucrose) groups in Experiment 6.

Analysis of the remaining shock days, 21-26 and 27-32, revealed no additional significant effects or interactions involving group.

Discussion

This experiment permitted within subject comparions of ethanol consumption under different circumstances. Groups of rats consumed either ethanol or sucrose in two drinking environments that differed in terms of location, illumination, odour, sound, andcage type. Sessions in one of these drinking environments were always followed by return to the home cage (safe-paired) and sessions in the other were always followed by a shock session in another box (shock-paired). Neither consumption during the initial exposures to each environment nor consumption during the baseline period differed between groups or between environments, which indicates that the groups and environments started out equivalent. During the 12 exposures to each environment after solutions were introduced there was a significant group by exposure by environment interaction. According to predictions from the Tension Reduction Hypothesis, ethanol consumption should have been consistently higher in the shock-paired than in the safe-paired drinking environment. However, this difference was only apparent when comparing the eleventh exposure to each drinking environment. When there were significant differences between groups (in either drinking environment during exposures

4-15, 16-18 or 19-20), the Sucrose group always consumed more fluid than the Ethanol group. This is consistent with my earlier findings (Experiments 1-5). After the ethanol and sucrose solutions were switched, there were no further effects or interactions involving group. The consumption data failed to provide consistent support for the tension reduction view. The introduction of supplementary water in the home cages appeared to have little effect on any measure except the initial daily home cage water consumption. Giving the animals extra water at the end of the day still meant that the animals were thirsty when they are first placed in the drinking environments.

As before, ethanol consumption might be expected to have the effect of reducing fear even if this is not what is motivating consumption. This possibility was once again assessed by looking at fecal bolus production in the drinking and shock environments. Defecation was near-zero in both drinking environments, which provides no support for differential fear (in either the Ethanol or Sucrose groups). Although bolus production is much higher in the shock boxes, there is still no consistent effect of group. This last finding might suggest that the group differences in bolus production observed in previous experiments were attributable to differences in fluid consumption.

Switching from a fixed time to a variable time shock schedule had little effect. It is possible that at the time the schedule was switched, the rats were already as afraid as

they could be in the shock boxes. After all, they had already experienced 18 shock sessions.

EXPERIMENT 7

In previous experiments, rats that had consumed ethanol before shock defecated less and froze less than rats that had not consumed ethanol. This pattern of results was consistent with the first tenet of the tension reduction hypothesis, that ethanol reduces tension (Cappell & Herman, 1972). However, tension reduction is not the only possible explanation. The lower level of expressed fear in our ethanol animals might have been due to an analgesic effect of ethanol (Pohorecky, 1981). If this were the case, then for animals that consumed ethanol prior to shock, the shocks might have felt less painful and for this reason might have evoked less conditioned fear. Ethanol may have interfered with the acquisition of fear rather than reducing its expression. The confound between fear reduction and pain reduction could be dealt with in a number of different ways. One possibility would be to establish the conditioned fear prior to the introduction of ethanol. This was done in Experiment 6, in which there were three shock sessions before the introduction of ethanol. Further, in both Experiments 5 and 6, the fluid available in the drinking environment was switched late in the experiment so that there were some animals that had a lot of shock experience prior to consuming ethanol. Establishing fear prior to introducing ethanol allowed for an assessment of the effect of ethanol on fear

rather than on the acquisition of fear. However, if when ethanol was introduced it were available prior to shock sessions (as in the previous experiments) there would still be the possibility that changes in behaviour were due to its analgesic properties. That is, if consumption of ethanol made the shocks less painful then the fear response that had been initiated before ethanol's introduction might have been expected to extinguish gradually. This problem could be avoided if ethanol was only made available on days when shock was not being presented. In the next two experiments, ethanol was introduced only after fear (as measured by increased bolus production and freezing) had been established and ethanol was not available on days when shock was administered.

In all of my previous experiments, the rats were fluid deprived, which raised the possibility that consumption in the drinking environment(s) was motivated by thirst. In Experiment 5, water consumption in the drinking environment was reduced more by preloading than was consumption of either ethanol or sucrose. While this observation suggested that thirst might not have been the only factor motivating ethanol and sucrose consumption it still seemed to be true that thirst was one of the factors motivating consumption. In order to further reduce the influence of thirst on consumption in this experiment the rats were not fluid deprived. They had free access to water in their home cages.

When bolus production was monitored (Experiments 3-6), levels were much lower in the drinking environment(s) than in the shock boxes. This observation suggested that the rats were not fearful in the drinking environment(s). If they did not experience anticipatory fear at the time when they had access to ethanol, then it could be argued that they could not have been expected to consume ethanol to reduce their fear (even though any ethanol that they did consume might have had that effect in the shock box). In order to make sure that the animals were experiencing fear at the time when they had access to ethanol, in the next two experiments the rats had access to fluids in the shock boxes. Other researchers have allowed their rats to consume ethanol in the shock boxes (for example (Bond, 1978; Kinney & Schmidt, 1979; Merrimen, 1996; Volpicelli et al., 1982) but in all of their experiments ethanol was available on the same days that shocks were being delivered so that any effects that did occur might have been based on analgesic effects of ethanol. These experiments monitored fluid consumption but did not measure the effects of ethanol on fear behaviour (except Merrimen, 1996 who measured bolus production). Only Merrimen included a control group that received an isocaloric solution.

It seemed very unlikely that the rats would drink in the shock boxes under the shock parameters used in the earlier experiments (0.8 mA, 2-s shocks on FT or VT 60-s schedules for 1 hour). I wanted to use shock parameters that would produce a moderate

level of freezing. If there was too much shock, the rats would have frozen continuously and this would have been incompatible with drinking. With too little shock, there would have been no fear to reduce. In addition to concern over the amount of freezing, I was also concerned about creating a shock schedule such that the likelihood of drinking being punished by shock was minimized (Bond, 1978). It seemed that one way to accomplish this would be to group the shocks at the end of the session, thereby giving the rats a shock-free period at the beginning of the session in which to drink. Such a pattern of consumption would have the added benefit of allowing the pharmacological effects of ethanol to take place while the animal was still in the shock box.

In order to determine a set of shock parameters that would produce an optimal level of freezing, I conducted a 7-day pilot experiment in which groups of rats (n = 11 per group) were exposed to a daily 30-min session in the shock box which was alternately a no-shock or a shock (1-, 5- or 15-Shocks) session. Water was available during all sessions. The experiment began with a no-shock day so that the animals would become accustomed to drinking in this environment before they received any shocks. The number of fecal boli deposited during each daily session was recorded. For each session, freezing was measured every 5 s during five 1-minute scoring intervals (minutes 2, 7, 15, 21, and 29). On each of the three shock days, the shocks occurred at different times, however, the

following "rules" were observed. The shock delivered to the 1-Shock group coincided with one of the shocks delivered to the 5-Shock group. The shocks delivered to the 5-Shock group coincided with five of the shocks delivered to the 15-Shock group. No shocks were delivered during minutes 21 or 29 or during the 5 seconds immediately preceding these minutes.

Percent freezing during the 5 scoring intervals on the 4 no-shock days (days 1, 3, 5, and 7) is shown in Figure 41. Freezing generally increased across scoring intervals and across sessions. Freezing on shock days is not shown since my ultimate purpose was to examine the effects of ethanol consumption on behaviour on days when shock was not administered. A 3 x 4 x 5 group by no-shock day by scoring interval mixed ANOVA revealed no significant effects or interactions involving group. The number of fecal boli deposited in the shock boxes on no-shock days is shown in Figure 42. A 3 x 4 group by no-shock day mixed ANOVA showed a significant group by no-shock day interaction, $F(6.90) = 2.810$, $p < .05$. The simple main effect of group was significant only during the second no-shock day, $F(2,30) = 10.249$. The 1-shock group produced significantly fewer boli than the average of the 5- and 15-shock groups, $F(1,30) = 19.957$. The 5- and 15-shock groups did not differ from each other. The simple main effect of no-shock day was significant for all three groups. All of the groups showed significant fear as indicated by

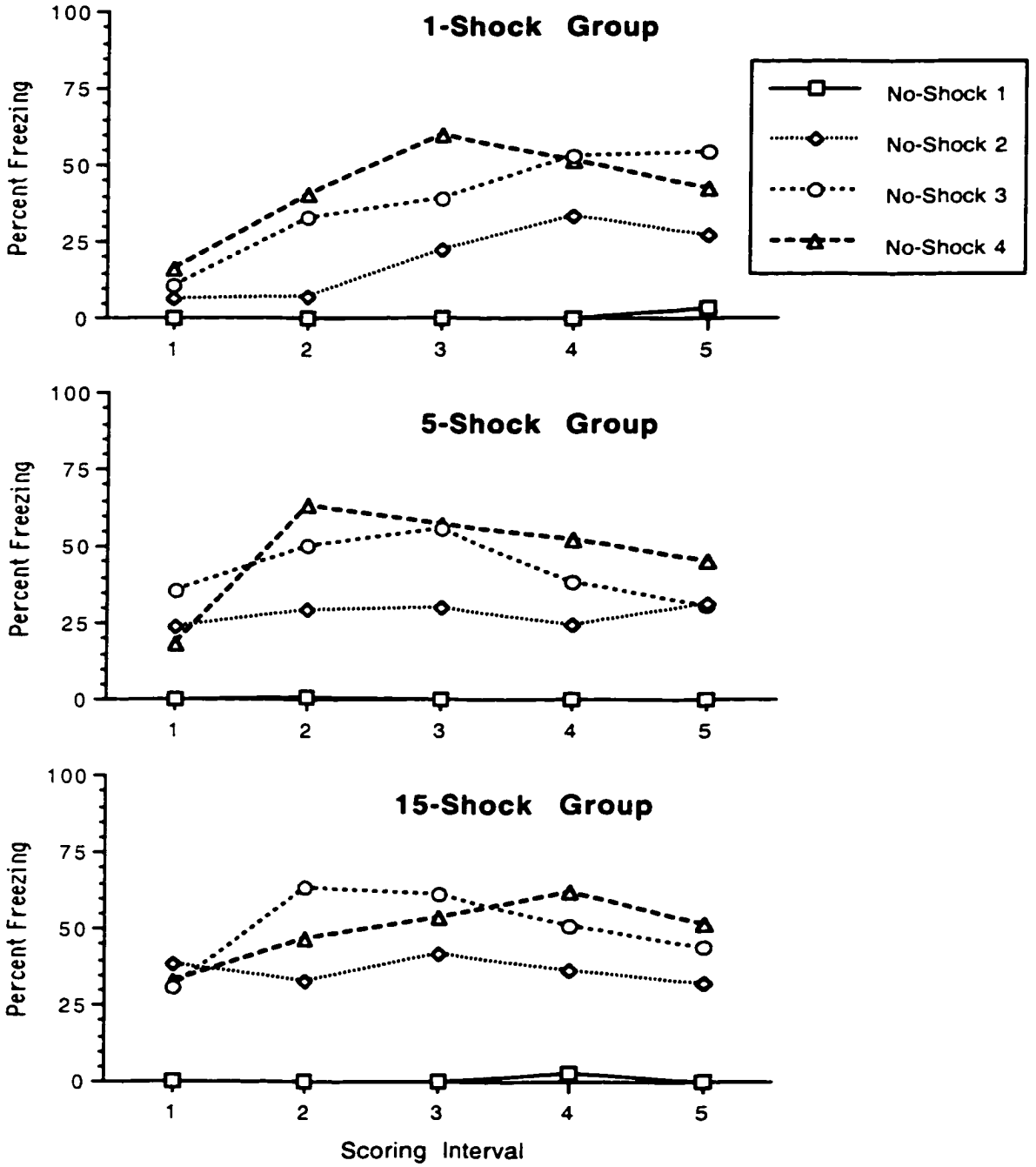

Figure 41: Mean percent freezing during the five scoring intervals of each no-shock day by the 1-Shock, 5-Shock, and 15-Shock groups during the pilot experiment of Experiment 7.

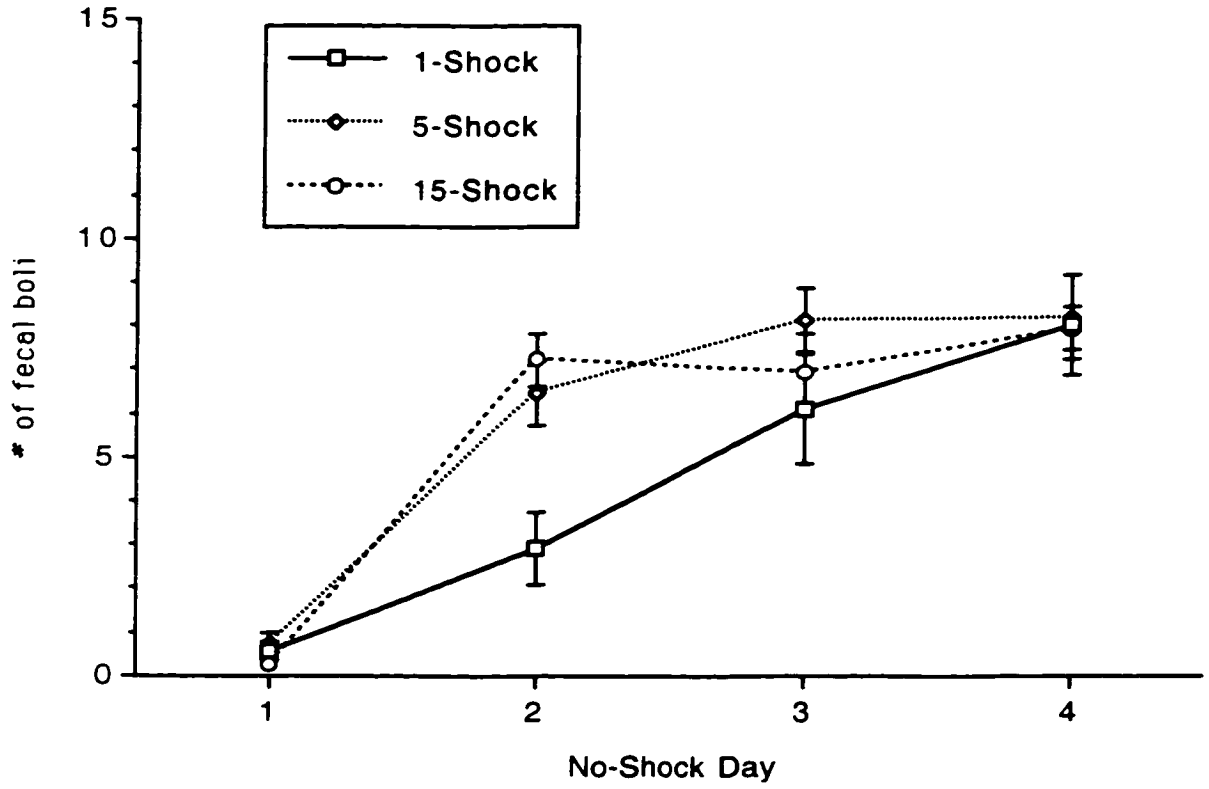

Figure 42: Mean (± SEM) number of fecal boli deposited in the shock boxes on no-shock days by the 1-Shock, 5-Shock, and 15-Shock groups during the pilot experiment of Experiment 7.

non-zero values for freezing and bolus production. Since there were no consistent behavioural differences between groups in either measure, the 1-shock schedule was selected for the subsequent experiments. There was no indication that there would be any gain associated with using more shocks. Consumption of water in the shock boxes was negligible (group means between 0.901 and 3.540 ml/kg) for all three groups on all of the no-shock days. In order to increase the level of consumption in the shock boxes, rats in the main experiment were approximately 23 hours water deprived at the beginning of their first no-shock session. I reasoned that if they were thirsty on their first exposure to the shock boxes that they would have more incentive to drink. Once they had drunk in the shock boxes they might have been expected to continue drinking there even when they were not so thirsty.

The main experiment was similar to the pilot experiment in that water was available during no-shock and shock sessions on alternate days. Instead of having three shock groups, this experiment included a 1-shock and a no-shock group. After the third shock day, shocks were discontinued and the extinction phase began. During the four days of extinction, instead of having water to drink in the shock boxes, the rats had access to one of four concentrations of ethanol or to one of four isocaloric sucrose solutions. Thus, the main experiment was a 2 x 2 x 4 factorial design with two levels of shock (1-

Shock or No Shock), two fluids (ethanol and sucrose), and four concentrations (highest, high, medium and low). The four concentrations of ethanol were 1.25, 2.5, 5, and 10% (v/v). The 5 and 10% ethanol solutions were the same as had been used in Experiments 1 and 2 and Experiments 3-6, respectively. The lower concentrations of ethanol should have allowed the rats to achieve an intoxicating dose of ethanol without achieving a debilitating dose since there was a greater volume difference between these doses at lower concentrations.

The six days of alternating no-shock and shock days should have allowed the acquisition of fear. Ethanol was introduced after shock was discontinued so that any effects it had on freezing or bolus production could not have been attributed to analgesia. The rats were not fluid deprived, so the role of thirst as a motivator of ethanol consumption was minimized. However, if thirst were motivating consumption, then the volume of consumption should have been relatively constant across concentrations of both solutions. If consumption were motivated by the caloric value of the solutions then consumption by ethanol and sucrose groups should have been equivalent for a given dose but the volume of consumption should have been inversely related to concentration. If ethanol consumption were motivated by tension reduction then consumption should have been higher by ethanol groups that had been shocked than by those that were not

shocked. The amount of absolute ethanol consumption (g/kg of absolute ethanol) should have been similar across concentrations. Behaviourally, if ethanol reduced tension then the ethanol groups should have extinguished their fear more quickly than animals that had not consumed ethanol. Once shocks were discontinued, the level of freezing and bolus production should have declined in all shock groups, but these reductions should have been faster for the ethanol groups.

This experiment was intended to run in four replications of 32 animals each, with 2 animals in each of the 16 cells per replication. However, examination of the data from the first 32 animals (presented below) indicated that consumption in the shock boxes was too low to warrant continuing the experiment as originally planned. The procedure was revised for the final three replications (see Experiment 8).

Method

Subjects

The subjects in this experiment were 32 male Sprague Dawley rats from Charles River Canada. At the start of the experiment, average body weight was 381.25 g, with a range between 260 and 422 g. Rats were individually housed in 18 x 24 x 17.5 cm Wahmann hanging cages under 16 h/8 h light/dark cycles. Half of the animals were

housed in a room with lights on at 5:00 a.m. and the remaining half were housed in a room with lights on at 8:00 a.m. All experimental sessions occurred during the light part of the cycle, beginning between 3.5 and 5.5 hours after lights on. The rats had ad lib access to Purina rat chow in their home cages throughout the experiment. Water was freely available in the home cage except during the 24 hours prior to the first and last days of the experiment.

Apparatus

Shock Boxes. Experimental sessions were conducted in the same four operant chambers that had been used in the previous experiments although they were removed from their sound attenuating shells. Levers were removed and drinking tubes inserted in their place. The drinking tube protruded into the box approximately 5 cm and ended approximately 7 cm above the floor. The boxes were located in two rooms, and within each room, two boxes were mounted side by side, 7 cm apart. During any particular session, the two rats in each pair of boxes were in the same shock condition. During sessions, the rooms were dimly lit and white noise masked any external sounds.

Recording Equipment. Segments of each session were videotaped using Panasonic WV-BP330 black and white cameras with Panasonic WV-LA9C3B automatic iris lenses connected to Panasonic AG-2500 videocassette recorders and SONY KV-

20M40 Trinitron televisions. An auditory signal, presented once every 5-s, was placed directly on the videotapes to facilitate scoring of the tape.

Delivery of shock, operation of videocassette recorders, and the pacing signal were controlled by an Apple IIe computer located in an adjoining room.

Fluids. Four ethanol and four sucrose solutions were used in this experiment, in addition to tap water. The highest concentrations used were a 10% (v/v) ethanol solution made with 0.1% (w/v) saccharin and a calorically equivalent 14.02% (w/v) sucrose solution made with 1% (v/v) acetic acid (both as described in Experiment 3). The remaining solutions (high, medium, and low concentrations) were dilutions of this first pair. The high, medium, and low concentration ethanol solutions were 5% (v/v) ethanol with 0.05% (w/v) saccharin (as described in Experiment 1), 2.5% (v/v) ethanol with 0.025% (w/v) saccharin, and 1.25% (v/v) ethanol with 0.0125% (w/v) saccharin. All of the ethanol solutions were made from 95% ethanol and tap water. The high, medium, and low concentration sucrose solutions were 7.01% (w/v) sucrose with 0.5% (v/v) acetic acid (as described in Experiment 1), 3.505% (w/v) sucrose with 0.25% (v/v) acetic acid, and 1.7525% (w/v) sucrose with 0.125% (v/v) acetic acid. Solutions were always mixed the night before they were needed so that they would be at room temperature when they were presented to the animals.

Procedure

Between the arrival of the rats in the laboratory and the start of the experiment, the rats were handled two or three times per week. Animals were randomly assigned to groups (1-Shock or No Shocks), and to squads of four. Within each squad, there were two rats in each of the 1-Shock and No Shock groups. Each squad consisted of one 1-Shock ethanol rat, one No Shock ethanol rat, one 1-Shock sucrose rat, and one No Shock sucrose rat. Within a squad all of the rats had access to the same fluid concentration (highest, high, medium, or low).

Each daily experimental session began with rats being removed from their home cages, weighed, and placed in a carrier with their squad mates for transport to the operant chambers. The animals were placed in the operant chambers after the drinking tubes had been inserted. At the end of the 30-min session, rats were again placed in the carrier and returned to their home cages.

For all groups, there were 10 daily sessions in the shock boxes. For rats in the 1-Shock groups, the first 6 sessions were alternating no-shock and shock sessions with water available. The single shock (0.8 mA, 2-s) was delivered at 23:17, 26:08, and 16:07 minutes into the first, second and third shock session, respectively. For rats in the No Shock groups, the first 6 sessions were identical to each other. During the last four

sessions water was replaced by the ethanol and sucrose solutions in the shock boxes (although water continued to be available in the home cages). No shocks were delivered during these extinction sessions. As in the pilot experiment, segments of each daily session (minutes 2, 7, 15, 21, and 29) were videotaped for subsequent scoring. While the rats were in the shock boxes, their home cage water bottles were refilled and/or replaced as required.

Water bottles were removed from the home cages approximately 24 hours prior to the first daily session in order to increase the likelihood that the rats would drink in the shock boxes. After this, rats had ad lib access to water in their home cages until after their ninth session in the shock boxes. The rats were water deprived between the ninth and tenth sessions to see if this would increase their fluid consumption in the shock boxes.

Measures. Rats were weighed prior to their daily sessions in the shock box. Fluid consumption in the shock boxes during no-shock and extinction sessions was recorded in ml and in ml/kg. Water consumption in the home cage during the 23.5 hours between sessions was also measured in ml and in ml/kg. The number of fecal boli deposited in the shock boxes on no-shock and extinction days was recorded for each rat. Data for shock days were not reported.

The videotapes were scored using a time sampling procedure. Each time the auditory signal was presented (once every 6 s) the behaviour of each rat was scored as freezing, not freezing, or obstructed view (if the rat had its back to the camera and its head was not visible). Freezing was defined as the absence of visible movement of the body and vibrissae, except for movement necessitated by respiration (Maes, Fidler & LoLordo, 1996). Videotapes were mainly scored by one of two observers, one who was aware and one who was not aware of the group membership of individual rats. In order to check reliability, both observers scored some of the tapes for one day. On this day, observers agreed on 89.78% of a total of 900 samples.

Results

Since there were only 2 animals per group in this experiment, it was not possible to analyse all of the possible factors. However, some examination of the data was possible. Several features stood out. First of all, the rats gained weight over the three no-shock days of the experiment and continued to gain weight over the first three extinction days. The rats lost weight between the third and fourth extinction sessions (when home cage water bottles were removed). Consumption in the shock boxes was very low except during the first no-shock day and the fourth extinction day when the animals were fluid deprived. Figure 43 shows consumption in the shock boxes on no-shock and extinction

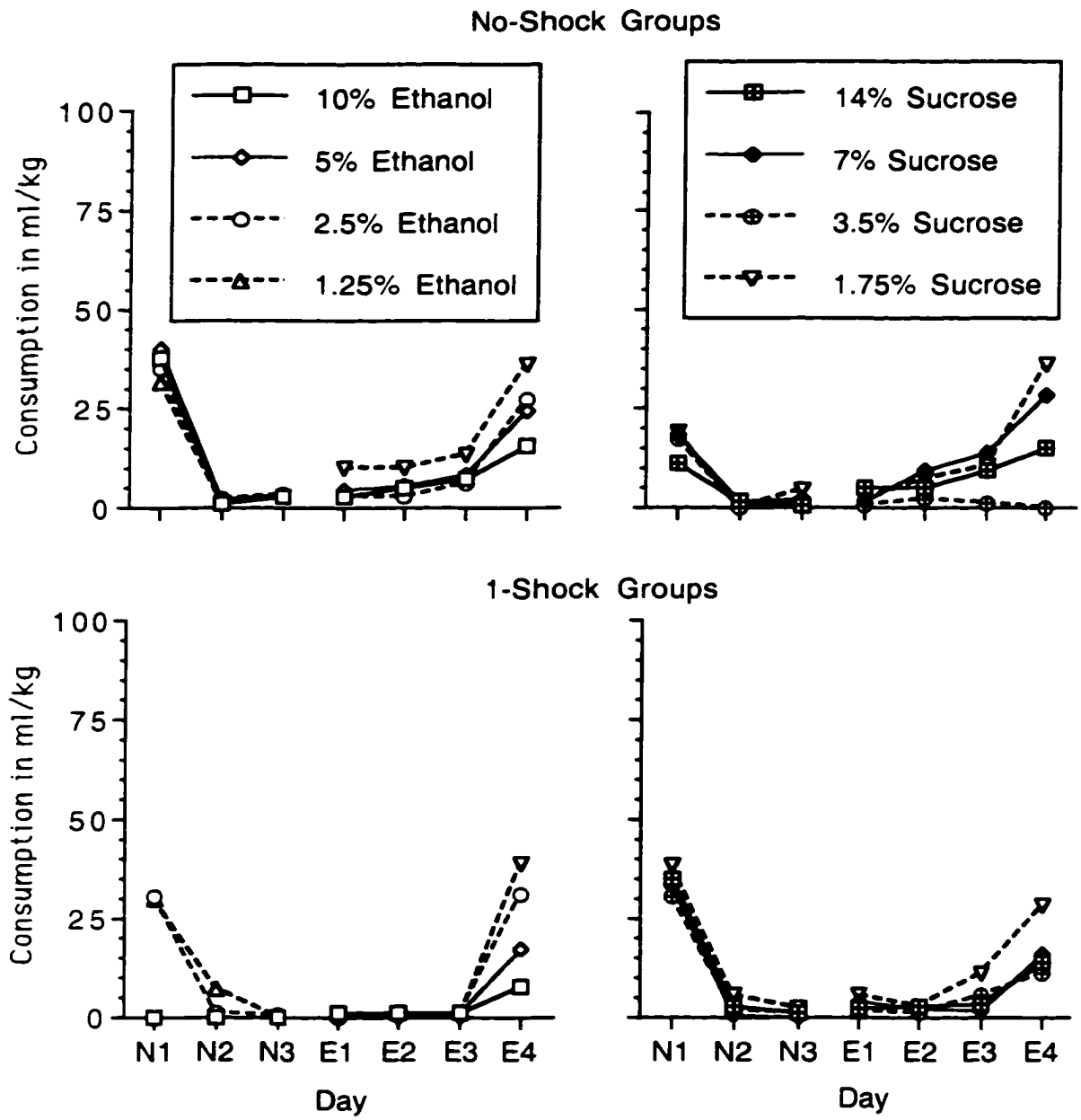

Figure 43: Mean consumption in ml/kg during sessions on no-shock days and extinction days by the No-Shock (upper panels) and 1-Shock (lower panels) groups that had access to one of four ethanol (left side) or sucrose (right-side) solutions during extinction in Experiment 7. All groups drank water on no-shock days.

days by the No Shock groups in the upper panels and by the 1-Shock groups in the lower panels. The left and right panels show consumption by groups that had ethanol and sucrose, respectively, in the shock boxes during extinction. On no-shock days, consumption did not differ between No Shock and 1-Shock groups. However, on extinction days rats in the No Shock condition (upper panels of Figure 43) consumed more fluid than rats in the 1-Shock condition (lower panels of Figure 43), $F(1,23) = 6.550$, as revealed by a 2 x 4 group by extinction day mixed ANOVA. Values that were missing due to leaky bottles were not replaced.

The percent freezing during no-shock and extinction sessions for the five scoring intervals combined is shown in Figure 44. A 2 x 3 group by day mixed ANOVA revealed that the 1-Shock groups froze more than the No Shock groups during no-shock days, $F(1,30) = 18.362$. There was also a significant effect of day, $F(2,60) = 35.678$, and a significant interaction between group and day, $F(2,60) = 10.394$. The simple main effect of day was significant for both the No Shock and 1-Shock groups, $F(2,30) = 9.561$ and 26.622. The simple main effect of group was significant only during the third no-shock day, $F(1,30) = 14.916$. It was not until the third no-shock day that the 1-Shock group froze more than the No Shock group. Similar analysis for extinction also showed significant effects of group, $F(1,30) = 34.204$, and day, $F(2,60) = 10.432$, as well as a

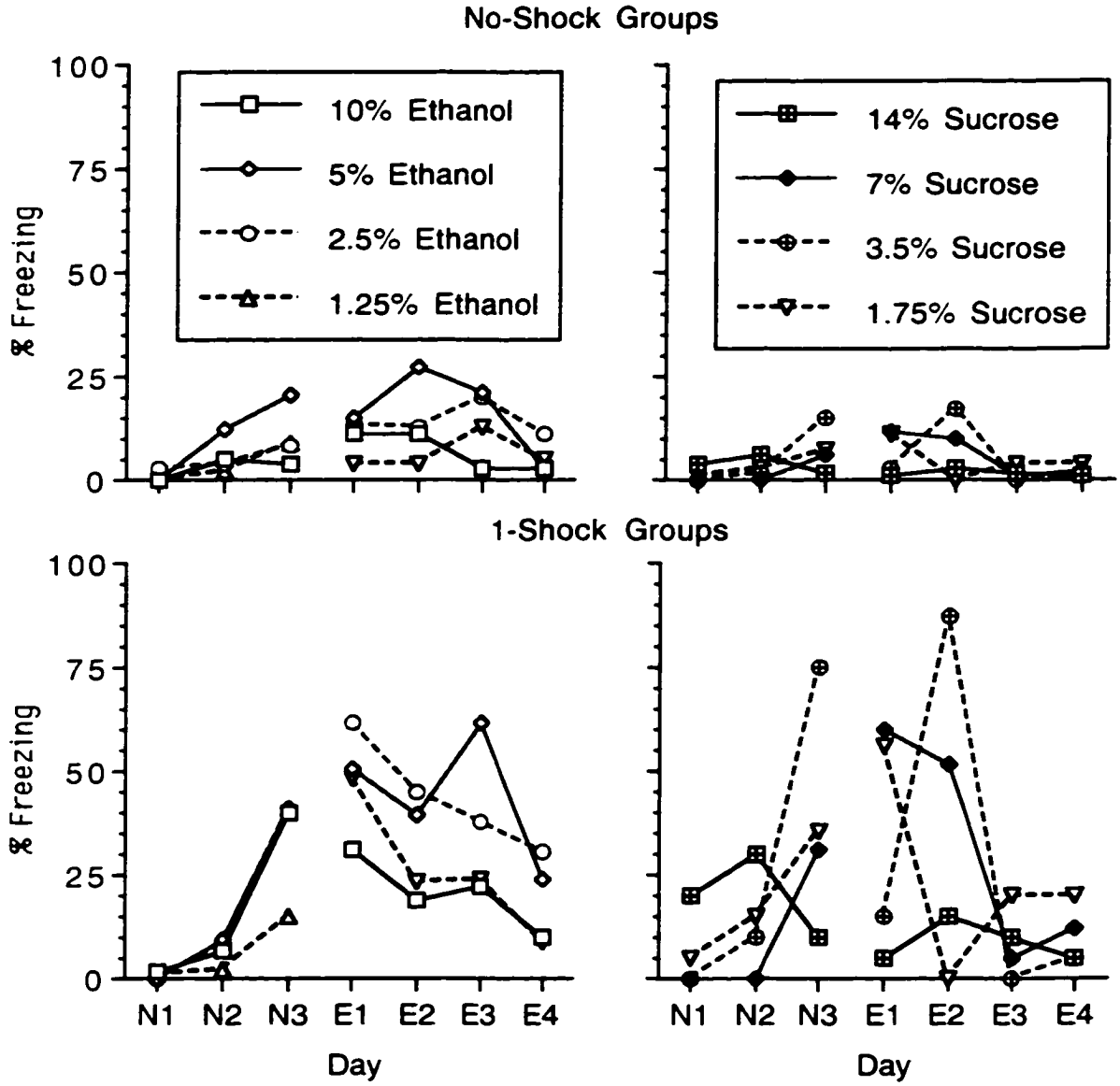

Figure 44: Mean % freezing during no-shock and extinction sessions by the No-Shock (upper) and 1-Shock (lower) groups that drank ethanol (left side) or sucrose (right-side) solutions during extinction in Experiment 7. All groups drank water on no-shock days.

significant interaction between them, F(2,60) = 4.277. The 1-Shock group froze more of the time than did the No Shock group and freezing generally decreased across extinction days. The simple main effect of extinction day was significant for both the No Shock and 1-Shock groups, F(3,45) =3.238 and 8.451. The simple main effect of group was also significant for each extinction day. The 1-Shock group always froze more than the No Shock group.

The number of fecal boli deposited in the shock boxes during sessions on no-shock and extinction days is shown in Figure 45. A 2 x 3 shock condition by day mixed ANOVA on the no shock days showed significant effects of shock condition, F(1,30) = 33.231, day, F(2,60) = 26.144, and a significant interaction between the two, F(2,60) = 26.126. The simple main effect of day was significant only for the 1-Shock groups, F(2,30) = 30.312. Bolus production did not differ between no-shock days 1 and 2. Defecation was significantly higher than the average of the first two no-shock days on the third no-shock day, F(1,30) = 60.235. The simple main effect of group was significant for the second and third no-shock days, F(1,30) = 4.667 and 46.394. Rats in the 1-Shock groups defecated more than rats in the No Shock groups. Similar analysis for the extinction days also revealed significant effects of shock condition, F(1,30) = 59.500, and day, F(3,90) = 24.088, and a significant interaction between the two, F(3,90) = 23.157.

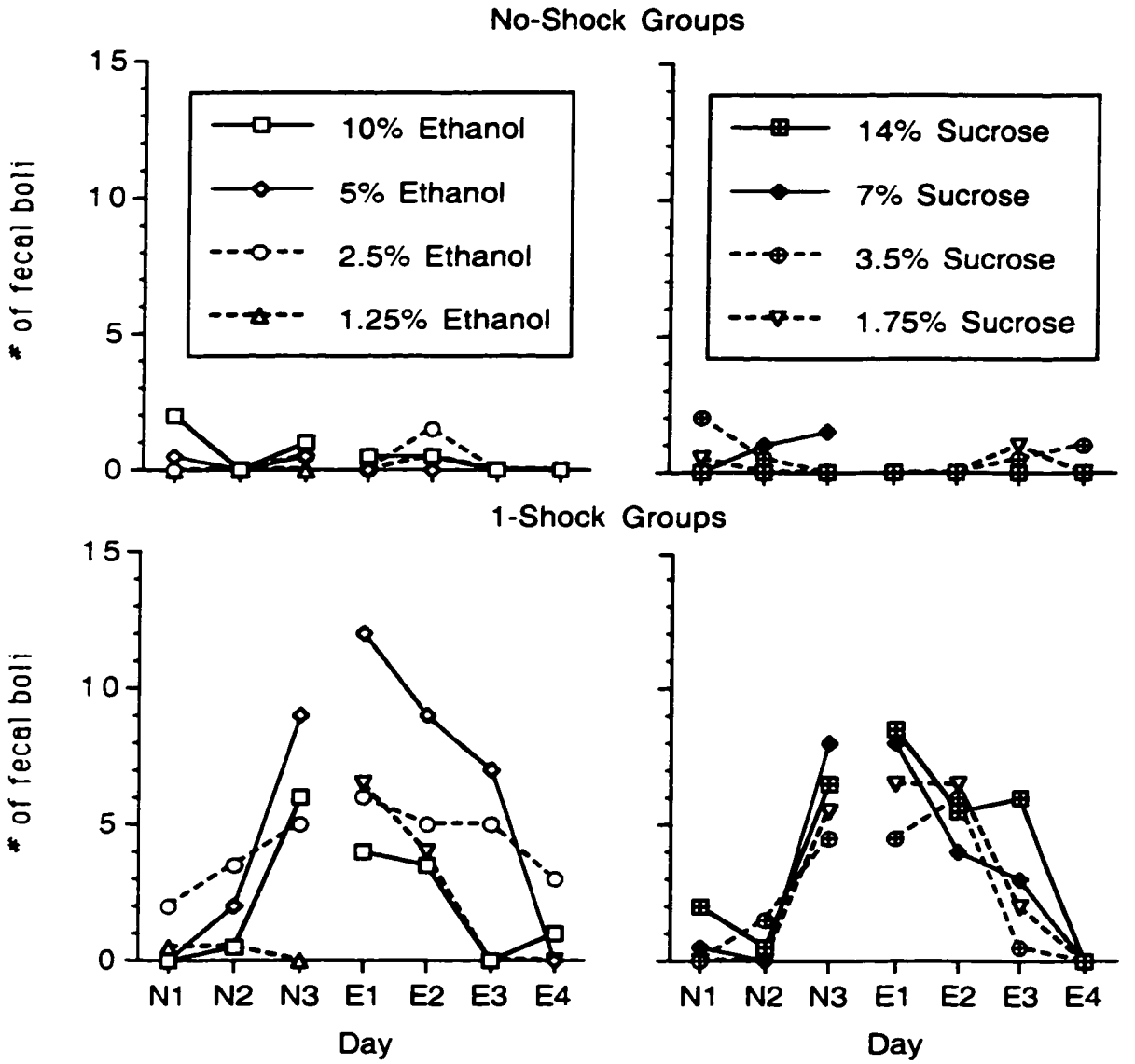

Figure 45: Mean number of fecal boli deposited in the shock boxes during no-shock and extinction sessions by the No-Shock (upper) and 1-Shock (lower) groups that drank ethanol (left side) or sucrose (right-side) solutions during extinction in Experiment 7. All groups drank water on no-shock days.

The simple main effect of day was significant only for the 1-Shock groups, $F(3,45) = 24.715$. Bolus production was lower on the fourth than on the first extinction day, $F(1,45) = 64.263$. The simple main effect of group was significant for the first three days of extinction, $F(1,30) = 61.79$, 43.967, and 12.485. On each of these days, the 1-Shock groups defecated more than the No Shock groups.

Discussion

Even though there were not sufficient animals in each fluid and concentration condition to address questions about the effects of ethanol on conditioned fear, this experiment did produce some interesting results. First of all, there was converging evidence from the freezing and defecation measures that the 1-Shock groups were more fearful than the No Shock groups. The 1-Shock groups froze more and defecated more than the No Shock groups. Even more encouraging, these differences emerged over time. There was very little freezing or defecation by any groups on the first no-shock day but by the third day 1-Shock groups were different from the No Shock groups. There was also evidence for extinction. Both freezing and defecation decreased across extinction days for the 1-Shock groups. These findings suggested that if the animals could be induced to consume larger volumes of fluid in the shock boxes then it would be possible to assess whether ethanol had any effects on conditioned fear.

EXPERIMENT 8

Experiment 7 was designed to address both tenets of the tension reduction hypothesis: (1) that ethanol reduces tension, and (2) that rats learn to drink ethanol for its tension reducing effects. In that experiment the animals had ad lib access to water in their home cages in order to minimize the role of thirst as a factor motivating consumption in the shock boxes. Unfortunately, consumption in the shock boxes (by the non-thirsty rats) was insufficient to produce any effects. In order to increase consumption in the shock boxes (at the cost of re-introducing thirst as a factor motivating consumption) access to water was restricted in the current experiment. The rats had access to fluid in the shock boxes and also had access to water in their home cages for one hour, one hour after their sessions. By restricting access to water in this way, consumption in the shock boxes should have increased enough so that the first tenet of the tension reduction hypothesis could be evaluated. Using the 1-shock schedule from Experiment 7, conditioned fear (as demonstrated by increased freezing and bolus production) was established prior to the introduction of ethanol and sucrose solutions. Thus this experiment investigated the effect of ethanol on an established fear response.

On the days when shock was presented, the single shock was always delivered during the second half of the session. Thus it was likely that the thirsty rats would drink

early in the session. This pattern of drinking was essential to ensure that ethanol, when it was introduced, could have its effect before the end of the session. Delivering shocks late in the session should have increased freezing late in the session, thereby allowing for the observation of fear-reducing effects of ethanol. Behaviour of Shocked groups was compared to that of No Shock groups.

During extinction, half of the previously shocked and half of the previously unshocked groups had access to ethanol in the shock boxes. The remaining half of the groups had access to isocaloric sucrose solutions. These four groups were repeated for the three concentrations of ethanol. If ethanol does reduce tension, then we would have expected to see a bigger (and faster) reduction in freezing behaviour and bolus production for the Ethanol-Shock groups than for the Sucrose-Shock groups. If thirst and hunger were the factors that motivated consumption then we would not have expected to see any differences in consumption between the Ethanol and Sucrose groups (or between the Shock and No Shock groups). If thirst were the primary motivator, we would have expected the volume of consumption to stay relatively constant across shock condition, fluid, and concentrations. If its post-ingestive effects motivated ethanol consumption, then the rats should have regulated their level of absolute ethanol consumption regardless of its concentration. Further, if ethanol were being used for tension reduction then

consumption by the Ethanol-Shock group should have been greater than consumption by the Ethanol-No Shock group, at least until their respective levels of fear were the same.

Method

Subjects

The subjects in this experiment were 96 experimentally naïve adult male Sprague Dawley rats run in three replications, with 32 rats per replication. Rats were individually housed as in all of the previous experiments. All of the rats were housed under a 16-h/8-h light/dark cycle. Half of the rats were housed in a room with lights on at 5:00 a.m. and the rest in a room with lights on at 8:00 am. All experimental sessions began between 3.5 and 5.5 h of lights on. Purina rat chow was freely available in the home cages. Fluid access was as described in the Procedure section.

Apparatus

Shock boxes and video recording equipment were all as described in Experiment 7. Three concentrations of ethanol and sucrose were used in this experiment and these concentrations corresponded to the high, medium, and low concentrations that were used in Experiment 7.

Procedure

Water bottles were removed from the rats' home cages approximately 24 hours before the first experimental sessions. From then on, daily fluid access was restricted to the 30-min experimental session and 1 h of supplementary water in the home cage beginning one hour after the experimental session. Home cage water bottles were returned following the hour of supplementary water on day 10 (the fourth extinction day), so the animals were non-deprived for the fifth extinction day.

Like Experiment 7, this experiment consisted of daily 30-min sessions in the shock boxes. Five minutes of each session (minutes 2, 7, 15, 21, and 29) were videotaped for subsequent scoring. The first six days alternated between no-shock and shock sessions and the final five days were extinction sessions. A single footshock (0.8 mA, 2-s) was delivered during each of the shock sessions. Shocks were delivered at 23:17, 26:08, and 16:07 minutes into the first, second and third shock sessions, respectively. Only half of the rats, the 1-Shock groups, were shocked. The remaining rats, the No Shock groups, received no shocks. For these rats the first 6 sessions were identical. Water was available to all of the rats during the first six shock box sessions.

During extinction half of the rats from the 1-Shock group and half of the rats from the No Shock groups had access to ethanol during their experimental sessions. The

remaining rats had access to sucrose during shock box sessions on extinction days. These four groups (Ethanol-Shock, Ethanol-No Shock, Sucrose-Shock, and Sucrose-No Shock) were further subdivided so that one third of the rats in each group (n = 8) had access to each of the high, medium, and low concentration of the solution. Within each of the three replications of the experiment there were 2 or 3 rats from each of the final 12 groups.

Measures. Rats were weighed daily prior to their sessions in the shock boxes. Fluid consumption during shock box sessions and during supplementary water sessions in the home cage was measured in ml and in ml/kg. Consumption of absolute ethanol in g/kg was also calculated. The number of fecal boli deposited in the shock boxes was recorded for no-shock, shock, and extinction sessions. Freezing was scored from the videotapes as described in Experiment 7. The number of incidences of freezing per scoring interval was converted to % freezing. Data for shock days were not reported. A scorer who was unaware of individual animals' group membership scored 607 of the 1056 individual sessions. Of these 607 sessions, 212 were animals in No Shock groups and 395 were animals in Shock groups.

Separate analyses were performed on the data for the no-shock days, the first four extinction days, and the fifth extinction day (the only day when rats were not water deprived). Three between-subject factors were included in all analyses: shock condition

(No Shock and 1-Shock), fluid (ethanol and sucrose), and concentration (high, medium, and low). Within subjects factors included day (1-3 or 1-4 for no-shock or extinction analyses) and scoring interval (for the freezing data).

In order to assess the magnitude and direction of change from no-shock days during extinction, a difference score was calculated for each extinction day (no-shock 3 minus extinction) for each measure. The resulting difference scores were then analysed as described above. Since extinction values were subtracted from no-shock values, positive difference scores represented values that were lower during extinction while negative difference scores indicated values that increased during extinction. Larger absolute values represented a greater magnitude of change and zero values represented no change from the third no-shock day.

Results

Mean body weight for the 6 No Shock and 6 Shock groups prior to shock box sessions on no-shock and extinction days is shown in Figure 46. A 2 x 2 x 3 x 3 shock condition by fluid by concentration by day mixed ANOVA for the no-shock days revealed a significant effect of day, $F(2,168) = 9.943$, and a significant interaction among all four factors, $F(4,168) = 2.650$, $p < .05$. Body weight on the second no-shock day was

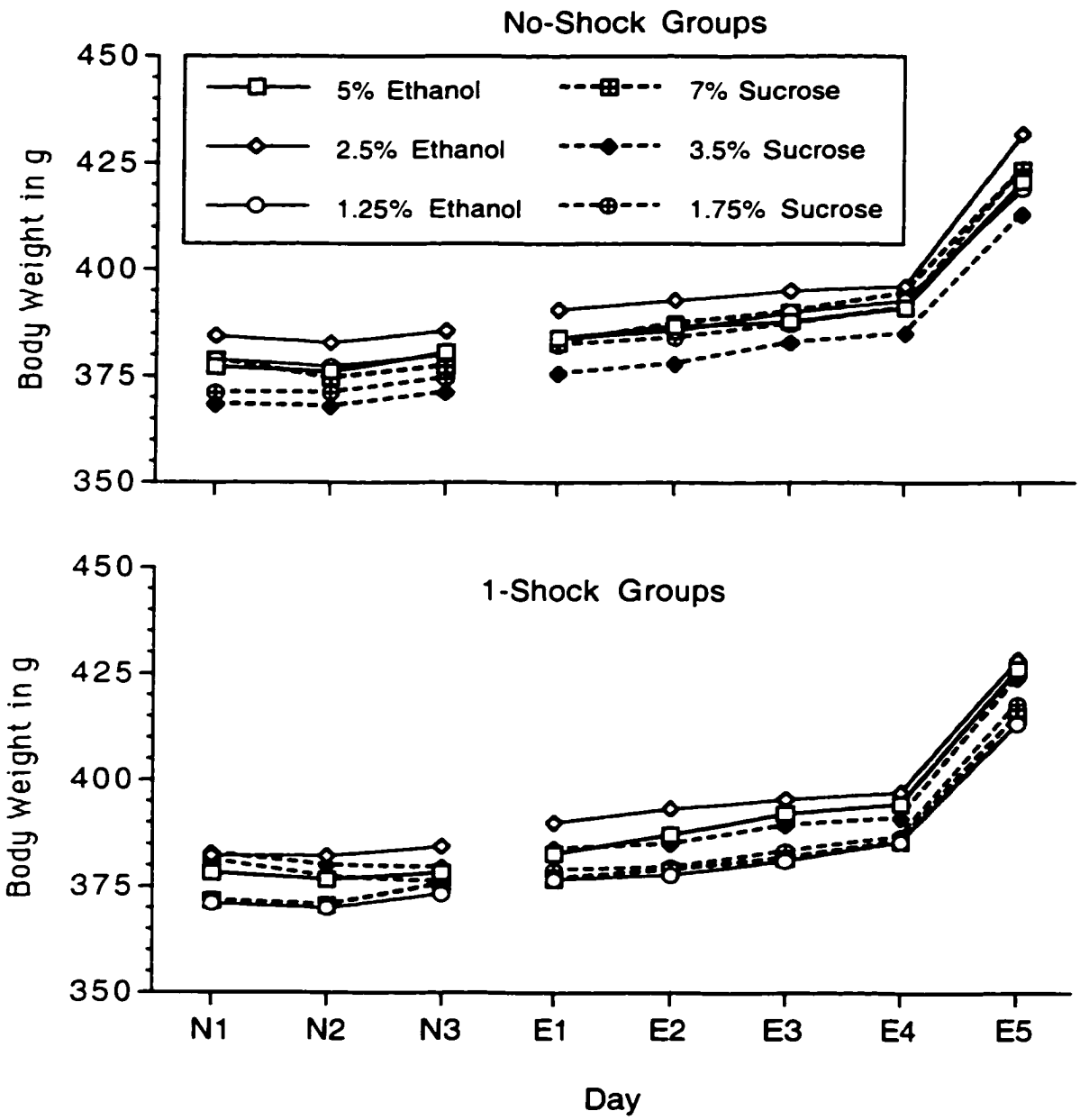

Figure 46: Mean body weight in grams of the six Shock (top) and six No Shock (bottom) groups (n = 8 per group) prior to the three no-shock (N) and five extinction (E) sessions in Experiment 8. Animals were water deprived except for the fifth extinction day.

lower than on the average of the first and third no-shock days, $F(1,168) = 17.755$. Body weights on the first and third no-shock days were not significantly different.

Analysis of the body weight data for the first four extinction sessions revealed a significant effect of day, $F(3,252) = 211.856$. Body weight increased across these four extinction sessions, with the result that body weight on the fourth extinction day was significantly greater than on the first extinction day, $F(1,252) = 551.661$. The interaction between concentration and day was also significant, $F(6,252) = 2.538$, $p < .05$. Simple effect analysis revealed simple main effects of day for each of the high, medium, and low concentrations, $F(3,84) = 72.416, 62.737, 81.218$. For each concentration, means comparisons showed that the rats weighed more on the fourth extinction day than on the first extinction day. The simple main effect of concentration was not significant on any of the first four extinction days. The rats were much heavier prior to the fifth extinction session (when they were not water deprived) but analyses revealed no significant effects or interactions.

Mean consumption in the shock boxes during no-shock and extinction sessions by the six No-Shock and six Shock groups is shown in Figure 47. All groups consumed water during sessions on the three no-shock days. Values were missing for 19 individual sessions (of a total of 1056) due to leaky bottles. These values were replaced by group

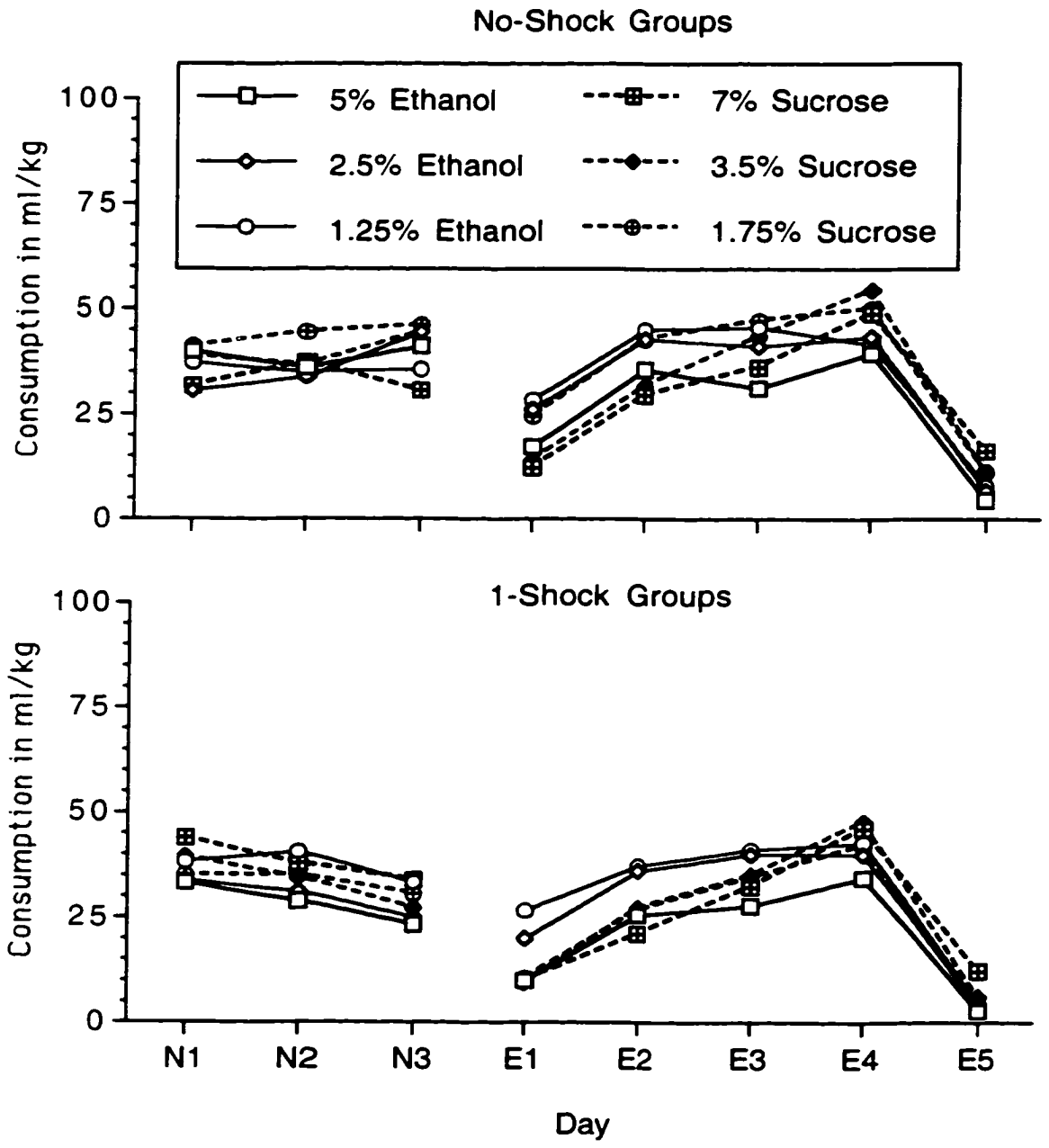

Figure 47: Mean consumption of water during no-shock (N) sessions and of ethanol or sucrose during extinction (E) sessions in the shock boxes by the six Shock (top) and six No Shock (bottom) groups (n = 8 per group) in Experiment 8. Animals were water deprived except for the fifth extinction day.

means. Analysis for the no-shock days revealed a significant effect of shock condition, $F(1,84) = 4.698$. Rats in the No Shock groups consumed more water on no-shock days than the rats in the Shock groups. There was also a significant interaction between shock condition and day. $F(2,168) = 6.529$. Simple effect analysis revealed a significant simple main effect of day for the Shock groups, $F(2,94) = 5.692$. For the Shock groups consumption during the third no-shock session was significantly lower than the average of the first two sessions, $F(1,94) = 10.178$. Consumption during the first two sessions did not differ. There was also a simple main effect of shock condition for the third no-shock day, $F(1,94) = 14.072$. Consumption was higher by the No Shock groups than by the Shock groups. Unfortunately, even though all of the groups were consuming water during the no-shock sessions there was a significant interaction among shock condition, fluid, and concentration, $F(2,84) = 4.079$, $p < .05$.

Over the course of extinction I expected to see the emergence of differences in consumption between Shock- and No Shock-Ethanol and Sucrose groups. Although there were significant effects of shock condition, $F(1,84) = 8.542$, concentration, $F(2,84) = 5.337$, and day, $F(3,252) = 236.769$, the main effect of fluid (ethanol versus sucrose) was not significant. The groups that had never been shocked consumed more fluid than the groups that had been shocked. Consumption by the high concentration groups was lower

than the average of the medium and low concentration groups, $F(1,84) = 9.942$. Consumption increased across the first four days of extinction. There were significant interactions between fluid and day, $F(3,252) = 25.080$, and between concentration and day, $F(6,252) = 2.946$, but not between shock condition and fluid.

On the fifth day of extinction, when the rats were no longer fluid deprived, consumption in the shock boxes was much lower. Analyses revealed a significant main effect of shock condition, $F(1,84) = 13.817$, and fluid, $F(1,84) = 17.405$, and a significant interaction between fluid and concentration, $F(2,84) = 6.170$, but not between shock condition and fluid or among shock condition, fluid, and concentration. Groups that had never been shocked consumed more fluid in the shock boxes on the fifth extinction day than did groups that had been shocked. Sucrose groups consumed more fluid than ethanol groups.

The change in shock box consumption from the third no-shock session for each extinction session (N3 - E1, N3 - E2, N3 - E3, N3 - E4, and N3 - E5) is shown in Figure 48. The Shock-Ethanol groups, in particular, were expected to increase their consumption over the course of extinction. Such an increase would have corresponded to decreasing difference scores. Over all 12 groups, difference scores did decrease significantly across the first four days of extinction. The effect of day was significant, $F(3,252) = 236.769$

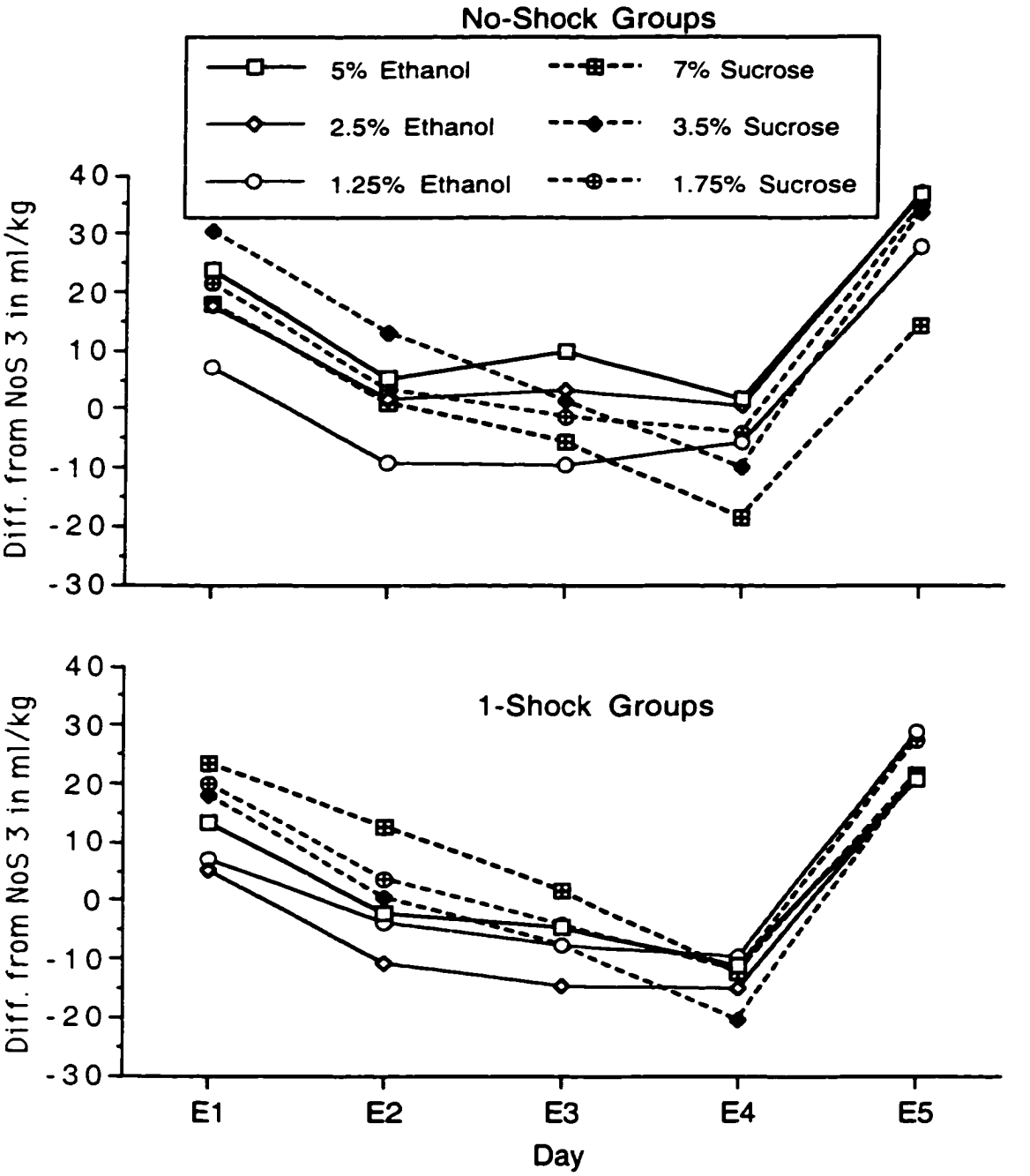

Figure 48: Mean difference in ml/kg between consumption during no-shock 3 and each extinction session (N-E) for the six No-Shock (top) and six Shock groups (bottom) in Experiment 8.

and means comparisons showed that difference scores for the fourth extinction session were significantly lower than for the first extinction session, $F(1,252) = 651.595$. Over the first four days of extinction there were significant interactions between fluid and day, $F(3,252) = 25.080$, and between concentration and day, $F(6,252) = 2.946$, but there were no significant interactions involving shock condition.

Examination of the difference scores for shock box consumption during the fifth extinction session revealed a significant effect of shock condition, $F(1,84) = 4.985$, $p < .05$. The mean difference score for the No Shock groups, 30.676, was significantly greater than that for the Shock groups, 23.644. That is, Shock groups showed less suppression (relative to no-shock 3) than the No Shock groups.

The consumption of absolute ethanol in g/kg by the 3 No Shock- and 3 Shock-Ethanol groups is shown in Figure 49. If the intake of absolute ethanol were being regulated then no concentration effects would have been expected. Over the first four days of extinction there were significant effects of shock condition, $F(1,42) = 5.997$, $p < .05$, ethanol concentration, $F(2,42) = 70.543$, and day, $F(3,126) = 75.175$. Groups that had never been shocked consumed more absolute ethanol than groups that had been shocked. The High concentration groups consumed more absolute ethanol than the average of the Medium and Low concentration groups, $F(1,42) = 110.401$. The Medium concentration

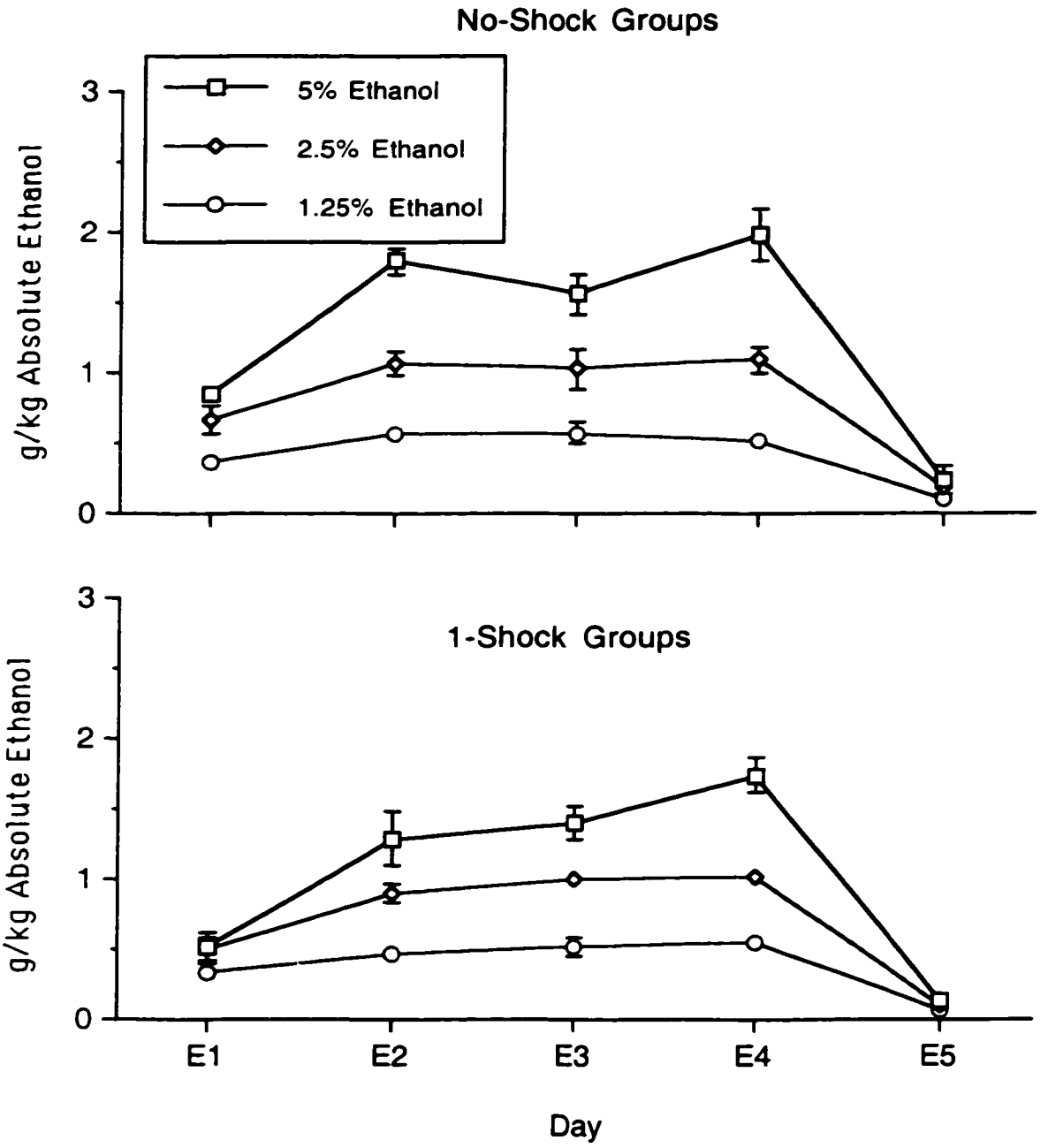

Figure 49: Mean (± SEM) consumption of absolute ethanol in g/kg during the five extinction sessions in the shock boxes by the High, Medium, and Low concentration No Shock and Shock Ethanol groups in Experiment 8.

groups consumed more absolute ethanol than the Low concentration groups, $F(1,42) = 30.686$. Consumption increased across the first four extinction sessions. The interaction between day and concentration was significant, $F(6,126) = 16.044$, but the interactions between day and shock condition and among day, shock condition and concentration were not. Examination of the absolute consumption of ethanol during the fifth extinction session revealed no significant effects or interactions.

Water consumption in the home cage during the daily supplementary water period of the no-shock and extinction days is shown in Figure 50. There was no supplementary water period on the fifth extinction day since the animals had free access to water on that day. A 2 x 2 x 3 x 3 shock condition by fluid by concentration by day mixed ANOVA for the no-shock days revealed a significant main effect of day, $F(2,168) = 16.186$. Consumption during the supplementary water period increased across no-shock days. The interaction between shock condition and day was also significant, $F(2,168) = 4.724$, $p < .05$. Simple effect analysis showed significant simple main effects of day for both the No Shock and the Shock groups, $F(2,84) = 3.568$ ($p < .05$) and 16.373. For the No Shock groups, consumption increased significantly from no-shock 1 to no-shock 2, $F(1,84) = 4.736$. For the Shock groups, average consumption during the supplementary water period on the first two no-shock days was lower than on the third day, $F(1,84) = 31.009$.

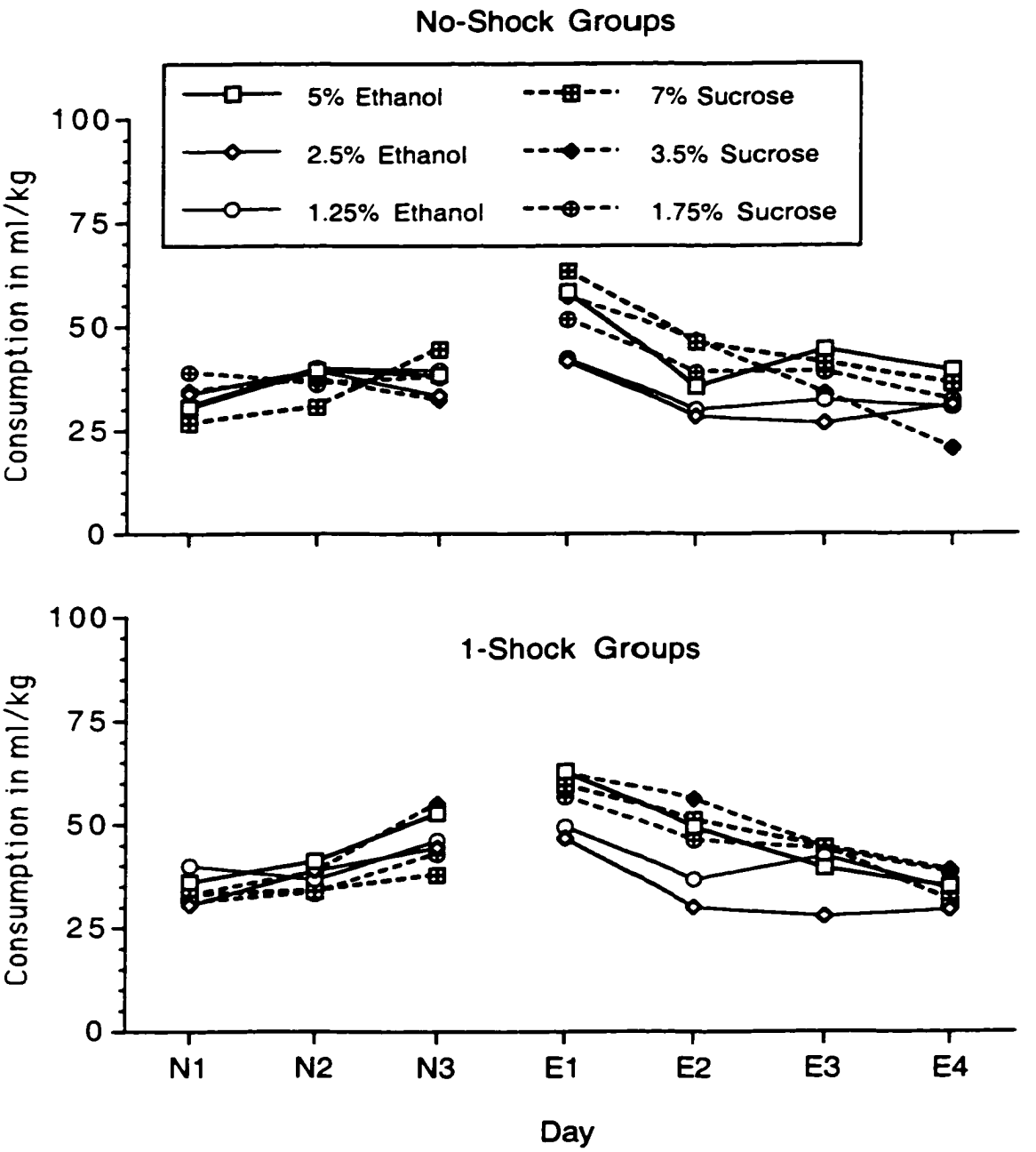

Figure 50: Mean consumption of water in ml/kg in the home cage during the no-shock (N) and extinction (E) sessions by the No-Shock and Shock groups in Experiment 8.

The simple main effect of shock condition was significant only for the supplementary water session following the third no-shock session, $F(1,84) = 10.387$. Consumption by the No Shock groups was lower than consumption by the Shock groups. Supplementary water consumption following extinction sessions 1-4 was analysed using a 2 x 2 x 3 x 4 shock condition by fluid by concentration by day mixed ANOVA. This analysis revealed significant main effects of shock condition, fluid, concentration, and day, $F(1,84) = 6.571$, 13.977, $F(2,84) = 6.532$, and $F(3,252) = 95.673$. The Shock groups consumed more supplementary water than the No Shock groups and the Sucrose groups consumed more supplementary water than the Ethanol groups. Means comparisons showed that consumption by the high concentration groups was significantly greater than the average of the medium and low concentration groups, $F(1,84) = 12.675$. The medium and low concentration groups did not differ from each other. Supplementary water consumption decreased across days (as consumption in the shock boxes increased). Analysis also revealed significant interactions between fluid and concentration, $F(2,84) = 3.305$, $p < .05$, between fluid and day, $F(3,252) = 8.787$, and between concentration and day, $F(6,252) = 2.768$.

The number of fecal boli deposited in the shock boxes by the six No Shock and the six 1-Shock groups during the three no-shock and five extinction sessions is shown in

Figure 51. To the extent that bolus production was an index of fear, over the three no-shock days a difference should have emerged between Shock and No Shock groups. Analysis confirmed that the Shock groups defecated more than the No Shock groups, $F(1,84) = 27.772$. There was also a significant effect of day, $F(2,168) = 19.811$. Defecation increased across sessions. The interaction between shock condition and day was significant, $F(2,168) = 19.271$. Simple effect analysis showed a significant simple main effect of day only for the Shock groups, $F(1,168) = 26.332$. These groups defecated more on the second no-shock day than on the first no-shock day, $F(1,168) = 12.585$, and more on the third day than on the average of the first two days, $F(1,168) = 40.078$. The simple main effect of shock condition was significant for both the second and third no-shock days, $F(1,84) = 16.041$ and 36.366. On each of these days, the Shock groups defecated more than the No Shock groups. In spite of the fact that all of the groups consumed only water during the no-shock sessions, there was also a significant effect of concentration, $F(2,84) = 4.749$, $p < .05$, and a significant interaction between shock condition and concentration, $F(2,84) = 5.231$.

Across extinction sessions bolus production by No Shock groups was not expected to change. Over the same period, bolus production by the Shock groups was expected to decrease. According to the tension reduction hypothesis, fear (as measured by

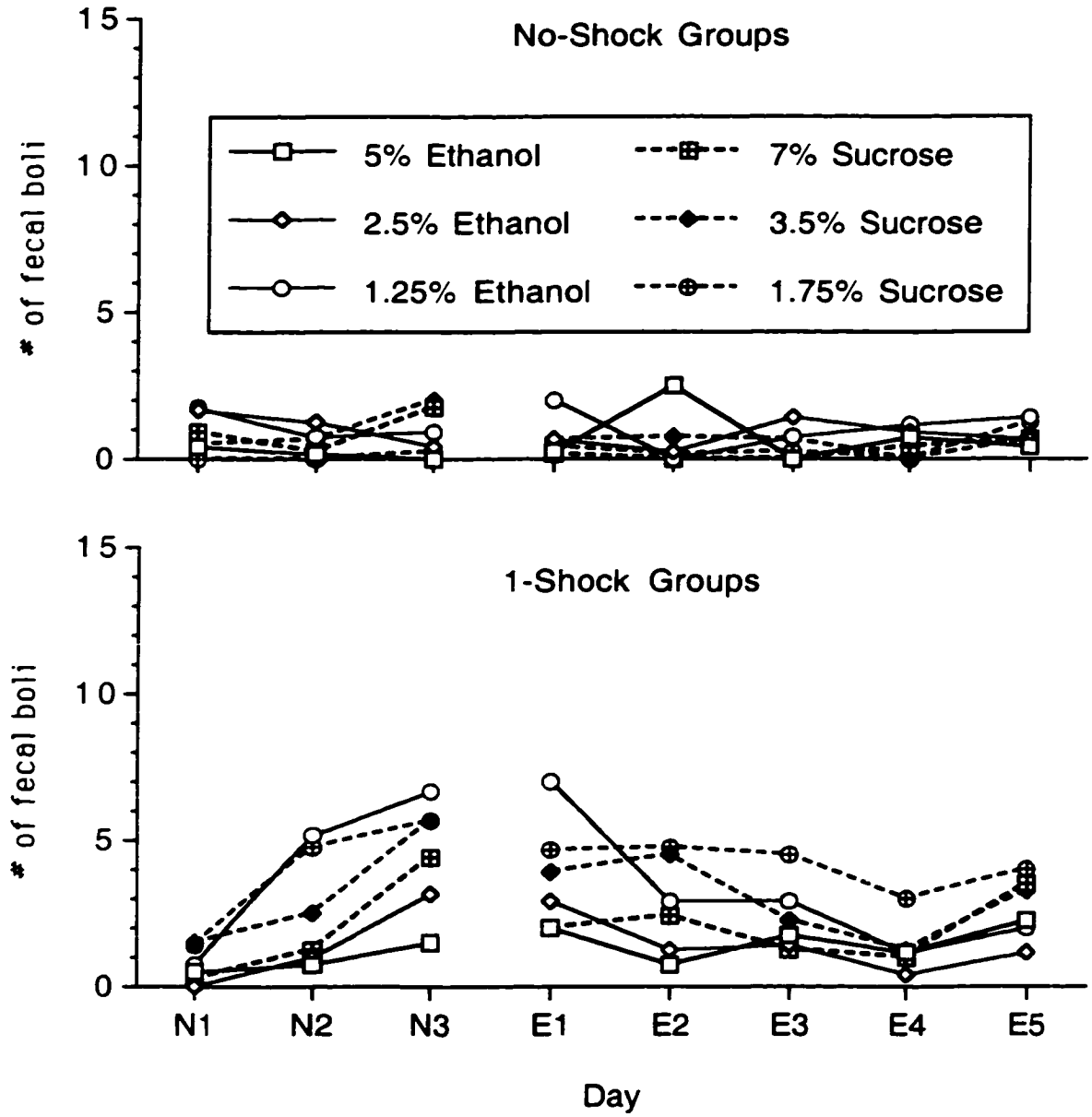

Figure 51: Mean number of fecal boli deposited in the shock boxes during no-shock (N) and extinction (E) sessions by the No Shock (top) and Shock (bottom) groups in Experiment 8.

bolus production) should have decreased more quickly and to a larger extent in the Shock-Ethanol groups than in the Shock-Sucrose groups. Of particular relevance to these predictions were the significant main effect of shock condition, $F(1,84) = 27.934$, and the significant interaction among shock condition, fluid, and day, $F(3,252) = 3.105$, $p < .05$. Shock groups defecated more than No Shock groups. The shock condition by day interaction was significant for both the Ethanol and the Sucrose groups, $F(3,126) = 5.853$ and 5.301. The simple main effect of day was significant for the Shock-Ethanol and Shock-Sucrose groups, $F(3,63) = 10.187$ and 7.470. Defecation by the Ethanol-Shock and Ethanol-No Shock groups differed only during the first extinction session, $F(1,42) = 15.958$. By comparison, the Sucrose-Shock groups defecated more than the Sucrose-No Shock groups during each extinction session, $F(1,42) = 19.737$, 25.503, 11.089, and 8.910.

Analysis of bolus production during the first four extinction sessions revealed additional main effects of concentration, $F(2,84) = 3.578$, and day, $F(3,252) = 10.706$. High concentration groups defecated less than the average of the medium and low concentration groups, $F(2,84) = 4.192$, $p < .05$, which did not differ from each other. Bolus production decreased across sessions. Interactions between shock condition and concentration, $F(2,84) = 3.294$, $p < .05$, between shock condition and day, $F(3,252) =$

8.238, between fluid and day, $F(3,252) = 2.806$, $p < .05$, between concentration and day, $F(6,252) = 2.720$, $p < .05$, and among fluid, concentration, and day, $F(6,252) = 2.673$, $p < .05$, were all significant.

On the fifth day of extinction, there were significant effects of shock condition, $F(1,84) = 18.517$, and fluid, $F(1,84) = 4.426$, $p < .05$. The No Shock groups defecated less than the Shock groups and the Ethanol groups defecated less than the Sucrose groups. There was also a significant interaction between shock condition and fluid, $F(1,84) = 4.033$, $p < .05$. Simple effect analysis revealed a significant main effect of shock condition only for the Sucrose group, $F(1,42) = 15.323$. Sucrose-Shock groups defecated more than Sucrose-No Shock groups. The simple main effect of fluid was significant only for the Shock groups, $F(1,42) = 5.221$, $p < .05$. Groups that consumed ethanol defecated less than groups that consumed sucrose.

In order to assess the change between bolus production during the third no-shock day and each day of extinction, difference scores were calculated for each extinction day (N3-E1, etc.). These difference scores are shown in Figure 52. Little change in bolus production from no-shock day 3 was expected for the No Shock groups. For the Shock groups, the difference scores should have increased across extinction sessions, especially for the Shock-Ethanol groups. As expected the difference scores were bigger for the

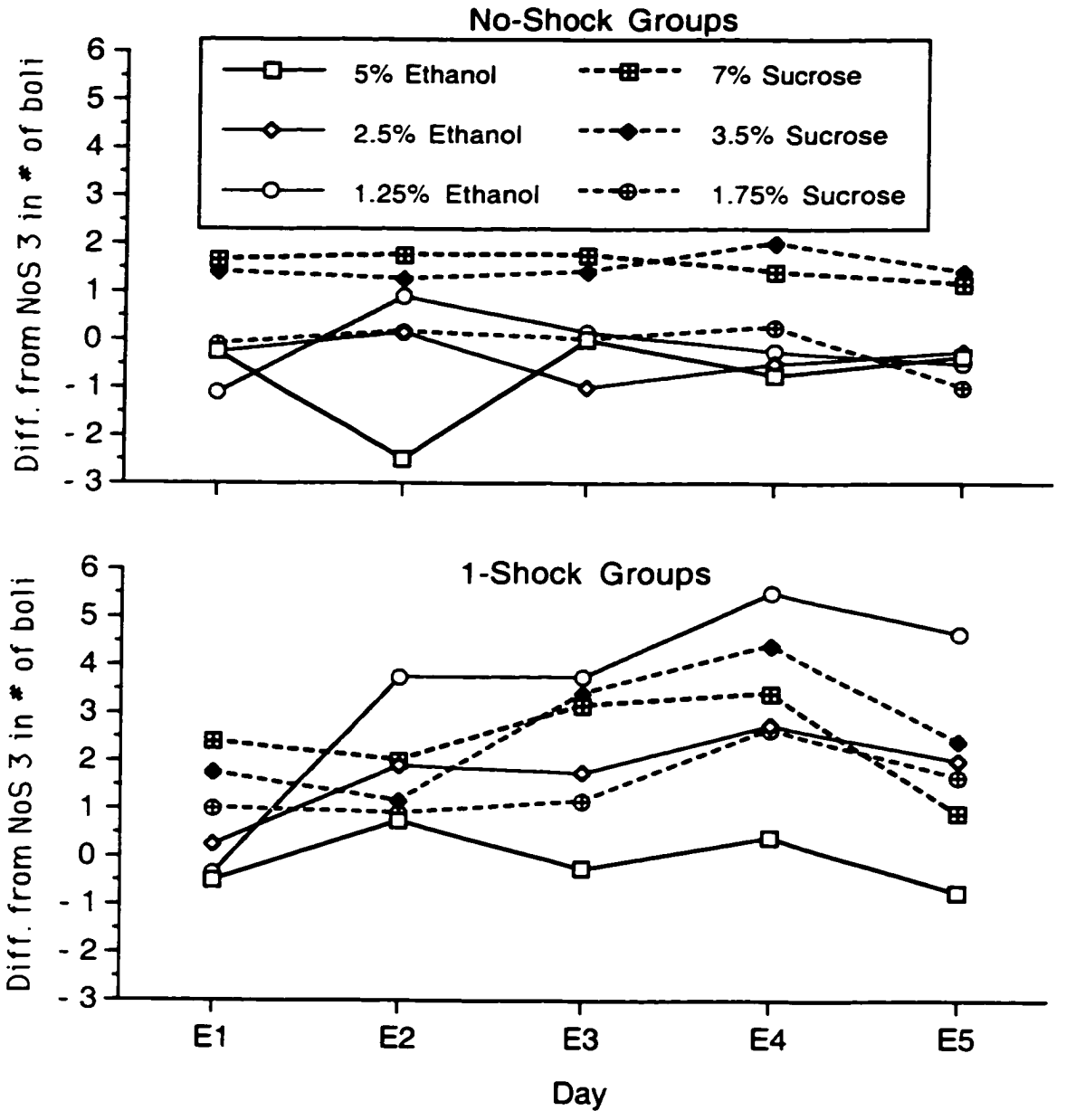

Figure 52: Mean difference in number of fecal boli between no-shock 3 and each extinction session for the No Shock (top) and Shock (bottom) groups in Experiment 8.

Shock groups than for the No Shock groups, $F(1,84) = 8.792$. There was also a significant effect of extinction day, $F(3,252) = 10.706$. The magnitude of difference scores increased over the four extinction days. There was a significant three-way interaction among day, shock condition, and fluid, $F(3,252) = 3.105$, $p < .05$. In accordance with my predictions, the day by fluid interaction was significant only for the Shock groups, $F(3,126) = 4.269$. However, difference scores increased across days for both Shock-Ethanol and Shock-Sucrose groups, $F(3,63) = 10.187$ and 7.470. A difference between No Shock-Ethanol and Shock-Ethanol groups emerged over days. The difference scores were bigger for the Shock-Ethanol groups than for the No Shock-Ethanol groups during the second, third, and fourth extinction sessions, $F(1,42) = 6.979$, 4.893, and 15.584. Differences between Shock- and No Shock-Sucrose groups emerged more slowly. The difference scores for the Shock-Sucrose groups were not significantly bigger than for the No Shock-Sucrose groups until the fourth extinction session, $F(1,42) = 6.238$. The interaction among shock condition, fluid, concentration and day was not significant. Significant interactions between fluid and concentration, $F(2,84) = 3.170$, $p < .05$, between day and shock condition $F(3,252) = 8.238$, between day and fluid, $F(3,252) = 2.806$, $p < .05$, between day and concentration, $F(6,252) = 2.720$, $p < .05$, and among fluid, concentration and

day, $F(6,252) = 2.673$, $p < .05$ were not directly relevant to predictions from the Tension Reduction Hypothesis.

Examination of the difference scores relating defecation during the fifth extinction session to defecation during the third no-shock session revealed a significant effect of shock condition, $F(1,84) = 7.787$. Defecation by the No Shock groups was unchanged from no shock 3 whereas the Shock groups decreased their defecation in the fifth extinction session. There were no significant effects or interactions involving fluid. The interaction between shock condition and concentration was significant, $F(2,84) = 3.813$, $p < .05$. The simple main effect of shock condition was significant for the Low concentration groups, $F(1,28) = 11.689$. The Low-Shock groups decreased their bolus production from no-shock 3 to extinction 5 and the Low-No Shock groups did not.

Percent freezing for each scoring interval during the three no-shock (left panels) and five extinction sessions (right panels) is shown in Figure 53(A-D). Figures 53A and 53B show the percent freezing by the three No Shock-Ethanol (53A) and the three No Shock-Sucrose groups (53B). Percent freezing for the Shock-Ethanol and Shock-Sucrose groups is shown in Figures 53C and 53D. In each case, freezing by the group consuming the High concentration is presented in the top panel with freezing by the group

Figure 53 (A-D): Mean percent freezing during each scoring interval for no-shock (left) and extinction (right) sessions during Experiment 8. A) Ethanol-No Shock groups; B) Sucrose-No Shock groups; C) Ethanol-Shock groups; D) Sucrose-Shock groups. In each case, the top, middle and bottom panels showed freezing by groups that consumed the High, Medium, and Low concentrations of ethanol or sucrose during extinction.

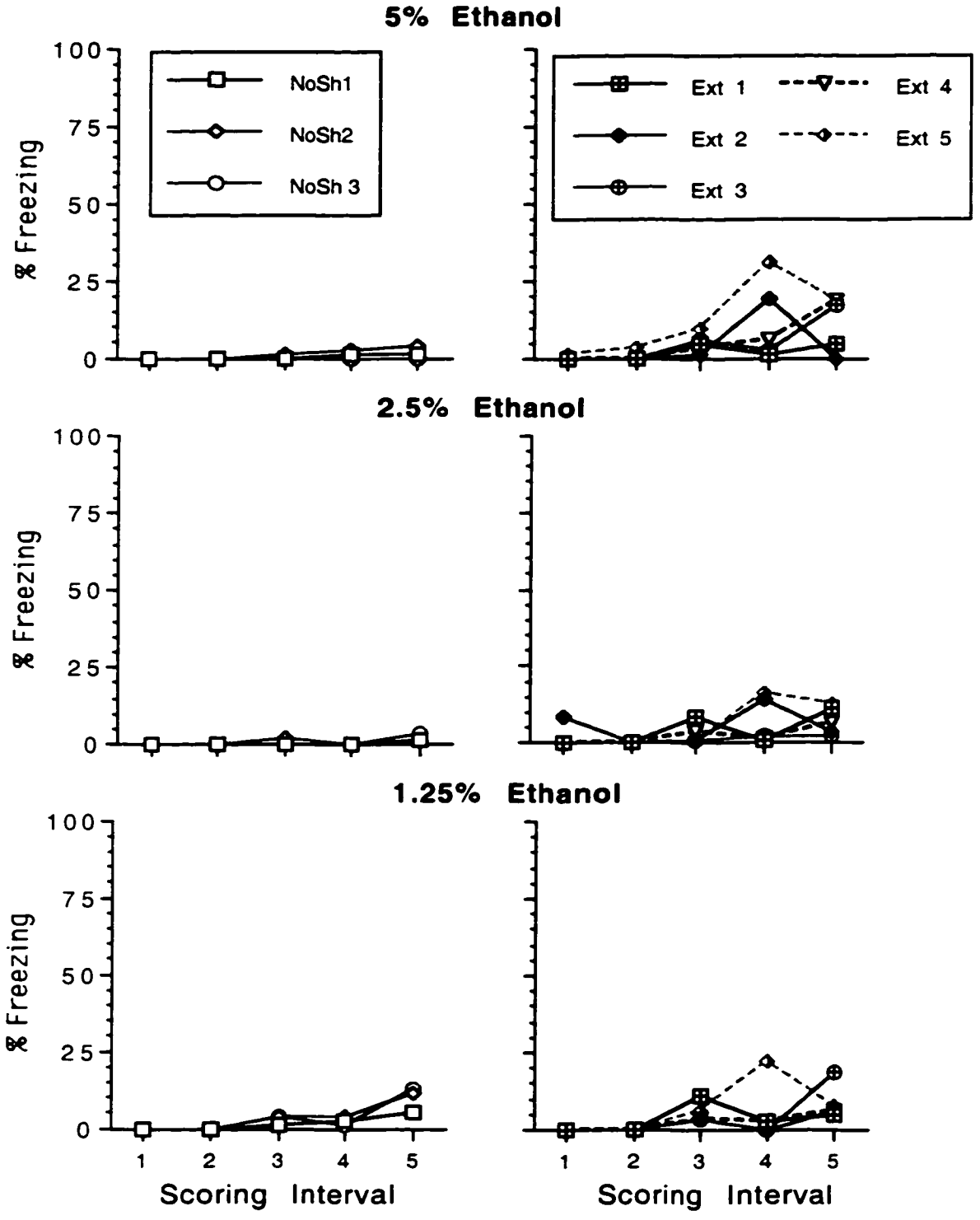

Figure 53A: No Shock-Ethanol groups

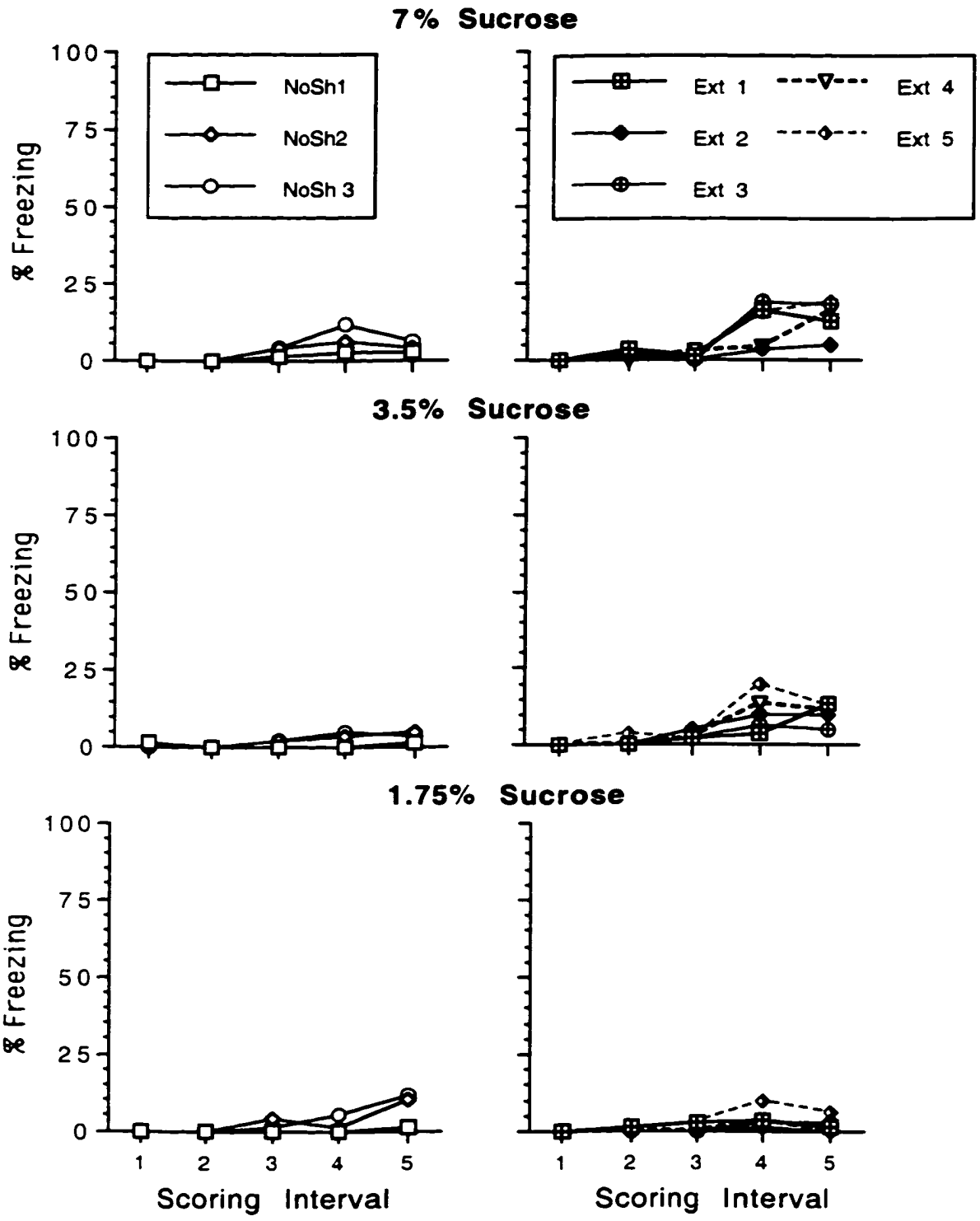

Figure 53B: No Shock-Sucrose groups

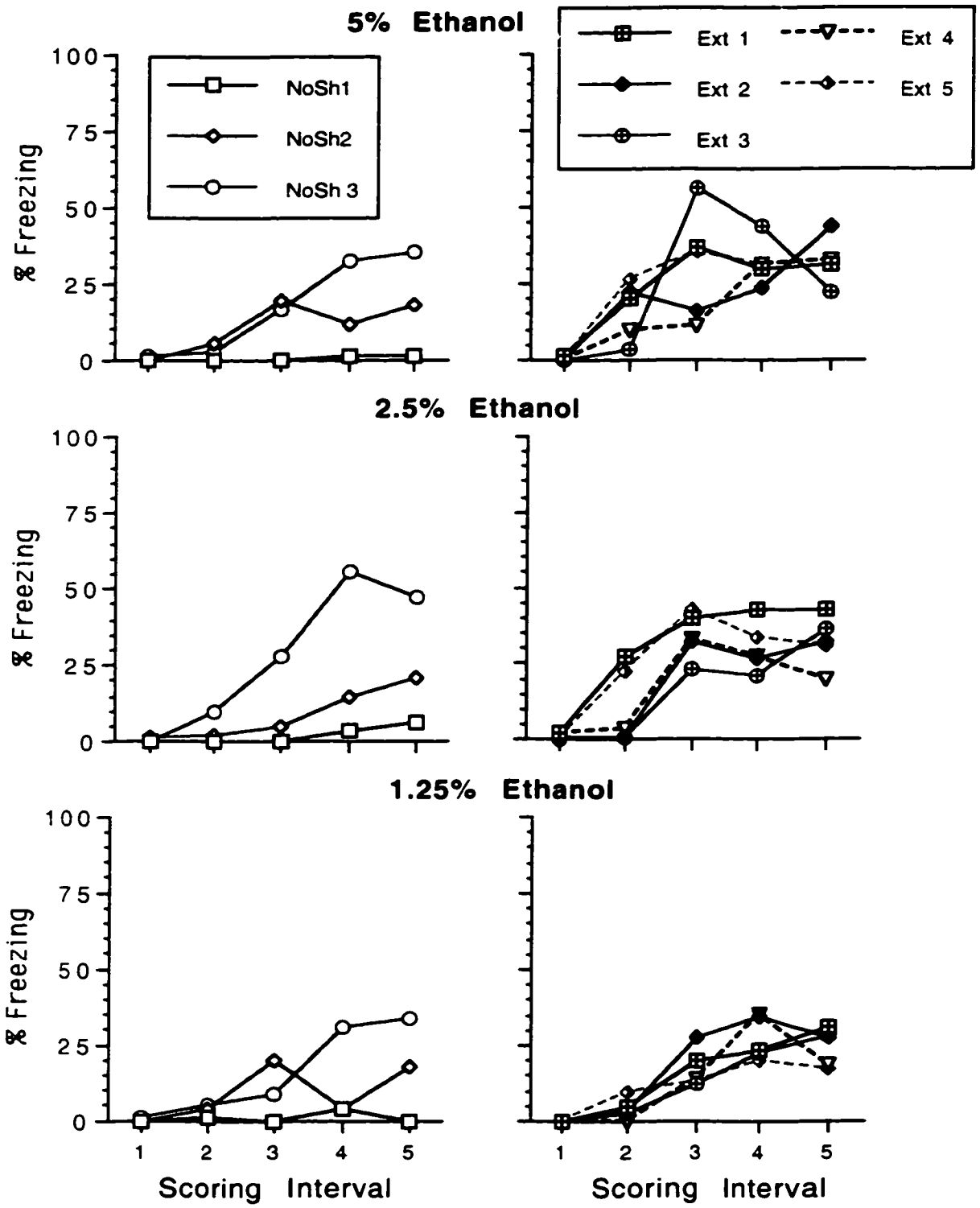

Figure 53C: Shock-Ethanol groups

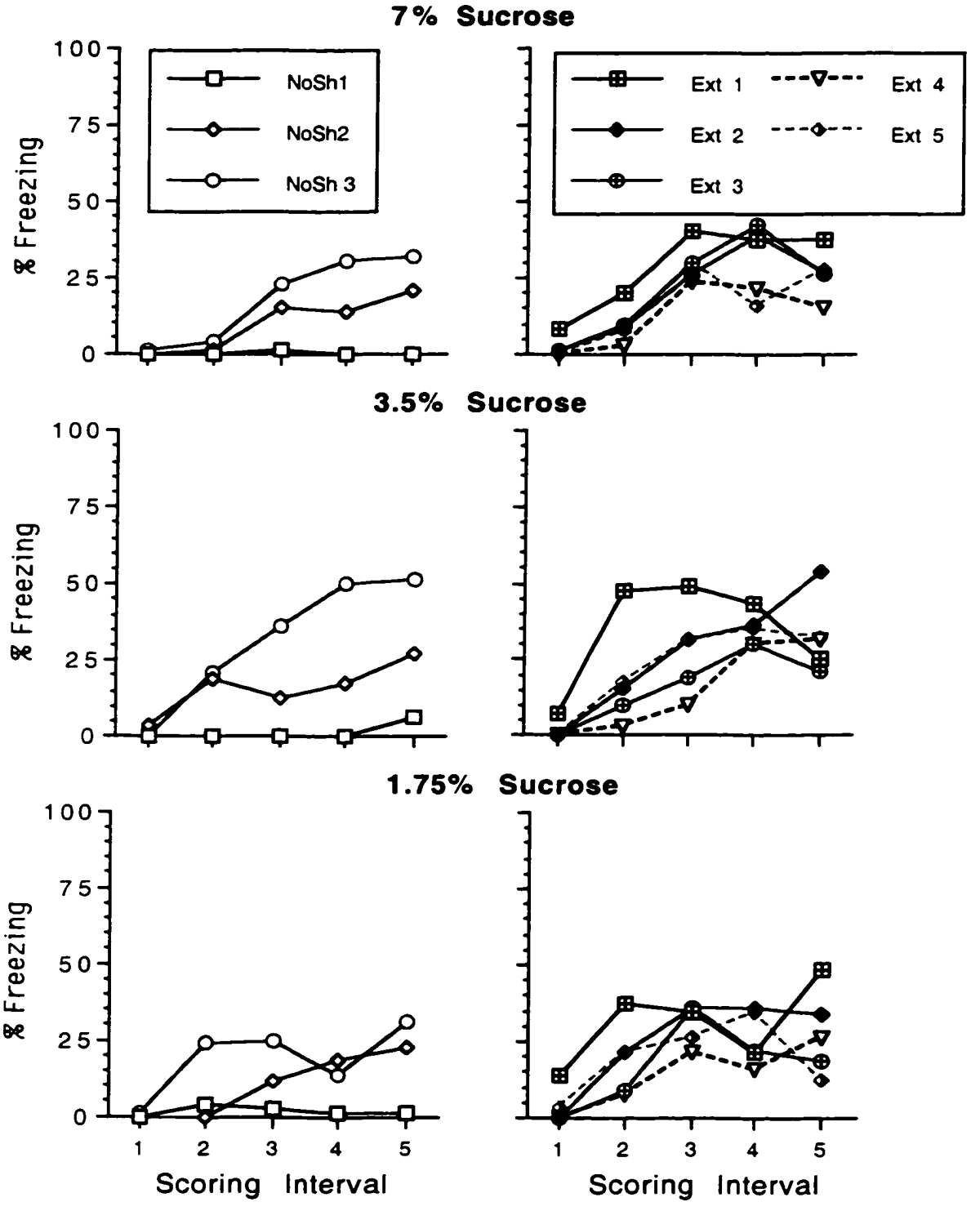

Figure 53D: Shock-Sucrose groups

consuming the Medium concentration solution in the center and freezing by the group consuming the Low concentration solution at the bottom.

Examination of freezing during the three no-shock sessions revealed significant effects of shock condition, $F(1,84) = 106.903$, day, $F(2,168) = 93.993$, and scoring interval, $F(4,336) = 47.334$. No Shock groups froze less than Shock groups. Freezing increased across scoring intervals and across no-shock sessions. The interactions between shock condition and day, $F(2,168) = 68.837$, between shock condition and scoring interval, $F(4,336) = 18.822$, between day and scoring interval, $F(8,672) = 12.701$, and among shock condition, day and scoring interval, $F(8,672) = 7.920$ were all significant. The simple main effect of day was significant for the Shock and No Shock groups, $F(2,84) = 90.698$ and 5.944. In each case, freezing was lower during the first no-shock session than during the second, $F(1,84) = 181.218$ and 9.257. Freezing during the third no-shock session differed from average freezing during the first two sessions only for theShock group, $F(1,84) = 140.875$. The simple main effect of shock condition was significant on the second and third no-shock days, $F(1,84) = 36.072$ and 126.479. In both cases, freezing was higher by the Shock groups than by the No Shock groups.

The simple main effect of scoring interval was significant for both the No Shock and Shock groups, $F(4,168) = 14.542$ and 35.773. Freezing increased across scoring

intervals for both shock conditions. The simple main effect of shock condition was significant for the last four scoring intervals, $F(1,84) = 22.721$, 35.616, 53.626, and 60.497. For each interval, the Shock groups froze more than the No Shock groups. The simple main effect of scoring interval was significant for each of the first, second, and third no-shock sessions, $F(4,336) = 5.284$, 15.369, and 32.538. Freezing always increased across the session. The simple main effect of day was significant for the second, third, fourth, and fifth scoring intervals, $F(2,168) = 6.887$, 21.642, 40.938, and 36.835. For each interval, freezing was lowest during no-shock day 1 and highest during no-shock day 3.

Examination of the freezing data for the three no-shock sessions also revealed a number of significant interactions involving fluid and/or concentration in spite of the fact that all of the animals were drinking water during these sessions. There were significant interactions between shock condition and concentration, $F(2,84) = 5.314$, between day and concentration, $F(4, 168) = 3.059$, $p < .05$, among day, shock condition and concentration, $F(4,168) = 3.839$, and among scoring interval, shock condition and concentration, $F(8,336) = 2.022$, $p < .05$.

Freezing during each scoring interval for the five extinction sessions is shown in the right-hand panels of Figures 53(A-D). Freezing during the first four extinction sessions was examined separately from freezing during the fifth session since the animals

were under a different deprivation state for the fifth session. Freezing by the No Shock groups was not expected to change across extinction sessions. However, freezing by the Shock groups should have diminished over days. Predictions from the tension reduction hypothesis are that freezing should have diminished more quickly and more completely for the Shock-Ethanol groups than for the Shock-Sucrose groups. Shock groups did freeze more than No Shock groups, $F(1,84) = 98.895$, and freezing did diminish across days, $F(3,252) = 10.497$, although freezing continued to increase across scoring intervals, $F(4,336) = 56.816$. The main effects of fluid and concentration were not significant and neither were the interactions among day, shock condition and fluid or among day, shock condition, fluid and concentration. The significant interactions between day and shock condition, $F(3,252) = 10.247$, between scoring interval and shock condition, $F(4,336) = 23.617$, and among day, scoring interval and shock condition, $F(12,1008) = 2.064$, $p < .05$ were not relevant to my predictions based on the tension reduction hypothesis since fluid was not involved.

Examination of freezing during the fifth extinction session revealed significant effects of shock condition, $F(1,84) = 23.611$, and scoring interval, $F(4,336) = 20.915$, and a significant interaction between the two, $F(4.336) = 5.464$. The Shock groups still froze more than the No Shock groups and freezing increased from the first to the fourth scoring

interval. The main effect of fluid was not significant nor were any of the interactions involving fluid.

Difference scores were calculated for each scoring interval of each extinction session in order to assess freezing during extinction relative to the third no-shock session (no-shock 3 minus extinction). These difference scores are shown for the six No Shock groups and six Shock groups in Figures 54 (A and B). In each case, difference in freezing by the Ethanol groups is on the left and difference in freezing by the Sucrose groups on the right. The uppermost panels show the difference scores for groups that consumed the High concentration of each solution. The middle panels show the difference scores for groups that consumed the Medium concentration of each fluid and the bottom panels show difference scores for the groups that consumed the Low concentration of each fluid.

Freezing by the No Shock groups was not expected to be different during extinction from what it had been during the third no-shock session. As a result, difference scores for the No Shock groups should have been near zero over the course of extinction. By comparison, Shock groups, particularly Ethanol-Shock groups, were expected to freeze less as extinction progressed. Thus the difference scores for Ethanol-Shock groups should have increased across extinction sessions, resulting in a significant interaction

Figure 54 (A and B): Mean difference from no-shock 3 in percent freezing for each scoring interval for the five extinction sessions during Experiment 8. A) No Shock groups; B) Shock groups. In each case, the top, middle and bottom panels showed freezing by groups that consumed the High, Medium, and Low concentrations of ethanol (left) or sucrose (right) during extinction.

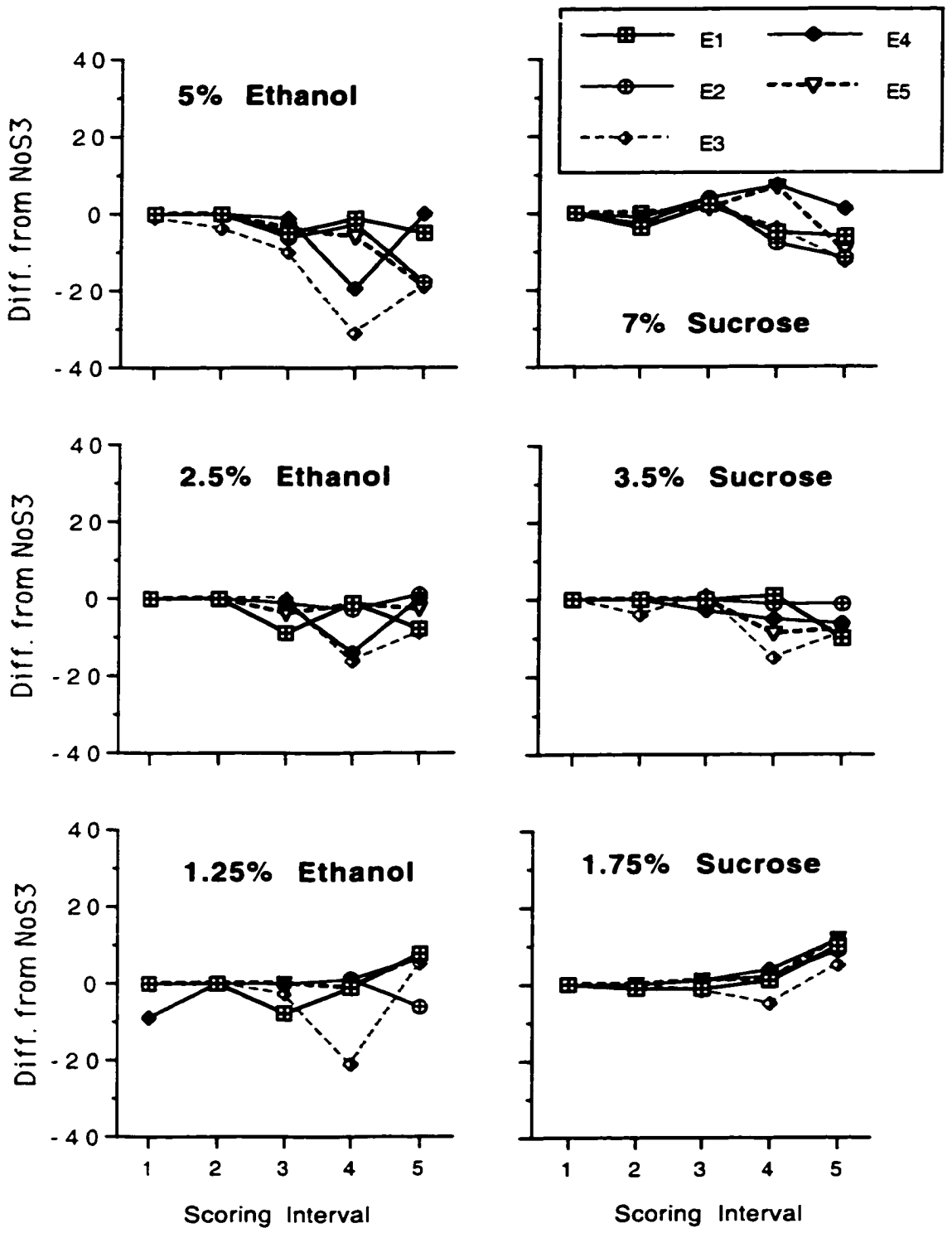

Figure 54A: No Shock groups

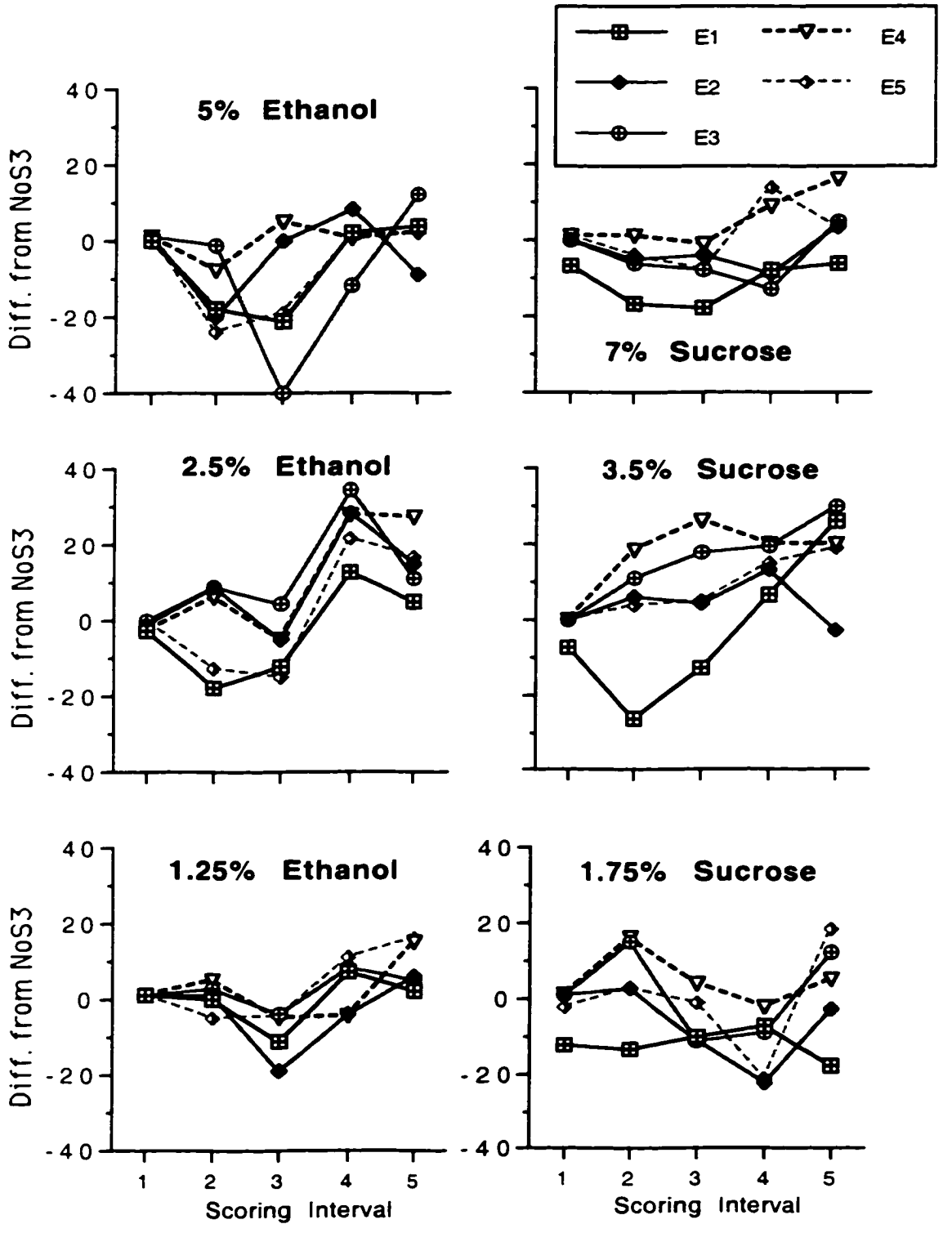

Figure 54B: Shock groups

among shock condition, fluid and day. This interaction was not significant, nor were any other interactions involving fluid.

There is evidence for extinction of freezing. The main effect of day was significant, $F(3,252) = 10.497$. Difference scores increased across the first four extinction sessions. Further, the interaction between day and shock condition was significant, $F(3,252) = 10.247$. Only for the Shock groups were the difference scores greater for the fourth extinction session than for the first, $F(3,126) = 12.454$. Difference scores for the Shock groups were greater than those for the No Shock groups in the third and fourth extinction sessions, $F(1,84) = 6.363$ and 15.136.

The main effect of concentration, $F(2,84) = 4.163$, was significant as were the interactions between shock condition and concentration, $F(2,84) = 5.702$, between scoring interval and shock condition, $F(4,336) = 2.684$, $p < .05$, and among day, scoring interval, and shock condition, $F(12, 1008) = 2.064$, $p < .05$. Since none of these interactions involve fluid, and therefore are not relevant to my predictions, they are not analysed further.

Examination of the difference scores relating freezing during the fifth extinction session to freezing during the third no-shock session (N3 - E5) revealed a significant effect of shock condition, $F(1,84) = 6.501$, $p < .05$, and a significant interaction between

scoring interval and shock condition, $F(4,336) = 6.942$. Freezing by the no-shock groups was higher during the fifth extinction session than it had been on no-shock 3. The Shock groups froze less. The difference scores for the No Shock group were lower than for the Shock groups. There were no significant effects or interactions involving fluid.

The correlations between total freezing and bolus production were examined for the six shock groups during the third no-shock session (when all groups had access to water in the shock boxes) and for the first extinction session (during which ehtanol and sucrose were introduced). During the third no-shock session the correlations were .641, .5038, .0566, -.082, .57, and .1783 for the high, medium, and low concentration ethanol groups and the high, medium, and low concentration sucrose groups respectively. There was a big range in correlations during this session in spite of the fact that all of these group were treated identically. During the first extinction session, the correlations between total freezing and bolus production were -.168, .6957, .0164, .5397, -.136, and .6049 (in the same sequence as above). Correlations for the last no-shock session did not predict correlations on the first extinction session.

Discussion

In Experiment 7, fluid consumption in the shock boxes was too low to permit an assessment of whether ethanol reduced tension. During Experiment 8, fluid deprivation

was re-introduced with the result that consumption during shock box sessions in this experiment was substantially higher than in the previous experiment. Further, casual observation suggested that I was successful in getting the rats to drink early in the shock box sessions. Most of the rats began to drink as soon as they were placed in the shock boxes and it is certainly true that more rats were drinking during the first than during the fifth scoring interval.

During both no-shock and extinction sessions fluid consumption was higher by the No Shock groups than by the Shock groups. Over the four extinction sessions, consumption of ethanol and sucrose increased. The main effect of fluid was not significant and neither was the interaction between shock condition and fluid, nor the interaction among shock condition, fluid and concentration. Thus, there was no evidence for a differential increase in consumption by the Shock-Ethanol groups. The second tenet of the Tension Reduction Hypothesis, that rats learn to drink ethanol for its tension reducing effects was not supported. The only effect on consumption that was attributable to the shock manipulation was the lower consumption by Shock groups relative to No Shock groups and this difference was apparent regardless of whether the animals were consuming water (during no-shock sessions), or ethanol or sucrose. The difference scores relating consumption during each extinction session to consumption during the third no-

shock session also failed to reveal any interactions involving both fluid and shock condition. Consumption of sucrose was suppressed (relative to water consumption on no-shock 3) more than ethanol consumption during the first two extinction days.

Absolute ethanol consumption was higher by the Shock groups than by the No Shock groups and consumption was inversely related to concentration. There was no indication that the rats were attempting to regulate either their dose of ethanol or their caloric intake from ethanol. Instead, it seemed that total volume of fluid was more important. There were no significant interactions involving shock condition. Over all of the measures of fluid consumption, there was no evidence that animals learned to consume ethanol for its tension reducing effects.

Regardless of whether the rats were consuming ethanol specifically for its tension reducing effects, ethanol could have exerted such effects. Conditioned fear was established prior to the introduction of ethanol and sucrose. The Shock groups defecated more and froze more than the No Shock groups. Defecation and freezing by the Shock groups increased across no-shock days (which reflected their shock exposures on intervening shock days). During extinction, the interaction among shock condition, fluid, and day was significant. Defecation decreased across extinction sessions for both the Shock-Ethanol and the Shock-Sucrose groups. Defecation by the Shock-Ethanol groups

had decreased to the level of the No Shock-Ethanol groups by the second extinction session. By comparison, the Shock-Sucrose groups defecated more than the No Shock-Sucrose groups during all five extinction sessions. These observations could indicate that fear extinguished more fully for the Shock-Ethanol groups than for the Shock-Sucrose groups which could indicate that ethanol was reducing tension.

Examination of freezing behaviour revealed not only significant effects of shock condition, but also a significant effect of scoring interval. Freezing increased across scoring intervals within sessions and overall across sessions for the three no-shock sessions. Although total freezing diminished across the extinction sessions for the Shock groups, within sessions freezing continued to increase across scoring intervals. For the freezing measure (or for the freezing difference score), there were no significant effects or interactions involving fluid. There was no evidence from the freezing measure for differential rates of extinction mediated by the intake of ethanol versus sucrose. The result from my experiment was contrary to that of another experiment (Stromberg & Hammond, 1997) following virtually identical shock exposure. Swiss Webster mice treated with ethanol (1.2 g/kg i.p.) prior to being placed in an environment in which they had previously been shocked froze more than mice treated with saline. Level of freezing did not differ between ethanol and saline groups that had never been shocked. Shock

exposure was virtually identical in the two experiments. The discrepancy between these experiments could result from species differences, or differences between oral consumption (my experiment) and i.p. injections of ethanol (Stromberg & Hammond, 1997).

The results of this experiment provided validation for the 1-Shock schedule being sufficient to produce a significant level of fear (relative to unshocked controls), as measured by both bolus production and freezing. Despite this finding, there is no consistent relationship between bolus production and total freezing. One mechanism that could produce marked variation between the correlations of the two measures among groups treated identically would be differential sensitivity of the two measures to fear. Specifically, freezing might be a more sensitive measure of fear that is bolus production. Examination of the data revealed no evidence for this mechanism.

This experiment provided little support for either tenet of the tension reduction hypothesis. The defecation data suggested that fear might have extinguished more rapidly in animals that consumed ethanol. However, without convergence from the freezing measure the evidence is not compelling. The evidence is equivocal as to whether or not ethanol reduces tension. Certainly there is no evidence to support the second tenet of the hypothesis, that animals learn to consume ethanol for its tension reducing effects. The

groups expected to be feeling tension (Shock groups) did not consume more ethanol than groups expected to be relatively stress-free.

GENERAL DISCUSSION

The consumption data failed to support either the tension reduction or the relief hypothesis. In Experiment 1 one group of rats had access to ethanol (or sucrose) immediately after shock sessions, a period when they would have been expected to feel relief. However, ethanol consumption by this group did not differ from ethanol consumption by a group that had access to ethanol (or sucrose) on no-shock days. Sucrose consumption was always higher than ethanol consumption but also did not differ between groups. In Experiment 2, when fluid access occurred immediately before shock sessions, a period when the rats would have been expected to be anxious, the pattern of consumption was strikingly similar to the pattern observed in Experiment 1. Ethanol consumption did not increase across cycles for any of the groups in the first two experiments. There was no evidence for increased consumption (across days) during periods either when the rats should have been anxious because they were anticipating shock or when they should have been relieved because shocks had been discontinued.

In Experiment 2, placement in the drinking environment occurred both before shock sessions and on no-shock days. The situation for the rats was further complicated by the fact that the fluid available in the drinking environment was different on different days. In Experiments 3 and 4, placement in the drinking environments only occurred in

the hour before shock sessions and different groups of rats had access to ethanol, sucrose, and water in the hour before shock sessions. Consumption was higher by the Water group than by the Sucrose group and both of those groups consumed more than the Ethanol group. The Ethanol group was the only one that increased its consumption across cycles. It might have been tempting to interpret this increase as being in accordance with tension reduction but it would be difficult to reconcile that interpretation with the fact that ethanol consumption was higher by a home cage ethanol group (which was included in Experiment 3). A number of studies have shown that individually housed rats consumed more ethanol than group housed rats (Hannon & Danlon-Bantz, 1976; Parker & Radow, 1974; Roske et al., 1994; Wolffgramm, 1990; Wolffgramm, 1991). This has been explained as an effect of stress due to social deprivation in the individually housed rats. To relate this to Experiment 3, rats in the home cage ethanol group would have been experiencing whatever stresses were associated with being individually housed. In order for tension reduction to hold, then the stresses associated with being individually housed must have been greater than the stresses associated with being individually housed, handled seven times per day, and subjected to a 1-hour shock session. Even if this were to be believed, it still would not explain why the two ethanol groups consumed less fluid

than the Sucrose and Water groups. The lower ethanol consumption might have reflected minimum levels of fluid consumption required by biological necessity.

Experiment 5 was conducted in order to determine to what extent thirst motivated consumption in the earlier experiments. Rats had access to fluid in the drinking environment under different levels of fluid deprivation. Thirst seemed to be one factor motivating fluid consumption in the drinking environment since all of the groups consumed less fluid in the drinking environment prior to shock on days when they had been preloaded than on days when they were not preloaded with water. Thirst appeared to account for more of the observed ethanol than sucrose consumption, since ethanol consumption was more suppressed than sucrose by preloading. Although thirst did not fully account for ethanol consumption, there was no evidence that ethanol was being consumed for its pharmacological effects (including tension reduction).

Experiment 6 was conducted in order to assess whether ethanol was more reinforcing during signals for a noxious event than in the absence of such signals. Consumption was higher by the Sucrose group than by the Ethanol group until after the fluids were switched. Ethanol consumption did not increase across cycles whether it was the original fluid or the one that was introduced during the twenty-first exposure to each drinking environment. There were no consistent consumption differences between the

shock-paired and safe-paired drinking environments. Again, the consumption data were inconsistent with tension reduction.

In Experiments 7 and 8, access to ethanol (or sucrose) occurred in the shock boxes during extinction. In Experiment 7, rats had free access to water in their home cages and consumption in the shock boxes was negligible. In Experiment 8, No Shock groups consumed more fluid than Shock groups in the shock boxes during no-shock and extinction days. Absolute ethanol consumption was higher by the Shock groups than by the No Shock groups. There was no evidence for a differential increase in consumption by the Shock-Ethanol groups as would have been required to support the tension reduction theory.

In previous experiments that used a shock schedule similar to the one used in Experiments 1-6 (Caplan & Puglisi, 1986; Fidler & LoLordo, 1996; Volpicelli et al., 1986; Volpicelli et al., 1982; Volpicelli et al., 1990) rats had access to fluid 23 hours per day. Rats in these experiments did not consume sufficient ethanol to maintain continuous intoxication, an amount estimated at 212 ml/kg of 5% ethanol or 106 ml/kg of 10% ethanol (Mills et al., 1977). It is possible that the discrepant results of these experiments arose from the fact that different amounts of consumption occurred just before or just after the shock sessions. In my experiments fluid access was restricted to a one hour

period immediately after (Experiment 1) or immediately before (Experiments 2-6) a one hour shock session in order to increase the likelihood that animals would consume a pharmacologically significant dose of ethanol in temporal proximity to the shock session. In spite of the fact that the rats in Experiments 1-6 and 8 consumed amounts of ethanol expected to be pharmacologically significant there was no evidence that tension reduction or relief reinforced their consumption.

Rather than consuming ethanol to obtain reinforcement from tension reduction or relief it seems more likely that much of the consumption observed in these experiments was reinforced by thirst reduction. In support of this contention consider the suppressed consumption of fluid (ethanol, sucrose, or water) in the drinking environment on days when the rats had access to fluid during a pre-loading period in Experiment 5. More compelling still is the negligible consumption in the shock boxes by non-deprived rats in Experiment 7.

My rats did not appear to consume ethanol in order to obtain its tension reducing effects but this does not mean that the ethanol that they did consume did not have a tension reducing effect. Bolus production and freezing were measured in order to assess whether ethanol did reduce tension. Higher levels of each should have been associated with higher levels of fear. Bolus production was recorded for both drinking and shock

boxes beginning part-way through Experiment 3. In Experiments 3 through 6, bolus production was very low in the drinking environments. This observation could explain the lack of support for the tension reduction hypothesis in these experiments. According to the tension reduction hypothesis, animals would have been expected to learn to drink ethanol when they were experiencing anxiety in order to obtain its tension reducing effects. If the rats were not experiencing any anxiety in the drinking environment then they could not be reinforced (by tension reduction) for consuming ethanol. In spite of the fact that there was a perfect correlation between a particular drinking environment and shock sessions (in Experiments 3-6), there were always handling cues that separated the two environments. In Experiments 7 and 8, these cues were omitted since the drinking environment and the shock box were one and the same. Unfortunately, it was not possible to determine the temporal distribution of defecation in these experiments.

Defecation in the shock boxes was measured beginning late in Experiment 3. If ethanol reduced tension, then rats that consumed ethanol prior to (or during) shock sessions would have been expected to produce fewer boli than rats that did not consume ethanol. Bolus production was consistently higher in the shock boxes than in the drinking environment, which suggests that the rats were more fearful in the shock boxes than they had been in the drinking environment. During the last six shock sessions of Experiment 3

(the only shock sessions for which these data were available) the Ethanol group defecated less than the Sucrose and Water groups. In Experiment 4, there were no group differences until the sixth shock session. After this time, the Ethanol group defecated less than the other groups (except during cycle 9). During Experiment 5, on days with no preloading, when ethanol consumption was higher, bolus production in the shock box was lower. On days with no preloading, defecation by the Ethanol group was lower than by the Sucrose and Water groups. After fluids were switched so that most rats were consuming ethanol, more fecal boli were produced on preloading days (when consumption was lower) than on no-preloading days (when consumption was higher). In Experiment 6, there was no evidence that animals that had consumed ethanol defecated less whether ethanol was available prior to shock on shock days 4-20 or 21-32. Shock groups defecated more than No Shock groups and defecation increased across no-shock days in Experiments 7 and 8. Defecation decreased across extinction days. This pattern is what would be expected with acquisition and extinction of fear by the Shock groups. In Experiment 8, defecation decreased across extinction days for both Shock-Ethanol and Shock-Sucrose groups. Defecation by the Shock-Ethanol groups had decreased to the level of the No Shock-Ethanol groups by the second session. Even after five extinction sessions the Shock-Sucrose groups defecated more than the No Shock-Sucrose groups.

The shock box defecation data were generally consistent with tension reduction. In Experiments 3-5, rats that had consumed ethanol defecated less than rats that had consumed Sucrose or Water. In Experiment 5, defecation was lower when ethanol consumption was higher (on no preloading days). In Experiments 3 and 4, ethanol was introduced immediately before the first shock session so that the rats were always under the influence of ethanol during shock sessions. There is evidence that ethanol can have analgesic properties at some doses (Brick et al., 1976; Cunningham & Brown, 1983). If my rats had consumed enough ethanol to produce analgesia then the shocks would have been less severe for these animals than for the animals that did not consume ethanol. If the shocks were less severe then there would have been less conditioned fear in these animals. The lower rate of bolus production by the ethanol group would be consistent with less conditioned fear. The rats from Experiment 4 were used in Experiment 5 so if ethanol interfered with fear conditioning in Experiment 4 this would also influence defecation in Experiment 5. In Experiments 6-8 shock was introduced prior to the introduction of ethanol so ethanol could not have interfered with the acquisition of fear conditioning. In Experiment 6 there was no difference in defecation between ethanol and sucrose groups, which lends support to the idea that ethanol interfered with acquisition of fear in the earlier experiments. This result might suggest that once fear had been

conditioned then reducing the perceived magnitude of the stressor (as would be the case if ethanol were an analgesic) had no impact on expression of fear. In Experiments 7 and 8 (in which many fewer shocks were administered than in Experiments 1-6) ethanol was introduced after the cessation of shock so analgesic properties of ethanol could not reasonably be invoked to explain any changes in defecation that occurred. Defecation appeared to extinguish more rapidly for Ethanol groups than for Sucrose groups.

Another alternative to tension reduction is that ethanol slowed gastric motility which could have resulted in lower defecation by Ethanol groups. The easiest way to assess the possibility that ethanol had effects on gastric motility would have been to compare the bolus production of Shock and No Shock groups (in Experiment 8). If ethanol reduced gastric motility then bolus production should have been reduced in both Shock and No Shock groups relative to the last no-shock day when all groups had consumed water. Unfortunately, on the third no-shock day, defecation by the No Shock group was already near zero, so it was not possible to determine whether ethanol consumption would have reduced defecation even without shock.

I am not aware of any research that has specifically addressed the effect of orally consumed ethanol on bolus production by fluid deprived Sprague Dawley rats. However, no significant correlation was observed between ethanol consumption and defecation for

non-deprived Sprague Dawley rats in the absence of stress (Tobach, 1957). There is evidence from an in vitro preparation that ethanol depressed phasic antral contractions which are important for normal emptying of solids from the stomach (Sanders & Bauer, 1982) and that higher concentrations of ethanol caused tonic contraction of antral muscle which might enhance emptying of liquids from the stomach (Sanders & Bauer, 1982). In CDR and Swiss Webster mice gastrointestinal transit of a charcoal meal was inhibited by prior injections of ethanol (i.p. or s.c.), particularly in the small intestine (Scroggs, Abruzzo & Advokat, 1986). However, it is not clear what these studies have to say about my results. From none of these studies is it clear how long after ethanol consumption effects on gastric transit would have been expected or what the effect should have been on food that was already in the system (since my rats had no opportunity to eat in the drinking environment or shock boxes).

Another measure of conditioned fear, freezing, was observed to a limited degree in Experiment 5 and for all sessions in Experiments 7 and 8. Since freezing was expected to be an index of fear, higher levels of freezing were interpreted as indicators of higher levels of fear. To the extent that ethanol reduced tension, rats that consumed ethanol should have spent less time freezing (and therefore had lower % freezing scores) than rats in other groups. For the small number of animals and sessions for which freezing data

were available in Experiment 5 the data appear to be consistent with tension reduction. Rats in the Ethanol groups froze less than rats in the Sucrose and Water groups. In Experiment 7, there was not enough fluid consumption in the drinking environment to assess effects of fluid consumption on freezing. In Experiment 8, Shock groups froze more than No Shock groups, suggesting that the 1-shock schedule was successful in evoking fear. Also, freezing decreased across extinction sessions which suggests that fear was extinguished by placements in the shock boxes with no shocks. There was no indication that ethanol was any more effective in reducing fear (as measured by freezing) than sucrose.

Ethanol may have effects on locomotor activity that are irrelevant to states of tension or relief. If ethanol suppressed locomotor activity then freezing might have been expected to increase regardless of shock treatment. Conversely, if ethanol enhanced locomotor activity then freezing would have been expected to decrease regardless of shock treatment. Freezing by the No Shock groups was near zero prior to the introduction of ethanol so it would not have been possible to identify increased activity in these groups (using this measure). There was no evidence of decreased activity (increased freezing) by No Shock groups so it is unlikely that changes in freezing by the Shock groups are attributable to effects of ethanol on general activity. Experiments that have

investigated the effect of ethanol injections on locomotor activity by Sprague Dawley rats have reported either no effect (Erickson & Kochhar, 1985; Frye & Breese, 1981) or decreased activity (Frye & Breese, 1981). The absence of differences between Ethanol- and Sucrose-Shock groups in Experiment 8 were contrary to those of another experiment which investigated the relationship between shock, freezing, and ethanol (Stromberg & Hammond, 1997). Mice injected with ethanol prior to placement in a box where shocks had previously been delivered froze more than saline injected controls. Level of freezing did not differ between ethanol and saline treated unshocked mice.

There is little support within this series of experiments for either the tension reduction hypothesis or the relief hypothesis. In spite of the fact that the experiment was designed to maximize ethanol consumption in temporal proximity to the shock session the observed pattern of fluid consumption was not consistent with predictions from either hypothesis. The two measures of conditioned fear, freezing and bolus production, were examined to assess whether ethanol reduced tension. Although bolus production tended to be lower in groups consuming ethanol this effect could be adequately explained without invoking tension reduction. Freezing data differentiated between Shock and No Shock groups but failed to differentiate between Ethanol and Sucrose groups. The lack of

convergence between these two measures is another reason not to invoke tension reduction.

Water deprivation was reported to facilitate freezing during an extinction test in an environment where shock had previously been administered (Maren & Fanselow, 1998). If water deprivation enhanced fear conditioning then both bolus production and freezing should have been higher in Experiment 8 than in Experiment 7. This did not appear to be the case. In Experiment 7, when the rats were non-deprived, mean bolus production by the Shock rats on the no-shock days was 0.6, 1.1, and 5.6. In Experiment 8, when the rats were deprived, mean bolus production by the Shock rats on no-shock days was 1.0, 2.8, and 5.21. Percent freezing scores for the Shock groups on no-shock sessions in Experiment 7 were 1.51, 7.25, and 28.8. The comparable values for Experiment 8 were 1.1, 10.9, and 21.8. These data provide no support for facilitation of freezing or bolus production by water deprivation. However, the rats drank at the beginning of the session and froze at the end of the session so at the instant that freezing was being measured the deprivation state in the two experiments might not have been very different.

Although there is little support within this series of experiments for either the tension reduction hypothesis or the relief hypothesis this does not necessarily mean that there are not other organisms, temporal arrangements, ethanol concentrations, shock

parameters, and/or stressors which might support either hypothesis. Each hypothesis is phrased in rather vague terms, making no specific statements about when ethanol might be expected to have its effects (how long before or after the fear-eliciting stimulus); how long the effects might be expected to last; what the effective doses might be; etc. Non-specific effects of ethanol on locomotor behaviour and gastric motility as well as reinforcement from repletion or thirst reduction would have to be eliminated before invoking other explanations for observed patterns of ethanol consumption. This might be accomplished by including multiple control groups (for example no-shock ethanol groups or shock and no-shock groups with access to isocaloric solutions) and by collecting multiple measures of conditioned fear. Much of the previous work has lacked appropriate control groups and/or measures. Earlier experiments (for example see (Fidler & LoLordo, 1996; Volpicelli et al., 1986; Volpicelli et al., 1982; Volpicelli et al., 1990) have failed to take any measure of conditioned fear which would have allowed a determination of whether animals that consumed ethanol actually exhibited less fear or anxiety. The time course of consumption should also be monitored (especially in experiments where fluid is available over a long period of time) in order to determine the temporal relationship between stress and ethanol. Whatever the deficiencies of the current set of experiment or previous ones, it seems clear that if ethanol is an anxiolytic, its potency as an anxiolytic is

not great, perhaps because as dose increases, the potency of side effects that interfere with tension reduction increases (Cappell, 1987).

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