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Licking and liking: The assessment of hedonic responses in rodents

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Affective processes are a key determinant of behaviour: At its simplest, liked stimuli are approached while disliked stimuli are avoided. Although assessing hedonic responses in nonverbal animals can be difficult, one relatively tractable approach relies on detailed analyses of rodents' consummatory behaviour. Rodents typically produce rhythmic sets of licks that can be grouped into clusters on the basis of the intervals between licks. The mean number of licks in a cluster (cluster size) is directly related to the concentration of palatable and unpalatable solutions. These relationships suggest that lick cluster size might be a useful index of an animal's hedonic reaction to the solution being consumed. I begin by reviewing studies of conditioned flavour preference and aversion that support the idea that lick cluster size can provide useful information about rats' hedonic reactions. I then describe how this methodology has been used to address previously intractable issues in the investigation of contrast effects as well as revealing an analogue of effort justification effects that, in humans, are commonly explained in

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terms of cognitive dissonance reduction. Finally, I consider how lick analysis might provide information about hedonic responses in animal models of human psychiatric disorders. In all these cases, *how* an animal did something was particularly informative about *why* it was doing it.

Keywords: Consumption; Lick microstructure; Learning; Affect.

The first proper experiment I ever ran was an investigation of context effects in taste aversion learning. While the theoretical details are no longer relevant, in practical terms it involved placing rats in boxes and measuring how much they drank. Twenty years later, I am still doing essentially the same thing! However, the examination of consumption is somewhat at the periphery of comparative psychology, and comparative psychology is (arguably) at the periphery of psychology as a whole. So why persevere with consumption studies? And why, despite being involved in other research topics that might be considered more "accessible"—such as face processing (e.g., Mundy et al., 2009; Mundy, Honey, & Dwyer, 2007) or debunking stories about animal reasoning (e.g., Dwyer & Burgess, 2011; Dwyer, Starns, & Honey, 2009)—should I choose consumption as the topic for my EPS Prize Lecture? I trust the answers to such questions will be apparent by the time I am finished.

At a first glance, consumption appears to be one of the simplest and most basic behaviours in an animal's repertoire. Consummatory behaviours are present from birth onwards and form the basis of nutrient intake throughout the lifespan—something that is an interesting topic in its own right as any failure of nutrient ingestion has catastrophic effects. However, consumption is not simply an affectively neutral means for introducing nutrients into the digestive system; it also reflects the contribution of both learnt and unlearnt food likes and dislikes. In turn, this reflects the more general fact that affective processes are a key determinant of behaviour: Generally, liked stimuli are approached while disliked stimuli are avoided. Indeed, given the obvious difficulties in asking nonhuman animals directly about what they do or do not like, examining the voluntarily consumption of a substance can provide valuable information about whether, or how much, they like it. For example, the fact that rats subject to chronic mild stress (a model for depression) consume relatively little sucrose has been taken as an indication that they do not like the taste of sucrose and in turn that this reflects the presence of a state of anhedonia (e.g., Papp, Willner, & Muscat, 1991). That said, it is apparent after the most cursory inspection of the literature that the total amount of a substance consumed is an end point measure that is influenced by a number of factors (from motivational differences to motor effects) and so cannot be taken as an unambiguous measure of liking for the substance being consumed (or any other single variable for that matter).

It has long been known that, for various carbohydrates, the amount of solution voluntarily consumed is related to the concentration by an inverted U-shaped function whereby maximal consumption (of the solution, not the carbohydrate solute) occurs at moderate concentrations, and relatively little is consumed at either very high or very low concentrations (e.g., Richter & Campbell, 1940). Despite tasting different and containing different quantities of nutrients, low and high concentrations of sucrose elicit the same amount of consumption, and so total intake measures alone cannot tell us what is actually driving the consumption. The recognition that overall intake provides only a relatively crude tool to examine the underlying causes of consummatory behaviour has led to many decades of pioneering and painstakingly detailed research examining the microstructure of ingestive behaviours (e.g., Davis, 1998; Grill & Norgren, 1978a). Although this research has taken place somewhat on the margins of mainstream psychology, it has provided a rich variety of tools with which to investigate consumption. Here I review some of my applications of these tools.

Microstructural analyses of consumption

One of the earliest demonstrations that *how* consumption occurs provides particularly useful information about *what* is controlling that consumption came from the detailed observation of rats' orofacial behaviours as a factor of the type of solution to which they were exposed (Grill & Norgren, 1978a). In short, Grill and Norgren observed that exposure to the sweet taste of sucrose elicited a response sequence including rhythmic mouth movements and tongue protrusions, while the bitter taste of quinine elicited a different set of behaviours including gaping, chin rubbing, and forelimb flailing. Although Grill and Norgren based their discussion on the nature of the solutions being consumed, subsequent analysis revealed that the relevant behaviours can be divided into "appetitive" and "aversive" patterns (for a review, see Berridge, 2000). Critically, this division into appetitive and aversive behavioural sequences is not a fixed function of the solutions themselves but instead reflects the current state and/or the previous experiences of the animal in question. For example, pairing sucrose with gastric malaise (induced by the injection of lithium chloride, LiCl) results in sucrose eliciting the aversive response sequence typically associated with quinine (e.g., Pelchat, Grill, Rozin, & Jacobs, 1983). Moreover, exposure to concentrated saline normally elicits a mixture of aversive and appetitive responses, but this swaps to an entirely appetitive sequence when animals are tested in a state of salt deprivation (e.g., Berridge, Flynn, Schulkin, & Grill, 1984).

One particularly attractive feature of this "taste reactivity" procedure is that it has admirable face validity. The orofacial responses of numerous mammals (from rodents to human infants via a number of different primates) has been characterized, and several generalizable features have emerged: In particular, the gaping response to bitter tastes that humans find aversive is conserved across species, as is the tongue protrusion response to sweet tastes. Indeed, placed side by side, images of human infants and laboratory rodents reacting in obviously analogous fashions to novel sweet and bitter tastes are particularly striking (for a review, see Berridge, 2000). Given its face validity, it is unsurprising that the taste reactivity procedure has provided insights into topics ranging from the mechanisms underpinning taste preferences and aversions (e.g., Myers & Sclafani, 2001b; Parker, 2003) to the brain and pharmacological mechanisms mediating hedonic reactions (e.g., Berridge, 1996; Gray & Cooper, 1995). However, despite these past, and continuing, successes, there are several features of the taste reactivity procedure that limit the generality of its use. Most theoretically relevant is that fact that this procedure essentially allows a set of reactions to be classified as appetitive, aversive, or a mixture of the two and so tends to afford largely qualitative distinctions. More practically, the original technique was based on the passive infusion of fluid into a rat's mouth via a surgically implanted cannula, and while free-feeding analyses are possible, these can reduce the technique's effectiveness. The technique is also particularly labour intensive as it requires test subjects to be individually recorded and the video images hand-scored while being played back in slow motion. Along with creating practical and ethical obstacles to the widespread implementation of the technique, such issues also create a theoretical problem in that taste reactivity analyses are typically restricted to a very small sample of consumption and thus tend to provide a "snapshot" of any hedonic reaction rather than one that naturally tracks across time.

Around the same time as the taste reactivity method was instigated, a separate research tradition was being developed, which focused on the use of automated equipment to record and analyse licking behaviours in rodents and relating the microstructure of that behaviour to the variables influencing consumption (e.g., Davis, 1989; Davis & Levine, 1977; Davis & Smith, 1992). One of the key observations to emerge from this work was that rats ingest fluids in sustained runs of rapidly occurring rhythmic licks (referred to here as *clusters*) separated by pauses of varying length. Crucially, the mean number of licks per cluster (lick cluster size) is not random, but instead is lawfully related to the nature of the solution being consumed: Lick cluster size shows a positive, monotonic relationship to the concentration of palatable fluids such as sucrose (e.g., Davis & Smith, 1992; Spector, Klumpp, & Kaplan, 1998), while lick cluster size decreases

monotonically with increasing concentrations of unpalatable quinine solutions (e.g., Hsiao & Fan, 1993; Spector & St John, 1998). Importantly, lick cluster size is not simply some proxy for total consumption as lick cluster size dissociates from total consumption in that maximum levels of consumption occur at moderate concentrations of palatable solutions such as sucrose while maximum lick cluster sizes occur at high concentrations. The fact that larger lick cluster sizes are associated with the consumption of solutions that can be thought to be palatable and lower lick cluster sizes with less palatable or aversive solutions suggests that this variable can be taken as an indication of the hedonic response to the solution consumed. It should also be noted that lick cluster size is not the only microstructural variable that has been shown to correlate with the nature of the solution. Some of the earliest analyses of licking microstructure revealed that initial lick rates also bear a positive monotonic relationship with the concentration of sugars and a negative relationship with the concentration of quinine (e.g., Davis & Levine, 1977).[1]

The idea that lick cluster size might be a good indicator of the hedonic evaluation of the solution being consumed is reinforced by the fact that a variety of manipulations influence lick cluster size in ways analogous to their effects on taste reactivity. In addition to being influenced in similar ways by the simple administration of different tasting solutions like sucrose and quinine, both lick microstructure and taste reactivity show similar effects of learning. For example, pairing an otherwise palatable taste with LiCl results in it eliciting lick cluster sizes similar to those seen when drinking quinine (e.g., Baird, John, & Nguyen, 2005; Dwyer, 2009), while the same treatment results in sweet tastes eliciting aversive taste reactivity patterns like those typically elicited by quinine (e.g., Pelchat et al., 1983). In addition, benzodiazepine administration, which is thought to enhance hedonic reactions to foods in humans (Haney, Comer, Fischman, & Foltin, 1997), enhances lick cluster size (e.g., Higgs & Cooper, 1998) and appetitive taste reactivity responses (Gray & Cooper, 1995). Moreover, although the preservation of microstructural variables across species is less well documented than with taste reactivity responses, there is at least some evidence that foods that are reported by people to differ in palatability can also produce variations in microstructural parameters during consumption by people that are similar to those seen in rats (e.g., Bellisle, 1989). Thus, taste reactivity and lick microstructure analyses seem broadly sensitive to the same manipulations, which is consistent with the idea that they can both be taken as indicators of an animal's hedonic reactions.

Dopamine, divergence, and disconnection

Despite the general agreement between taste reactivity and lick microstructure analysis methods, there is one critical area of divergence pertaining to the effects of dopamine manipulations. Typically, manipulating dopamine function through either pharmacological or other means is reported to have no meaningful effects on hedonic reactions as assessed by taste reactivity (for reviews see, Berridge, 1996, 2000; Berridge & Robinson, 1998). In contrast, treatment with dopamine antagonists has repeatedly been reported to influence the structure of licking behaviours and, in particular, to lower the size of licking clusters in ways that suggest a reduction in hedonic responses (e.g., D'Aquila, 2010; Galistu et al., 2011; Schneider, Davis, Watson, & Smith, 1990; Smith, 2004; Smith & Smith, 2010). This difference is potentially of critical theoretical significance, for the reduction in the lick cluster size (and other effects on licking structure) produced by dopamine antagonists has been interpreted as strong support for Wise's "anhedonia" hypothesis of dopamine function (e.g., Wise, 1982, 2008). In contrast, the

[1] Initial lick rate and lick cluster size are generally influenced in similar ways by most manipulations and thus provide overlapping information about hedonic reactions. While such converging evidence is particularly useful, the fact that observations of the initial rate of drinking are, by definition, restricted to a subset of a total consumption in a session means that this measure has some limits on the generality of its use. Thus the remaining discussion focuses on the size of licking clusters as it has been the primary tool for my research in this area.

absence of effects of dopamine manipulations on taste reactivity is the cornerstone of the idea that dopamine underpins the incentive salience of rewards and not hedonic processes (for reviews see, Berridge, 1996, 2000; Berridge & Robinson, 1998).

Before discussing the implications of such results for the analysis of hedonic processes in rodents, it should be noted that the discrepancy in empirical results might be more apparent than it is real. That is, taste reactivity responses have been reported to be influenced by the administration of dopamine antagonists (Leeb, Parker, & Eikelboom, 1991; Pecina, Berridge, & Parker, 1997), and the decrease in appetitive taste reactivity responses produced by dopamine antagonists was seen most clearly later in testing. This is the same temporal pattern of results as that originally reported for instrumental behaviours that led to the genesis of the anhedonia hypothesis (Wise, Spindler, Dewit, & Gerber, 1978). Turning to lick microstructure analyses, these too show a similar pattern whereby the effects of dopamine antagonists are seen either late in extended test sessions or following a series of test sessions (e.g., D'Aquila, 2010; Smith & Smith, 2010). In addition, taste reactivity methods are typically applied to short samples of behaviour (because scoring longer samples is particularly time consuming and adds little power in and of itself), and lick microstructure analyses often reflect extended or repeated short tests (because the data are more reliable when from larger samples of licking). If the effects of dopamine antagonists on reward processing are most pronounced when testing is extended, then, as typically applied, taste reactivity methods might well underestimate such effects, while lick microstructure methods would naturally cover this delayed period of effect. In short, despite perceptions to the contrary, manipulations of dopamine function do not show a fundamental dissociation between taste reactivity and lick microstructure methods.

Noting these similarities saves the idea that taste reactivity and lick microstructure methods are both sensitive to similar underlying mechanisms from empirical challenge. But it does not resolve the theoretical division between interpretations of dopamine's role in reward processing in which the results of lick analysis techniques have been taken as supporting the idea that dopamine played a critical role in hedonic processes while those from taste reactivity methods have been taken as supporting the incentive salience hypothesis. Ideally, this would be addressed by a body of research aimed at comparing the methods used and resolved by discussions (in print) between researchers using these techniques. Unfortunately, despite the importance of resolving this critical theoretical issue, no such body of research exists, and so it is not possible to offer here any conclusive analysis of the role of dopamine in reward processing that reconciles the theoretical differences noted above. Moreover, this disconnection is not restricted to research on dopamine, and it is quite striking, to me at least, that despite similarities in general research topics and results, there is remarkably little contact between research traditions involving taste reactivity and those analysing the microstructure of licking at any level. Indeed, in a landmark review examining taste reactivity analyses, Berridge (2000) cited not a single lick microstructure study (the same is true of a recent book encompassing similar material, Kringelbach & Berridge, 2010), while few of the major summary papers on lick microstructure analysis mention any work on taste reactivity (e.g., Davis, 1998; Davis & Smith, 1992; Spector et al., 1998). There are, of course, scattered exceptions to this general rule of mutual neglect (e.g., Davis, 1989; McCaughey, 2008) but I am aware of only one pair of papers that explicitly used both types of technique to directly provide converging evidence on a single topic (Myers & Sclafani, 2001a, 2001b). Why there is such a disconnection between what appear to be complementary research traditions is entirely beyond me.

This general disconnection in research traditions acknowledged, there is at least some scope for hope of a resolution (both of the general disconnect between research traditions and the role of dopaminergic mechanisms in reward processing). Berridge's (1996, 2007) suggestion that dopamine is involved in reboosting reward value through prior contact with the reward has been partially adopted by D'Aquila (2010) in analysing the interaction

between D1 and D2 dependent mechanisms in reward evaluation based on lick analysis methods.

Regardless of this theoretical dispute and its potential resolution, detailed analyses of both taste reactivity and lick microstructure have revealed lawful relationships between the manner in which solutions are consumed and the nature of those solutions. Comparisons between species suggest that these lawful relationships reflect the hedonic responses to the solutions being consumed. Moreover, the effects of learning on both taste reactivity and lick microstructure measures suggest that both are sensitive to the current value of the solutions, rather than being some fixed function of their physical properties. For practical reasons, my own research has focused on the analysis of licking microstructure rather than the use of taste reactivity methods, although I will note where my own work has overlapped with taste reactivity studies from other labs.

What is it like to be a rat?

The evidence described above provides, for me, good reasons to believe that analyses of the microstructure of consumption provide insights into hedonic reactions. But others will disagree. Thus, before moving on to consider the application of lick analysis methods in more detail, I would like to digress briefly into philosophy in order to answer one conceptual challenge to this type of research. Nagle's (1974) classic work "What is it like to be a bat?" has been cited in support of the idea that it is simply impossible to know anything about the subjective experiences of a species other than our own. The obvious corollary is that it is invalid to claim that lick analysis (or any other tool) could be providing information about something as subjective as hedonic experiences in nonhuman animals. Indeed, there are some circumstances in which the details of a consummatory response are obviously independent of subjective experience. For example, decerebrate rats can show at least some modulation of both taste reactivity and lick microstructure responses (Grigson, Kaplan, Roitman, Norgren, & Grill, 1997; Grill & Norgren, 1978b). Thus the motor movements being observed can occur in situations where no subjective hedonic experience is likely. But just because the behavioural responses we are interested in can occur in decerebrate animals does not rule out the idea that such responses typically reflect hedonic experiences in animals that retain the competence to have affective reactions.

Now, I happen to disagree with Nagle's (1974) contention that it is impossible to know anything about what it is like to be a bat (or any other species for that matter) and with the wider conclusion he seeks to draw from this idea—that it is not possible to provide an explanation of mental phenomenon in physical/biological terms. But as I am on safer ground talking about psychology than I am debating philosophy, I will restrict myself here to the application of Nagle's views to the particular issue of whether the microstructural analysis of licking can provide any information about hedonic reactions. To this end, I would contend that it does not actually matter whether one believes that it is possible to know anything about the subjective experience of another animal (including other human animals) in the present case. Remember that lick cluster size is, in general, lawfully related to the nature and concentration of the solution being consumed. Thus changes in lick cluster size are normally indicative of changes in the solution. If the solution is held constant, then a manipulation that produces a change in lick cluster size is producing an effect analogous to the effect that would be seen if the solution had actually changed.

Take the example of taste aversion learning. Here, pairing a sweet taste with LiCl results in a change in lick cluster size from large to small, the same change that would have been produced by a change from a sweet to a bitter solution (e.g., Baird et al., 2005; Dwyer, 2009). But as the solution itself remains sweet, the change must be due to some effect on the animal being tested. As the primary driver of lick cluster size is the nature of the solution, then a change in the licking response without a change in the solution licenses the inference that the change is in the *perception* or *evaluation* of the solution.[2] Nothing in this chain of inferences requires an understanding of

any particular subjective states present in the animal being observed, merely that whatever that experience might be is directly analogous to the experience produced by physically changing the solution. As we already know the lawful relationships between the nature of the solution and the details of the consummatory response, then observing changes in lick cluster size (or taste reactivity for that matter) provides information about the factors influencing that response. One might go on to suggest that because humans would find a switch from sweet to bitter an unpleasant experience, then the same change in rats would also be unpleasant, and thus we do know something about their subjective experience. I would support some variety of this claim, but it is not necessary to agree with me on this last assertion to appreciate that an analysis of the microstructure of consummatory behaviour provides information about the factors driving that consumption.

Having, hopefully, put to one side philosophically inspired questions regarding the use of behavioural observations to inform the analysis of hedonic reactions, I would like to return to more empirical concerns. Initially, I review my early studies of licking microstructure in learnt flavour preferences and aversions. In addition to providing valuable insights into the mechanisms underpinning such learning, these studies also reinforce the idea that lick microstructure analyses provide information over and above that which can be gained from the study of consumption alone. This review also includes an examination of extinction, where lick microstructure analyses have revealed some of the limits of hedonic responses as controllers of ongoing behaviour. Next, I consider simultaneous contrast, where the additional information gained from lick microstructure analysis was critical in answering a question that has been open since the effect was first observed almost 40 years ago. Then I turn to studies in which the analysis of lick microstructure provided the first direct evidence that effort influences reward value. Finally, I return to the analysis of animal models of human psychiatric disorders such as depression and schizophrenia. Here, the examination of lick microstructure has informed the search for analogues of the "anhedonia" that is thought to be a key symptom of such disorders.

Cluster size and consumption as dissociable measures in preference and aversion

As discussed above, lick analysis techniques potentially provide their most interesting data in situations where the nature of the solution presented to an animal is held constant, and other manipulations produce changes in the microstructure of the licking behaviours elicited. While the different effects of varying solution concentration on lick cluster size and consumption provide a sound basis for the idea that these are dissociable measures, these manipulations involve changes in the solution being presented. Ideally there would be evidence that this dissociation extends to situations where the nature of the solution is held constant. The analysis of learnt flavour preferences and aversions provides this evidence.

As already noted, conditioned taste aversions based on the administration of LiCl result in a rejection of the flavour paired with LiCl as well as a change in the way that flavour is consumed. However, taste aversions can also be based on a variety of other manipulations, such as pairing with shock or drugs such as amphetamine (e.g., Pelchat et al., 1983; Zalaquett & Parker, 1989). Interestingly, although paring a palatable flavour with such things leads to a reduction in consumption, it does not influence the taste reactivity responses elicited by the flavour itself. This dissociation supports the suggestion that there is a distinction between "true" taste *aversions*, where experience of a flavour in advance of nausea leads to the acquisition of a disgust response and a change in the palatability of the flavour, and mere taste *avoidance* based on the fact that the flavour

[2] This inference requires that other influences on the licking response, such as motor effects, are ruled out. In practice, this means that other microstructural variables (such as the interval between licks within a cluster) should be unaffected by the manipulation that is influencing cluster size. This criterion is met in all the examples I discuss here.

predicts the occurrence of some unpleasant (but not nausea-inducing) event (e.g., Parker, 2003; Pelchat et al., 1983). In addition to supporting this theoretical account, the dissociable effects of LiCl and amphetamine on taste reactivity and consumption provide a direct demonstration that they are not merely different measures of the same single underlying process. If this dissociation extended to the analysis of licking microstructure, it would help confirm that lick microstructure and total consumption are independent.

We (Dwyer, Boakes, & Hayward, 2008) addressed this question by examining a particularly simple conditioned taste aversion design. Rats in the experimental conditions received saccharin followed by either LiCl or amphetamine injections, while control animals received unpaired presentations of saccharin and either LiCl or amphetamine. Relatively weak doses of LiCl and amphetamine were used in order to ensure that there was at least some consumption of the test solution following taste aversion training. All animals were subsequently tested for their response to saccharin. Paring with either LiCl or amphetamine resulted in a reduction in the consumption of saccharin, and this effect was, if anything, larger in the amphetamine-paired condition. Pairing saccharin with LiCl resulted in low lick cluster sizes on test (relative to the unpaired control condition). However, lick cluster sizes for saccharin were unaffected by pairing with amphetamine (relative to controls), despite the fact that consumption was greatly reduced. This is exactly the pattern of results seen when LiCl- and amphetamine-based taste aversions were examined using taste reactivity analyses (Zalaquett & Parker, 1989), reinforcing the idea that taste reactivity and lick microstructure are complementary techniques reflecting similar aspects of behaviour and adding to the previous evidence that taste avoidance can be based on both conditioned nausea (e.g., with LiCl) and conditioned fear (e.g., with amphetamine). Moreover, in an additional experiment, pairing saccharin with wheel-running activity reduced both consumption and lick cluster size, which is similar to the results obtained with LiCl-based aversions. This provided the first direct evidence that activity-based taste aversions are at least partially based on nausea induced by wheel exposure (as suggested by Lett & Grant, 1996) and exemplifies the contribution that examining licking microstructure can make to the analysis of the mechanisms underpinning consumption.

As well as speaking to the mechanisms underpinning taste aversions, the fact that pairing saccharin with amphetamine reduced consumption but not lick cluster size, while pairing saccharin with LiCl reduced both, provides direct evidence that there are manipulations that have dissociable effects on these measures even when the nature of the test solution is held constant. This dissociation suggests that lick microstructure variables and total consumption are at least partially independent. However, the possibility remains that the dissociation observed could simply be due to a difference in sensitivity whereby cluster size was less susceptible to some conditioning effects than was total consumption. To complete the double dissociation and truly demonstrate the independence of the two measures requires a demonstration that some manipulation can influence lick cluster size at the same time as leaving total consumption unaffected. The research on flavour preference learning, to be described next, makes just this point.

Typically, pairing a neutral flavour with a solution such as sucrose results in a preference for that flavour when it is subsequently encountered alone. However, the consumption of flavours previously paired with sucrose is affected by the concentration of sucrose in which that flavour is presented during test (e.g., Harris & Thein, 2005; Sclafani, 2002). For example, having paired one flavour with 30% sucrose (the CS+, where CS is the conditioned stimulus) and a second flavour with 5% (the CS–), Harris and Thein observed that rats consumed more of the CS+ than the CS– when they were both presented in 5% sucrose but that the preference was reversed when they were presented in 30% sucrose. One interpretation of these results is that pairing a flavour with sucrose allows it to activate the representation of sucrose, and this adds to the representation of sucrose that is directly activated by the presence of sucrose itself—that is, presenting a sucrose-paired flavour is analogous to raising the

perceived concentration of sucrose. Remember that the relationship between the concentration of sucrose and the amount consumed is described by an inverted U. Given this, manipulations that increase the perceived concentration of sucrose should act to increase consumption when the baseline concentration is low (i.e., on the ascending limb of the function) but act to decrease consumption when the baseline concentration is high (i.e., on the descending limb of the function), and there should be little effect when the baseline concentration is moderate (i.e., in the middle of the function where consumption is relatively unaffected by concentration). This accords to the pattern of results described by Harris and Thein (2005; see also, Sclafani, 2002). Most importantly for my current concerns, this relationship between preference and baseline concentration means that there will be situations in which overall consumption of flavour preference CS+ and CS– should be relatively similar.

Now, consider that the typical relationship between carbohydrate solution concentration and lick cluster size is positively monotonic. In this case, any manipulation that increased the perceived concentration should result in an increase in cluster size, regardless of the baseline concentration. That is, learning-dependent manipulations of the perceived concentration of a test solution should have dissociable effects on consumption and lick cluster size at moderate to high baseline concentrations. The very first of my published experiments using lick analysis techniques (Dwyer, 2008) assessed this prediction. An initial experiment demonstrated that, in food-deprived rats, consumption of maltodextrin (a hydrolysed starch product containing a mixture of polysaccharides, along with small quantities of mono- and disaccharides) was maximal in a broad peak around 8–16% concentrations and that cluster size increased monotonically as concentration was moved from 2% to 16% (see also, Davis, 1996). The critical second experiment began with a training phase where one initially novel Kool Aid flavour (the CS+) was mixed with 16% maltodextrin, and a second flavour (the CS–) was mixed with 2% maltodextrin. During test, each of the CS+ and CS– flavours was presented when mixed with either 2% or 16% maltodextrin. This judicious choice of parameters should have arranged for there to be no difference in the total consumption of the CS+ and CS– when mixed with 16% maltodextrin, but that the cluster size during consumption of the CS+ would be higher than that of the CS– (while both consumption and cluster size should be larger for the CS+ and CS– when they were mixed with 2% maltodextrin). This was exactly the pattern of results observed. That is, combining a conditioned cue for maltodextrin with the presentation of maltodextrin itself had effects that were analogous to increasing the perceived concentration of maltodextrin: increases in consumption at low, but not high, concentrations, and increases in lick cluster sizes at all concentrations.

Taken alongside the results previously observed with sucrose, these results reinforce the idea that conditioned flavour preferences allow the CS to elicit activity in the representation of the US (unconditioned stimulus) solution. That is, pairing the neutral CS flavour with a US solution results in the formation of an association between the CS flavour and the representation of the US solution, and the subsequent preference for the CS depends (at least partially) on the CS being able to activate this US representation. The fact that larger lick cluster sizes were observed during consumption of the CS+ than during consumption of the CS– also suggests that flavour preference learning produces an increase in the hedonic evaluation of the CS+, which is the converse of the conditioned decrease in hedonic reaction produced by pairing a flavour with LiCl. This is consistent with observations from taste reactivity studies of flavour preference learning (e.g., Forestell & LoLordo, 2003; Myers & Sclafani, 2001b). Moreover, as noted above, a demonstration of changes in lick cluster size in the absence of accompanying changes in consumption is particularly relevant to establishing that these are independent measures. The results obtained when the CS+ and CS– were combined with 16% maltodextrin during test provide precisely this demonstration.

Considered together, my early studies of flavour preference and aversion comprise a double

dissociation between licking microstructure measures and overall consumption, thus demonstrating their independence. This dissociation reinforces the idea that microstructural measures provide information over and above that which can be gleaned from an analysis of consumption alone. More concretely, some taste aversions resulted in changes in lick cluster size that are analogous to moving from a pleasant to an aversive taste, while flavour preference conditioning results in changes that are analogous to moving from a neutral to a palatable taste. Not only does this speak to the mechanisms underpinning learnt preferences and aversions but it is also consistent with the idea that they genuinely do produce changes in hedonic experience: Rats appear, quite literally, to like flavours that are associated with palatable nutrients and dislike flavours associated with illness.

Licking and extinction

Having demonstrated that the analysis of the microstructure of licking is a valuable tool, I now want to consider some of the ways in which I have applied it. Having begun by arguing that affective processes are a key determinant of behaviour, it is appropriate to note that they are not the only factor, and thus we should also consider the limits of hedonic responses in controlling learned preferences and aversions. Indeed, while lick microstructure analyses confirmed that preference and aversion learning can produce changes in hedonic responses, they have also revealed that acquired hedonic responses are not the only factor involved. This is particularly evident when considering the effects of repeatedly presenting the conditioned cue alone following training (extinction). Typically, nonreinforced exposure to a conditioned stimulus during extinction results in a decrease in the size (or probability) of the learnt response, and as extinction is extended, the learnt response will no longer be elicited (Rescorla, 2001). While extinction of flavour learning is certainly possible (e.g., Delamater, 2007), under some circumstances both learnt flavour preferences (e.g., Harris, Shand, Carroll, & Westbrook, 2004) and conditioned taste aversions (e.g., Bevins, Jensen, Hinze, & Besheer, 1999) can be remarkably resistant to extinction treatments. This resistance has been attributed to conditioning producing a permanent change in the palatability of, or hedonic reaction to, the conditioned stimuli. For example, Harris et al. (2004) suggested that the learnt enhancement in the hedonic reaction to a flavour cue paired with sucrose is independent of the flavour–sucrose association, and so even if extinction broke that flavour–sucrose link, learnt positive hedonic reactions would maintain a preference for the flavour when presented alone.

While these ideas seem plausible, the analysis of licking microstructure during extinction suggests that they are not correct. Pairing the sweet taste of fructose with LiCl produced both a reduction in the size of licking clusters and a reduction in consumption, but while consumption never recovered to the level of controls despite extended fructose-alone presentations, the reduction in cluster size was completely removed (Dwyer, 2009). That is, learned changes in consumption proved more resistant to extinction than did learned changes in hedonic responses as indicated by lick microstructure analyses. Similarly, pairing either almond or rose odours with concentrated sucrose produced a persistent preference for the sucrose-paired odour presented alone, but the enhancement in lick cluster size produced by preference training rapidly returned to control levels during extinction (Dwyer, Pincham, Thein, & Harris, 2009). While both conditioned preferences and aversions can produce shifts in hedonic responses, these do not appear to be permanent and thus cannot support the resistance to extinction. Moreover, long-lasting preferences can be conditioned to aversive sour and quinine tastes (Drucker, Ackroff, & Sclafani, 1994) even though such conditioning does not change the taste reactivity response to such tastes from aversive to appetitive patterns (Myers & Sclafani, 2003). While the mechanism(s) underpinning the persistence of conditioned flavour preferences and aversions remain to be confirmed, one possibility is suggested by considering the distinction between preparatory and consummatory responses (e.g., Konorski, 1967): Preparatory behaviours, particularly approach and avoidance, might be relatively

insensitive to extinction because they are somewhat remote from the nonexperience of the reinforcer, while consummatory behaviours, which presumably include hedonic reactions, occur exactly when the expected, but absent, reinforcer would have been experienced.

Simultaneous contrast as a "sensory" process

It has long been known that the response to a given stimulus is not a fixed function of that stimulus but instead is governed by previous and current exposure to other similar stimuli. For example, consider two identical roast chicken dinners. As they are the same, one would suppose that the response to them would also be the same. However, if one was eaten soon after a meal in a restaurant that has three Michelin stars, and the other was eaten soon after a cheap takeaway burger, then contrast processes would result in the latter being perceived as more positive than the former. Although contrast effects occur in a variety of situations and for a variety of reasons, I will concentrate on what Charles Flaherty described as *simultaneous contrast* (for reviews see Flaherty, 1982, 1996). Here, alternating exposure to two different solution concentrations in a single session (e.g., 4% and 32% sucrose) results in changes in their consumption compared to control conditions where only one concentration was presented (e.g., two bottles both containing 4% sucrose, or two bottles both containing 32% sucrose). In this example, consumption of 32% sucrose would typically be higher when it was consumed in alternation with 4% sucrose than when consumed in alternation with 32% sucrose (a positive contrast effect), while consumption of 4% sucrose would typically be lower when it was consumed in alternation with 32% sucrose than when consumed in alternation with 4% sucrose (a negative contrast effect).

Although the earliest reports of simultaneous contrast (e.g., Flaherty & Avdzej, 1974; Flaherty & Largen, 1975) were relatively uncommitted with respect to the mechanisms responsible for the effect, Flaherty soon proposed that it was primarily sensory in nature (Flaherty, 1982). That is, experiencing a high concentration of sucrose alongside a lower concentration would result in it literally tasting sweeter than it would otherwise (with a corresponding reduction in the perceived sweetness of the lower concentration). This was a view he was to maintain over many years (e.g., Flaherty, 1996; Flaherty & Rowan, 1986), and there is a certain degree of circumstantial evidence to support it. For example, there is an exponential function relating the ratio of the two concentrations being consumed to the ratio of licks emitted by rats for each solution (Flaherty & Kaplan, 1979; Flaherty & Sepanak, 1978), and the same function describes the relationship between human judgements of relative sweetness to the ratio between two sucrose concentrations (e.g., Moskowitz, 1970; Stevens, 1969). In addition, a sensory locus for the effect is consistent with the fact that numerous brain or pharmacological manipulations thought to influence "central" processes have no effect on simultaneous contrast (e.g., Flaherty, Becker, & Driscoll, 1982; Flaherty, Lombardi, Kapust, & D'Amato, 1977; Flaherty & Meinrath, 1979; Flaherty, Wrightson, Deptula, & Duston, 1979; Reilly, Bornovalova, & Trifunovic, 2004; Reilly & Pritchard, 1997) despite often having effects on other contrast procedures.

However, all of the evidence cited for the idea that simultaneous contrast relies on sensory mechanisms is indirect. Moreover, there is at least some evidence that might question whether peripheral/sensory mechanisms are sufficient to explain simultaneous contrast. Grigson et al. (1997) note that in some unpublished experiments, neither introducing a water rinse between the to-be-compared stimuli, nor alternating between stimuli that are processed by separate gustatory receptors, completely abolished simultaneous contrast. As both of these manipulations should interfere with purely sensory comparisons between solutions, their lack of effect suggests that more central mechanisms are involved. That said, these data have been reported only in conference form and may not be entirely reliable, as Flaherty (1982) cites a further unpublished study suggesting that water rinses do interfere with contrast.

The vast majority of studies on simultaneous contrast rely on endpoint measures of total consumption such as the total number of licks produced. As noted already, simple consumption

measures can be affected by numerous mechanisms, and so these consumption-only studies would admit multiple explanations. In contrast, lick analysis measures would seem particularly well suited to testing the idea of sensory comparisons. If simultaneous contrast depends on changes in the perceived concentration of the test solutions, then simultaneous contrast should produce changes in lick cluster size that are analogous to actual changes in the solution concentrations. Prior to my own work in this area, there had been only two reports of the effects of simultaneous contrast on lick cluster size (Fagen, Rycek, Ritz, & Shoemaker, 1983; Grigson et al., 1997). Unfortunately, these studies produced conflicting results: Fagen et al. (1983) reported that lick cluster size was increased in positive contrast and decreased in negative contrast, while Grigson et al. (1997) reported contrast effects that were not accompanied by changes in lick cluster size. In order to resolve these inconsistencies in both published and unpublished experiments, we (Dwyer, Lydall, & Hayward, 2011) reexamined the effects of simultaneous contrast treatments on licking microstructure both with and without a change in the quality of the test solutions.

The first two experiments from Dwyer et al. (2011) established that we could produce reliable contrast effects using both the "classic" design (e.g., Flaherty & Largen, 1975) and a novel discrete-trial alternative. In both cases, positive and negative contrast effects in consumption were accompanied (respectively) by increases and decreases in lick cluster size. That is, the changes in lick cluster size were exactly those that would have been produced by increases and decreases in the perceived concentration of sucrose—just as would be expected if simultaneous contrast relied on sensory mechanisms. But what of the effects of preventing sensory comparisons by examining contrast between stimuli that are processed by separate sensory systems? Here we examined sucrose and maltodextrin. Although maltodextrin is quite bland tasting to humans, rats appear to particularly like the "poly" taste produced by maltodextrin. However, they process this taste entirely separately to the sweet taste of sucrose (for reviews see Sclafani, 1987, 2004). The design of the critical experiment (Dwyer et al., 2011, Experiment 3) is shown in Table 1. Consider first the 8 → 8 same condition where rats received the test bottle (containing either 8% sucrose or 8% maltodextrin) immediately after a sample bottle containing the same solution. This corresponds to the control condition as there is no contrast. In the 2 → 8 same condition, rats received the test bottle (containing either 8% sucrose or 8% maltodextrin) immediately after a sample bottle containing the same type of solution, but at a lower concentration (2%). This is a positive contrast condition. Now turn to the 2 → 8 different condition. In this case, the sample and test bottles contained different solution types (either sucrose to maltodextrin or maltodextrin to sucrose). There is still an "upshift" between sample and test bottles (because an 8% solution is more valuable than a 2% solution

Table 1. *Design of cross-solution contrast study*

Condition		*Sample bottle*	*Test bottle*
2 → 8	Same	2% sucrose	8% sucrose
		2% maltodextrin	8% maltodextrin
8 → 8	Same	8% sucrose	8% sucrose
		8% maltodextrin	8% maltodextrin
2 → 8	Different	2% sucrose	8% maltodextrin
		2% maltodextrin	8% sucrose
8 → 8	Different	8% sucrose	8% maltodextrin
		8% maltodextrin	8% sucrose

Note: Dwyer et al., 2011, Experiment 3. The numbers in the condition names refer to the concentration in the sample and test bottles, respectively. For the same conditions, both the sample and test bottles contained the same solution (this was sucrose on half the trials and maltodextrin on the remainder), while for the different conditions the sample and test bottles contained different solutions (for half the trials sucrose was the sample solution and maltodextrin the test solution, with this reversed for the remaining trials). Each test session consisted of six trials where the sample bottle was presented for 60 s followed immediately by the test bottle for 60 s. There was an interval of 120 s between trials. All trials within a test session were from a single condition, and all rats were tested four times in each condition (twice with sucrose as the test solution and twice with maltodextrin as the test solution).

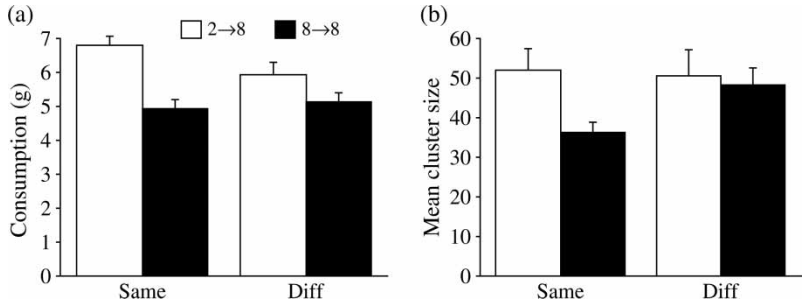

Figure 1. *Reports the data from the test bottle in cross-solution contrast (Dwyer et al., 2011, Experiment 3) as a function of session type (2 → 8 or 8 → 8) and contrast condition (same or different). Rats had access to the sample bottle immediately prior to access to the test bottle. The sample bottle contained a 2% or 8% solution, respectively, in the 2 → 8 and 8 → 8 conditions, and the test bottle held an 8% solution in all conditions. Same refers to conditions where both sample and test bottles contained the same solution (either sucrose or maltodextrin), while diff (different) refers to conditions where sample and test bottles contained different solutions (one sucrose and one maltodextrin). Panel A shows consumption (with* SEM*), and Panel B shows lick cluster size (i.e., mean number of licks per cluster with* SEM*).*

regardless of whether the solution is sucrose or maltodextrin); however, if simultaneous contrast relies on entirely sensory mechanisms, then there should be no effect of this comparison since the two solutions are processed by separate sensory pathways (relative to the 8 → 8 different control to allow for any nonspecific effects of exposure to two different solution types in a single session).

The results are shown in Figure 1 (consumption in Panel A, cluster size in Panel B). Taking first the consumption data in the same conditions, here there is a typical contrast effect in that there was more consumption of 8% sucrose or maltodextrin when it followed 2% sucrose or maltodextrin, respectively, than when it came after an 8% solution. The contrast effect on consumption was reduced, but not entirely abolished, in the different conditions. This residual contrast effect in the different conditions is not consistent with an entirely sensory locus for the simultaneous contrast effect. However, the residual cross-solution contrast effects might simply reflect the fact that 2% solutions would be less satiating than 8% solutions, and so consumption from the test bottle would be more attenuated by general satiety in the 8 → 8 different than in the 2 → 8 different conditions. That said, satiety cannot be the only contribution to contrast effects on consumption because it should have the same effect regardless of whether the comparison was within a solution type (same conditions) or between different solutions types (different conditions). Turning to the cluster size data, it is apparent that in the same conditions, a positive contrast effect in consumption is accompanied by a corresponding increase in cluster size. There was no sign of such an effect on cluster size in the different conditions, despite the residual contrast effect on consumption. That is, within-solution comparisons produced changes in cluster size that were consistent with perceived changes in the concentration of the test solution, but this was not seen for between-solution comparisons.

Taken together, these results are consistent with the idea that simultaneous contrast produces a perceived change in the concentration of the test solutions, exactly as Flaherty (1982) suggested, but that differences in the level of satiety produced by solutions of different concentrations can also contribute to contrast effects on consumption.[3]

But why should comparison across concentrations of a single solution type lead to changes

[3] The idea that positive contrast increases the consumption of 8% sucrose (or 8% maltodextrin) might initially seem inconsistent with the inverted-U relationship between concentration and consumption referred to previously. However, it should be noted that this relationship is typically observed only when exposure is far longer than the 60-s periods used here, and any inconsistency would also require that 8% solutions represented the peak of the inverted U (which they are not always).

in perception? Perhaps the most obvious possibility is habituation or adaptation. In the absence of reinforcement, responses elicited by a particular stimulus will generally reduce across repeated exposures (e.g., Groves & Thompson, 1970; McBurney, 1972; Whitlow, 1975). This general principle is reflected in a reduction in lick cluster sizes over repeated exposures to sucrose. In a previously unreported pilot study, rats were given either three 60-s exposures to 4% sucrose or three 60-s exposures to 32% sucrose (with a 10-s interval between exposures). As can be seen in Table 2, mean lick cluster size reduced over repeated exposure across both solutions, $F(2, 34) = 7.11$, $p = .003$. In addition, lick cluster sizes were lower overall for 4% than for 32% sucrose, $F(1, 17) = 28.61$, $p < .001$, while the reduction over exposure was larger with 32% sucrose than with 4% sucrose, $F(2, 34) = 3.81$, $p = .032$. That is, exposure to both low and high concentrations of sucrose produced an adaptation of the lick cluster response measure, and this adaptation was greater for the higher concentration of sucrose. So, if higher concentrations of a solution produce greater sensory adaptation than do lower concentrations, then the simultaneous contrast effect can be explained as a by-product of adaptation or habituation (with an additional contribution from general satiety). Of course, adaptation and habituation are themselves processes that require explanation, and saying that simultaneous contrast is a product of adaptation is not a complete account. But any mechanism that allows for greater adaptation/habituation to be produced by stronger stimuli and requires a high level of stimulus specificity would be sufficient.

Table 2. *Adaptation and lick cluster size*

Concentration	*Trial 1*	*Trial 2*	*Trial 3*
4% sucrose	33.6 (3.6)	23.3 (1.9)	19.7 (2.5)
32% sucrose	154.9 (31.0)	124.1 (25.3)	59.8 (7.5)

Note: Shows cluster size data (with *SEM*) for 4% and 32% sucrose as a factor of trials within a session. Trials were 60 s in length and were separated by 10 s. All 18 rats were exposed to both 4% and 32% sucrose in separate sessions (with the order of presentation counterbalanced).

In summary, analysing the microstructure of licking confirmed that sensory mechanisms contribute to simultaneous contrast, but that this was not the only contributing factor as general satiety also plays a role. In turn, these sensory mechanisms can be reduced to processes of adaptation or habituation. This is partially, though not completely, consistent with Flaherty's 30-year-old hypothesis that simultaneous contrast was entirely the result of adaptation-based sensory mechanisms. Having looked at an example where lick analysis helped to close an issue that had been open for over 30 years, I would now like to turn to an example of where a serendipitous observation of lick cluster data brought an issue to my attention that was entirely outside my normal interests.

Effort justification in rats: A segue into serendipity and Morgan's canon

"Effort justification" is the term for the phenomenon whereby people place greater value on things that they have worked harder for than things that have "come easy" (even when the outcome of different levels of effort is the same). In one classic demonstration of this effect, Aronson and Mills (1959) found that people who undertook a "severe" initiation as part of joining a reading group rated the group more highly than people who had not done so. Such effort justification can be explained in terms of cognitive dissonance reduction (Festinger, 1957). That is, expending a large amount of effort for an outcome perceived to be of little value places an action (paying a high cost) in a dissonant relationship with an attitude (that the outcome is not valuable). One way to reduce this dissonance is to change the attitude and increase the perceived value of the outcome. Described in this way, dissonance theory seems to require the involvement of somewhat complex cognitive mechanisms (e.g., to support the awareness of the attitude, the relevant actions, and the discrepancy between them). However, the possibility that simpler mechanisms might support dissonance reduction processes has also been considered. Indeed, Festinger coauthored a book (Lawrence & Festinger, 1962) that sought to

explain a variety of animal behaviours in terms of dissonance reduction. Although no precise account of the rat's cognitive processes was offered, they were explicitly described as "nothing very elaborate" (Lawrence & Festinger, 1962, p. 37). Should dissonance truly influence the behaviour of nonverbal animals like the rat, then it either questions whether any complex cognitive mechanisms are required for dissonance reduction or suggests that the cognitive capacity of such animals extends beyond what is typically supposed.

While Lawrence and Festinger (1962) clearly raised this theoretical issue, the empirical evidence they considered was almost completely irrelevant. For example, they found that animals that had to run up a steeply inclined ramp to reach food persisted in running once the food reward was removed for longer than did animals that had run on a shallow incline. This resistance to extinction was attributed to an effort-justification-based increase in the perceived value of the reward supporting greater learning during training. Unfortunately, as was clearly explained by Mackintosh (1974), resistance to extinction is actually a very poor indicator of reward value and instead is directly related to the discriminability of the learning and test situations. As all the experiments reported by Lawrence and Festinger rely on resistance to extinction measures, then there is simply no evidence to support their contention that dissonance-based variations in reward value are involved. Interestingly, Mackintosh's critique focuses of the application of dissonance theory to partial reinforcement effects and so does not actually note any evidence that is directly inconsistent with effort justification effects existing in rats. Nevertheless, the idea has effectively faded from the view.

It was against this background that Emma Lydall made a striking observation on rats she was training to lever press for access to sucrose reward as part of her PhD research (Lydall, 2011). As the requirement of the number of lever presses for sucrose access increased, the size of lick clusters when consuming the sucrose also increased. In other words, it seemed that the higher effort implied by higher response requirements was resulting in more positive hedonic responses to the reward. I was initially sceptical and instead suggested it may simply have been some artefact of the animals becoming familiar with the equipment. Fortunately, Emma was persistent as well as observant and found that there was a reliable effect whereby higher response requirements were associated with higher lick cluster sizes. Moreover, as this relationship was based on a direct assessment of the rats' response to the reinforcer, it was not open to the critique of previous studies of effort justification in rats. That said, there was one obvious confound remaining: Higher response requirements entail longer intervals between instances of reinforcer availability; in turn, longer intervals between exposures to sucrose would provide more opportunity for adaptation to dissipate. That is, the apparent effect of effort on the hedonic response to sucrose reward might reduce to a simple effect of interreinforcer interval on adaptation to sucrose.

In order to test this possibility we (Lydall, Gilmour, & Dwyer, 2010b) arranged an experiment where some rats were required to press a lever either 50 (high effort) or 10 (low effort) times for access to sucrose. Other rats were simply presented with sucrose at the same time as those that were working for the rewards. That is, the yoked animals had the same interval between rewards as the "master" animals, but did not differ in terms of the effort required to obtain them. The results were clear: Differences in interreinforcer interval did have an effect on cluster size in the yoked animals, but this was far smaller than the effect produced by differences in effort. Moreover, a reanalysis of the preliminary studies considering only the first reinforcer gained during a given session (thus eliminating any effect of interreinforcer interval) also revealed that lick cluster sizes were higher following high response requirements than low ones (albeit that this was a relatively small effect). In short, rats genuinely do seem to like sucrose more if they have had to work hard for it than if they have not.

But why should effort influence reward value? The current experiments are perfectly consistent

with the idea that rats found responding on high schedule requirements to be dissonant with a moderate evaluation of the reward and thus reevaluated the reward as of greater value. Dissonance reduction can clearly explain the current results just as it can the effects of undergoing a severe initiation ceremony. But other accounts are also possible. One refers to the idea of "within-trial" contrast whereby the value of a stimulus is evaluated relative to the motivational state experienced immediately prior to its presentation (see, Clement, Feltus, Kaiser, & Zentall, 2000). On the (entirely reasonable) assumption that pressing a lever 50 times induces a more negative state than does pressing the same lever 10 times, then the value of sucrose would receive a greater contrast-dependent boost in its value under high- than under low-effort conditions. Another account refers to the idea of dishabituation, whereby adaptation effects can be disrupted by exposure to an irrelevant stimulus following adaptation (e.g., Whitlow, 1975). Again, assuming that 50 lever presses are more effective at producing dishabituation than 10 lever presses, then the high-effort condition should be less subject to the effects of repeated exposure to the sucrose reinforcer (although this cannot explain any effort effects seen on the first trial of a session).

Although the current experiments allow no conclusive experimentally based separation between these accounts, more general considerations suggest a way forward. In response to the rampant anthropomorphism of the late 19th-century, Lloyd Morgan proposed a general principle for the analysis of the behaviour of nonhuman animals: "In no case may we interpret an action as the outcome of the exercise of a higher psychical faculty, if it can be interpreted as the outcome of one which stands lower in the psychological scale" (Morgan, 1894, p. 53). In light of Lloyd Morgan's canon, the simpler accounts of adaptation or contrast (for which we already have direct evidence) should be preferred. Alternatively, one might remain within the framework implied by Lloyd Morgan's canon if it was accepted that dissonance reduction can be supported by very simple cognitive processes in nonverbal animals (cf. Lawrence & Festinger, 1962). Regardless, our demonstration that rats display effort justification effects does not support the conclusion that they possess complex cognitive capacities commonly held to support cognitive dissonance processes. Before leaving this topic, although Lloyd Morgan's canon explicitly only applies to nonhuman animals (for which we do not have a well-founded prior belief that they possess at least some complex cognitive processes) our studies of rats suggest that dissonance-like effects might (sometimes) be due to very simple processes. In turn, this highlights the possibility that effects attributed to dissonance reduction in humans might also be derived from very simple mechanisms under some circumstances (see also, Egan, Santos, & Bloom, 2007). Whatever the explanation, the important point to remember is that analysing licking microstructure provided the first direct evidence that, for rats, the effort required to acquire a reward influences its value.

"Anhedonia" in animal models of depression and schizophrenia?

I now want to explore some of the benefits that can be gained from using licking analyses as a tool for investigating animal models of psychiatric conditions. Animal models have been one of the key tools in the study of the biological mechanisms underpinning various psychiatric disorders and in the discovery and development of clinically effective treatments (e.g., Lewis & Lieberman, 2000; Lipska & Weinberger, 2000; McArthur & Borsini, 2006; Morris, Cochran, & Pratt, 2005). Despite this past success, there remain significant questions over the fidelity and predictive validity of such models (e.g., Lipska & Weinberger, 2000; McArthur & Borsini, 2006; O'Neil & Moore, 2003). One part of this concern relates to the issue of whether the affective or emotional aspects of disorders can be behaviourally assessed, or even be considered to exist at all, in animals. Take for example, anhedonia (a diminished interest or pleasure in rewarding stimuli), which is a core symptom in depression as well as being a feature of other conditions such as schizophrenia (American Psychiatric Association, 2000).

Several experimental models of depression, in particular chronic stress (e.g., Kompagne et al., 2008; Rygula et al., 2005; Sanchis-Segura, Spanagel, Henn, & Vollmayr, 2005; Willner, 2005), result in reduced sucrose consumption. Using a reduction in the voluntary consumption of sucrose as an indicator of anhedonia has some surface validity: If there is genuinely a reduction in the pleasure associated with such a stimulus then it should not support high levels of consumption. But despite the attractive simplicity of this assay, there are some very serious problems in interpreting changes in sucrose consumption as an index of anhedonia. For example, while a reduction in the voluntary consumption of sucrose is consistent with the presence of an anhedonic state, the same reduction could also be produced by changes in motivational state or in sensory capacity.

A far stronger case could be made for the idea that chronic stress produces a state analogous to anhedonia if this treatment were to produce changes in *how* sucrose was consumed, rather than simply whether it was consumed. That is, if chronic stress in rats really does model depression, then lick cluster sizes (or taste reactivity responses) during the consumption of sucrose should be reduced. To date, there is no published research on this issue, so to illustrate the potential for microstructural analyses to contribute to this area, I describe some pilot data collected with Laura Peronace using the psychosocial cage change stressor (PCCS) model developed during her PhD research (Peronace, 2007). Each day, rats were quasirandomly placed in a new cage alone, in a pair, or in a cage of three rats, while being provided with bedding that had been soiled by separate groups of rats. Although PCCS has not been directly validated as a rodent model of depression, it is quite similar to those used previously in that it entails repeated disruption of the social environment in which the rats live (cf. Rygula et al., 2005), and this disruption is unpredictable (cf. Kompagne et al., 2008). Moreover, PCCS has been shown to produce elevation of corticosterone levels, attenuate bodyweight gain, and have a negative impact on measures of reproductive function (Peronace, 2007).

Thirty-two male hooded Lister rats were acclimatized to drinking 5% sucrose in 20-minute sessions (this occured in drinking cages that have been described previously; Lydall, Gilmour, & Dwyer, 2010a) prior to being separated into a stress and a control group (matched for bodyweight and drinking parameters). The stress group received 16 days of the PCCS treatment described above, while the control group remained housed in stable pairs for the same period and were handled daily. All animals had free access to food and water in their home cages throughout the treatment period. Following treatment, all animals were then given a 20-minute test session with 5% sucrose. Unfortunately, equipment failure meant that licking data were unavailable for nine of the animals. Table 3 shows the data from the remaining rats in terms of the change between the baseline and test periods. Although the PCCS treatment clearly had an effect, as it acted to attenuate weight gain during the stress period, there was no reduction in the amount of sucrose consumed compared to controls. Despite this, the size of licking clusters was significantly lower in the stress than in the control conditions. Although the preliminary nature of this data requires that no firm conclusions be drawn, the reduction in lick cluster size is the pattern that

Table 3. *Effects of social stress in rats on consumption and weight*

	Control (N = 10)	*Stress (N = 13)*	*t(21)*
Weight gain (g)	39.1 (1.6)	30.5 (2.7)	2.56 (0.018)
Consumption change (g)	–2.5 (1.0)	–2.0 (1.1)	0.37 (0.716)
Cluster size change	3.2 (4.9)	–11.9 (2.9)	2.77 (0.012)

Note: Shows mean weight, consumption, and cluster size data (with *SEM*) for the control and stress groups. The data shown represent the change from the prestress baseline to the poststress measurement (post – pre). Also shown are the *t* test results for the comparisons between stress and control (with *p* values). Animals in the stress group received 16 days of chronic social stress (daily changes in number and identity of cage mates and daily provision of soiled bedding from other rats). Control animals remained pair-housed with no disruption to their home-cage environments. There were no differences between the stress and control groups at the prestress baseline, largest $t(21) = 1.1$, $p = .28$.

would be expected if chronic stress were to produce an analogue of anhedonia as experienced in depression.

But what of schizophrenia? This disorder is characterized by a heterogeneous presentation of "positive", "negative", and "cognitive" symptoms (Morris et al., 2005), with anhedonia included amongst the negative symptoms. One common modelling approach has been driven by the fact that glutamatergic dysfunction, particularly N-methyl D-aspartate (NMDA) receptor hypofunction, has been directly linked to schizophrenia. Antagonism of NMDA receptors can produce behavioural and cognitive deficits in healthy volunteers that closely mimic schizophrenia (e.g., Javitt & Zukin, 1991; Lahti, Weiler, Michaelidis, Parwani, & Tamminga, 2001) and also exacerbate psychosis in schizophrenic patients (e.g., Lahti, Koffel, Laporte, & Tamminga, 1995). Repeated administration of low doses of the NMDA antagonist phencyclidine (PCP) is purportedly unrivalled for modelling the nonpsychotic symptoms of schizophrenia (e.g., Morris et al., 2005; Pratt, Winchester, Egerton, Cochran, & Morris, 2008) because it produces a reduction in glucose utilization and blood flow in the prefrontal cortex (hypofrontality; Cochran et al., 2003). This hypofrontality can be exhibited in schizophrenia and correlates with deficits in cognitive function (e.g., Buchsbaum et al., 1990). In addition to such pathological changes, chronic PCP treatment produces a range of behavioural effects in rodents, including cognitive deficits (e.g., Dunn & Killcross, 2006), changes in social behaviour (e.g., Jenkins, Harte, McKibben, Elliott, & Reynolds, 2008; Sams-Dodd, 1999), and sensitization to the effects of amphetamine on locomotor behaviour (e.g., Jentsch, Taylor, & Roth, 1998). Despite this, neither chronic nor acute PCP treatment reduces the hedonic response to the sweet taste of sucrose (Lydall et al., 2010a), questioning whether any analogue of anhedonia is present (see also, Jenkins, Harte, & Reynolds, 2010).

The most obvious conclusion to be drawn from the fact that chronic stress results in the reduction of lick cluster size, while PCP-based treatments do not, is that the PCP treatments that have been examined only produce an incomplete model of schizophrenia, excluding at least anhedonia and possibly the negative symptoms entirely. But before uncritically accepting this idea, it should be noted that there have been suggestions within the clinical literature that anhedonia (as commonly conceived) may not be a true symptom of schizophrenia at all. For example, it is possible to distinguish between consummatory and anticipatory pleasure, the former occurring at the time of an experience and the latter in expectation of events in the future (e.g., Gard, Gard, Kring, & John, 2006; Wolf, 2006). Critically, anticipatory pleasure seems more severely affected in schizophrenia with consummatory pleasure left relatively intact (Gard, Kring, Gard, Horan, & Green, 2007). Delay discounting studies reinforce the idea that schizophrenics have deficits in their ability to anticipate the value of future rewards (Heerey, Bell-Warren, & Gold, 2008; Heerey, Robinson, McMahon, & Gold, 2007). It is thus possible that our failure to find evidence of consummatory anhedonia in rats following PCP treatment may reflect an absence of this symptom in the disorder itself rather than a deficiency in the modelling approach. Foussias and Remington (2010) have also argued against anhedonia as a core symptom of schizophrenia, suggesting instead that the apparent anticipatory pleasure deficit in schizophrenia (Gard et al., 2007) is closely related to dysfunctions in motivation and goal-directed behaviour. They therefore proposed a model in which avolition (lack of motivation) is the core negative symptom of the disorder. Although controversial, these ideas would suggest that a valid model of schizophrenic negative symptomatology should produce motivational rather than hedonic deficits. We are currently examining anticipatory and motivational processes alongside hedonic reactions following PCP treatment and other putative models of schizophrenia—for example, "MAM" (methylazoxymethanol) treatment, Featherstone, Rizos, Nobrega, Kapur, & Fletcher, 2007; or manipulations of the DISC-1 (disrupted-in-schizophrenia-1) gene, Li et al., 2007—which will hopefully help establish exactly what "anhedonia" might mean with respect to schizophrenia and its animal models.

In summary, the results of the chronic stress study confirm the utility of applying lick microstructure

analyses to animal models for psychiatric disorders by demonstrating that this treatment reduces the hedonic reaction to palatable sucrose, suggesting that a genuine analogue of anhedonia is being produced. In contrast, such effects have yet to be observed with rodent-based models for schizophrenia. This may be due to deficiencies in the models themselves, or perhaps it may point to a mischaracterization of the nature of anhedonia as a symptom of schizophrenia. Future studies using microstructural analysis methods should help to determine which of these possibilities is the case.

Conclusions

I began by saying that my reasons for addressing such an unfashionable and seemingly peripheral topic as the microstructural analysis of rodent drinking patterns for this article should be apparent by the time I was finished. The range of (somewhat disparate) studies where licking microstructure provided critical information that would have been unobtainable by the examination of consumption alone clearly demonstrates the capacity for microstructural analyses to contribute to the resolution of otherwise intractable issues. Each of these examples alone would be reason enough to have dwelt on the microstructure of consummatory behaviour. But more generally, the experiments discussed here (and the many other examples of microstructural analysis I have not mentioned) speak to a more general conclusion—*how* an animal does something can be particularly informative about *why* it is doing it. In short, a careful examination of consumption belongs at the centre, rather than the periphery, of comparative psychology (and psychology as a whole)!

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REFERENCES

American Psychiatric Association (2000). *Diagnostic and statistical manual of mental disorders*, (4th ed., text rev.). Washington, DC: Author.

Aronson, E., & Mills, J. (1959). The effect of severity of initiation on liking for a group. *Journal of Abnormal and Social Psychology*, *59,* 177–181.

Baird, J. P., John, S. J. S., & Nguyen, E. A. N. (2005). Temporal and qualitative dynamics of conditioned taste aversion processing: Combined generalization testing and licking microstructure analysis. *Behavioral Neuroscience*, *119,* 983–1003.

Bellisle, F. (1989). Quantifying palatability in humans. *Annals of the New York Academy of Sciences*, *575,* 363–375.

Berridge, K. C. (1996). Food reward: Brain substrates of wanting and liking. *Neuroscience and Biobehavioral Reviews*, *20,* 1–25.

Berridge, K. C. (2000). Measuring hedonic impact in animals and infants: Microstructure of affective taste reactivity patterns. *Neuroscience and Biobehavioral Reviews*, *24,* 173–198.

Berridge, K. C. (2007). The debate over dopamine's role in reward: The case for incentive salience. *Psychopharmacology*, *191,* 391–431.

Berridge, K. C., Flynn, F. W., Schulkin, J., & Grill, H. J. (1984). Sodium depeletion enhances salt palatability in rats. *Behavioral Neuroscience*, *98,* 652–660.

Berridge, K. C., & Robinson, T. E. (1998). What is the role of dopamine in reward: Hedonic impact, reward learning, or incentive salience? *Brain Research Reviews*, *28,* 309–369.

Bevins, R. A., Jensen, H. C., Hinze, T. S., & Besheer, J. (1999). Taste quality and extinction of a conditioned taste aversion in rats. *Animal Learning & Behavior*, *27,* 358–366.

Buchsbaum, M. S., Nuechterlein, K. H., Haier, R. J., Wu, J., Sicotte, N., Hazlett, E., et al. (1990). Glucose metabolic-rate in normals and schizophrenics during the continuous performance test assessed by positron emission tomography. *British Journal of Psychiatry*, *156,* 216–227.

Clement, T. S., Feltus, J. R., Kaiser, D. N., & Zentall, T. R. (2000). "Work ethic" in pigeons: Reward value is directly related to the effort or time required to obtain the reward. *Psychonomic Bulletin & Review*, *7,* 100–106.

Cochran, S. M., Kennedy, M., McKerchar, C. E., Steward, L. J., Pratt, J. A., & Morris, B. J. (2003). Induction of metabolic hypofunction and neurochemical deficits after chronic intermittent exposure to phencyclidine: Differential modulation by antipsychotic drugs. *Neuropsychopharmacology*, *28,* 265–275.

D'Aquila, P. S. (2010). Dopamine on D2-like receptors "reboosts" dopamine D1-like receptor-mediated

behavioural activation in rats licking for sucrose. *Neuropharmacology*, *58,* 1085–1096.

Davis, J. D. (1989). The microstructure of ingestive behavior. *Annals of the New York Academy of Sciences*, *575,* 106–121.

Davis, J. D. (1996). Microstructural analysis of the ingestive behavior of the rat ingesting polycose. *Physiology & Behavior*, *60,* 1557–1563.

Davis, J. D. (1998). A model for the control of ingestion —20 years later. In A. R. Morrison & S. J. Fluharty (Eds.), *Progress in Psychobiology and Physiological Psychology* (Vol. 17, pp. 127–173). San Diego, CA: Academic Press.

Davis, J. D., & Levine, M. W. (1977). Model for control of ingestion. *Psychological Review*, *84,* 379–412.

Davis, J. D., & Smith, G. P. (1992). Analysis of the microstructure of the rhythmic tongue movements of rats ingesting maltose and sucrose solutions. *Behavioral Neuroscience*, *106,* 217–228.

Delamater, A. R. (2007). Extinction of conditioned flavor preferences. *Journal of Experimental Psychology: Animal Behavior Processes*, *33,* 160–171.

Drucker, D. B., Ackroff, K., & Sclafani, A. (1994). Nutrient-conditioned flavor preference and acceptance in rats: Effects of deprivation state and nonreinforcement. *Physiology & Behavior*, *56,* 701–707.

Dunn, M. J., & Killcross, S. (2006). Clozapine but not haloperidol treatment reverses sub-chronic phencyclidine-induced disruption of conditional discrimination performance. *Behavioural Brain Research*, *175,* 271–277.

Dwyer, D. M. (2008). Microstructural analysis of conditioned and unconditioned responses to maltodextrin. *Learning & Behavior*, *36,* 149–158.

Dwyer, D. M. (2009). Microstructural analysis of ingestive behaviour reveals no contribution of palatability to the incomplete extinction of a conditioned taste aversion. *Quarterly Journal of Experimental Psychology*, *62,* 9–17.

Dwyer, D. M., Boakes, R. A., & Hayward, A. J. (2008). Reduced palatability in lithium- and activity-based, but not in amphetamine-based, taste aversion learning. *Behavioral Neuroscience*, *122,* 1051–1060.

Dwyer, D. M., & Burgess, K. V. (2011). Rational accounts of animal behaviour? Lessons from C. Lloyd Morgan's canon. *International Journal of Comparative Psychology*, *24,* 349–364.

Dwyer, D. M., Lydall, E. S., & Hayward, A. J. (2011). Simultaneous contrast: Evidence from licking microstructure and cross-solution comparisons. *Journal of Experimental Psychology: Animal Behavior Processes*, *37,* 200–210.

Dwyer, D. M., Pincham, H. L., Thein, T., & Harris, J. A. (2009). A learned flavor preference persists despite the extinction of conditioned hedonic reactions to the cue flavors. *Learning & Behavior*, *37,* 305–310.

Dwyer, D. M., Starns, J., & Honey, R. C. (2009). "Causal reasoning" in rats: A reappraisal. *Journal of Experimental Psychology: Animal Behavior Processes*, *35,* 578–586.

Egan, L. C., Santos, L. R., & Bloom, P. (2007). The origins of cognitive dissonance: Evidence from children and monkeys. *Psychological Science*, *18,* 978–983.

Fagen, J. W., Rycek, R. F., Ritz, E. G., & Shoemaker, G. E. (1983). Effect of varying sucrose concentration on macrobehavioral aspects of licking in the rat. *Journal of General Psychology*, *109,* 181–187.

Featherstone, R. E., Rizos, Z., Nobrega, J. N., Kapur, S., & Fletcher, P. J. (2007). Gestational methylazoxymethanol acetate treatment impairs select cognitive functions: Parallels to schizophrenia. *Neuropsychopharmacology*, *32,* 483–492.

Festinger, L. (1957). *A theory of cognitive dissonance*. Stanford, CA: Stanford University Press.

Flaherty, C. F. (1982). Incentive contrast—A review of behavioral-changes following shifts in reward. *Animal Learning & Behavior*, *10,* 409–440.

Flaherty, C. F. (1996). *Incentive relativity*. New York, NY: Cambridge University Press.

Flaherty, C. F., & Avdzej, A. (1974). Bidirectional contrast as a function of rate of alternation of two sucrose solutions. *Bulletin of the Psychonomic Society*, *4,* 505–507.

Flaherty, C. F., Becker, H. C., & Driscoll, C. (1982). Conditions under which amobarbital sodium influences contrast in consummatory behavior. *Physiological Psychology*, *10,* 122–128.

Flaherty, C. F., & Kaplan, P. S. (1979). Gustatory contrast in rats. *Chemical Senses & Flavour*, *4,* 63–72.

Flaherty, C. F., & Largen, J. (1975). Within-subjects positive and negative contrast effects in rats. *Journal of Comparative and Physiological Psychology*, *88,* 653–664.

Flaherty, C. F., Lombardi, B. R., Kapust, J., & D'Amato, M. R. (1977). Incentive contrast undiminished by extended testing, imipramine, or chlordiazepoxide. *Pharmacology, Biochemistry, and Behavior*, *7,* 315–322.

Flaherty, C. F., & Meinrath, A. B. (1979). The influence of scopolamine on sucrose intake under absolute and

relative test conditions. *Physiological Psychology*, *7,* 412–418.

Flaherty, C. F., & Rowan, G. A. (1986). Successive, simultaneous, and anticipatory contrast in the consumption of saccharin solutions. *Journal of Experimental Psychology: Animal Behavior Processes*, *12,* 381–393.

Flaherty, C. F., & Sepanak, S. J. (1978). Bidirectional contrast, matching, and power functions obtained in sucrose consumption by rats. *Animal Learning & Behavior*, *6,* 313–319.

Flaherty, C. F., Wrightson, J., Deptula, D., & Duston, C. (1979). Chlordiazepoxide does not influence simultaneous gustatory contrast. *Bulletin of the Psychonomic Society*, *14,* 216–218.

Forestell, C. A., & LoLordo, V. M. (2003). Palatability shifts in taste and flavour preference conditioning. *Quarterly Journal of Experimental Psychology*, *56B,* 140–160.

Foussias, G., & Remington, G. (2010). Negative symptoms in schizophrenia: Avolition and Occam's Razor. *Schizophrenia Bulletin*, *36,* 359–369.

Galistu, A., Modde, C., Pireddu, M. C., Franconi, F., Serra, G., & D'Aquila, P. S. (2011). Clozapine increases reward evaluation but not overall ingestive behaviour in rats licking for sucrose. *Psychopharmacology*, *216,* 411–420.

Gard, D. E., Gard, M. G., Kring, A. M., & John, O. P. (2006). Anticipatory and consummatory components of the experience of pleasure: A scale development study. *Journal of Research in Personality*, *40,* 1086–1102.

Gard, D. E., Kring, A. M., Gard, M. G., Horan, W. P., & Green, M. F. (2007). Anhedonia in schizophrenia: Distinctions between anticipatory and consummatory pleasure. *Schizophrenia Research*, *93,* 253–260.

Gray, R. W., & Cooper, S. J. (1995). Benzodiazepines and palatability—Taste reactivity in normal ingestion. *Physiology & Behavior*, *58,* 853–859.

Grigson, P. S., Kaplan, J. M., Roitman, M. F., Norgren, R., & Grill, H. J. (1997). Reward comparison in chronic decerebrate rats. *American Journal of Physiology: Regulatory Integrative and Comparative Physiology*, *273,* R479–R486.

Grill, H. J., & Norgren, R. (1978a). The taste reactivity test. I. Mimetic responses to gustatory stimuli in neurologically normal rats. *Brain Research*, *143,* 263–279.

Grill, H. J., & Norgren, R. (1978b). The taste reactivity test. II. Mimetic responses to gustatory stimuli in chronic thalamic and chronic decerebrate rats. *Brain Research*, *143,* 281–297.

Groves, P. M., & Thompson, R. F. (1970). Habituation: A dual-process theory. *Psychological Review*, *77,* 419–450.

Haney, M., Comer, S. D., Fischman, M. W., & Foltin, R. W. (1997). Alprazolam increases food intake in humans. *Psychopharmacology*, *132,* 311–314.

Harris, J. A., Shand, F. L., Carroll, L. Q., & Westbrook, R. F. (2004). Persistence of preference for a flavor presented in simultaneous compound with sucrose. *Journal of Experimental Psychology: Animal Behavior Processes*, *30,* 177–189.

Harris, J. A., & Thein, T. (2005). Interactions between conditioned and unconditioned flavor preferences. *Journal of Experimental Psychology: Animal Behavior Processes*, *31,* 407–417.

Heerey, E. A., Bell-Warren, K. R., & Gold, J. M. (2008). Decision-making impairments in the context of intact reward sensitivity in schizophrenia. *Biological Psychiatry*, *64,* 62–69.

Heerey, E. A., Robinson, B. M., McMahon, R. P., & Gold, J. M. (2007). Delay discounting in schizophrenia. *Cognitive Neuropsychiatry*, *12,* 213–221.

Higgs, S., & Cooper, S. J. (1998). Effects of benzodiazepine receptor ligands on the ingestion of sucrose, intralipid, and maltodextrin: An investigation using a microstructural analysis of licking behavior in a brief contact test. *Behavioral Neuroscience*, *112,* 447–457.

Hsiao, S., & Fan, R. J. (1993). Additivity of taste-specific effects of sucrose and quinine—Microstructural analysis of ingestive behavior in rats. *Behavioral Neuroscience*, *107*(2), 317–326.

Javitt, D. C., & Zukin, S. R. (1991). Recent advances in the phencylidine model of schizophrenia. *American Journal of Psychiatry*, *148,* 1301–1308.

Jenkins, T. A., Harte, M. K., McKibben, C. E., Elliott, J. J., & Reynolds, G. P. (2008). Disturbances in social interaction occur along with pathophysiological deficits following sub-chronic phencyclidine administration in the rat. *Behavioural Brain Research*, *194,* 230–235.

Jenkins, T. A., Harte, M. K., & Reynolds, G. P. (2010). Effect of subchronic phencyclidine administration on sucrose preference and hippocampal parvalbumin immunoreactivity in the rat. *Neuroscience Letters*, *471,* 144–147.

Jentsch, J. D., Taylor, J. R., & Roth, R. H. (1998). Subchronic phencyclidine administration increases mesolimbic dopaminergic system responsivity and augments stress- and psychostimulant-induced hyperlocomotion. *Neuropsychopharmacology*, *19,* 105–113.

Kompagne, H., Bardos, G., Szenasi, G., Gacsalyi, I., Harsing, L. G., & Levay, G. (2008). Chronic mild stress generates clear depressive but ambiguous anxiety-like behaviour in rats. *Behavioural Brain Research*, *193,* 311–314.

Konorski, J. (1967). *Integrative activity of the brain.* Chicago, IL: University of Chicago Press.

Kringelbach, M. L., & Berridge, K. C. (2010). M. L. Kringelbach & K. C. Berridge (Eds.), *Pleasures of the brain*. New York, NY: Oxford University Press.

Lahti, A. C., Koffel, B., Laporte, D., & Tamminga, C. A. (1995). Subanesthetic doses of ketamine stimulate psychosis in schizophrenia. *Neuropsychopharmacology*, *13,* 9–19.

Lahti, A. C., Weiler, M. A., Michaelidis, T., Parwani, A., & Tamminga, C. A. (2001). Effects of ketamine in normal and schizophrenic volunteers. *Neuropsychopharmacology*, *25,* 455–467.

Lawrence, D. H., & Festinger, L. (1962). *Deterrents and reinforcement: The psychology of insufficient reward.* Stanford, CA: Stanford University Press.

Leeb, K., Parker, L., & Eikelboom, R. (1991). Effects of pimozide on the hedonic properties of sucrose—Analysis by the taste reactivity test. *Pharmacology, Biochemistry, and Behavior*, *39,* 895–901.

Lett, B. T., & Grant, V. L. (1996). Wheel running induces conditioned taste aversion in rats trained while hungry and thirsty. *Physiology & Behavior*, *59,* 699–702.

Lewis, D. A., & Lieberman, J. A. (2000). Catching up on schizophrenia: Natural history and neurobiology. *Neuron*, *28,* 325–334.

Li, W., Zhou, Y., Jentsch, J. D., Brown, R. A. M., Tian, X., Ehninger, D., et al. (2007). Specific developmental disruption of disrupted-in-schizophrenia-1 function results in schizophrenia-related phenotypes in mice. *Proceedings of the National Academy of Sciences of the United States of America*, *104,* 18280–18285.

Lipska, B. K., & Weinberger, D. R. (2000). To model a psychiatric disorder in animals: Schizophrenia as a reality test. *Neuropsychopharmacology*, *23,* 223–239.

Lydall, E. S. (2011). *Palatability and animal models of schizophrenia* (Unpublished PhD thesis). Cardiff, UK: Cardiff University.

Lydall, E. S., Gilmour, G., & Dwyer, D. M. (2010a). Analysis of licking microstructure provides no evidence for a reduction in reward value following acute or sub-chronic phencyclidine administration. *Psychopharmacology*, *209,* 153–162.

Lydall, E. S., Gilmour, G., & Dwyer, D. M. (2010b). Rats place greater value on rewards produced by high effort: An animal analogue of the "effort justification" effect. *Journal of Experimental Social Psychology*, *46,* 1134–1137.

Mackintosh, N. J. (1974). *The psychology of animal learning.* Oxford, UK: Academic Press.

McArthur, R., & Borsini, F. (2006). Animal models of depression in drug discovery: A historical perspective. *Pharmacology, Biochemistry, and Behavior*, *84,* 436–452.

McBurney, D. H. (1972). Gustatory cross adaptation between sweet-tasting compounds. *Perception & Psychophysics*, *11,* 225–227.

McCaughey, S. A. (2008). The taste of sugars. *Neuroscience and Biobehavioral Reviews*, *32,* 1024–1043.

Morgan, C. L. (1894). *An introduction to comparative psychology*. London, UK: Walter Scott Publishing Co.

Morris, B. J., Cochran, S. M., & Pratt, J. A. (2005). PCP: From pharmacology to modelling schizophrenia. *Current Opinion in Pharmacology*, *5,* 101–106.

Moskowitz, H. R. (1970). Ratio scales of sugar sweetness. *Perception & Psychophysics*, *7*(5), 315–320.

Mundy, M. E., Honey, R. C., Downing, P. E., Wise, R. G., Graham, K. S., & Dwyer, D. M. (2009). Material-independent and material-specific activation in functional MRI after perceptual learning. *Neuroreport*, *20,* 1397–1401.

Mundy, M. E., Honey, R. C., & Dwyer, D. M. (2007). Simultaneous presentation of similar stimuli produces perceptual learning in human picture processing. *Journal of Experimental Psychology: Animal Behavior Processes*, *33,* 124–138.

Myers, K. P., & Sclafani, A. (2001a). Conditioned enhancement of flavor evaluation reinforced by intragastric glucose: I. Intake acceptance and preference analysis. *Physiology & Behavior*, *74,* 481–493.

Myers, K. P., & Sclafani, A. (2001b). Conditioned enhancement of flavor evaluation reinforced by intragastric glucose: II. Taste reactivity analysis. *Physiology & Behavior*, *74,* 495–505.

Myers, K. P., & Sclafani, A. (2003). Conditioned acceptance and preference but not altered taste reactivity responses to bitter and sour flavors paired with intragastric glucose infusion. *Physiology & Behavior*, *78,* 173–183.

Nagle, T. (1974). What is it like to be a bat? *The Philosophical Review*, *83,* 435–450.

O'Neil, M. F., & Moore, N. A. (2003). Animal models of depression: Are there any? *Human Psychopharmacology: Clinical and Experimental*, *18,* 239–254.

Papp, M., Willner, P., & Muscat, R. (1991). An animal model of anhedonia—Attenuation of sucrose consumption and place preference conditioning by chronic unpredictable mild stress. *Psychopharmacology*, *104,* 255–259.

Parker, L. A. (2003). Taste avoidance and taste aversion: Evidence for two different processes. *Learning & Behavior*, *31,* 165–172.

Pecina, S., Berridge, K. C., & Parker, L. A. (1997). Pimozide does not shift palatability: Separation of anhedonia from sensorimotor suppression by taste reactivity. *Pharmacology, Biochemistry, and Behavior*, *58,* 801–811.

Pelchat, M. L., Grill, H. J., Rozin, P., & Jacobs, J. (1983). Quality of acquired responses to tastes by *Rattus norvegicus* depends on type of associated discomfort. *Journal of Comparative Psychology*, *97,* 140–153.

Peronace, L. A. (2007). *A two-part investigation of the biopsychosocial model in male reproductive health* (Unpublished PhD thesis). Cardiff, UK: Cardiff University.

Pratt, J. A., Winchester, C., Egerton, A., Cochran, S. M., & Morris, B. J. (2008). Modelling prefrontal cortex deficits in schizophrenia: Implications for treatment. *British Journal of Pharmacology*, *153,* S465–S470.

Reilly, S., Bornovalova, M., & Trifunovic, R. (2004). Excitotoxic lesions of the gustatory thalamus spare simultaneous contrast effects but eliminate anticipatory negative contrast: Evidence against a memory deficit. *Behavioral Neuroscience*, *118,* 365–376.

Reilly, S., & Pritchard, T. C. (1997). Gustatory thalamus lesions in the rat: 3. Simultaneous contrast and autoshaping. *Physiology & Behavior*, *62,* 1355–1363.

Rescorla, R. A. (2001). Experimental extinction. In R. R. Mowrer & S. B. Klein (Eds.), *Handbook of contemporary learning theories* (pp. 119–154). Mahwah, NJ: Lawrence Erlbaum Associates.

Richter, C. P., & Campbell, K. H. (1940). Taste thresholds and taste preferences of rats for five common sugars. *Journal of Nutrition*, *20,* 31–46.

Rygula, R., Abumaria, N., Flugge, G., Fuchs, E., Ruther, E., & Havemann-Reinecke, U. (2005). Anhedonia and motivational deficits in rats: Impact of chronic social stress. *Behavioural Brain Research*, *162,* 127–134.

Sams-Dodd, F. (1999). Phencyclidine in the social interaction test: An animal model of schizophrenia with face and predictive validity. *Reviews in the Neurosciences*, *10,* 59–90.

Sanchis-Segura, C., Spanagel, R., Henn, F. A., & Vollmayr, B. (2005). Reduced sensitivity to sucrose in rats bred for helplessness: A study using the matching law. *Behavioural Pharmacology*, *16,* 267–270.

Schneider, L. H., Davis, J. D., Watson, C. A., & Smith, G. P. (1990). Similar effect of raclopride and reduced sucrose concentration on the microstructure of sucrose sham feeding. *European Journal of Pharmacology*, *186,* 61–70.

Sclafani, A. (1987). Carbohydrate taste, appetite, and obesity—An overview. *Neuroscience and Biobehavioral Reviews*, *11,* 131–153.

Sclafani, A. (2002). Flavor preferences conditioned by sucrose depend upon training and testing methods: Two-bottle tests revisited. *Physiology & Behavior*, *76* (4–5), 633–644.

Sclafani, A. (2004). The sixth taste? *Appetite*, *43,* 1–3.

Smith, G. P. (2004). Accumbens dopamine mediates the rewarding effect of orosensory stimulation by sucrose. *Appetite*, *43,* 11–13.

Smith, G. P., & Smith, J. C. (2010). The inhibitory potency of SCH 23390 and raclopride on licking for sucrose increases across brief-access tests. *Physiology & Behavior*, *101,* 315–319.

Spector, A. C., Klumpp, P. A., & Kaplan, J. M. (1998). Analytical issues in the evaluation of food deprivation and sucrose concentration effects on the microstructure of licking behavior in the rat. *Behavioral Neuroscience*, *112,* 678–694.

Spector, A. C., & St John, S. J. (1998). Role of taste in the microstructure of quinine ingestion by rats. *American Journal of Physiology: Regulatory, Integrative and Comparative Physiology*, *274,* 1687–1703.

Stevens, S. S. (1969). Sensory scales of taste intensity. *Perception & Psychophysics*, *6,* 302–307.

Whitlow, J. W. J. (1975). Short-term memory in habituation and dishabituation. *Journal of Experimental Psychology: Animal Behavior Processes*, *104,* 189–206.

Willner, P. (2005). Chronic mild stress (CMS) revisited: Consistency and behavioural–neurobiological concordance in the effects of CMS. *Neuropsychobiology*, *52,* 90–110.

Wise, R. A. (1982). Neuroleptics and operant behaviour: The anhedonia hypothesis. *Behavioral and Brain Sciences*, *5,* 39–53.

Wise, R. A. (2008). Dopamine and reward: The anhedonia hypothesis 30 years on. *Neurotoxicity Research*, *14,* 169–183.

Wise, R. A., Spindler, J., Dewit, H., & Gerber, G. J. (1978). Neuroleptic-induced anhedonia in rats: Pimozide blocks reward quality of food. *Science*, *201,* 262–264.

Wolf, D. H. (2006). Anhedonia in schizophrenia. *Current Psychiatry Reports*, *8,* 322–328.

Zalaquett, C. P., & Parker, L. A. (1989). Further evidence that CTAs produced by lithium and amphetamine are qualitatively different. *Learning and Motivation*, *20,* 413–427.