reinforcement on behavior is now supported by much data. The suggestion that "incentive motivational stimuli . . . themselves have a blunted impact" under the influence of neuroleptics is still equivocal, however; this point will be discussed below. In this commentary consideration will also be given to the possibility that two independent types of learning are involved in most operant test situations and that neuroleptics impair only one of them.

To account for the observation of slowed responding in animals treated with neuroleptics prior to the presentation of the first reinforcer (Gray & Wise 1980; Phillips & Fibiger 1979; Tombaugh, Anisman & Tombaugh 1980), Wise suggests that neuroleptics blunt the effects of conditioned incentive stimuli as well as primary incentive stimuli (reinforcers). Consistent with this hypothesis is the frequently reported finding that animals placed on extinction while under the influence of neuroleptics respond less than neuroleptic-treated animals that continue to receive food pellets (Ettenberg, Cinsavich & White 1979; Gray and Wise 1980; Mason, Beninger, Fibiger & Phillips 1980; Phillips & Fibiger 1979; Tombaugh et al. 1980). Thus responding of the former animals is decreased because of blunted effects of the conditioned incentive stimuli present in the test environment whereas the latter animals respond more presumably because of the additive effects of (blunted) conditioned incentive stimuli and (blunted) primary incentive stimuli.

Although the extended anhedonia hypothesis can account for additivity of the effects of dopamine receptor blockade and extinction, other data are not as easily reconciled with this idea. It has been repeatedly shown in many laboratories that the level of unconditioned activity is decreased in a dose-dependent manner by neuroleptics (see reviews by Cook & Kelleher 1963; Costall & Naylor 1979; Ungerstedt 1979). In these experiments unconditioned activity is assessed in test environments that have not had explicit primary incentive stimuli associated with them. Therefore, the incentive stimuli (primary or conditioned) that are responsible for eliciting activity in undrugged animals and that are blunted by neuroleptics are entirely hypothetical. Adherence to the extended anhedonia hypothesis requires the postulation of hypothetical incentive stimuli controlling unconditioned responding.

An alternative to Wise's suggestion is the hypothesis that dopaminergic neurons are involved in two different processes. One is the well-documented role in mediating the behavioral effects of primary incentive stimuli and the other is a role in general behavioral arousal. According to this hypothesis neuroleptics will always produce a dose-dependent decrease in operant behavior, whether it is conditioned or unconditioned; additionally, neuroleptics will blunt the effects of primary incentive stimuli. The latter effect would of course lead eventually to the diminished effectiveness of conditioned as well as primary incentive stimuli because conditioned incentive stimuli lose their effectiveness when associated with blunted primary incentive stimuli.

The advantage of this two-function hypothesis is that it does not require the assumption that hypothetical incentive stimuli control unconditioned behavior. This hypothesis, like the extended anhedonia hypothesis, predicts the additive effects of neuroleptics and extinction as well as the apparent performance decrement observed prior to presentation of the first food pellet in neuroleptic-treated animals trained on schedules of partial reinforcement. Another advantage of this alternative hypothesis is that it predicts the often-observed lack of symmetry in transfer effects (Mason et al. 1980; Tombaugh et al. 1980; Tombaugh, Tombaugh & Anisman 1979), an observation inconsistent with the anhedonia hypothesis. Thus, transfer from extinction to neuroleptic-plus-reinforcement is observed because the effect of dopamine receptor blockade on general behavioral arousal favors the expected cumulative decrease in

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Wise's conclusion that dopaminergic neurons constitute a critical link in the circuitry that subserves the effects of

responding. On the other hand, transfer from neuroleptic-plus-reinforcement to extinction fails because the motor effects of neuroleptics are absent during the extinction session.

The second point of this commentary concerns the possibility that there are two independent types of learning. This general position can be outlined as follows. One type of learning is stimulus-stimulus associative learning and occurs independently of dopamine function. The other is incentive motivational learning and does require dopamine to be established; however, once established, the latter type of learning can influence behavior for a time even when dopamine function is blocked. Following is a consideration of this hypothesis in the light of Wise's article.

There are several lines of evidence supporting the hypothesis that stimulus-stimulus associative learning occurs even when dopamine function is blocked. Here, however, the response being studied is critical to the conclusions drawn. When classical conditioning of autonomic responses supposedly indicative of "fear" (e.g., defecation) is studied, even large doses of neuroleptics fail to impair learning of the response (Hunt 1956; Posluns 1962). Unconditioned responses such as defensive burying (Pinel & Treit 1978) and exploration, which involve elaborate motor sequences, are suppressed by neuroleptics (Ahlenius, Engel & Zoller 1977; Beninger, MacLennan & Pinel 1980) but if a neuroleptic-treated rat receives prod-shock pairings or exposure to a maze and is then tested for associative learning 24 hours later while undrugged, clear evidence of learning is found. Thus, contrary to Wise's conclusion, classical conditioning does occur in neuroleptic-treated animals as evidenced by burying (Beninger, MacLennan & Pinel 1980) and latent learning (Ahlenius et al. 1977) studies. Similarly, neuroleptics block the performance of avoidance responses but not tone-shock associative learning (Beninger, Mason, Phillips & Fibiger 1980).

Intact dopaminergic function is required for incentive motivational learning to occur. This form of learning involves the acquisition by neutral stimuli of the ability to elicit operant responses and could be termed stimulus-response associative learning. Employing appetitive conditioning procedures, Wise & Schwartz (1981) showed that the acquisition of this type of learning is impaired by neuroleptics; corroborative data were provided by a number of investigators (Beninger & Phillips 1980; Davis & Smith 1975). Similarly, in tasks involving aversive stimuli the incentive motivational consequences of negative reinforcement are blocked by neuroleptics (Beninger, Mason, Phillips & Fibiger 1980). However, to infer, as Wise does, that neuroleptic-treated animals have failed to learn stimulus-stimulus associations is incorrect. Thus it has been shown that, although animals receiving tone-food pairings while under the influence of neuroleptics fail to show any preference for the tone when subsequently tested in extinction (indicaing that neuroleptics blocked incentive learning), they do learn the tone-food (stimulus-stimulus) association (Beninger & Phillips 1981). Similarly, animals failing to avoid shock while under the influence of neuroleptics nevertheless do learn the tone-shock association (Beninger, Mason, Phillips & Fibiger 1980b; Posluns 1965).

It is perhaps worth noting that the failure of incentive learning observed in animals with disrupted dopamine function can be overcome by strong external stimuli (cf. Teitelbaum, Schallert, DeRyck, Whishaw & Colani 1980). Thus, although neuroleptic-treated rats fail to avoid, they readily escape when shock is presented (Posluns 1965). Animals treated with intranigral 6-hydroxydopamine successfully learn a brightness discrimination in an electrified Y-maze (Price & Fibiger 1975); this finding indicates that stimulus-stimulus associative learning did occur and appropriate responses could be made when impaired stimulus-response associative learning was compensated by shock. The Ranje and Understedt (1977a; 1977b) underwater swim maze study actually does show similar results, although the authors conclude otherwise (see Beninger, Mason, Phillips & Fibiger 1980 for further discussion of this point). Wise's conclusion that these animals did not learn the discrimination is not supported by the data.

Finally, it should be noted that although incentive conditioning fails to occur if naive animals begin training with dopamine function blocked (as discussed above), conditioned incentive stimuli can maintain responding for a time in neuroleptic-treated animals. This, of course, is the now classic extinction-like effect seen in neuroleptic-treated animals, as discussed extensively by Wise. One of the most elegant demonstrations of this phenomenon was by Franklin and McCoy (1979) who showed that apparently extinguished neuroleptic-treated animals reinitiate responding when presented with a conditioned incentive stimulus. This study, and the many experiments now showing the neuroleptic extinction effect, raise the intriguing possibility that during incentive conditioning dopaminergic neurons mediate a synaptic change that can for a time maintain responding even when dopamine function is severely impaired.