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SMYTHIES' USE OF THE 'SIDMAN SCHEDULE' AS AN ANIMAL TEST FOR HALLUCINOGENIC DRUG EFFECTS

DEAR SIR,

Smythies *et al.* (1969) have described in this *Journal* the use of a 'Sidman' avoidance schedule for the behavioural analysis of psychotropic drugs. Although this method and similar ones may be quite useful from a pharmacological point of view in differentiating between drug effects, the particular technique adopted by Smythies *et al.* raises a number of problems for any behavioural interpretation of such effects. Moreover, we feel it may also make interpretation of the drug action rather suspect.

The technique used by Smythies (see also Smythies *et al.*, 1967) is labelled as a 'Sidman' schedule. This it most certainly is *not*. If it were, there would, of course, be the advantage of a considerable behavioural literature to which reference could be made. 'Sidman avoidance' is, by definition, *non-discriminated* avoidance, i.e. there is no external stimulus to warn the animal of impending shock (Sidman, 1966). A more usual avoidance learning paradigm is *discriminated avoidance*, where an external stimulus (e.g. a light) is presented prior to the onset of shock. The Bovet-Gatti-Smythies' technique combines both these paradigms—and confusion results. At the simplest level, it allows the animal to solve this problem in two distinct ways: (i) in a 'Sidman' way—timing its avoidance response by estimating the time elapsed since the previous response; or (ii) by timing its response from the onset of the light. Thus, either all rats use only one of these strategies, in which case the programming of the other is redundant; or, rats could use either strategy—some using one, some the other. Since disruption of performance by a drug is likely to be related to the particular strategy used (i.e. to what the animal has learned), a correct interpretation of the data will obviously depend on knowledge of which strategy is being used.

The basic Sidman-type *timed* schedule is further disrupted in this situation by the fact that a shock is given every 10 seconds if the rat fails to respond within 30 seconds (and thereby reset the cycle). These shocks stop if the lever is pressed, and the cycle restarts. The light does not come on during the first 10 seconds of the cycle. The problem is that the animal may learn at least two 'tricks': (i) to respond to the lever *in the dark* within 10 seconds of the last response; (ii) to respond to the lever (preferably late on during the period when the *light is on*) to avoid or postpone the shock. These responses may not be compatible—probably explaining, in part, why training takes such a long time (three months or so) and never reaches complete efficiency. It is difficult in these circumstances to envisage what the animal learns about its environment—is the light a 'danger' signal (as in normal discriminated avoidance) in forewarning of shock, or is the dark period a 'safe' period, since the animal is never shocked when the light is off? Furthermore, an animal could be so 'upset' by initial shock that it might adopt a strategy of fairly frequent responding such that the light never comes on, nor are there further shocks. Apart from a high rate of responding, this, at least, is a successful strategy since the animal continually avoids shock. It could, however, contribute to the slow progress of training.

A further problem concerns the *criteria involved in training* animals on this schedule. Although Smythies *et al.* give very few details, we would argue that it is important to specify such criteria. For instance, are the animals trained until they reach some particular level of efficiency, say 75 per cent efficient responding (where an 'efficient' response is one made during the CS light period)? In view of the differential strategies involved in this schedule (as mentioned previously), individual rats are likely to achieve such a criterion level after quite different training periods. Thus previous experience in the situation could well be markedly different between rats. Or, are animals trained until they have achieved some level of stable performance (e.g. where the 'efficient' response rate remains approximately stable over some specified period of, say, two weeks)? This would produce 'stable' animals, but the response rates at which they stabilize may be quite different. They could also stabilize after shorter or longer periods of training.

Smythies *et al.* make the point, of course, that each animal is used as its own control (saline-drug comparison). Nevertheless, because animals may be quite different with respect to efficiency level, stability, length of training, etc., we would not be too impressed

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with the resulting drug profiles, unless they were replicated over a reasonable sample of animals. Certainly, from an interpretational point of view, it would be important to determine, for instance, whether the performance of a 'highly efficient' rat was affected by a particular drug in the same way as that of a relatively inefficient animal (even though both might be stable responders). The converse of this question (same efficiency level, but different stabilities or training periods) is also an important issue.

Shock further complicates any interpretation of the data. The number of shocks an animal receives in a given session will be solely determined by the 'efficiency' of the animal's responding. A drug which disrupts efficient responding may do so in one of two main ways—either by slowing down responding (thereby increasing the number of late responses) or by increasing the response rate (with more premature responding and fewer late responses). In the former case the animal will receive more shocks, and in the latter situation fewer shocks. As the session continues, we are therefore uncertain as to whether the animal's subsequent performance is being controlled by the drug or by the differential number of shocks administered. Indeed, it seems likely that these two factors will interact in a way which the Smythies' procedure cannot hope to describe. Furthermore, those drugs having analgesic effects will affect shock-avoidance performance in rather different ways, depending upon the degree of analgesia induced.

One way to control for some of the drug-shock interaction effects would be to eliminate the shock altogether from the saline/drug test sessions. In the case of reasonably efficient rats, their performance is almost wholly controlled by the threat of shock, rather than by the shock itself; and therefore switching off the shock will make very little difference to their performance (provided they have undergone substantial training with shock). It seems rather surprising that neither Bovet and Gatti nor Smythies *et al.* appear to have utilized this control.

To conclude: (i) it should be possible, with different time periods, etc., to make Smythies' technique more efficient in terms of training time per animal; (ii) to control for drug-shock interactions, non-shock test sessions should be run; (iii) it seems probable that either simple Sidman schedules or basic discriminated avoidance programmes would give similar results, which would then be easier to interpret (in the sense that learning strategies could be more precisely specified).

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Dear Sir,

Our paper was not originally intended to answer any of Lowe and Williams' theoretical interests. Initially, they have intimated that our method may be quite useful from a pharmacological point of view in differentiating between drug effects. This in fact is our only avowed interest, and we ask nothing more from the behavioural baseline which we have chosen. They then suggest that this particular technique would make interpretation of the drug action rather suspect. However, as is evident in the theoretical disposition of our paper, we are not concerned with explaining *why* a drug disrupts behaviour in a particular way or *why* certain sensory or behavioural mechanisms are altered in specific ways by certain compounds. The essential pragmatism of our work is geared to categorizing drugs into different classes based on behavioural effects which they may exhibit in common.

The psychopharmacologist using behaviour as a measure of drug effects is always in a dilemma. If he is interested in what effect the drug has on behaviour he must design a test with only one dependent variable according to the recommendations of Lowe and Williams. On the other hand if he is seeking to develop a test to categorize a new compound into one of a number of possible drug classes he will need to develop a test with several dependent variables so that a complex and informative drug 'profile' may be obtained. It is very difficult to try and combine these objectives, as Lowe and Williams ask us to do. Bovet and Gatti (1963) used this test for the purpose of drug screening, and we have developed it for this sole purpose. Thus our criterion was merely that the test should allow us to say whether a new drug synthesized was likely to be an hallucinogen, or to have an amphetamine-like action, or to be inactive. For this purpose no schedule less complex than a discriminative Sidman avoidance schedule will suffice —even though, as Lowe and Williams rightly point

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