Moreover, many of these findings appear to accord well with a physiological model of diminished behavioural inhibition (Fowles, 1980).

(b) Blackburn is correct in stating that Cleckley's criteria comprise items tapping both personal and social deviance. Nevertheless, Cleckley's criteria are weighted heavily in the direction of personality characteristics, and the extent to which the presence of a few social deviance items results in aetiological heterogeneity remains an empirical question. Although there is admittedly little research on this issue, the results of at least one investigation suggest that Cleckley-defined psychopaths with relatively low rates of social deviance resemble those with higher rates of social deviance on a number of psychometric, clinical, and familial variables (Widom, 1977).

(c) The only evidence that Blackburn cites in support of his third contention is a cluster analysis (Blackburn & Maybury, 1985) in which criminals satisfying Cleckley's criteria fell into two groups, both characterised by a lack of empathy and affection, but differing in their degree of aggression and impulsivity. Nevertheless, heterogeneity at the phenotypic level does not preclude homogeneity at the genotypic level, and there is no evidence that these two clusters are distinguishable on external or biological validating variables. Moreover, as the authors themselves point out (p. 385), the less impulsive and aggressive group was considerably older, raising the possibility that the two clusters were an artefact of the decline of psychopaths' criminal behaviour with age. This 'burn-out' phenomenon has recently received empirical support (Hare *et al*, 1988). Finally, Blackburn points out that because some of Cleckley's criteria overlap with those of several DSM–III personality disorders (e.g. histrionic and narcissistic), Cleckley's construct is probably broader than that of antisocial personality disorder. Although this may be the case, this does not necessarily imply that Cleckley's construct is aetiologically heterogeneous. An equally plausible alternative is that the DSM–III taxonomy of personality disorders has failed to "carve nature at its joints", and that a single diathesis underlies several Axis II syndromes (Lilienfeld *et al*, 1986).

By referring to psychopathic personality and DSM–III antisocial personality disorder as "synonyms" (p. 505), Blackburn may unintentionally give readers the mistaken impression that the former is generally operationalised in terms of socially deviant behaviour. In fact, the most influential criteria sets for psychopathic personality, particularly that of Cleckley, rely primarily on personality characteristics, and are thus largely immune from the criticisms that Blackburn raises. Moreover, Cleckley's criteria yield a syndrome with theoretically meaningful and well replicated psychophysiological correlates. Finally, Blackburn's assertion that individuals identified on the basis of Cleckley's criteria are aetiologically heterogeneous has yet to be demonstrated. Thus, Blackburn's proclamation that the concept of psychopathic personality is "ill-conceived" and "should be discarded" (p. 511) is premature and misleading.

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Positive Symptoms of Schizophrenia

SIR: We are pleased that our paper suggesting an approach to the neuropsychology of schizophrenia (Frith & Done, *Journal*, October 1988, **153**, 437–443) has proved a stimulus sufficient to drive some to put pen to paper. Lo (*Journal*, March 1989, **154**, 414–415) has presented a most interesting account of certain ways in which the dopamine system might break down in psychosis. Since, however, he does not relate these mechanisms directly to symptoms, we feel unable to give useful comments on his account.

As is only to be expected, most writers have readily identified the weakest part of our account, as regards the positive symptoms of schizophrenia. We can explain quite well positive symptoms concerning action (e.g. delusions of control, passivity experiences), but not nearly as well, symptoms concerning communication (delusions of reference, third person

hallucinations, etc.). We are currently preparing an account which specifically addresses these problems of communication. We suggest that recent experiments on early childhood autism (implicating the lack of a 'theory of mind') may be relevant to those positive symptoms of schizophrenia that concern communication. Briefly, analogous to the problems of monitoring their intentions, the patients may also have problems in monitoring the intentions of others.

In addition to concisely pinpointing our weakness, Timney (*Journal*, February 1989, **154,** 268) implies that the symptoms we can explain are among the less common. However, in a survey of 242 patients with a first episode of schizophrenia carried out by our colleagues Eve Johnstone and Fiona Macmillan, delusions of control (34%) and of thought insertion (27%) were almost as frequent as verbal hallucinations to the subject (48%) and third person hallucinations (32%). We would agree with Ring (*Journal*, February 1989, **154,** 268) that depersonalisation and derealisation should be fundamental experiences in schizophrenia which increase with the increasing severity of positive symptoms. In Johnstone & Macmillan's survey they are indeed no more common than the classic positive symptoms (30% and 40% respectively). Maybe these experiences pale into insignificance beside the more extremely positive symptoms and are simply not reported.

Adams (*Journal*, March 1989, **154,** 416–418) describes an elegant hierarchy of monitoring symptoms derived from Hofstadter. She does not, however, indicate how this more complex account might be experimentally tested. She is wrong to suggest that in Frith (1987) questioned the validity of Crow's distinction between type I and type II schizophrenia. Indeed, it was stated that "negative symptoms represent a primary disease process rather than a secondary coping strategy".

In contrast, Klemperer (*Journal*, March 1989, **154,** 415–416) considers that our account is unnecessarily complicated. She suggests that we are wrong in believing that hallucinations are actions rather than percepts. It is by now well established that perception is not a passive process of stimulus reception, but an active one requiring, among other things, the generation and testing of hypotheses. Our account does not deny that hallucinations might be percepts. In fact, we are suggesting that hallucinations occur when the patient misperceives his own actions. Such misperceptions can also give rise to the false beliefs that are the basis of delusions. Klemperer suggests that a much simpler explanation of these phenomena is that they occur because of biochemical and structural abnormalities. We feel that this explanation has little predictive value and, furthermore, represents a form of dualism that is most unsatisfactory. It is implied there are independent physical and mental systems that communicate with one another. As a result, an abnormal signal from the physical system causes an abnormal experience in the mental system. We believe that the mental and physical systems are not independent entities, but different descriptions of the same system. Thus there are physical processes in the brain that exactly correspond with mental processes in the mind. Our approach to the neuropsychology of schizophrenia is an attempt to find ways of matching up these two descriptions.

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Internal monitor defect in schizophrenia

SIR: Frith & Done (*Journal,* October 1988, **153,** 437–443) put forward the theory of 'internal monitor defect' to explain the symptoms of auditory hallucination, delusion of control, and thought insertion in schizophrenia, and rejected the 'defective filter' theory. They postulated that these symptoms arose from the failure of the internal monitor system in registering the self-initiated actions (subvocal speech/act/thoughts) and hence labelling them as originating from an external agent. While I find this quite convincing, I have to point out that such a theory is no superior to the 'defective filter' theory is explaining other positive symptoms in schizophrenia, such as delusional perception, formal thought disorder, and delusion of reference. For delusions of reference at least, I find that the defective filtering out of insignificant and irrelevant external stimuli is a better explanation than the faulty labelling of "switch elicited by irrelevant stimulus". Symptoms like delusional perception and loss of reality testing indicate that there are defective thought processes, involving not only the labelling of ownership, but the actual logical deduction and interpretation of perception and thoughts.

Furthermore, I do not agree with the authors in saying that the monitor system is itself intact in schizophrenia. The classic symptoms of ambitendency (which Bleuler considered as a manifestation of the ambivalence of will), negativism, automatic obedience, and forced grasping seen in catatonic schizophrenia would be explicable only by a defective monitor failing to carry out its usual function of regulating the stimulus intention and the willed intention and deciding on which one to follow first.