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EDITORIAL

A critique of biological psychiatry[1]

Biological psychiatry has established itself as a fully fledged branch of medical science. Originally a subject which developed at the borderline where psychiatry meets the biological sciences (especially endocrinology, neurochemistry and clinical biochemistry), it has now budded-off, taken root, and developed its own journals, theories, practices and proofs: even its own training.

In what follows, I use the term biological psychiatry in this restricted sense, which gives the subject a fairly short history: the great bulk of research has been done since the 1970s. Biological psychiatry can be defined as the attempt to discover biological correlates of psychiatric disorder, with the aim of establishing aetiology, therapy and prognosis. This is a much narrower definition than that of Samuel Guze in his recent article in *Psychological Medicine* (1989), where he uses the term to refer to all that is valid and useful in the realm of psychiatric practice – in effect, if it is not biological then it is not good psychiatry. Guze's article is a highly 'whiggish' account describing the impressive progress of psychiatry up until now, with a confident extrapolation of this trend into the future. While differences in the scope of definition mean that the two essays are not strictly comparable they nevertheless adopt a clearly different standpoint, and I will contrast my views with those of Professor Guze where it helps to clarify them.

Biological psychiatry is not without its critics, and any 'whiggish' history of progress requires a 'revisionist' response which calls this progress into question. It seems timely to take a sceptic's view of things, to play the devil's advocate perhaps. I am not so impressed as Professor Guze by the supposed progress in this field, and I am also concerned that we may be adopting a mistaken way of interpreting biological data in psychiatry. In this editorial I aim to take on board some of the less flattering things said about us by fellow biologists, psychologists and philosophers. This will be seen to lead on to philosophical considerations of the scope and status of biological psychiatry, with the aim of discovering how best to interpret the subject.

Aside from its innate attractions, common to all branches of science, the ultimate promise of biological psychiatry is that it will, sooner or later, lead to improved therapy. My critique of biological psychiatry springs from the simple observation that the most important physical therapeutic advances (roughly speaking, in order, the development of ECT, phenothiazines, tricyclics and lithium) were discovered more or less by accident and before the advent of biological psychiatry as a distinct speciality. In fact the therapeutic advances were themselves the major stimulants of theory and subsequent biological research: for example the discovery of phenothiazines led to the dopaminergic hypothesis of schizophrenia, while tricyclics and MAOIs led to the amine hypothesis of depression. In other words, theory has been highly effective at fitting itself to past experiments, but relatively useless at predicting the results of future experiments.

My perspective is therefore coloured by a sense of disappointment, rather than pride, at the achievements of the subject. And my aim is to explore some of the reasons which lie behind this sluggish rate of progress.

The first problem is the sheer difficulty of doing experiments in biological psychiatry. This is the purist's reductionist critique, and is widespread among basic biological scientists, particularly those who work on single cells. The story runs that because humans are not animals (let alone single cells), we cannot do the experiments we want or need to. For example, it is not desirable to give highly toxic drugs, leave patients untreated to study the natural course of illness, or to kill people in order to reach inaccessible tissues such as the brain: these are the ethical constraints. Then there is the lack

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of accepted animal (or cellular) models for psychiatric illness, making the usual reductionist analogies highly speculative: this is the difference in kind between psychiatric and physical illness.

It is even suggested that humans are too complicated for current biological science to make any sense of, and there does seem to be an important insight in this view. To concentrate for a moment on the dominant area of biological psychiatry, the application of neuroscience, our basic approach to studying brain function dates back to the days when there were only three or four neurotransmitters. The application of neuroscience to psychiatry is often justified by the hope that we might be able to trace the relevant neural pathways and build up a complete picture of their interactions: the goal of mapping behaviour on to structural and functional changes. This did not seem unrealistic a few years ago. Such maps have been constructed for some of the very simple lower animals which have just a 'few' nerves (about a million). For example, Guze refers to work on the sea snail by Kandel & Schwartz (1985), where simple forms of behaviour were correlated with specific synaptic events.

However, the discovery of another fifty or more neurotransmitter (or neuromodulator) substances, combined with the almost unimaginable complexity of interactions between them, must surely have raised the 'neural mapping' goal to a qualitatively different level of difficulty. Each passing year makes neuroscience more complicated and less useful. It now seems impossible even to conceive of mapping and inter-relating the known neuromodulatory pathways, and the validity of this approach must be considered highly dubious.

What this boils down to is that neuroscience is awaiting the development of a new paradigm, a whole new way of theorizing, experimenting and predicting about the brain. One important feature of such a paradigm, aside from its fruitfulness in stimulating scientific activity, must be its radically greater simplicity (Kuhn, 1970).

Another contemporary criticism which deserves mention comes from those cognitive neuropsychologists who are concerned primarily with the building of 'models' of memory, language use, and other aspects of cognition (Shallice, 1989; Marshall, 1989). The 'modellers' criticism dates back at least to the great French physiologist Claude Bernard (1865), who warned that the use of group averages in medicine 'leads, so to speak, necessarily to error... The greatest obstacle to applying calculation to physiological phenomena is still, at bottom, the excessive complexity which prevents their being definite and comparable one with another... Averages must therefore be rejected, because they confuse, while aiming to unify, and distort while aiming to simplify. Averages are applicable only to reducing very slightly varying numerical data about clearly defined and *absolutely simple* cases'. This stricture applies with particular force to psychiatry, where the great diversity of symptoms contained within our psychiatric syndromes (even when tight diagnostic categories are used) means that the 'average schizophrenic' is perhaps even more of a mythical beast than the so-called 'classical cases' of other branches of medicine. As Bernard says, we 'thus have a description that will never be matched in nature'.

The modelling critics are suspicious of the way that group averages are used to build up a description of an average individual. It goes without saying that such an individual (with his 1·9 legs, 2·4 children etc.) does not actually exist; but more crucially, the subtle individual differences – the fine detail, or what Bernard calls the 'biological character of phenomena' – of the actual individual subject is lost in the averages: and it has been found that it is just this fine detail which is essential for effective model building. For example, Bernard warns against the averaging of variable or pulsatile phenomena (such as is done for many measurements of neurotransmitter and hormone function) to produce a single, steady state value. Instead, 'when dealing with complex and variable experiments, we must study their various circumstances, and then present our most perfect experiment as a type, which, however, still stands for true facts'. This is the procedure adopted in several other branches of contemporary neuroscience as well as cognitive neuropsychology: electrophysiology and genetic linkage studies spring to mind. Old though Bernard's observations are, they have not been superseded, and biological psychiatrists are among those who could benefit most from considering them.

These are not the only, or even the most important criticisms. The major problem with the

approach of biological psychiatry is far more fundamental, and has essentially to do with the interpretation of its findings. It is useful to reflect on the implicit rationale or philosophy underlying much biological psychiatric research: the idea that psychiatric illness is caused by alterations in neurotransmitter function. This view is so firmly embedded in most research programmes that it seldom surfaces to consciousness, and we forget that there is in fact no direct evidence to link any specific psychiatric diagnosis with a neurotransmitter change.

This type of thinking is an example of reductionism: the view that the bigger and more complex is explained by the smaller and simpler; and that the smaller and simpler is more fundamental than the bigger and more complex. Science becomes divided into hierarchical descriptive levels (Rose, 1987): at the top are big, complex subjects like anthropology and social psychology, then below this (getting more fundamental) are such disciplines as behavioural psychology and psychiatry, then physiology, chemistry, materials physics, particle physics etc. etc. The thinking resembles peeling off the skins of an onion to find a core of dense and dependable truth.

One point at which this scheme can be challenged is to ask whether these levels are inter-related in such an hierarchical manner or whether instead, as I believe, they are merely different ways of doing science, different paradigms, each with its own provenance and not impinging upon the other levels in any causal fashion. I would argue that the different 'levels' are better regarded as autonomous disciplines. They are different sciences with different purposes, vocabularies and ways of doing things. To solve a problem within any particular discipline (or level) it does not make sense to use the methods of other disciplines. It is just not relevant to the purpose.

What constitutes a descriptive level? It is a network of vocabulary within which we can theorize and explain. The higher complex levels are not explained by the lower simple levels; they are simply different ways of describing the same thing. Explanations and causes only make sense within individual disciplines, they can only be pursued within each level of activity. It follows that to explain psychiatric illness in terms of biochemistry (for example) is not to describe its underlying cause, but to redescribe it (Rose, 1987). The individual human organism is in question. Either we can describe its abnormal behaviour and how to normalize it in terms of a medical vocabulary (equals psychiatry); or we can describe its cerebral structure, its neurochemical make-up, its pattern of heat distribution, its atomic structure, or whatever we want – but we are not talking about the cause of its behaviour, we are not explaining the behaviour. The organism is unchanged and nothing has been said about causes or explanations. We have no vocabulary to link these.

In philosophical terms, I maintain that different sciences (or different paradigms of the same science) are often incommensurable, which means that they cannot be reduced to a common vocabulary. They are not saying the same things in different ways, but are actually about different things. The pragmatist philosophers, whose most influential living spokesman is Richard Rorty (1982), prefer to regard different vocabularies (e.g. different sciences) as being just more-or-less-useful ways of achieving specific ends, and having no necessary inter-relationship. One upshot of this is that the so-called mind–brain problem is dissolved. Notions of free will, for example, are concerned with matters of morality and responsibility, while concepts related to neurotransmitters are concerned with biochemical descriptions of the brain: the two vocabularies are used for quite different purposes and it is a misunderstanding to try to explain one using the other.

In contrast, Guze quotes with approval recent works of philosophy which purport to build links between the brain sciences and philosophical problems related to the mind (e.g. Churchland, 1986). The assumption is that increased knowledge of, say, brain structure and synaptic function, will inform questions such as whether or not we have free will, or whether we are morally responsible for our actions. Along with McGilchrist (1987), I would regard this whole enterprise as a category error based on a confusion between the aims of philosophy and neuroscience; and a peculiar kind of materialism which assumes the priority of objects quite detached from any consideration of our interactions with them (i.e. how we know anything about 'the world' at all).

As McGilchrist says in his review of Churchland's book: '... How the brain works and what it is are not just different questions but different sorts of question'. Similarly, I regard Shakespeare's insights into psychology as different in kind from the insights of behavioural psychologists, and

both of these as different again from the neuroscientific correlates which may be occurring at the same time. *Hamlet* is both simply not and not simply grist to the neuroscientist's mill. Art, philosophy and the sciences are all valid approaches for their different purposes, but they cannot be combined, and we will end up talking what Wittgenstein meant by 'nonsense' if we try to combine them.

So long as biological psychiatrists stay within the descriptive levels, they can make an important contribution to their discipline; it is as rational to study endocrinology or neurochemistry in psychiatric patients as in any other group, and it is likely that there will be interesting differences in the biological mechanisms identified. And it is rational to talk in terms of correlations; but given the complexity of biological systems it would not be at all surprising if correlations, by themselves, turned out to be practically useless for the purposes of learning about aetiology, therapy and prognosis.

Scientific specialities are not God-given, eternal, immutable; they just happen to be the way we divide things up at present with particular purposes in mind so that we can understand and predict them. There is no reason why we should not develop entirely new disciplines which cut across the presently conceived boundaries and link the present descriptive levels. A new discipline would have to put all its components onto a single non-hierarchical plane; none would be more fundamental than another because if we are interested in constructing an explanatory chain of events then all the links must be of equal importance, of an equally fundamental nature. Each science is a system of linked metaphors. To combine sciences we need a new system of metaphors: there is no point in mixing the metaphors.

Kuhn has commented (1970) that when a paradigm is dubious – when inconsistencies begin to build up, when good predictions are not forthcoming; when, in other words, things are not working as well as they used to – then at such times we will expect to see an increase in the 'philosophical' thinking about the subject. This represents the blind gropings for another paradigm. I believe that biological psychiatry is at just this point in its development, and that we should expect and welcome widespread philosophical debate as we search for new and better ways to look at the human brain.

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