It's all done with smoke and mirrors. Or, how to create the illusion of a schizophrenic brain disease

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One of the more intriguing aspects of the "schizophrenia" literature is the discrepancy between the strength of the belief that "schizophrenia is a brain disease" and the availability of direct supporting evidence; even those who hold the belief admit that there is no direct evidence for it (e.g. Chua and McKenna, 1995; McGrath and Emerson, 1999; American Psychiatric Association, 2000). This raises the question of why the belief seems so reasonable and credible. Or, to put it another way, how is the presentation of "schizophrenia as a brain disease" managed in such a way that the absence of direct evidence will not be noticed or not seem important? These questions are important not least because the belief has profound implications for research and intervention. For example, the US National Institute for Mental Health's "next steps for schizophrenia research" focused - in this order- on genetics, neuroimaging, post-mortem studies, developmental neurobiology and clinical trials (Hyman, 2000). In line with this biological emphasis, drugs may be seen as the "natural" and inevitable treatment, with non-physical interventions being seen - to use Tarrier et al.'s (2000) own description of their CBT - as "adjunct" therapies. ("Adjunct" is defined by the Oxford English Dictionary as "a subordinate or incidental thing".)

In this paper, I shall discuss some of the main ways in which the credibility and reasonableness of the belief in schizophrenia as a brain disease is created and maintained; before I do that, however, it is important to note that this belief obviously implies a prior belief in "schizophrenia" and, since "schizophrenia" is consistently presented as a diagnosable illness which causes bizarre behaviour and mental experiences, the scene is set for acceptance of the idea of schizophrenia as brain disorder, albeit one whose precise nature is unknown. This in itself is perhaps a powerful enough mechanism to account for the credibility of the belief, but there are other mechanisms which are worth discussing, for at least two reasons. First, those who want to disseminate alternative models of psychotic behaviour and experience may be dispirited by the sheer persistence of the belief in schizophrenia as a brain disorder and want to reflect on some possible reasons for this persistence; second, those who are open to alternative models may still find themselves pulled between these and the apparent credibility of the belief in schizophrenia as a brain disease. One further point should be emphasised. I'm not suggesting that any of the mechanisms I'll discuss are planned or even consciously used. On the contrary, at least some of them might seem simply like "doing science". I would argue, however, that it is difficult to over-estimate the threat presented by criticisms of the biological basis of schizophrenia and of the idea of schizophrenia itself, and that it would be naive not to expect defensive and anxiety-reducing measures to be (consciously or unconsciously) taken.

Creating the Impression of a brain disorder...
...by assertion

One of the most popular and direct ways of making "schizophrenia" seem like a brain disease is simply to assert that it is, leaving us in the awkward position of questioning the judgement of apparent experts. The assertions may be made almost in passing (e.g. "with a brain disease like schizophrenia. .." - McGrath, 2000) or more directly (e.g. "The recognition that schizophrenia is an organic brain disease. .." - Iverson 1997). Alternatively, and more subtly, it may be agreed that there is no direct evidence but with the clear implication that such evidence is bound to appear. What is notable about many of these assertions is the failure to mention any of the detailed and extensive criticisms which have been made of genetic and biological research on "schizophrenia" (see, e.g. Lidz et al., 1981; Lidz and Blatt, 1983; Rose et al., 1984; Bentall, 1990a; Chua and McKenna, 1995; Ross and Pam, 1995; Boyle, 1990; 2002; Sieben, 1999.) It is difficult to overstate the importance of uncritical assertion in presenting "schizophrenia" as a brain disorder: the claims are often made in secondary sources where readers cannot directly evaluate the data on which the claims rely; and, by failing to mention criticisms, those who make the assertions create an impression of a truth which has never been challenged and is beyond challenge. This silence ; about criticism also deprives readers of information about sources which might offer a different view. Interestingly, when criticisms or references to lack of evidence are made in traditional sources, they are much more likely to be about biological than genetic research. The most plausible reason for this is that the claims that "schizophrenia" is a genetic disorder can almost indefinitely justify the search for direct biological evidence and make its absence seem relatively unimportant. Criticism of genetic research, in conjunction with the lack of direct biological evidence, therefore presents a much more serious threat to the belief in schizophrenia as a brain disease than does criticism of only biological research. In fact the latter, provided it is accompanied by optimism about the future, might actually help to maintain an aura of scientific respectability without compromising the assertion that schizophrenia is a brain disease.

...by creating apparently meaningful associations

In order to support the assertion that schizophrenia is a brain disorder, researchers must provide evidence that a diagnosis of schizophrenia is reliably associated with particular biological events or processes and that these have a direct causal relationship to the behaviours and experiences which are called schizophrenia. In the face of the lack of such evidence, the impression of a causal association between schizophrenia diagnoses and biological processes is created and maintained in four main ways.

The first involves the generation of large amounts of data on possible associations between schizophrenia diagnoses and many different biological variables. Indeed, Bentall (1990b) has remarked that virtually every known brain region or brain chemical has, at one time or another, been claimed to be linked to "schizophrenia". Not only that, but every technological advance in the study of the brain is quickly recruited for the study of "schizophrenics", although it is notable that this research is largely atheoretical (Ross and Pam, 1995). The resulting trawl for associations unguided by theory is greatly facilitated by computer and other technology, which allows measurement of possible associations between schizophrenia diagnoses and large numbers of biochemicals, brain regions, brain functions and, now, chromosomes, in far less time than it would take to develop a constructive theory of why any particular association might be expected or meaningful. Instead, a spurious impression of meaningful associations may be created by the preferential publication of positive results as well as by the inevitable finding of chance associations.

But it is uncomfortable to rely on the mere existence of correlations between schizophrenia diagnoses and biological variables, important though they are in creating an impression of a biological disorder, because critics can quickly point out that the association may not be specific to "schizophrenia" or attributable to other factors. An important way of obscuring this problem or, at least, of avoiding providing data which would highlight it, is through the use of "normal" control groups. The choice of comparison group is obviously important in any research study because of its role in controlling for potentially confounding variables. In the case of "schizophrenia" it is particularly crucial because those given the diagnosis are "deviant" in many ways apart from their "schizophrenic" behaviour. There is, for example, a strong association between diagnoses of schizophrenia and substance abuse (Kosten and Ziedonis, 1997) and between substance abuse and traumatic brain injury (McGuire and Priestley, 2002). Not only that, but following their diagnosis people routinely receive drugs with profound biological and psychological effects (Day and Bentall, 1996). Those diagnosed as schizophrenic may also have had earlier physical interventions for complaints of anxiety and depression, common precursors to a diagnosis of schizophrenia. Andreasen et al.'s (1982) study of ventricular enlargement, for example, reported that 29 per cent of their "relatively young" sample of "schizophrenics" had received ECT. Lader et al. (1984) and Breggin (1990) have also reported a relationship between structural brain abnormalities and the use of minor tranquillizers. It is not surprising that the use of "normal" comparison groups in "schizophrenia" research has been strongly criticised for decades, yet it remains a common practice; more appropriate comparison groups would include those without a diagnosis of schizophrenia but with a history of legal and illegal drug misuse, those who are very socially isolated, those with a diagnosis of severe depression or anxiety, the long-term unemployed, those who had obstetric complications and those with a history of ECT or minor tranquillizer use.

An impression of meaningful association between "schizophrenia" and biological variables is created, third, (and most often in secondary sources) by failing to specify the degree of overlap between "schizophrenic" and comparison groups and by misleadingly presenting group differences in a particular factor, say ventricle-brain ratio or dopamine levels, as if the attribute in question applied to every person with a diagnosis of schizophrenia and not to anyone else (for example, by claiming that "schizophrenics have enlarged ventricles"). The data themselves, however, show a very different picture. Lewis (1990), for example, reviewed 20 studies which compared lateral ventricle-brain ratios in people with a diagnosis of schizophrenia and "normal" controls and found only eight showing significant differences. Similarly, Andreasen et al. (1990) found that only six per cent of participants with a diagnosis of schizophrenia had ventricle-brain ratios more than two standard deviations outside the "normal" control group mean, and this figure might have been reduced still further had more appropriate comparison groups been used, particularly given the relationship between structural brain abnormalities and the use of major and minor tranquillisers. Similarly, the relationship between schizophrenia diagnoses and obstetric complications - one of the factors used to present "schizophrenia" as a "neuro-developmental disorder" - is not only relatively weak, and even weaker for females than males (Hultman et al., 1999), but there is little consistency in the type of complication suggested as associated with "schizophrenia" or evidence that any association is specific to "schizophrenia".

Finally, if schizophrenia is to appear to be a biological disorder, then researchers need to demonstrate not only a reliable association between schizophrenia diagnoses and some biological factors, but also to demonstrate that these factors are direct causal antecedents of "schizophrenia". (I am using causal" here in the sense suggested by Joffe (1996): that in the presence of a particular factor the probability of a certain outcome is increased and we have no reason to believe that both are dependent on a third variable.) This point is crucial. For example, there is a relationship between congenital severe facial disfigurement and social anxiety, but we do not consider social anxiety to be a biological disorder linked to neurodevelopmental processes, because we are aware of the complex and indirect relationships between social anxiety and the organic phenomenon of disfigurement.

There is, however, no evidence of a causal relationship between schizophrenia diagnoses and any genetic or biological event or process. Instead, the weak, variable and difficult to interpret associations between schizophrenia diagnoses and biological variables are subtly and not so subtly transformed to apparently causal relationships through language rather than evidence. For example, associations have been presented as progress in understanding the underlying neurobiology of schizophrenia, as support for neurodevelopmental theories of aetiology of schizophrenia, as part of our knowledge of the biological basis of schizophrenia, as reflecting causes active early in life and as part of the "strong case" for placing the beginnings of pathogenesis in the pre- or peri-natal period (Woods, 1998; Hultman et al., 1999; Jones and Tatrant, 2000; Tsuang et al., 2000; Lobato et al., 2001).

Taken together, these mechanisms create the misleading impression of an evidence base which is constantly being "built up" by the findings of new research, which is far stronger than it actually is and whose interpretation is entirely straightforward.

...by managing non-biological associations

Falloon (2000) has remarked that "paradoxically, the evidence for specific pathophysiological factors in major mental disorders is rather weak, whereas the research findings on stress factors such as family stress and life events, are extremely robust" (p.188). Of course, there is a paradox here only if we believe in schizophrenia as a brain disorder; otherwise, the paradox lies only in the large imbalance in attention paid to the two kinds of factors. Nevertheless, Falloon's remark suggests that if "schizophrenia" is to be convincingly presented as a biological disorder, then the "robust" research findings on its association with non-biological factors must somehow be managed in such a way as to maintain the primacy of biology. I'll briefly consider four ways in which this is achieved. The first is by presenting the association between schizophrenia diagnoses and social factors as consequential rather than antecedent or causal. For example, it is well established that schizophrenia diagnoses are associated with the lowest social classes and most disempowered social groups (Gomme, 1996) Aro (1995), for example, found that the risk of psychiatric hospitalization was usually two to four times higher for the lowest than the highest educated social groups, but that this socio-economic gradient was steepest of all for schizophrenia diagnoses. This well-replicated association, however, has consistently been presented as part of a "downward drift" in which "having schizophrenia" causes people to perform less well in education and employment. The argument, of course, is reasonable: it is difficult to achieve if you are tormented by voices or cannot be bothered to get up in the morning. But the causal argument is also reasonable and has empirical support which is not often mentioned in the literature (Link et al., 1986, Muntaner et al., 1991). Instead, what is notable is the speed and persistence with which the consequential argument was and is advanced, in marked contrast to the tendency of uncritically presenting "association as cause" in the case of biological research. Similarly, there is a well-established association between being readmitted to hospital (relapse) following a diagnosis of schizophrenia, and certain very negative patterns of family interaction, known as high expressed emotion and characterised by overinvolvement and intrusiveness, and negative, hostile and critical comments (Leff and Vaughn, 1981). Following complaints from relatives that they were being blamed for their relative's "schizophrenia", researchers quickly offered reassurance that "high expressed emotion" did not cause "schizophrenia" but only influenced its course. It was also argued that the patients' "schizophrenic" behaviour had elicited these negative behaviours from relatives (Kavanagh, 1992). There , fact, no clear evidence that such behaviour had suddenly appeared in response to a relative's "schizophrenia", although the argument is not implausible (see Patterson et al., 2000); there was, however, evidence from longitudinal studies (Doane et al., 1981; Goldstein, 1987) that patterns of negative interaction very similar to "high expressed emotion" had long preceded a diagnosis of schizophrenia. Again, what is striking is the speed and enthusiasm with which the non-causal and consequential arguments were adopted, in spite of the lack of evidence which favoured them over a causal argument.

These, however, are just two examples of a phenomenon - consequential thinking about social factors - which is so pervasive in the "schizophrenia" literature that it becomes difficult to think in any other way. Virtually all negative aspects of the lives of those diagnosed as schizophrenic, such as substance abuse, unemployment, social isolation, poverty and disrupted relationships, are routinely presented as consequences of "having a serious mental disorder". Of course the assumption is plausible and partly valid - hence its credibility. But it discourages us from asking in any systematic way whether some of these factors could have caused the "mental disorder" or whether, for example, substance abuse and psychosis might not both be ways of reacting to or trying to deal with very aversive life circumstances.

A second way of managing associations between schizophrenia diagnoses and social factors is through the idea of vulnerability; indeed the vulnerability-stress model of "schizophrenia" is now so popular as to have assumed the status of truth. McGlashan and Johannessen (1996), for example, claimed that "biological vulnerability is necessary for the development of psychosis but it is seldom sufficient in itself' (p.204).

The vulnerability-stress hypothesis - widely interpreted as implying biological or genetic vulnerability - has proved to be an extraordinarily useful and effective mechanism for managing the potential threat to biological models of "schizophrenia" presented by the association between the diagnosis and non-biological factors. The usefulness of the hypothesis lies partly in its lack of specificity - since the nature of the claimed vulnerability has never been discovered, anything can count as an instance of it. Its usefulness also lies in its seeming reasonableness (who could deny that biological and psychological or

social factors interact?) and its inclusiveness (it encompasses both the biological and social - surely better than focusing on only one?) while at the same time it firmly maintains the primacy of biology, not least through word order, and potentially de-emphasizes the environment by making it look as if the "stress" part of the vulnerability-stress model consists of ordinary stresses which most of us would cope with, but which overwhelm only "vulnerable" people. We are thus excused from examining too closely either the events themselves or their meaning to the "vulnerable" person.

The association of schizophrenia diagnoses with non-biological factors is managed, thirdly, by what might be called a double standard of presentation, whereby more critical comments are made about and more evidence demanded for, social than biological theories. Warner (2000), for example, under the heading "Poor parenting does not cause schizophrenia" claimed that "there is no evidence, even after decades of research, that family or parenting problems cause schizophrenia" and that "such theories have seldom been adequately tested" (p.9, 10). By contrast, his discussion of biological research, under the heading of "The brain in schizophrenia" was entirely uncritical and ended with the conclusion (echoing the vulnerability-stress model) that "These findings suggest that in schizophrenia there is a deficit in the regulation of brain activity by interneurones so the brain over-reacts to the many signals in the environment" (p.l0) .

A final way of de-emphasizing the links between schizophrenia diagnoses and environmental factors is simply to convert the environment to biology. McGorry (2000) has provided a striking example of this process in his argument that "[low] vitamin D also provides a possible explanation for the increased risk of schizophrenia in second generation dark-skinned migrants who have moved to live in cooler climates (their skin is less efficient at producing vitamin D)" (61; parenthesis in original). There is no mention here that these "cooler climates" are peopled with light-skinned people with a long history of subjugating those with darker skins and that "dark-skinned migrants" are often exposed, from birth in the case of the second generation, to high levels of racism and social disadvantage.

This, however, is only one example of a much more general phenomenon. Littlewood and Lipsedge's (1982; 1997) analysis of ethnicity and psychosis provided an important lead in developing alternatives to biological accounts of "schizophrenia"; they acknowledge, however, that little of substance has been achieved (Littlewood and Lipsedge, 1997). One reason for this is that "race" has simply been converted back to a biological variable. A search of recent literature on "race", ethnicity and "schizophrenia" produced very few studies in which "race" was analysed as a social construct which mediates psychological experience. Instead, much of the research focused on biology and genetics (e.g. "race" differences in neuroleptic response; genetic linkage in southern African families). And the trend is likely to continue if the research direction favoured by Lewine and Coudle (1999) is followed. They argued that "despite [National Institute of Mental Health] efforts to facilitate the study of women and minorities in schizophrenia research, there is a significant lack of information about race differences in brain morphology and neuropsychologi4ral functioning in schizophrenia".

...by privileging biology

All of the factors discussed so far can be seen as ways of privileging biology in relation to "schizophrenia", but two further ways of achieving this are worth mentioning. The first is through the manipulation of lists: wherever there is a list, for example of "risk factors" or research directions, then biology will almost always predominate, whether numerically or in word order. For example, in McGlashan and Johannessen's (1996) list of around 55 supposed "vulnerability markers" for schizophrenia, only three could be said to be definitely social or interpersonal. And, as I noted earlier, the NIMH "next steps in schizophrenia research" focused on genetics, neuroimaging, post-mortem studies, developmental neurobiology and clinical trials.

A second way of privileging biology is through the frequent use of medical (and only medical) analogies. For example, "schizophrenia" has often been compared to diabetes, as "a syndrome whose causes are un-known" while discussion of the prevention of "schizophrenia" frequently includes comparisons with the prevention of infectious diseases or lung cancer. The importance of these medical analogies lies not only in their power to reinforce the idea of "schizophrenia" as a companion biological disorder, but in their borrowing of the credibility and success of medicine to create an impression of hope and optimism which would be very difficult to achieve simply through the biological literature on "schizophrenia" itself.

How can these mechanisms be challenged or at least balanced? Two obvious ways are, first, to question biological research much more closely (e.g. What were the control groups? How much did their results overlap with the "schizophrenic" group? What other factors might account for the results?) and, second to produce lists in which social and interpersonal factors predominate or are mentioned first. But we can also take every opportunity to insert causal thinking into discussions of social factors, to point out that behaviour and experience can cause biology as well as the other way round (Harrop et al., 1996) and to highlight the many different ways in which associations between brain and behaviour might be interpreted. For example, obstetric complications may be (weakly) associated with a diagnosis of schizophrenia not because of their effects on people's brains but because of their effects on their lives, through their links with social disadvantage, educational difficulties, possible bullying and social rejection, lower employment prospects and so on. In other words, we need to foster accounts of psychosis which do not privilege biology but which do greater justice to the research data and to the reality of people's lives.

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