The following exchange of views between Robert Harris and George W. Brown was originally published in Medical Sociology News Volumes 5 and 6, between January 1978 and January 1979. The four parts are published here, together, for the first time.

WHY I AM BROWNED OF WITH QUANTITATIVE METHODOLOGY

A comment on Brown, Birley, Wing (1972) and Vaughn and Leff (1976)

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Recently, two papers have appeared in the British Journal of Psychiatry which together claim to have established a new fact to which psychiatrists are enjoined to pay regard when making discharge decisions (Brown, Birley and Wing, 1972; Vaughn and Leff, 1976). The first of these studies purports to have found a causal relationship between the nature of the affective environment of discharged schizophrenics and their readmission to hospital. The second is a replicative study which substantiates what the authors call the unequivocal nature of the results of the first. On the face of it the results do indeed appear quite convincing. However, more detailed examination reveals that what the authors have demonstrated is a common-sense observation which I suspect has always been recognised by psychiatrists' and others concerned with the discharge and readmission of psychiatric patients.

Of course if researchers wish to spend their time scientising common sense judgements that is entirely their own affair. It becomes a matter of debate when their findings are presented as scientific facts which should be taken into account by practitioners when deciding upon disposal options. Furthermore, when a common-sense judgement masquerades as a scientific fact, it gains a spurious authority which cannot be gainsaid by available put-downs such as 'that's just an opinion', or 'that is generally true, but it doesn't apply in this case' and so forth. My personal commitment in addressing the following comments towards these studies is that I regard the form of scientism which they represent to be a positive hindrance to our understanding of schizophrenics and their families. Vaughn and Leff's study stands or falls on the merits of the earlier research carried out by Brown et al. My remarks, therefore, are directed entirely to this. The principal findings reported by Brown et al. is shown by the following 2 x 2 contingency table. I address to this table two questions: 1. How was it produced? 2. What does it mean?

Relationship of relatives' emotion to relapse in the 9 months after discharge

Expressed emotion of relatives	No Relapse	Relapse	% Relapse
High	19	26	58
Low	47	9	16

P<.001

(From Brown et al. 1972, p. 246)

The table shows a statistically significant direct relationship between the level of emotion expressed by members of a household group living with a discharged schizophrenic patient towards that patient and the patient's relapse. The higher the expressed emotion, the more likely is the patient to relapse. Relatives' expressed emotion is an overall index arrived at by combining three indicators of emotional response towards the discharged patient. Assessment of the relative strength of these indicators were made by analysing data produced in a lengthy family interview. The three indicators are: critical comments, hostility and the emotional over-involvement of the relative. Relatives were categorised as expressing high or low emotion according as to whether or not they made seven or more critical comments about the ex-patient, expressed or did not express hostility about him or showed marked or low emotional over-involvement with him.

The most significant of these indicators in demonstrating high expressed emotion is the first, i.e. seven or more critical comments. This accounted for 35 of the relatives in that category. Hostility accounted for two and emotional over-involvement for five. The three remaining numbers of the high EE group were added following a joint interview, i.e. an interview where the patient was present. The criteria for allocating relatives into the high or low EE groups following this interview were different from those used following the joint interview. In particular, two or more critical comments were sufficient for allocation into the high EE group. This criterion added one relative, marked over-involvement added two, and hostility none. Hence 37 out of 45 patients lived with relatives who were assessed as being critical or hostile towards them.

Thus half of the answer to my first question, 'how was the association produced?', is as follows: Certain discharged patients and their relatives were interviewed. What they said at the interview and how they said it was judged to show hostility, over-involvement and criticism. If there was judged to be ***any*** hostility or ***marked*** over-involvement or ***seven or more*** critical comments the relatives were said to be expressing high emotion. To complete the answer to the first question we need to know how relapse was measured. Brown simply says that relapse was judged using all available information. Presumably this includes interview material and in the case of readmitted patients, hospital records. Twenty nine out of thirty five relapsed patients were readmitted. So much for how the table was produced. Let us now consider what it means. First of all, what do Brown and his colleagues think it means? They are in no doubt:

> *"…a high degree of expressed emotion is an index of characteristics **in** the relatives which are likely to **cause** a florid relapse of symptoms, independently of other factors such as length of history, type of symptomatology or severity of previous behaviour disturbance" (Brown et al. p 242. My emphasis)*

Brown and his colleagues believe they have discovered a causal relationship between EE and relapse. Furthermore, EE is a measurement of characteristics in the relatives. It is thus a psychogenic variable. Later on in the same paper, however, Brown and his colleagues speak of the level of EE as 'an enduring potential characteristic of the relative's behaviour towards the patient' (p 246). I am not at all sure that I know what they mean by an enduring potential characteristic of behaviour. Perhaps they mean that EE in an index of personality characteristics which dispose or cause the relative to behave in a particular way towards the patient. This interpretation accords well with the first statement quoted above. However, my interpretation does imply that the second statement is elliptic and that this ellipticism obscures the model of relatives' behaviour on which Brown's study appears to be based. The implicit model seems to be this: Relatives have inside then certain characteristics. When exposed to a certain stimulus - the patient - these characteristics are activated. Once

activated they cause the relative to behave in a particular way which, in turn, causes the patient to relapse. Diagrammatically Brown's model of relatives' behaviour seems to be this:

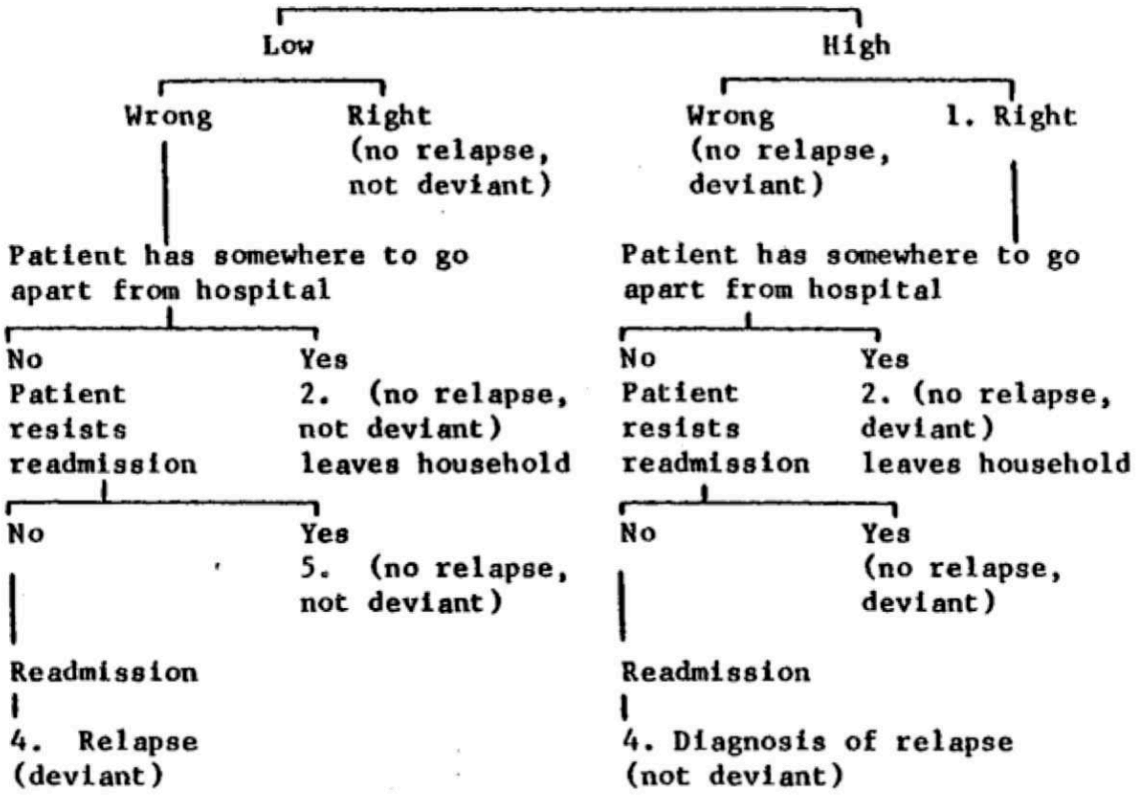
As has been frequently pointed out explanations like this deny the volitional aspects of human action and for that reason alone must be rejected as satisfactory explanations of that action. There in no denying the fact, however, that the strong statistically verified relationship shown in the table above remains. If Brown's explanation for the relationship, i.e. one based on a mechanistic view of man, is inadequate, then what might be a more adequate explanation?

An alternative way of accounting for the association between EE and relapse is given in diagram 1. This diagram is not to be seen as a flow chart if by that is meant a sequential series of stages. Rather it is an attempt to render intelligible, a process (schizophrenic relapse) by seeing it as the praxis of relatives and patients and others with whom they come into contact, particularly psychiatrists. This way of seeing is, of course, that recommended by Laing and Esterson (1971).

As Brown himself states, 'expressed emotion has a highly negative connotation' (p 253). In other words, when Brown assesses a high level of expressed emotion he is saying, in effect, that the relatives don't like the patient very much. They are critical of him and hostile towards him.

Brown relies upon his own and his interviewers' common-sense ability to recognise hostile and critical comments when they hear them, to see hostility in gestures and hear it in tones of voice. There is nothing exceptional in this ability. We all have it and we use it. However, neither we in our everyday lives nor Brown and his interviewers in their research activity are invariably right in judging that A dislikes B. The first point to notice then is that when Brown assesses high or low EE he may be wrong or right. That is to say, relatives may, in fact, like the patient when Brown assesses that they don't and vice-versa. I assume that within the families studied by Brown there is a state of affairs recognised by them by which Brown may be said to be right or wrong. It seems to me to be silly to deny that our common-sense judgements of relationships may be incorrect. Insofar as Brown believes his methodology to be valid (which he does) he is denying that he may be wrong. In a separate paper (Rutter and Brown, 1966) devoted to the issue of the reliability and validity of the research instruments used in the study discussed here, Brown and his co-author describe in great detail the procedures followed to ensure validity and reliability. For him, therefore, expressed emotion (dislike) exists entirely ***in and through the methodical practices by which it is recognised and described*** (Garfinkel, 1967).

Brown Assesses Expressed Emotion of Relatives



1. He is more likely to be right since we can recognise dislike when we see it. Note that intuitive judgements and feelings disallowed.
2. No reported cases in Brown's series.
3. Brown's paradox: High E.E.: Severe Disturbance, No relapse: Patient follows medical regimen.
4. Crucial assumption that a diagnosis of relapse is more likely following re-admission.
5. No relapse but not because low E.E. Such cases though assumed to support Brown's theory.

Thus, I accept that for Brown, relatives' dislike of patients exists in and through his methods of recognising and describing it. I do not accept that these methods constitute the totality of the affective components of the households he studies. In short, Brown may be wrong. He is, though, I submit, more likely to be right than wrong. This is ***not*** because of the excellence of his research instruments as Brown believes, but simply because both he, his co-authors and his 'hired hand' researchers are ordinary, competent members of society who can, as a matter of fact, recognise, a criticism when they hear it. But while Brown relies on common-sense to recognise a critical comment, at other points in his research design common-sense is thrown out of the window as being unscientific. For example, in treating critical comments as ordinal data he assumes that one critical comment carries as much censure as any others. Again, he denies the ambiguity of family relationships and characterises them as ***either*** hostile or ***not*** hostile. His interviewers were counselled against allowing their 'feelings' to interfere with their judgements of the affective aspects of the households they visited. All these offend common-sense. Thus, although Brown is likely to be right in his assessment of dislike as long as he relies on common-sense, insofar as he rejects common-sense, he is

more likely to be wrong. ***The more scientific he is, the more wrong he is likely to be.*** For reasons which will become plain as we proceed, I believe that the strong association between EE and relapse is due entirely to the greater likelihood that Brown is right when he assesses that a patient is not liked very much.

Let us assume that Brown ***is*** right when he assesses that a patient is not liked very much. Further, let's try and see the family situation from the point of view of the patient by imaginatively taking his position. If we are in a situation where we are not liked we may reach a point where we try to escape from it. Escape may involve physical removal from the situation or social withdrawal. The second is effective only as far as the others allow you to withdraw. Both forms of withdrawal require a level of command over scarce resources, namely accommodation, either a separate and private room in the household home, or accommodation outside the household home. We need to ask, therefore: Has the disliked patient somewhere to go apart from hospital? If the answer is 'Yes' the patient leaves the situation in either of the above senses. If the answer is 'No', we than have to ask: Does the patient resist readmission? If the answer is 'Yes' we have then to ask two questions. Firstly, what resources are available to the patient to resist readmission and remain sane in the household? Whatever the available resources, among them is likely to be one of the major tranquillisers routinely prescribed for discharged schizophrenics. Secondly, what is likely to occur in a household in which the patient is disliked and refuses to leave? My common-sense hunch would be that the level of conflict in the household is likely to increase. We would not be surprised, therefore, to find some cases of high dislike, relatives' reports of severe disturbance in the patient, the patient following the medical regimen (he takes tranquillisers because he himself defines his need for them) and no relapse (if he relapses the patient knows he will be readmitted). And this is precisely what we do find among Brown's data. Since Brown's implicit theory effectively denies human volition he regards this state of affairs as a paradox. We now see it not as paradoxical at all but as a state of affairs brought about by specifically human action.

The disliked patient, however, may not resist readmission and may indeed, actively seek it as a means of egress from the household. Should he be readmitted I hypothesise that ***either*** he will be assumed to have relapsed, ***or*** any assessment of his behaviour will be biased towards a finding of relapse once the patient has been readmitted. I regard this hypothesis as plausible on three counts: Firstly, relapse was assessed by Brown using all available information. Presumably this includes relatives' reports of the patient's behaviour. Relatives who dislike the patient are more likely to define the patient's behaviour in a way that renders it perceivable as symptomatic ***as a means of extruding him*** from the household. Secondly, the patent himself may mimic psychiatric symptoms as a means of escaping from the household (Braginsky and Braginsky, 1969). The belief of medical and nursing personnel that psychiatric patients can ***act insane*** is documented in the literature and the simulation of psychiatric symptoms is a recognised syndrome in clinical psychiatry: the Ganser syndrome. Thirdly, other research has shown psychiatric diagnosis to be manifestly influenced by social context. The patient's presence in the hospital ***requires*** psychiatric legitimation. A finding of relapse is, therefore, likely.

An important feature of this rendition of Brown's significant association is that the problem of cases which don't fit Brown's hypothesis disappears. Cases which deviate from Brown's hypothesis are cases either where his assessment of dislike is wrong or the patient has somewhere to go or the patient resists readmission.

It might of course, be argued that even allowing the plausibility of my rendition of Brown's research I have not disproved his hypothesis. This is perfectly true, but do we really need research of this kind to convince us that discharged patients living with people who don't like

them are more likely to be readmitted than those who live in a more sympathetic emotional atmosphere? If we do, we seem in great danger of substituting a mystifying welter of reified statistics for our common sense

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Science and Common Sense: A Reply

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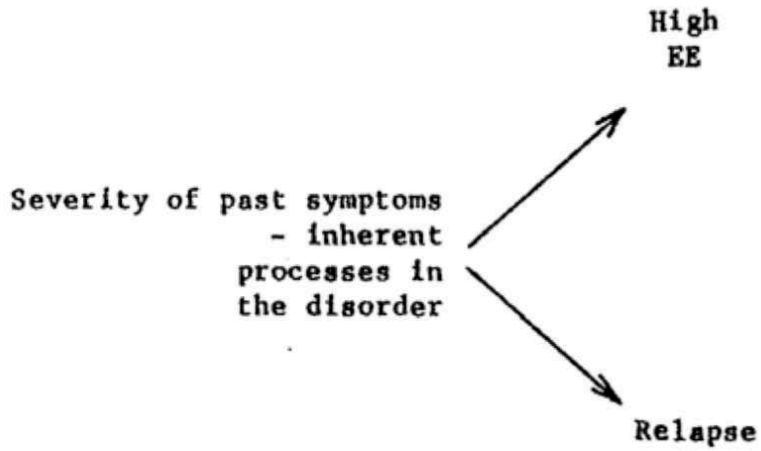
Harris's article in the January number of ***Medical Sociology News*** criticising the London research on schizophrenia depends throughout on a dichotomy between 'common sense' and 'science' which are at no point defined or explained. Moreover its attack is sometimes based on the argument that the work is common sense masquerading as science but elsewhere it is seen as rejecting common sense. This makes a reply difficult and only in his last sentence do we get a hint that his doubts stem from a suspicion of quantitative expression - 'a mystifying welter of reified statistics'.

Since at heart doing science consist of a persistent effort to seek as honestly as possible answers to particular questions about the world, it is the procedures used, not the answers obtained, that distinguish it from common sense. Moreover science does not expect to obtain a completely valid description of the world or a final understanding of it. A scientific study merely sets out to measure the world well enough to arrive at a plausible answer to questions. Measurement has only got to he good enough to achieve this. It is therefore silly for Harris to say that he does not accept our measures 'constitute the totality of the affective components of the households' we studied. Who ever claimed that they were? We claim no more than that they are relevant and valid enough to test whether the emotional atmosphere in the house influences the course taken by a schizophrenic disorder. The statement 'insofar as Brown believes his methodology to be valid (which he does) he is denying that he may be wrong' illustrates the depth of the confusion here. The term belief is used pejoratively: the appropriate idea would he a struggle to believe. Methodology consists of procedures and arguments that can at best influence, positively or negatively, a confidence in one's results. This is always a matter of degree - confidence can never be absolute. To repeat, methodological considerations are not concerned to achieve totally accurate or comprehensive measurement (an impossible task) but to arrange measurement in such a way that, given a particular set of results, the investigator can rule out as implausible interpretations of the results other than the one he believes to be correct. There is no question of claiming at any point infallible knowledge. Indeed, scientific knowledge is often best viewed in economic terms: given that effective research is expensive In terms of money and time, is the pursuit of a question justified? Should we go on and if so, in what way? The study under discussion is the third of a series and now over twenty years since the start of the programme, work on the role of expressed emotion (EE) is currently being carried out in the UK, USA, Denmark and India. When this work is complete we will be more sure or less sure of what we know: it is unlikely that our knowledge will remain unchanged.

Harris early in his paper seems to be convinced that our conclusions are no more than common sense and, I infer, that the work need not have been done. This is a historical and an intellectual misjudgement. When I first thought in terms of the influence of home atmosphere twenty years ago there was no question of the idea's general acceptance. There was a widespread belief in an inevitable endogenous process in schizophrenia and the extraordinarily disturbed and unusual condition of many chronic schizophrenic patients did not make this view absurd. Second, he ignores the plausible alternative interpretation of our

results and our long struggle to rule it out. This is that there in no causal link between degree of expressed emotion (EE) and relapse: that the patients‘ disorder determines both EE and relapse and the apparent causal link is spurious:



To ignore such major methodological questions is to trivialise the research and to underplay grossly the need to test ideas.

Harris is also muddled about the idea of causality. He questions the relevance of our interpretation because it denies 'the volitional aspects of human action and for that reason alone must be rejected as satisfactory explanations of that behaviour.' This again posts a dichotomy the elements of which are unexplained. We, in fact, make clear that those involved are not without some personal influence. The patient can, of course, take drugs. We also demonstrate that the schizophrenic patient when returning to the 'wrong' sort of home can, by reducing the amount of face-to-face contact in the home, much reduce its adverse effects. But this is not the same as saying that a patient necessarily realises the full implication of what he or she in doing. One young woman told us how she no longer lingered after a dance to make love behind the local hall, as she had learned that this brought back the voices in her head. This degree of understanding in probably uncommon but it is possible, and one consequence of developing knowledge of aetiological processes is the chance of incensing such self-awareness. But the general point is that in sociological research we can never assume we know the degree to which 'volitional' and ’deterministic' components are involved in a situation: it in an empirical issue to be settled for every situation anew. What we can be sure about is the uselessness of a general assertion of the kind made by Harris.

The matter in inherently complex. Having plans in not the same as being able to put them into effect; putting plans in to effect in not the same as achieving them and achievement will not necessarily lead to the emotions that we hoped would flow from the successful realisation of our plans. At best we are only partially and episodically in control of our experience. It is a travesty of this view to claim it is mechanical. I should add in this context that I am at a loss to understand his discussion on page eight where he discusses patients returning to a high EE home, who have been seriously disturbed before admission, who take drugs after discharge and who do ***not*** relapse. In interpreting this in so-called volitional terms he seems to suggest that the patient does not relapse because 'if he relapses the patient knows he will be readmitted.' This might be intended to mean that this is why they took drugs and this is the reason for them not relapsing. If so this is entirely consistent with our position; or it might be meant to suggest that patients can decide whether or not they will relapse. Our view is that schizophrenic patients do not have this control in the sense they can decide

whether or not to catch a bus. But they certainly do have potential for doing things that will lessen the chance of their getting into a situation where their experience of schizophrenic symptoms gets out of control. And I have no doubt that patients to varying degrees develop and utilise such 'knowledge'. If we assume that a phenomenon in entirely 'determined' or entirely 'voluntaristic' we will not only almost certainly be wrong, we are almost bound to rule out the development of effective social theory. Such theory in essence is about constraints and the degree to which they may be 'overcome'.

A more general point can be made. The argument illustrates a common fallacy - that of confusing procedures employed for methodological purposes with ideas held by investigators about the nature of the phenomenon they study. It is an though a scientist were accused of denying the existence of colour because he based his research on black and white photographs. Of course, it is possible that some are led astray - that they do doubt the existence of colour. But this cannot be used to impugn the method; the correct deduction in that we must work to obtain and retain a self-awareness of the status of our methods. Simple-minded dichotomies of the kind made by Harris obscure this vital issue.

Confusion seems in part to arise from his ideas about what we have measured. I quote: 'For him, therefore, expressed emotion (dislike) exists in and through the methodic practices by which it is recognised and described.' Once again Harris's argument rests on the assumption that science claims to encapsulate totally its subject - a straw man. And furthermore that if one's methods play a role in shaping one's results, this constitutes a reason for the blanket rejection of results. That by nuking operational assumptions one will inevitably be wrong. For example, that using 'critical comments' as though they were equivalent to each other must lead us astray. (In fact in our earlier papers on measurement we describe an ***overall*** measure of criticism not making this assumption - this gave much the same result in predicting relapse as the count of individual critical comments). This again implies a view that science is either right or wrong - a straw man epistemology. It also follows from such a view that only exhaustive description could avoid being wrong. Yet to invoke Garfinkel (as he does), every description has to be finite and limited in its selection of reality. The world exists only insofar as we are able to develop categories that describe it. We, of course, still need to deal with the accuracy of our measures and whether the process of abstracting has produced casual links where there are none. And given our measures survive such tests we need to go on to question the status of any theoretical interpretations that we have made.

I will deal with the issue of measurement inaccuracy and error first. Harris notes that 'intuitive judgements and feelings are not allowed' in our measurement of EE. We make it clear that they are. If we use observers to measure emotion there is no other way to proceed. While we systematise such judgements, we have never doubted that for this we use tacit knowledge and skills, developed and used in everyday life. There is evidence that we have managed to do this reasonably well (see Brown and Rutter, 1966; Rutter and Brown, 1966). Our belief that our results are not artefacts is strengthened by the measurement of expressed emotion before any relapse. I, in fact , know of nothing about our measurement procedures that suggests that the links we have obtained are not casual: that returning to a certain kind of home atmosphere often leads to a relapse that would not have otherwise occurred. But ideally experimental confirmation in still required.

The second issue of the theoretical status of what we were measuring and the interpretation of the reasons for the link between EE and relapse is more open. Harris seems unaware, however, that many of the theoretical concepts and measures in the social sciences involve ***dispositional concepts***: constructs that indicate that a person in likely to act in a certain way ***given a certain set of conditions***. We believe our measure of EE most likely reflects a disposition of the relative to act in a certain way towards the patient under certain conditions.

While this interpretation at present is speculative, Harris again manages to trivialise the matter. He, for example, equates high EE with dislike of the patient. But we already know enough to be confident that such a general interpretation will not do. For instance, extensive dissatisfaction with the patient was common; and yet unless dissatisfaction was associated with seven or more 'critical comments' it was unassociated with an increased risk of relapse. This result held however marked the dissatisfaction. Moreover, high emotional involvement was associated with relapse irrespective of criticism or hostility.

Harris also at this point ignores our broader theory about schizophrenia - that schizophrenic patients are particularly sensitive to too little or too much stimulation. That with too little stimulation they can develop the signs of extreme withdrawal, poverty of speech and even muteness, to be seen in old style chronic mental hospital wards; and that with too much stimulation they can develop florid symptoms, the latter developing in response to the experience of any marked emotion including joy and excitement. Although he may not agree, it is misleading to reduce our work to ‘coping with dislike' without dealing with these theoretical ideas about the nature of schizophrenia.

There are many other criticisms I could make. But I will restrict myself to one more: about the nature of schizophrenia. Implicit in Harris’s interpretations is the wish to deny the existence of schizophrenia and its manifestation in characteristic florid symptoms and disordered behaviour. His opening insistence on the common sense nature of our results appears to derive from this. For him relatives who dislike the patient are more likely to define hint behaviour in a way that renders it perceivable as symptomatic, the patient may mimic psychiatric symptoms, the patient’s presence in hospital can lead to the definition of symptoms as a means of legitimising his presence. In other words any interpretation other than one accepting that schizophrenic phenomena exist, cause great distress and present a major medical and social problem.

Recent community studies have been, as far as I am aware, completely in accord about the extent and seriousness of the symptoms and handicaps of discharged schizophrenic patients. If there is not a core of characteristic symptomatology at the heart of the disturbed behaviour of schizophrenic patients, I am at a loss to conceive of an explanation of what has been documented in these studies. That someone may be able to mimic a schizophrenic disorder in order to fool s hospital psychiatrist is irrelevant for this issue (and whether this has been done is in any case in some doubt - see Spitzer, 1976). My experience of schizophrenic patients has been that the majority have arrived in treatment only after persistent efforts by relatives and friends to deal with them as though there were little or nothing wrong. I would not wish to undervalue the effectiveness of such an attitude. It may help patient and family to cope. But to persist in such a view – that nothing essentially is wrong that everyday responses cannot put right - can lead to distressing, if not tragic consequences. Harris may well consider the implications of David Reed's account of his wife's schizophrenic illness in his book ***Anna***.

I will make a final and more general point. Underlying invective of the kind that Harris pours on science appears to be a rejection of attempts to sum up the complexly of the human condition in abstract, and perhaps numerically expressed, principles. This is a false fear. As Toulmin has made clear, even for the physical sciences, the actual complexity of the real world can defeat any straightforward practical use of its principles. Although Newtonian and later physics gives us a satisfactory explanation for the phenomenon of tides, the only way to predict the tides at Southend is to go there and measure them. Likewise, at best we may obtain principles of relevance to the course of a schizophrenic disorder. These will be fallible not only because to some degree they are bound to be inadequate but because we cannot possibly predict (or control) the contingent factors likely to impinge on the patients' life. A

patient may return to a ‘perfect’ home according to our principles, but an unexpected occurrence (say the return of the landlady’s son from sea) may transform the situation. This is why we will always need 'clinicians' to ’translate' any scientific principles we acquire; and this is why there will always be a place in the social sciences for intensive descriptions of the individual and his life. But to confuse either with the building of broader principles - and fallible knowledge - is pitifully misguided.

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SCIENCE AND COMMON SENSE: A rejoinder to Professor Brown

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I thank Professor Brown for his lengthy reply to my article in the January edition of Medical Sociology News. However, for all its length, Professor Brown has not answered satisfactorily my main criticism which is that his research bolls down to the observation that discharged schizophrenic patients living with relatives who do not like then are more likely to be readmitted to mental hospital than are those patients discharged to families where they are liked. Brown simply states that in equating High EE with dislike I manage ‘to trivialise the matter’.

My criticism was, and remains, that in calling criticism, hostility and emotional over-involvement 'expressed emotion' and according this manufactured psychogenic variable a causal status in 'relapse' Professor Brown manages to reify the matter.

The crux of the contention is whether I am justified in regarding High EE as basically an indicator of dislike. Suppose during a conversation the person you are talking with makes several critical remarks about an absent third person and/or indicates strong hostility towards that person. It would seem to me that you would have strong grounds for concluding that the person you were talking to did not like the other person very much.

Professor Brown reminds us that ‘high emotional over-involvement was associated with relapse irrespective of criticism or hostility’. Indeed it was, but this indicator of High EE added only 7 out of 45 families to the High EE subgroup. Moreover, the predictive power of this indicator was much less than 7 or more 'critical comments'. The association between High EE and relapse was therefore created predominantly by the families who were critical about the patient. To make the matter clearer suppose, for example, than an England football team were to win the World Cup by fielding a side containing nine Manchester United players*. Would we not conclude that this indicated the overall strength and depth of Manchester United rather than English football as a whole? And would we not suspect special pleading if a commentator played down the contribution of the Manchester United players compared with the two from elsewhere? The analogy must not be pushed however, for while 11 football players constitute what is recognisably a team, Professor Brown's High EE is a reified device which serves to mystify the relations between people.

Since ‘marked emotional over-involvement' did not materially affect Professor Brown's results we might justifiably ask why he retained it in his 'overall index of EE’. Could it be that had he left it out he would have been clearly seen to be wearing no clothes?

This particular research conducted by Professor Brown would probably best be left burled in the pages of the British Journal of Psychiatry were it not for the fact that a recent collection of ‘Basic readings in medical sociology' (Tuckett and Kaufert 1978) gives pride of place to an article by Brown and Rutter which describes the methods used in the study I have criticised and another by Vaughn and Leff which replicates Professor Brown’s research on EE, warts and all. Both articles are to be found in the opening section of the book under the heading 'Sociology as a Science', where presumably they are intended to stand as examples of the best scientific work available within medical sociology. Thus, the pretentions of those who

count, while losing sight of what it is they are counting, are enshrined, legitimated and objectified for consumption by the next generation of students.

* I am grateful to Bill Bytheway for suggesting this analogy.

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SCIENCE AND COMMON SENSE: A Final Reply

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In his rejoinder to my reply ‘Science and Common Sense’ to his original statement in the January number of Medical Sociology News, Harris has ignored my points and turns from broad criticism to a specific issue concerned with analysis of data. Since he shows here an equal penchant for seeing only what he wants to see, I am writing a brief and final reply.

He claims that the EE (expressed emotion) index is no more than a measure of dislike. He notes that only 7 of the 45 families high on EE are 'added' when high emotional over-involvement is taken into account and uses this to argue for the overwhelming importance of ‘dislike’ in the EE index. However, he fails to note that emotional over-involvement only produced relapse for patients returning to live with parents - a point emphasised in our paper. For these patients returning to parents the emotional over-involvement measure makes a quite sizeable contribution to the index. But this is not all. It is misleading to refer to patients ‘added’ to the index in the context of his argument as what is added depends on an arbitrary decision of what measure is considered first. If, in forming the index, we had first considered emotional over-concern rather than criticism and only ‘added‘ criticism at the second stage, double the number of patients would have been contributed by emotional concern measure to those high on EE. Of patients returning to high EE homes, 48% were included for criticism alone, 24% for emotional over-involvement alone and 28% for both criticism ***and*** over-involvement. There is therefore no justification in any ***theoretical*** interpretation of these results for giving priority to criticism. Harris confuses this point by selecting for his illustration mutually exclusive categories - you either play for Manchester United or someone else. Once this is accepted the issue of importance cannot be sorted out along the common sense lines advocated by Harris: and clearly over-concern is not the same phenomenon as dislike.

Harris by talking in general about dislike also manages to obscure another issue – that of of categorising ***degree*** of dislike. It was when only a particular number of critical comments were exceeded that criticism predicted relapse i.e. seven comments. Even if dislike is an important component of our measure (as it probably is) just how much and of what kind has still to be established.

Harris has largely rested his case again systematic measurement in social research by setting up a straw-man account of scientific activity. I earlier argued that his account is grossly misleading and I will not return to this. However, irrespective of the merits of my argument about scientific activity there is no doubt that much sociological measurement is inadequate. Harris appears to be concerned to denigrate the London measures because, without such work, it would be easier to equate these widespread shortcomings with a scientific approach. I am convinced that they have in fact nothing to do with science as such. They result largely from ignorance and the need to measure things on the cheap.

Since Harris has failed to sustain either the case against science or against our measures, he might, I suggest, consider the implications of his failure for current research in medical sociology.