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GIMMIC—or: The Self-Construed Mousetrap for Two Methodologists Trying to Find Their Logical Routes Through OMMON

Remarks on "Statistical manoeuvres in the dark" by M. Burisch and D. Rhenius

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Summary. The critical remarks made by Burisch and Rhenius regarding our concept of OMMON are examined in detail. It is shown that they themselves created the logical and methodological errors they attributed to the model—and claimed merit for their detection.

Sometimes one tends to wonder why there is so little exchange of ideas between the psychotherapy research *workers*, who so desperately struggle to get ground under their feet, and psychology's *real thinkers* (pure methodologists) who, of course, know how things ought to be done. This time we do not wonder.

Burisch and Rhenius—whose methodological wit we greatly admire—have confronted us with an elegantly formulated attempt to prove, in essence, the stupidity of our approach as such and in particular with regard to its logic and methodology. Their position reflects a remarkable and unexpected non—and misunderstanding particularly of the methodological aspects of our paper.

Our OMMON paper consists of three separate, though connected parts: a critical review of current diagnostic and assessment habits in symptom neuroses as far as their therapeutic relevance is concerned; an experimental investigation of multisymptomatology in different kinds of symptom neuroses; and finally, the proposition of one four-symptom model for different kinds of (symptom-)neuroses, with an additional suggestion on how to simulate hypothetically the course of time from a single assessment occasion. We regret, that neither this nor the purpose of our doing so has become evident to our colleagues.

Now let us examine in detail how Burisch and Rhenius themselves created the logical and methodological errors they attributed to the model—and claimed merit for their detection. To facilitate comparison of their critical remarks and our replies we shall follow the labelling of the sections of their paper.

1. Introduction

(1) As it goes without saying that "most neuroses are complex" we did not have to claim it. Instead, we investigated the specific, clinically derived hypothesis that certain "symptom-neuroses" are "multisymptomatic" neuroses, and we produced experimental evidence for this, even indicating that there seems to be a hierarchical order with regard to multisymptomatology among the investigated symptom-neuroses.

(2) The "main route" through OMMON, shown in our Fig. 1 and described on p. 373 (our paper) was not claimed to be *the* main route from low to high pathology. We stated "the main route with our patient sample... could be regarded as a characteristic route for many obsessive-compulsive developments, which start with obsessive-compulsive symptomatology." This was the simple conclusion from the fact, that obsessive-compulsive patients were by far the largest subgroup of our total sample.

Unfortunately, not having read our paper unbiased Burisch and Rhenius conducted a lot of superfluous statistics.

(3) Nowhere have we stated that the model "is capable of identifying the 'causes' of a particular symptom configuration". We claimed that the model allows the derivation of hypotheses about causal interdependencies of symptoms and their changes over time. Obviously, the causes of any symptomformation can, if at all, only be derived from careful (behavioural) analysis of current and developmental conditions of the individual patient. As we claimed, the model-derived hypotheses about causal symptom interdependencies and their changes over time are mainly to be used together with behavioural analysis in order to stimulate and to control clinically derived hypotheses and the respective treatment interventions. Then, the next assessment occasion will provide answers.

2. Analysis

(2.1.) We explained (our paper, p. 362 and 363) in detail why we chose the four "neurotic" symptoms four our model. There is absolutely no evidence whatsoever that there are generally high correlations between specific measures of psychopathology, like self-ratings on the symptoms scales used. Already our own data show that there is no general high correlation, but that correlations of ratings vary from one diagnostic group to the other (p. 367 and 369). Also, Burisch and Rhenius' indirect, "ex cathedra" dismissal of the rating scales used is contrasted by the fact that there are simply no better quality scales avail-

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able; further, it comes as a surprise to us that the authors regard themselves competent to comment on their clinical utility.

(2.2) In this section the authors—in spite of several unspecific, very general critical comments—arrive at the same conclusions as we did in our paper (p. 373): i.e. that the main route of our total sample is decisively influenced by the large over-representation of obsessive-compulsive patients in the total sample. So what? Even a simple eye-ball-analysis of the data in our Table 5 would have revealed to them that the vast majority (94 our of 121, or 77%) of the obsessive-compulsive patients are distributed over stages 1,5,11,15,16. Burisch and Rhenius' conclusion "the alleged 'main route' is not indicative of any conspicious ("obvious to the eye or mind"—Webster's 9th New Collegiate Dictionary, 1983) trend" thus is simply wrong.

Nevertheless, their replication of our results emphasizes an important question that is already inherent in our paper: If our "main" route is largely characteristic for obsessive-compulsive patients, why then are they positioned along this particular route and not equally distributed over all their theoretically possible six routes through OMMON: Assuming that only those patients who have above average (i.e. "+") obsessive-compulsive symptomatology at any stage go into the analysis, obsessive-compulsive patients could occupy five routes alternatively to our main route. All the six routes would have three stages together: 1,5 and 16, which have to be "passed" on the obsessive-compulsive course. But they could differ with regard to the two intermediate stages (11,15).

The assumption that only those patients who actually show above average (+) obsessive-compulsive symptomatology should go into the obsessive-compulsive "path" of OMMON was mainly made with regard to our intention to develop a *treatment-relevant* model. A patient with the clinical diagnosis of obsessive-compulsive neurosis, who shows rather low actual ratings on obsessive-compulsive symptomatology but high ratings e.g. in depression and social anxiety, probably does not need treatment for compulsions but needs intervention in other problem areas connected to the currently high ratings on the two other symptom scales. This does not say anything about the long-term usefulness of the clinical diagnosis. Our assessment allows a multisymptomatic here-and-now "snapshot" of actual complaints to support planning of immediate interventions. Another snapshot later in time may reveal a different situation. Of course, the same would apply to the other diagnostic groups.

The interpretation that we "estimate transition probabilities" between OMMON stages reflects yet another severe misunderstanding by Burisch and Rhenius. We did not use this concept at all. We construed the model on the main assumptions, the "developments" start with "no above average symptoms" (− − − −), that only one + or − can change in a given time interval and that the respective time intervals may vary extremely. These assumptions were introduced for mere theoretical reasons, in order to arrive at a logically consistent connection of the 16 stages. Whereas Burisch and Rhenius regard "the arrows in the model... (as) purely speculative" they in fact indicate the only logically possible connections of the stages under the assumptions made. This does not say anything about the practical usefulness of the model, which can only be shown by empirical data and their distribution over the model in repeated assessment; we are happy that Burisch and Rhenius did understand us at least in this regard, by repeating our own repeated claims in the original paper.

Even if the empirical data (which were, as we stated, not available at the time of publication of OMMON) did not confirm hypotheses derived from the model, this would only question its practical usefulness. As repeated measurements would only create a random sample of different, unknown individual speeds of change, results—confirming or disconfirming—would not allow conclusions regarding the logic of the model construction.

(2.3.)Also in this section the authors show surprising non- and misunderstanding of the methodological basis of our model.

Their statement that "the alleged snapshot can as well depict a still life" comes as quite a surprise. If they operationalize "still life" not as a chronically stable state, but as an intermittent stability in a long-term dynamic process, this does not contradict our assumptions for the model, which does not specify the time needed for change. If, on the other hand, they assume that a certain symptom combination could represent a chronic still life, they not only claim higher stability for "symptom" (state) ratings than is assumed for "personality" (trait) ratings—in fact they would claim that there can be life without movement.

Apart from this forgivable logical fault, there is also a serious logical misadventure in this section. The authors are victims of the error that their GIMMIC is constructed in analogy to OMMON. Their error confirms one of the basic wisdoms of clinical research: the quality of a study is decisively dependent upon the appropriateness of the clinical question to be investigated; only when this is assured, can methodology add particular quality. With GIMMIC Burisch and Rhenius unintentionally make the worthwhile, though premature suggestion of extending our model to general medicine; unfortunately, they wasted their idea on a "wrong" clinical question. Their construction of GIMMIC is by no means an analogy to OMMON. As we stated in our paper (p. 362 f.) we assume that the particability of such models largely depends on a selection of variables which all belong to the same "class"

The four symptoms in our model are all regarded (in psychotherapy and psychopathology) as indicators of neurotic disturbance. They can actually occur as monosymptoms and in any combination with each other; most importantly, each can influence any of the others causally with regard to intensity and frequency. Only under these clinically observable conditions does it make sense to construe a multivariate model like OMMON, when this is intended to support deduction of causal symptom interactions. If only one variable in such a model does not fulfil these requirements, any results from it are bound to create confusion.

Not having grasped this principle Burisch and Rhenius made the following mistakes: (1) They seem to have intended to create an analogue model of OMMON for diabetes and influenza (these thought to be analogues of e.g. phobia and depression), wrongly using identical symptom variables for these two kinds of "illness" which in fact are characterized by very different symptoms. Thus, they created a model for the detection of symptom interactions with four symptom variables of which at least one (fever) has never been observed to influence (or to be influenced by) the others directly. To avoid this basic mistake, Burisch and Rhenius would have had to gain information about four characteristic symptom variables that constitute two or more different "higher class" diagnostic entities in medicine. One could speculate about the feasibility of such an adventure and about the necessary steps to arrive at a

proper analogue model of OMMON, the symptom variables of which have been found to characterize different groups of symptom-neuroses, all of which, together with other subgroups, are subsumed under "neurosis". We prefer to leave this to our colleagues. (2) Table 2 in Burisch and Rhenius' paper seems to reflect their confusion with regard to operationalization of symptoms versus syndromes versus diseases.

GIMMICK "is an ingenious... new scheme" or "an important feature *that is not immediately apparent*" (Webster's Ninth New Collegiate Dictionary, Springfield, Mass. 1983)—is that what Burisch and Rhenius *really* wanted to imply for OMMON with their GIMMIC?

With regard to the latter definition we could then agree.

P.S. The empirical results with OMMON will be submitted for publication to this journal.

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