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Model Theory and Psychiatry

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Introduction

Human psychopathology investigation has too many difficulties, due, not only to the peculiar nature of the psychopathological fact, but also due to many other ethical characteristics which concur with it. This fact reduces the systematic and exhaustive research of those phenomena.

Apart from these difficulties, there are many others of a different nature. For example, the fact that psychopathological phenomena have no strict correspondance with the precise words and descriptions we use for them. This difficulty could be solved if we had operative definitions for every psychopathological fact. In this way, through those definitions, we could, perhaps, verify or refute the truth which is implied in our assertions by calling on functional operationalism. At the moment, however, these operational definitions are not available to enable an exact reference to all the very different psychopathological phenomena. And we have not got them, because we do not have the necessary controlled studies we should have had before their formalization.

All this means that the consequences and scope of future psychopathological investigations must, necessarily, depend on concepts, designs and procedures used in this field by the present state of contemporary science.

In fact, scientific progress is tied to — while at the same time it is dependent

on — the more or less rigorous control that can be established, referring as much to evaluation and/or reproduction of psychopathological facts as to experimental response of those very facts.

Even if psychopathological research cannot be reduced to just experimental methodology — discarding the valuable previous methodological orientations such as phenomenology, clinical description, etc. —, a great part of its growth, advance and development does have to come this way, as can be observed through the enormous progress made in the field of psychopharmacology.

In this peculiar methodological context it is not surprising that new explanatory models are raised in order to legitimize psychopathological phenomena, as we shall presently see.

Psychopathological Theories and Models

Any scientific model studied is, in the long run, nothing more than an inclusive and flexible organization, which coherently articulates the facts and the scientific hypotheses that may better explain its subject. We reach scientific theories through the rules that, deductively connected, regulate phenomenological behaviour.

On the other hand, those very rules can only be put into words through the outcomes whose veracity has previously been checked. From this perspective, rules are

theoretical propositions that summarize the obtained empirical generalizations. In some way, we must assert that we could hardly postulate a scientific law that was not previously based on one or several hypotheses and assumptions that support it.

Hypotheses can only be formulated with a certain effectivity if they are rooted in the answers that have been empirically given to the questions asked about certain facts. The juncture among those answers, which through observation and experimentation have been useful to verify or refute a certain fact — constitute the unavoidable net for the formulation of hypotheses. Thus every hypothesis is nothing more than the supposition offered at a certain moment to explain a natural phenomenon. Hypotheses must be formulated in the simplest possible way, and be very exactly and clearly defined in order to be refuted or verified, partially or totally.

We call postulations those suppositions that have not been verified and refutable hypotheses are also virtually assumed in this formulation, linked to observations, theorems and theoretical propositions that give shape to the scientific frame where this investigation takes place. The postulations must be very precisely formulated, be very few and as simple as possible (Polaino-Lorente 1987).

Neither theories nor models have to deal with the verification or refutation of hypotheses, nor with the capacity to predict the links between the variables used in the studied fact. The extent of their mission consists in giving us the knowledge we do not have about a certain fact at the particular moment.

In this way, they will show us the method of getting this knowledge so that, facing the fact successfully, it will finally give us its truth.

Neither theories nor models should deal with the final judgement on the studied fact. We must not forget that the final judgement sentence is not based on the best or worst intuitions of the researcher, but to man's judgement which is founded

on the concreteness of the obtained outcomes; those outcomes can also be contested and obtained by others, and their veracity counts as much as their verosimilarity among the researchers who make up a certain scientific community.

It is in the generic frame of what theories are where talk about a model can be meaningful.

Analogy and Models

In practice, it is impossible to understand what a model is without previously understanding what analogy is, for the model, being a schematic representation of reality, necessarily implies the setting of a certain analogy between the reality represented by it and the reality that it itself represents (Espert 1918).

From this perspective, there is always a nexus between the model and reality, that is to say, the setting of a certain relationship between them both.

And in a wide sense, that is exactly what we understand by analogy: the correlation between terms of two or many systems or orders, or, better still, the existence of a relation among every term of a system (model) and every term of another (psychopathological phenomenon).

We can equate analogy to the proportion of sameness or likeness of certain traits, characters or functions among several objects. In the last term, analogy is the attribution of the same predicates (variables, traits, symptoms, etc.) to several objects (model and psychopathological phenomenon). But this attribution must not be understood as a univocal determination of these objects but as the expression of the correspondance, similarity or correlation that occurs among them.

In fact, the very core of analogy is not the similarity in a strict sense, but here, the emphasis is placed on the existence of a certain similarity or proportion between two or more systems, the model being the expression of them (Ferrater-Mora 1979).

Analogy must also be differentiated from another concept: Univocity. Analogy is founded on the possibility of making relations between beings or substantially different facts. To be able to establish an analogy, the existence of a certain community among those beings or phenomena is necessary because, if not, the very analogy would be impossible due to its lack of foundation.

Univocity, on the contrary, is founded on the individuated possession of a common element among several beings or phenomena. The previous distinction shows, in a clear way, the nature of analogy – as opposed to univocity – as it is applied to research models. Nevertheless, the use of analogical models in clinical practice does not always prevent equivocity, as we will see later. This is because analogy is used on many different levels in clinical research as much to express two things or facts, as to refer to two expressions or terms.

In the particular case we are referring to, psychopathology, the calling to analogy that founds the models, can be even more equivocal. Note that in psychopathological models, symbols (expressions of expressions) and symbols of symbols must be frequently used, and that can easily lead to equivocity. On the other hand, the very equivocal nature of psychopathological facts – of which models are only sheer analogates – should condition the methodological analysis of the outcomes obtained through them, which is a procedure method very rare in the psychopathological field.

Finally, we must not forget that analogy itself, on which every psychopathological model is founded, is itself analogical, which complicates the veracity and the possibilities of generalization of the outcomes obtained in this scientific field even more.

Psychopathological Models; Concept, Validity, Utility and Limitations

The importance of models lies in their being an “operative arm” of theories, by

virtue of which we can empirically explore theories (Anguera 1977, Lehman 1977, Arnau 1979).

In some way, models are half-way between theories and reality, and, consequently, they mediate their relationships. But, as every reality is hypercomplex – and even more this one of psychopathology – it is logical that the model tries to represent it by oversimplifying it, that is to say, reducing the reality to what, according to his theoretical orientation, the author considers more pertinent in the advance in this progressive analysis. So, on the one hand, he gets his inspiration in theory, and on the other, he fits himself, in a limiting way, to the reality he is intending to study.

Whatever the case, the potential versatility of the model is based on its permitting a reductive but additional analysis of reality, and from the very starting point, from the perspective recommended by theory. Even if models are different from theories in some other way, the models are formed as sectorial and partial micro-theories of reality (Bunge 1972).

Models are also more sensitive and can be relatively modified by their own results, which may be the outcome of their use in the investigation of reality; on the contrary, theories are more resistant, less vulnerable to the action of the results obtained from investigation and which are generated through these. And this is because the validity of a theory about a psychopathological fact can be verified or refuted from very different perspectives and from very different models (clinical, experimental, epidemiological, etc.). That is why if what is furnished by an investigation through one of those models is not in accord with the theory that inspired it, it is likely that the model will be made again attending to what those findings are, trying to verify the same theory which is not, in any case, modified by this.

Even if every model has a trait (graphic or symbolic) of representation of the reality it tries to explain, they can, nevertheless, be distinguished from one another by

attending to their level of mathematical formulation. Analogical Models and Symbolic Models have been distinguished from this perspective (Achinstein 1969, Gibbs 1974). In Analogical Models, the representation of reality is more iconical and immediate and has a lower level of mathematical formulation. This is the case, for example, of enzymes which are represented by the biological model, since they are studied, as "the key and the lock".

Symbolic Models have, on the contrary, a higher abstract value and a much more mediate explanatory function due to their high level of mathematical formulation and to the abstraction level where they are. The stochastic models used for perception study are an example of this (Bowers 1973).

The reflexion regarding the validity of a model is one of the most relevant questions in the analysis of present scientific investigation. That is to say, some criteria must be chosen to find out if the model fits to what is modelled or not, as well as to find out the degree or intensity of this fitting. With validation we try to set the degree of fitness of the model to those traits of the real fact which we have tried to reproduce in it. In view of the fact that, for the design of the model, only some reality traits were chosen, traits which were necessarily very reduced, it is logical that the very validity of the model, being a representation of reality, would also be, necessarily, very reduced (Lehman 1977).

Because of the limitation itself of the validity of the model, the verification of its hypotheses does not make it really more valid, but just more useful. Instead, the validity of the theory is really a function which depends on the degree of checking or verification of the hypotheses in the theory.

The short scope of the results checked with regard to the validity of the model is logical. And that is because the model is not the reality, but merely an analogate to it, that is to say, something conjectural and ready for operative simulation, whose outcomes can be neither generalized nor com-

pletely nor precisely transferred to the reality that the model represents. Although models are relatively valid, it must be said that they are, all the same, relatively strong, because through them the validity of the theory, of which the model is just its operative arm, the powerful instrument to explore empirically the theory, is based.

Next I am going to summarize very briefly some of the advantages and disadvantages that the use of models has in psychopathological field research. Some of their main advantages are:

1. The model is the operative instrument that gives theory the empirical chance of checking its statements and general predictions, which are usually less concrete and more abstract.

2. The model allows some processes to stand out, which, although they take part in the behaviour development, cannot be directly observed.

3. The model provides a formal framework of explanation and comprehension to the theory, at the same time giving rules of meaning by which to interpret what is observed, and contributing in that way to decrease the entropy of the observation.

4. The model is useful in as far as it serves to represent, in a more graphic and comprehensive way, the complexity of what is real, using the heuristic value that it has, because it is an analogy of reality.

Among their main disadvantages we can point out the following:

1. The model can excessively simplify the reality it is supposed to represent, even to the point of not representing it at all, and fall into the equivocation: it is a question of simplifying the reality that the models claim to represent, but without deforming it because of this representation. The researcher can avoid falling into simplifying reductionism if he does not consider part (the outcome obtained in his research through that model) as the whole (what could really be concluded if those

very results came from the reality that the model claims to represent).

2. The results from the model are never isomorphic to those that come from reality. Thus they can never be generalized to the point of being attributed to reality as well. Excess referring to the generalization can easily be made, mainly when: *a*) we do not have the necessary semantic rules that assure the veracity of our logical inferences; *b*) we no longer have any more terms of comparison between the model and its reality (signs, symptoms and clinical syndromes) than those which are merely conventional and *c*) we have no criteria that could optimize the first isomorphization, hypothetically established between the characteristics of the model and the true properties that characterize the reality they claim to represent.

3. The contextual conditions (laboratory and/or experimental) where the work with models is carried out, can scarcely be extrapolated to ecological conditions (spontaneous, natural and not provoked) where the pathological behaviour that the model claims to represent, usually arise. The ecological validity of the conditions where the model works (Alvira et al. 1979) remain completely apart – to the point of it being impossible to compare the results obtained from reality and from the model – from the circumstances that are connatural to the spontaneous emergency of the studied pathological behaviour.

The characteristics, traits and peculiarities that define the model almost always constitute a constellation completely apart from the one that characterizes the pathological fact. Frequently, models work with experimental animals or simulated patients. In the first case there is a long and indefinite distance – as much as the one that characterizes the jump from one species to another – between the patients and the experimental animals where the illness to be studied is artificially caused. In these circumstances there is scarcely any normative criterium on which certain inference

rules could be founded, so that what is experienced by a species could be transferred and attributed to another species.

In the second case, even though here there is no distance inter-species, it is at least very problematical to consider that the events that occur in the simulated patients (experimental subjects of the model) can be generalized to the sick people who spontaneously suffer from those pathological disorders.

As can be observed, the use of models in psychopathology is not free from limitations and difficulties. Many of them can be precisely identified with what, according to another certain view, could be pointed out as advantages and good points of those models. Thus, they are like the two sides of a coin, the greatness and value of those procedures of investigation which, feigning reality, make the advantage of science possible because they facilitate more manipulation and closer control of certain outstanding variables.

Some Psychopathological Models

The employment of models has a long and uneven tradition in the psychopathological and psychiatric field, although the meaning given to the concept of model changes a lot from an author to another. In textbooks, reductionism in the use of this concept is very frequent, because this term is employed in such an abstract and ambiguous sense (medical model, conductist, psychodynamic, sociological, etc.) that the psychopathological theories almost overlap.

In the sense used up to now, the concept of model comes from Pavlov's works (1927 & 1961) and since then it has been used, although with a very different meaning, in some psychopathological phenomena.

In this last sense, the concept of model has been employed as an "experimental analogy" of the subject (experimental animals versus depressive patients), of inde-

pendent variables that make up the framework that characterizes a psychopathological phenomenon (unsolvable anagrams versus unsolvable problems of daily personal life), and, of dependent variables that form the symptomatological profile of a certain gnosiological entity (suspicion, self-references and hypersensitivity versus analogical systems at symbolic process level and computerized algorithms).

In spite of the doubtful analogy which could be made between those models and psychopathological reality, the fact is that their effectiveness — also in clinical application — has been satisfactory.

There are many examples of this. It is true that the models that were traditionally thought of were mainly directed to the study of phobias, learning in laboratory (Watson & Rayner 1920) and to the simulation of schizophrenic behaviour, through symptoms induced by psychedelic drugs (Hollister 1962). But it is also true that, in another field — in animal models of experimental psychopathology — the tradition of study through models has continued up to now. From Pavlov (1941) to Karsh (1970) and Masserman (1971), a long and uninterrupted tradition put the emphasis on many outstanding works in experimental neurosis research (Anderson et al. 1941, Liddell 1944 and 1956, Maier 1949 and 1956, Wilcoxon 1952, and Dmitruk 1974).

However, besides those classical works — well known to everybody — there are some more that, leaving behind the controversial and arguable field of neurotic behaviour, are one of the most important landmarks in the present development of experimental psychopathology. I am talking, of course, about the learned helplessness model, outlined by Seligman (1975), and later drawn up again by Abramson et al. (1980). There are many works of research carried out all over the world on this subject, not only because it is a new way to treat psychopathological research, but mainly because it has generated a completely new therapeutical range for the

psychological treatment of depression, whose effectiveness is quite satisfactory (Polaino-Lorente 1984, 1987).

Another field where research through models is being very fruitful is in Alcoholism. At this point it suffices to reread the now classical Eriksson, Sinclair & Kiianmaa (1980) work on alcoholism research in animal models, to prove the great future we may expect from these ways of approaching psychopathological investigation. The same thing can be said regarding other psychopathological problems such as obesity (Rodin 1983), minimal cerebral dysfunction (Sechner 1983) psychosomatic disturbances (Schwartz 1983, Weiss 1983), catatonia (Gallup & Masser 1983), or schizophrenia (Paul 1983).

Another fact to be borne in mind, as regards the training of psychiatrist learners, is the model of Artificial Paranoia, outlined by Colby (1967, 1975 and 1981) in the Neuro-Psychiatric Institute of Los Angeles University (UCLA). The author's intention was to find out how human thought works in paranoia. Not having a real algorithm of this kind of psychopathological thought, he made a theoretical model, based on a computerized and simulated algorithm. The model built in that way meant a partial analogical system, able to work at symbolic processing levels. The building of such a model means, in some way, the identification of the traits that describe paranoia best (suspicion, self-references, hypersensitivity, fright, hostility, stiffness), so that, once individualized, it was possible to build a theory using them, which could later be operativized in a computer (Polaino-Lorente 1987).

From this perspective, models have also been applied to the reproduction of symptoms (Miller, Rosellini & Seligman 1983), etiological cause (Sarason et al. 1980, Maser et al. 1983) and pathogenic mechanisms (Cosnier 1975). In spite of their utility in clinical investigation, however, it seems necessary to remember the following points:

1. There is scarcely any psychopathological entity that could be applied to other species where the symptoms, the course and treatment that characterize what happens to the human being could be efficaciously compared (Maher 1974, Balis 1978). That means that most of the psychopathological realities in human beings cannot be defined or modelled in transpecific terms without falling into a radical reductionism.

2. The possible analogy that could be made between dependent and independent variables that occur in a real psychopathological disturbance and those that are studied in experimental models, is, by now, a sheer attribution — attribution of analogy — that must be empirically verified, in spite of the efficacy that, in some cases, has resulted from the investigation of analogies.

3. The use of models has sometimes gone too far, either by isomorphing the different phenomena that occur in the model and in what is modelled, or by identifying the correlational method in the study of psychopathological phenomena with causal method, that is, as Toulmin (1970) says, mistaking reasons for causes.

Trying to make those phenomena that occur in the real world homogeneous with those artificially outlined in laboratories, in order to provide experimental psychopathology with the necessary rules of comparison by which to ratify this analogy, Abramson et al. (1978) have proposed a set of criteria. Thus, it is a question of searching for a more or less objective system of references, for an analogy that "is seldom a precise reproduction of spontaneous disturbances but that is useful to some degree as it imitates the phenomenon" (Minsky 1968).

I next summarize the questions asked by Abramson et al. (1978) with regards to this:

1. Is the experimental analysis of laboratory phenomenon enough to describe the

main traits of its causes, as well as its prevention and its remedies?

2. Are the close resemblances between the model and the spontaneously produced psychopathological facts convincingly proved?

3. What is, in physiology, the degree of resemblance among cause, remedy and prevention?

4. Does the laboratory model describe, in every case, a psychopathology produced in a natural way or just a subgroup of it? Is the phenomenon of laboratory a specific psychopathological model or does it represent general traits of the global psychopathological model which is going to be observed?

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