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Correspondence

Thoughts on research in homoeopathy

PAOLO BELLAVITE, University of Verona, Italy

The paper by Daniel Eskinazi[1] raises many questions and thoughts on research in homoeopathy. I totally agree with his 'questions' and with his analysis of the principle of similarity, but I disagree with some of his 'thoughts', particularly with the final pessimistic view about the role of scientific research in this field. Moreover, I take the opportunity of replying to some of the points raised on my paper,[2] previously published in this Journal. The critisisms are essentially based on the presumption that the experimental approach we follow does not give definite answers to the most important questions which are relevant for homoeopathy.

In my opinion, the fact that most of experimental evidence supporting the claims of homoeopathy (effects of low-dose/high dilutions, inverse effects in precise laboratory or animal models, bioelectromagnetics, etc.) failed to convince the sceptics is not a good reason for 'changing the research agenda' of basic research that has been followed in the last 10 or 15 years. The purpose of fundamental research is neither to 'convince the sceptics' nor to suggest 'how to choose a remedy' (this is the main object of clinical research), but to describe and possibly to understand the phenomena claimed by homoeopathy, using the experimental method. Experimental method is based on the assumption that any hypothesis should be testable, that is, measurements can be done to prove or disprove it. To do this, we need specific and carefully selected experimental models, which often, if not always, fail to grasp the wholeness of the phenomenon or of the object of investigation. If the object of investigation was 'Homoeopathy', we would never find an experiment suitable to prove it. We must distinguish several points, for example the problems of potencies, of individualization of cure, of quality of remedies, and so on. We have chosen to carry some experiments on the 'similia principle', but even in this case, there is not an experiment that can 'prove or disprove' the whole matter. We had to use a specific experimental model, and we have utilised leukocytes, that were already working well in our lab. This is simply how the science community proceeds. This is the so-called experimental method and it must hold also for homoeopathy, even if I acknowledge that not every claim of homoeopathy can be dealt with using this approach. The possible contribution of theoretical physics, of mathematics (complexity, chaos, fractals) or even philosophy to understanding this highly controversial medical discipline cannot be ruled out, as we have already pointed out.[3]

In my cited paper,[2] one can read: 'Of course, it is worth noting once again that this is not an explanation of therapeutic effects of homoeopathic drugs, but the demonstration of how the concept of similia principle can be explained on a cellular scale in a precise experimental model. Our experiments do not allow to understand the 'general' effect of high potencies on the whole organism, but it is conceivable that there are homoeopathic remedies which act through this model such as low-potency extracts or self-derived products (hormones, cytokines, organ extracts, antigens, etc).'

In our experience, opposite effects of the same agent (stimulating effects of inhibitors or inhibiting effects of substances which stimulate when employed at high doses) can be observed in several models, but the experimental conditions (doses, type of stimulant, cell treatment, cell function) must be carefully set in order to regulate the complex balance of receptors and transduction mechanisms. Therefore, these phenomena at the cellular level should be regarded not as an 'universal law' but as an expression of a possible behaviour of the living system when it is exposed to suitable conditions. We have also clearly stated that our theory is not the only model that can explain the occurrence of inverse effects at a cell level. One should consider, for example, the presence of various receptors, having different affinity and different coupling with effector systems, or the

induction of detoxification enzymes or heat-shock proteins (gene expression and enzyme activation).

Clearly, the 'classic' Hahnemann's principle is based on *symptom* similarity and we have said that the procedure finds its justification in the complexity of the homeostatic control and in the sensitivity of human organism, but at the level of simpler systems, the same principle may be expressed as a paradoxical reactivity to experimental stimuli and as measurable changes of specific biochemical variables. Since it is highly conceivable that homeostatic systems at various levels are organised in various hierarchies of regulatory mechanisms and that multiple communications exist between the different levels, it is also conceivable that the elucidation of the mechanisms of inverse effects at cellular level may be paradigmatic for the understanding of the similia principle at a more general level.

From our experiments on the regulation of leukocyte adhesion in cell culture and from a large body of biological knowledge that has accumulated in the last twenty years (receptors, transduction mechanisms, pathway of biological communication) we have also suggested a kind of generalisation of the similia principle/inverse effects. Our general model ('Regulation of stressed homeostatic networks') predicted that the homoeopathic approach—based on the use of low doses of carefully selected drugs that stimulate the homeostasis at multiple levels—may be more effective than conventional pharmacology when the complex, subtle and individual dynamics of the disease are considered.[2]

In our models, we have not considered the question of homoeopathic dilution and dynamization. However, we have written that 'assuming that information storage in water has a physical basis and that both biochemical and bio-electrical homeostasis exist in the body, it is possible to speculate that relevant signal transduction mechanisms like those here described are the target also of high-potency homoeopathic remedies. However, further studies are necessary to clarify this critical point of the theory.' So I entirely agree with the statement of Eskinazi that 'there are distinct molecular mechanisms for low and high concentrations.'

My personal view regarding research in homoeopathy is that it will never end up with a final explanation, because homoeopathy is such a large field that there is not an experiment, nor an experimental line, which can clarify everything and convince everybody. It is only through patient accumulation of knowledge and elimination of errors that we get nearer to the truth. I do not believe in 'research agendas' in general and they are particularly dangerous in homoeopathy at this stage, where the problems are so many and so open that every researcher should select his field, according to his experience and available instrumentation.

Finally, I have a fundamental thought that overlaps with many of the thoughts expressed by Eskinazi: does speaking of a 'research in homoeopathy' make sense? Why not speak simply of 'research which may have some relevance for homoeopathy'? As a matter of facts, basic research cannot be either homoeopathic or allopathic or anything else, simply because reality is both 'similar' and 'contrary', both 'mechanistic' and 'complex': with our studies, we see only a small part and a few aspects of it, from a single standpoint. I personally believe that researchers, who are supplying worthy experimental evidence of the various applications of similia principle are working for a united medicine.

References

1 Eskinazi D. Some questions and thoughts on research in homoeopathy. *Br Hom J* 1998; **87**: 33–38.
2 Bellavite P, Lussignoli S, Semizzi ML, Ortolani R, Signorini A. *Br Hom J* 1997; **86**: 73–85.
3 Bellavite P, Signorini A. *Homeopathy–A Frontier in Medical Science.* North Atlantic Book, Beverley, CA, 1995.

Address for correspondence:
PROFESSOR PAOLO BELLAVITE
Instituto di Chimica e Microscopia Clinica
Ospedale Policlinico
Laboratorio d'Analisi
Universita Degli Studi di Verona
37134 Verona
Italy

TXU (Anti CD-7)–Pokewood anti-viral protein as a potent inhibitor of HIV

MIMI IRWIN

Sir — I would like to bring the following paper to the attention of your readers:

TXU (Anti CD7) — Pokeweed Antiviral Protein as a Potent Inhibitor of Human Immunodeficiency Virus.[1]

Homoeopathic practitioners have long used the plant *Phytolacca americana.* In 1988, after Dr Peter Fisher suggested to me that Phytolacca might have a place in the treatment of AIDS, I wrote a paper; Acquired immunodeficiency syndrome: Is *Phytolacca americana* homoeopathic to the acquired immunodeficiency syndrome?[2] I concluded that the materia medica of Phytolacca shared some important features with the clinical presentation of AIDS. In my research for this paper I did not come across some previously published articles which described the antiviral activity of phytolacca against plant mammalian viruses.

It has been known since the 1970s that Pokeweed Antiviral protein (PAP) inhibits the transmission of tobacco mosaic virus in plants. Later it was found that PAP has an inhibitory effect on influenza virus, poliovirus, herpes simplex and HIV-1. PAP is a ribosome inhibitory protein which is derived from the leaves and seeds of *Phytolacca americana.*

When PAP is conjugated to antibodies specific to cell surface receptors the antiviral activity of PAP is much improved and highly cell selective. This conjugate inhibits HIV-1 replication at picomolar concentrations. Fortunately the proliferation of normal CD4 T cells is not inhibited at these concentrations. The conjugate has been used *in vivo* in mice and cynomolgus monkeys, with no significant side effects.

Yours faithfully

Mimi Irwin
17 Ngauruhoe St. Mt. Eden,
Auckland, New Zealand

References

1 Uckun FM, Chelstrom LM, Tuel-Ahlgren L et al. TXU (Anti-CD7)- Pokeweed Antiviral Protein as a Potent Inhibitor of Human Immunodeficiency Virus *Antimicrobial Agents and Chemotherapy.* 1998. 383–388.

2 Irwin MKS. Acquired immunodeficiency Syndrome: Is Phytolacca americana homoeopathic to the acquired immunodeficiency syndrome? *Br Hom J.* 1988; **77**: 219–223.