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How observations on oneself can be scientific

David A. Booth

School of Psychology, University of Birmingham, Edgbaston, Birmingham B15 2TT, U.K.

d.a.booth@bham.ac.uk **www.bham.ac.uk**

Abstract: The design and interpretation of self-experimentation need to be integrated with existing scientific knowledge. Otherwise observations on oneself cannot make a creative contribution to the advance of empirical understanding.

Seth Roberts is right to argue that experiments on oneself are unduly neglected in contemporary science. Unfortunately, however, **Roberts** has misapplied scientific method in the studies that he describes running on himself.

There are four types of flaw in his claims to have evidence for some effects of visual exposure, movement practices, or food selection on expressed mood, perceived sleep, symptoms of a cold, or body weight.

1. **Roberts'** self-observations are contaminated by effects of his knowledge of previous observations that he made of himself. There is little or no point in self-replication when the phenomena depend on perceptible stimulation or controllable action.

2. **Roberts'** manipulations are confounded by known influences that may provide explanations of his observations that conflict with his hypotheses.

3. Contrary to the argument by **Roberts**, the unexpectedness of an observation makes no contribution to the strength of the evidence. This is because, if flaws 1 and 2 were avoided by considering only a single observation, the surprise becomes logically indistinguishable from mere coincidence. Worse, a lifetime spent looking for surprises will collect an increasing number of spurious concurrences. For example, this is the basis of the very high proportion of supposed intolerances for foods that, on testing, prove to be misperceived (Booth et al. 1999; Knibb et al. 1999).

4. In the case of his most "weighty" conclusions, **Roberts'** theory has been refuted by extensive prior research.

It is with some grief that I see **Roberts** spoil his case for self-study, because I began my research in molecular neuroscience and my education in cognitive psychology with experiments on myself.

My initial brain/mind interest was the neurochemistry of psychosis. In that context, I once ate nothing but a large bar of chocolate for lunch and analysed its metabolic products, to show that the origin of a compound seen more often in hospital patients diagnosed with schizophrenia was less likely to be their brains than the boxes of chocolates that visitors gave to them more often than to the nurses who served as the control group in that project (Booth & Smith 1964). The printer's block for the key figure still sits on my office shelf, labelled "In Memoriam: experimenter as his own subject" – although the nausea that I suffered after eating a half-pound of chocolate was clearly not fatal! Note that my metabolism (or the hospital visitors' gifts) could not be affected by my perceptions or my actions, given that I kept the chocolate down.

Eight years previously, I worked by myself through a little book of experiments in psychology (Valentine 1949). My memory is of laboriously training out the Müller-Lyer illusion and replicating the primacy and recency effects in recall of lists of words. Later, however, I found that I had a capacity for direction of attention sufficient for observable dissociative effects as autosuggested movement. So I became aware that directed forgetting could modulate that curve of deficits in serial recall. No one these days should estimate the size of an effect without comparing performance between people who know and don't know the correct hypothesis.

Some of my professional discoveries about hunger provide a basis for sympathy with **Roberts'** thesis but also for criticism of his examples. In some instances, I can't tell if personal experience stimulated the hypothesis, my theorising triggered the self-observations, both, or neither.

For example, Booth et al. (1970) provided the first (group) evidence that protein is better than carbohydrate in a meal at keeping hunger at bay some hours afterwards. The finding has been replicated several times (most recently by Long et al. [2000]); indeed, the original pair of experiments was limited, like all single studies, and so needed to be extended by different designs (very differently by French et al. [1992]). The effect moreover may be the key to low-carbohydrate diets (like Atkins'): weight loss occurs only when energy intake is lowered, and reduction of hunger by the raised protein content may enable this self-restriction to be better sustained (Bravata et al. 2003). The autobiographical twist on this is that I had gained the impression that meals based on rice, even when I had eaten enough to feel very full, left me hungry again little more than an hour later. For a long while now, I have believed that I can prevent this (other) "Chinese restaurant syndrome" by including enough flesh food of some sort in the meal. Yet I can't tell if this effect is self-experimental evidence for the hunger-delaying effect of protein or autosuggestion from – or valid application of – my theory of late mobilisation and utilisation of assimilated amino acids through the alanine cycle.

On **Roberts'** ideas about sleep, he pleads that the basis of his anticipatory awakening is "surely not expectation." Yet it is likely that he was aware that he was not going to have his usual breakfast: I regularly wake early when I know I have something unusual to do when I get up. Quite apart from autosuggestion, **Roberts** fails to allow for some obvious

mechanisms. For example, fruit eaten in the evening could induce earlier waking by filling the bladder more than drier foods do.

The most disastrous moves made by **Roberts** deploy the notion of a “set point” for body weight. This concept of a reference value is simply redundant when there are opposing negative feedback functions (Booth et al. 1976; Peck 1976). Furthermore, even for body temperature regulation, the hypothalamus only has countervailing networks for heat production and dispersal – no 37°C-setting neurones. The urgent scientific issues about obesity are the mechanisms by which a person can most easily lose more energy than they gain during and between meals (Blair et al. 1989; Booth 1998): “set” points that move (!) divert attention from the real scientific problems. Similarly mind-numbing is the unoperationalised notion cited by **Roberts** that flavour-calorie associations increase “palatability” (Booth 1990; Conner et al. 1988).

Basic mistakes undermine these designs and interpretations. One example will have to suffice here: sucrose is a compound of fructose and glucose and so is useless as a control for fructose. Indeed, a lot of fructose without glucose is poorly absorbed and the resulting upset could reduce hunger (Booth 1989, p. 249). **Roberts** can swap anecdotes with his readers for a very long time, but scientific understanding is not advanced until a literature-informed hypothesis is tested between or within groups in a fully controlled design shown to be double-blind.

To conclude, personal experience can be a good way to get new ideas. Deliberate manipulation of the environment and keeping an eye open for unusual consequences may accelerate the generation of hypotheses. Yet the only way that science progresses with new ideas is to test novel hypotheses against existing theories in a competent design. Individualised analysis of complex performance (Booth et al. 1983; 2003; Booth & Freeman 1993) is also grossly neglected but requires adequate design and aggregated data. Don’t compare conclusions; find out about mechanisms.

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