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On function, cause, and being Jerry Hogan's student

Martin Daly[a,b,*]

[a] Department of Psychology, Neuroscience & Behaviour, McMaster University, Canada
[b] Department of Anthropology, University of Missouri, United States

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ABSTRACT

Jerry Hogan has forcefully maintained that cause and function are distinct questions, and that attempts to integrate them are conceptually muddled. I dissent from his view, maintaining that causal analysis is conducted in the shadow of premises about function, and that bringing functional ideas out of the shadows facilitates the generation of fruitful causal hypotheses. This is not to suggest, however, that cause–function muddles are non-existent; I agree with Hogan that they are both common and mischievous.

This article is part of a Special Issue entitled: In Honor of Jerry Hogan.

In January, 1967, I enrolled in Jerry Hogan's upper-level undergraduate seminar in animal behaviour at the University of Toronto. Then in his third year as a faculty member, Jerry was like no professor I'd ever met. He had strong opinions, forcefully expressed, and some idiosyncratic ideas about the authors that we students needed to read: Tinbergen and Lehrman and Robert Hinde's textbook, sure, but also Jakob von Uexküll, T.C. Schneirla, and Zing-Yang Kuo. Jerry would have been intimidating, except for the fact that he also encouraged us to be skeptical of received wisdom and whooped with delight whenever a student mustered an articulate criticism of some eminent greybeard's ideas. His enthusiasm was infectious, and his collegial treatment of students was a revelation.

Jerry and his wife Lidy Hogan-Warburg then lived with their two young sons in a modest townhouse in the east Toronto suburb of Flemingdon Park, to which the keener members of our small class sometimes travelled in the evening to continue classroom discussions, drink beer, and play Frisbee in the parking lot under the street lights. Other regulars were Patrick Colgan (1944–2004), later to be a Professor of Biology at Queen's University, fish ethologist, and director of research programmes for Canada's Royal Botanical Gardens, and Roy Patterson, now the head of Cambridge University's Centre for the Neural Basis of Hearing; we were just three of the many students whose interests and ambitions Jerry shaped.

I had already accepted admission to graduate studies in physiological psychology (which was not yet known as "neuroscience") at McGill University in the coming fall. But after a few weeks of Jerry's seminar meetings, I had lost my desire to insert electrodes into rats' brains. I wanted to become an ethologist like Jerry. So I fled McGill with a quick M.A. and returned to Toronto in September 1968 to become his PhD student. I never regretted it.

1. Can theories of function help us generate good causal hypotheses?

Jerry Hogan pursued postdoctoral studies with Gerard Baerends, and he has always called himself an ethologist. But his PhD was in psychology, and it seemed to me that a psychology department was his natural home. His research focused on problems of cause (*sensu* Tinbergen, 1963) and ontogeny, and while he granted that function and phylogeny were also important, it always seemed a rather grudging concession. He apparently thought that these topics were scientifically intractable, and that anyone who persisted in trying to study them wasn't thinking very clearly.

The philosopher Eliot Sober has proposed that psychological science's contempt for explanations in terms of adaptive function derives from physics envy: the lesson experimental psychologists drew from the victory of Newtonian physics over Aristotelian teleology was that "a science progresses by replacing teleological concepts with ones that are untainted by goals, plans, and purposes" (Sober, 1983, p. 115). That was certainly Jerry's view. He admired B.F. Skinner, notwithstanding the attacks that behaviourism drew from ethologists whom Jerry also admired, and he sometimes disparaged the early stirrings of the "cognitive revolution" for backsliding into vitalism. He was similarly unimpressed by the rise of behavioural ecology and sociobiology, despite having done some early work in the area himself (Kruijt and Hogan, 1967) and despite having assisted with Lidy's classic field study of ruffs (Hogan-Warburg, 1966). He thought that muddling cause and function was endemic to the work of behavioural ecologists and

* Correspondence to: Department of Psychology, Neuroscience & Behaviour, McMaster University, Canada. Tel.: +1 5732286900.
E-mail address: daly@mcmaster.ca

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sociobiologists, and he had no trouble finding published examples to justify his scepticism. We, his students, took to heart the lesson that failing to distinguish cause from function was a source of much confusion, but we were never entirely persuaded that he hadn't thrown a baby out with the bathwater.

David Sherry completed his PhD under Jerry's supervision a few years after me, and decades later, he wrote an essay entitled "Do ideas about function help in the study of causation?" (Sherry, 2005). His answer was "yes", and I agree. (Jerry is surely disappointed in both of us.) This essay can be read as an elaboration on Sherry's discussion, which concluded as follows: "When the function of behaviour is known, and even when there is a plausible hypothesis about function, the details of any proposed causal hypothesis are constrained. Causal explanations must meet design criteria that are set by the function of behaviour." (Sherry, 2005, p. 449). My only quarrel with this conclusion is that the language of "constraint" suggests that functional arguments merely narrow the scope of causal investigation, and omits their *enabling* role. A functional theory grounded in the logic of natural selection doesn't just *constrain* the set of causal hypotheses worth investigating, although it certainly does do that, as witness the many blind alleys that social psychologists have stumbled into thanks to their penchant for arbitrary a-Darwinian functional theories (Daly, 2011). What a good functional theory can also do is point the way to worthwhile hypotheses about causal processes that would otherwise never have arisen. Let me try to make that case with reference to an experimental example.

Wedekind et al. (1995) recruited a group of male students to wear fresh T-shirts for two nights, and a group of female students to later sniff the T-shirts and rate their odours on scales of intensity and (un)pleasantness. The students were furthermore genotyped for three MHC (major histocompatibility complex) loci, and the shirts that the women were asked to rate had been worn by men who were either genetically similar to the rater (having 3 or 4 of a possible six alleles at the typed loci in common) or genetically dissimilar (having one allele or none in common). Finally, for purposes of analysis, raters who were taking oral contraceptives were distinguished from those who were not. The principal findings were that naturally cycling women rated the odours of "genetically dissimilar" men as significantly more pleasant than those of "genetically similar" men, whereas those who were "on the pill" did precisely the opposite.

This study has been deservedly influential (garnering 703 Google Scholar citations as of 21 June, 2014); its results have been upheld and variously extended. But why conduct such a study in the first place? Why hypothesize that a man's MHC genotype might be a determinant of women's responses to his odour? Why hypothesize that any such effect would be self-referential, depending on the woman's own genotype? And why ask whether the women were using oral contraceptives? The answer to all these questions is, of course, that the specific causal hypotheses that were tested in the experiment were inspired by a set of hypotheses about the adaptive functions of women's affective responses to male odours: that affective response to male odours plays a role in mate choice; that MHC-dissimilar partners should be preferred because homozygosities at MHC loci are detrimental to offspring health; and that oral contraceptives induce an infertile psychophysiological state in which the primary function of women's affiliative preferences lies in the realm of social support, not mate choice.

Note that the Wedekind experiment never pretended to be a "test" of these functional hypotheses, although the results certainly enhance their plausibility. The hypotheses that *were* put to test were strictly causal ones: that self-similarity at certain genetic loci would influence women's affective responses to male odours, and that a rater's hormonal state would modulate her affective responses. In a sense, this is everyday psychological research: discovering hitherto unknown causal impacts on psychological processes is what experimental psychology is all about. However, a psychologist without a Darwinian overview would never have asked the right questions. Discovering these particular effects required familiarity with functional theorizing about the adaptive challenges inherent in mate choice.

So functional theories certainly *can* facilitate the generation of worthy hypotheses about causal mechanisms. But might there be an even bigger lesson? Mayr (1983) has argued that *every* important discovery in physiology and other mechanistic fields of biology has been crucially grounded in the researchers' interpretations of the functions and concomitant "design features" of the systems under study. Before Harvey deduced that the heart's primary function is that of a pump, for example, no progress could be made in understanding its actions. And there could be no neuroscience – that quintessentially mechanistic approach to explaining the causation of behaviour – prior to the relatively recent discovery that information processing is what nerve tissue is *for*. If Mayr is correct in claiming that it is scarcely possible to investigate cause *without* making implicit assumptions about function, then a proscription on explicit functional theorizing amounts to an insistence that those assumptions remain covert and unexamined (Daly and Wilson, 1995). That can't be a good thing.

2. Functional theories as task analyses

Jerry Hogan never bought these arguments. A forceful published statement of his views is to be found in Hogan (1994), where he maintains that "cause and function are logically distinct" (p. 12) and then complains that "the biological definition of function as 'consequences that have been selected' and current biological usage of the phrase 'ultimate cause' both imply some sort of causal relationship between cause and function, a relationship that I maintain is teleological (and un-Darwinian)". Lest one wonder exactly what he means by "teleology", he approvingly quotes the American Heritage Dictionary's definition: "the belief that natural processes are not determined by mechanism but rather by their utility in an overall natural design".

Arguably, this teleologist is a straw man. I have never met a behavioural ecologist (or anyone else) who subscribed to such an either-or opposition of "mechanism" versus "utility". What really rankled Jerry, I believe, were suggestions that studies of "mere" causation in the absence of an explicit selectionist rationale were dull, a conceptual, even pointless. And such suggestions did indeed abound in the hubristic early days of the "sociobiological revolution". But things soon improved.

Consider Charnov's (1976) "marginal value theorem", one of the classic theoretical models in behavioural ecology. The problem that Charnov posed is how a forager could best exploit a resource distributed in discrete depletable patches. According to his elegant model of the problem, the optimal solution, if one is to maximize rate of return, is to leave partially depleted patches to seek fresh ones when the instantaneous rate of food-getting from the present patch equals the highest gross rate of return that can be attained over the total time spent foraging within patches plus travelling between and/or searching for patches.

A number of studies "testing" Charnov's theory were soon forthcoming. In some cases, the data matched its predictions fairly closely, in others not (Pyke, 1984). But how close a fit is close enough? The basic theory doesn't provide an expected distribution of errors. Mechanistic "details" that it omits include such formidable problems as how foragers should derive an estimate of a patch's instantaneous rate of yield from encounters that are distributed stochastically, and how they should induce the habitat-specific expected inter-patch travel time. Almost

immediately, researchers realized that these issues could not simply be set aside, because unless one addressed them, it was impossible to specify what would constitute a potentially falsifying test of the theory. Do departures from prediction reflect a mere shortfall in the information available to guide behavioural decisions, or the omission of some crucial consideration from the optimality analysis?

This question inevitably led to hypotheses about information processing and decision rules, hypotheses that were contrived to predict causal mechanisms from functional considerations of what would be optimal. How close to optimal performance could a forager get by using different patch departure rules, and which ones were actually being used (Krebs, 1978; Green, 1984)? How would the answers change if memory loads and computational demands were treated as costs? Optimal foraging theorists found it necessary to address issues like the optimal investment of time in information gathering (Stephens and Krebs, 1986) and the form of optimal forgetting functions for obsolescing information (Healy, 1992). And as these issues were tackled, it became impossible to classify work in the area as being about cause *or* function. The topic had become the interplay between cause *and* function: what would an optimal cognitive programme for solving this patch-foraging problem look like, what decision processes do animals actually use, and if the actual processes don't seem well-structured for solving the problem, what have we overlooked?

A common mistake is to think of models like Charnov's marginal value theorem as directly testable theories about how animals will behave. They are better construed as instances of what Marr (1982) called "task analyses": theoretical characterizations of the essential features of some adaptive problem that animals regularly confront in nature, and that we might expect natural selection to have equipped them to solve. Cosmides and Tooby (1987) argue that these evolved solutions are often best characterized at a cognitive level, as algorithms for information processing and behavioural decision making, and it seems to me that this sort of theorizing is highly compatible with – indeed, essentially identical to – the sort of causal analysis of "behaviour systems" that Hogan (1994, 2005) advocates. Nor is it just in foraging theory that behavioural ecology has come around to this cognitivist focus on causal mechanisms. The same thing has happened, for similar reasons, in theory and research about parental care (Daly and Wilson, 1988), predation avoidance (Lima and Dill, 1990), and kin recognition (Krupp et al., 2011).

Hogan (2005, Fig. 2) portrays a 40-year rise in studies of function in the journal *Animal Behaviour*, and a mirror-imaged decline in studies of causation, trends that he laments. But the zero-sum aspect of these apparent trends derives from his having classified each paper as being about one or the other. I don't see the chasm between causal and functional analyses that Jerry sees, and I believe that if he were to examine the ostensible studies of function again, he would find that a great many used functional considerations to generate causal hypotheses that were then put to test by standard experimental methods.

3. Cause–function muddles

In conclusion, I must be clear about one thing: the arguments above do not constitute a dismissal of the problem of failures to distinguish cause from function. Such muddles are real, and they have not gone away. In the literature on evolution and human behaviour, for example, "fitness maximization" is regularly invoked as an "explanation" for observed patterns of behaviour, even in evolutionarily novel circumstances in which we have no warrant for expecting that the operation of evolved psychological mechanisms should maximize fitness, and in which such maximization is clearly not being achieved (Symons, 1989).

A double-layered example of cause–function confusion occurs in the first sentence of the Wikipedia entry entitled "kin selection" (or at least in today's version, 21 June, 2014): "kin selection is the evolutionary strategy that favours the reproductive success of an organism's relatives, even at a cost to the organism's own survival and reproduction". Kin selection is *not* a "strategy", not even in the metaphorical sense in which jockeying for position on a lek (say) *is* a component of a "reproductive strategy". It is instead a theoretical characterization of the natural selective process. Maynard Smith (1964) did a great disservice to the field of social evolution when he rebranded Hamilton (1964) inclusive fitness theory as "kin selection", for two main reasons. First, this term has been misunderstood (e.g. by Wilson, 1975) as referring to a level of selection intermediate between "group selection" and "individual selection", when it does not. Individual selection is the systematic differential reproduction of types of individuals, and group selection is the systematic differential reproduction of types of groups, but Hamilton's theory is *not* about the differential reproduction of types of "kins". Secondly, the term is routinely misapplied to an hypothesized *product of* "kin selection", namely action that favours kin ("nepotism"), rather than being applied appropriately to the population genetical selective process. This has inspired a lot of muddled language and at least some muddled thinking, including frequent statements to the effect that some action is (or is not) "motivated by kin selection". Whatever it may be, "kin selection" is *not* properly a motivational concept.

These are the sorts of muddles that Jerry Hogan has decried. But can they be blamed, as he suggests, on teleological thinking that misguidedly equates an attribute's "function" with what it has evolved to achieve? I don't think so. Defining function in terms of past selective consequences has been standard practice in evolutionary biology for over half a century, and for good reason: fortuitous beneficial effects would otherwise have the same status as genuine adaptations, and Dr. Pangloss would have been correct in interpreting his nose as a device for holding his glasses up. This is not to deny that making inferences about selection in the past is tricky, but Williams (1966) provided us with a roadmap for identifying adaptive function that has served evolutionists well. It is also worth noting that cause–function confusions are far more prevalent in the secondary literature (see, e.g. Park, 2007) than in works by behavioural ecologists. Blaming behavioural ecologists for those muddles seems to me to be no more justified than blaming students of behavioural development for the persistence of inane statements about "nature versus nurture".

After completing this essay, I received the paper with which this special issue begins (Hogan, 2014). After some thought, I don't believe that this latest statement of Jerry's position requires any substantial change in what I have written above, but I do feel compelled to react to one point. Hogan (2014, proof page 7) accuses me, among others, of arguing mistakenly "that natural selection is a mechanism, and therefore a cause of evolution". I fully agree that natural selection is *not* a mechanism (and I hope I never called it one!). "Mechanisms" are devices organized to accomplish specific functions, whereas natural selection is a statistical process with no function. By virtue of its nonrandom, cumulative action, however, this aimless process is very much "a cause of evolution", indeed *the* crucial causal process that brings functional mechanisms into existence and hones their "designs". That is Darwin's theory, and it's the only scientific explanation that we have – or need – for why adaptations exist.

Jerry Hogan encouraged his students to be argumentative, and I suppose I am. I have tried to emulate his excellent example in my treatment of my own students.

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