The Modal Theory of Function: Lessons from Molecular Biology

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Abstract

The modal theory of biological function (Nanay 2010) states that the token trait x of an organism has a function F iff if x were F-ing, this would contribute to the organism's fitness. Nanay claims that the modal theory has two main advantages: first, in contrast to etiological or systemic theories of function, the modal theory is trait-type independent; second, the modal theory explains malfunctioning without taking recourse to normativity. In this paper, I scrutinize the modal theory's counterfactual semantics and the applicability of the modal theory to molecular biology, neither of which has received much attention in recent criticisms. Based on a case study of the tumor-supressor gene p53's malfunctioning in human cancer (Vogelstein et al. 2000), I argue that the modal theory cannot fulfill its promise. First, I show that a function ascription to p53 requires individuating trait-types because tumor suppression crucially depends on the concentration of tokens of a certain trait-type, namely the type 'p53 protein'. Second, I reveal that by failing to ascribe the function of suppressing tumors to p53 ('if p53 were inducing apoptosis, this would contribute to the organism's fitness' is ceteris paribus false), the modal theory contradicts an established function ascriptions in molecular biology. Finally, I propose a revised modal theory capable of explaining (mal)function in molecular biology without taking recourse to normativity.
Nanay, Bence (2010): "A modal theory of function", Journal of Philosophy, 107(8), 412-431.
Vogelstein, Bert et al. (2000): "Surfing the p53 network", Nature, 408, 307-310.

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