Fourth European Advanced Seminar in the Philosophy of the Life Sciences

Function and Malfunction in the Biological and Biomedical Sciences and the Social Sciences

September 5th – 9th, 2016

KLI Klosterneuburg,

Austria

Participating institutions

Egenis, the Centre for the Study of Life Sciences, Exeter
Institut d'Histoire et de Philosophie des Sciences et des Techniques,
Paris-1 Panthéon - Sorbonne
KLI Institute, Klosterneuburg/Vienna
IUFE, Faculty of Science, University of Geneva
Department of Philosophy, Faculty of Humanities, University of Geneva
Department of Logic and Philosophy of Science, University of the Basque Country,
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Institute for Philosophy, University of Hannover

Directors

Jean Gayon (Paris) & Alvaro Moreno (San Sebastian)

FACULTÉ DES LETTRES
DÉPARTEMENT DE PHILOSOPHIE

Welcome

to the Fourth European Advanced School for the Philosophy of Life Sciences, which is organized by six top-level European institutions in the philosophy and history of the life sciences. EASPLS aims at fostering research, advancement of students, and collaborations in the field of the philosophy of the biomedical sciences. Meetings are held every other year. After a preliminary meeting in Gorino Sullam (Italy) in 2008, EASPLS met in Hermance near Geneva in 2010 and 2012, and in Klosterneuburg near Vienna in 2014. The present meeting is again hosted by the KLI in Klosterneuburg.

This year's seminar topic is "Function and Malfunction in the Biological and Biomedical Sciences and the Social Sciences." The schedule mixes presentations of senior researchers, post-doctoral researchers, and PhD students from thirteen countries and three continents. The best junior papers resulting from the meeting will be published in a thematic issue of an international journal in the field. Submissions will be subject to normal peer review.

We are delighted that you are able to participate in this seminar, and we wish you a productive and enjoyable stay!

Jean Gayon (Director EASPLS 2016)
Alvaro Moreno (Director EASPLS 2016)
Isabella Sarto-Jackson (Local Organizer EASPLS 2016)

Function and Malfunction In the Biological and Biomedical Sciences and Social Sciences

Functional ascriptions and functional explanation have been major topics in philosophy of science since the 1950s. A turning point was attained in 1973, when Larry Wright proposed his 'etiological theory of function', according to which 'The function of *X* is *Z* means (a) *X* is there because it does *Z*; (b) *Z* is a consequence (or result) of *X*'s being there.' [1] According to Wright, such a definition of function satisfied three requirements that were essential to him: (1) it offered a criterion for distinguishing a function from a mere effect; (2) it applied both to biology and to artifacts; (3) it was able to capture the normativity of functional ascriptions, that is the implicit assumptions that malfunction is always a possibility (a given object may have a function, and nevertheless be unable to accomplish that function).

Shortly after Wright's article, in 1975, Robert Cummins proposed a very different definition of function, according to which ascribing a function to something 'is to ascribe a capacity to it which is singled out by its role in an analysis of some capacity of a containing system'[2]. Contrary to the 'etiological' theory, which looks backwards, the 'causal role' theory of function looks forward. As Wright's concept of function, Cummins' concept applied both to biological and technical objects, but did not take into account normativity. In open opposition to Wright, Cummins insisted that functional ascriptions had nothing to do with the past history of a system, and should be understood exclusively from the viewpoint of the present organization of a system. Because Cummins also (and erroneously), reduced Wright's concept of function to that of 'selected effect', Wright's and Cummins' seminal papers were the origin of an ongoing debate between authors adhering to 'backward looking' or 'evolutionary' theories of function, and authors defending a 'forward looking' or 'dispositional' theories of function.

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[1] Larry Wright, "Functions", *The Philosophical Review*, 82 (1973): 139-168
[2] Robert Cummins, "Functional analysis", *The Journal of Philosophy*, 72 (1975): 741-765

Another philosopher who played an important role was Christopher Boorse, who proposed in 1976 to define function as the causal contribution of something to a goal in a teleological system[3]. This concept is close to Cummins's concept, but the originality of Boorse was to connect the debate of function with the debate on health and disease. For Boorse, function is a non-normative concept, itself part of a non-normative concept of disease and health: disease is no more than dysfunction; and health is 'typical functioning', defined in terms of available physiological knowledge and statistical normality[4]. Correlatively, for Boorse, in sum, normative issues come into play only for a subclass of human disease, which he calls 'illness'. Illness is disease plus subjective and social components[5]. Yet, since Boorse defines disease in terms of statistical abnormal functioning of a specific trait in comparison with the average functioning of traits of the same type in individuals of a concrete "reference class" (members of the same species, gender and age), and health, instead, as simply the absence of disease, its view raises many difficulties to establish a clear frontier between healthy (normal) and unhealthy (abnormal) levels of functioning without adducing subjective and arbitrary considerations. And in this sense, it is dubious that this approach can be really consistent with a biologically grounded theory of functions.

This debate on the other hand, goes beyond the domain of Life Sciences and affects in many aspects nuclear questions of the social sciences. In particular, the debate about functions and malfunctions has affected directly the philosophy of technology, questioning whether the biological theories of function (and malfunction) could or could not be applied to human made artifacts.

The purpose of the 4th EASPLS is to reassess the modern philosophical debate on function in the dual perspective of (1) malfunction (or dysfunction), and (2) with respect to the use of such concepts in both the biological and the social sciences, with a particular concern for the interrelations and interactions between these two fields. Applicants are expected to submit a title and an abstract that fit with this overall

[3] Christopher Boorse, "Wright on functions", *The Philosophical Review,* 85, (1976) : 70-86
[4] Christopher Boorse, "Health as a theoretical concept", Philosophy of Science, 44 (1977): 542-573
[5] Christopher Boorse, "On the distinction between disease and illness", *Philosophy and Public Affairs,* 5 (1975): 49-68.

scheme. Here is a list of particular questions illustrating the general question. This list should be taken as open rather than exhaustive.

- Should the concept of function leave room for normativity? If yes, how?
- How does this relate to reflections about malfunction?
- To what extent does the debate about health and disease in the philosophy of medicine meet with the function/malfunction debate?
- How can the social sciences contribute/have contributed to this debate?
- Speaking of malfunction seems to imply that there is something like “normal” functioning (Boorse); can this be an objective concept or not?
- What is the reference system for the concept of function/malfunction (*e.g.*, levels of organization below and above the organismal level)?
- To what extent is it appropriate to speak of function or malfunction in the social or economical sciences?
- Do we need a common concept of function and malfunction for the biological, social, and technological domains?
- Has the philosophical reflection about malfunction, dysfunction, and abnormality significantly evolved over the past 40 years?

The program at a glance

	Mon, 5 Sept.	Tue, 6 Sept.	Wed, 7 Sept.	Thu, 8 Sept.	Fri, 9 Sept.
Chairs		**Etxeberria**	**Moreno**	**Weber**	**Reydon**
9:35		**Moosavi** Discussion **Suarez** Discussion	**Nordenfelt** **Huber** ***commentary*** Discussion	**Parker** Discussion **Hernandez** Discussion	**Krohs** **Prevost** ***commentary*** Discussion
10:35			Break		Break
10:45		Break		Break	
11:00 11:10	**Registration**	**Round Table** **Dzah** **Hladky** **Rivelli** **&** **Moderation** **Jaeger**	**Roux** Discussion **Conley** Discussion **Canciani** Discussion	**Round Table** **Bonnin** **Hundertmark** **Passariello** **&** **Moderation** **Arnellos**	**Loison** **Concluding Remarks** & General Discussion Publication Plans
12:45	Lunch	Lunch	Lunch	Lunch	Lunch
Chairs	**Sarto**	**Nicholson**	**Glackin**	**Mossio**	
14:15 14:25	**Müller** **Welcome Address** **Moreno** **Opening Lecture** Discussion	**Reydon** **Karaca** ***commentary*** Discussion	**Mossio** **Betzler** ***commentary*** Discussion	**Glackin** **Asselin** ***commentary*** Discussion	
15:15	**Nicholson** **Anderson** ***commentary*** Discussion	**Weber** **Kornienko** ***commentary*** Discussion	**Etxeberria** **Canali** ***commentary*** Discussion	**Sarto** **Fernandez** ***commentary*** Discussion	
16:15	Break	Break	Break	Break	
16:40 17:15	**Lefèvre** Discussion	**Ardern** Discussion	**DiFrisco** Discussion	**Dieli** Discussion	

Detailed program

Monday, 5 September 2016

11:00 Registration

12:45 Lunch

14:15 **Gerd B. Müller**

Welcome Address

14:25 **Alvaro Moreno**

Opening Lecture

"What are Biological Malfunctions?"

15:00 Discussion

15:15 **Daniel Nicholson**

"Structure and Function: A Process-Centred View"

15:50 **Wesley Anderson**

Commentary

16:05 Discussion

16:15 Coffee break

16:40 **Victor Lefèvre**

"'On Defense of the Organizational Account of Ecological Functions"

17:05 Discussion

Tuesday, 6 September 2016

09:35 **Parisa Moosavi**

"Can the Biological Accounts of Function Be Applied to Human Morality?"

10:00 Discussion

10:10 **David Suarez Pascal**

"Function and Value: Calibrating Philosophical Theories of Function Through Their Evaluative Compromises"

10:35 Discussion

10:45 Coffee break

11:30 **Round Table Discussion**

"Function, Normativity, and Teleology in Biology and Technology"

Daniel Dzah

Michal Hladky

Luca Rivelli

Moderated by **Johannes Jaeger**

12:30 Discussion

12:45 Lunch

14:15 **Thomas Reydon**

"Kind-Formation for Functionally Defined Groups"

14:50 **Caglar Karaca**

Commentary

15:05 Discussion

15:15 **Marcel Weber**

"How Objective Are Biological Functions?"

15:50 **Aleksandra Kornienko**

Commentary

16:05 Discussion

16:15 Coffee break

16:40 **Zachary Ardern**

"'Evolution, Dysfunction, and Disease: A Response to Griffith and Matthewson"

17:05 Discussion

17:30 **Business Meeting** of the EASPLS Partners

Wednesday, 7 September 2016

09:35 **Lennart Nordenfelt**
"Functions and Health"
10:10 **Maximilian Huber**
Commentary
10:25 Discussion
10:35 Coffee break
11:00 **Étienne Roux**
"Normality, Function, Dysfunction in Biology: Function as a Variable"
11:25 Discussion
11:35 **Brandon Conley**
"Putting Dysfunction to Work in Functional Analysis"
12:00 Discussion
12:10 **Mark Canciani**
"The Organizational Theory of Functions and Higher-Level Functions"
12:35 Discussion
12:45 Lunch
14:15 **Matteo Mossio**
"The Organizational Account of Biological Functions"
14:50 **Riana Betzler**
Commentary
15:05 Discussion
15:15 **Arantza Etxeberria**
"On the Biological Organization of Pathologies: Functions, Relations and the 'Normal–Broken' View"
15:50 **Stefano Canali**
Commentary
16:05 Discussion
16:15 Coffee break

16:40 **James DiFrisco**

"Functional Explanation and Functional Equivalents"

17:05 Discussion

Thursday, 8 September 2016

09:35 **Christopher Parker**

"Two Varieties of Dysfunction in Mental Disorder"

10:00 Discussion

10:10 **Paola Hernandez Chavez**

"Cognitive Dysfunctions in Neuroscience: Stuck in Old Biology"

10:35 Discussion

10:45 Coffee break

11:30 **Round Table Discussion**

"Philosophical Theories of Biological Function"

Thomas Bonnin

Fabian Hundertmark

Alessandra Passariello

Moderated by **Argyris Arnellos**

12:30 Discussion

12:45 Lunch

14:15 **Shane Glackin**

"Mad Disease and Martian Disease"

14:50 **Jennifer Asselin**

Commentary

15:05 Discussion

15:15 **Isabella Sarto-Jackson**

"Plasticity of Functions: The Plasticity–Pathology Continuum of the Nervous System"

15:50 **Eva Labandera Fernandez**

Commentary

16:05 Discussion

16:15 Coffee break

16:40 **Anna Maria Dieli**

"Function as a Goal Oriented Behaviour: The Case of Cancer"

17:05 Discussion

17:30 Dinner at the KLI

Friday, 9 September 2016

09:35 **Ulrich Krohs**

"Tba"

10:10 **David Prévost-Gagnon**

Commentary

10:25 Discussion

10:35 Coffee break

11:00 **Laurent Loison**

Concluding remarks

General Discussion

12:30 **Publication Plans**

12:45 Farewell Lunch

Abstracts

Representing Causation in the Biological Sciences

Wesley Anderson *(KLI Klosterneuburg)*

Most philosophers and biologists work with models that describe phenomena that are inherently causal without providing counterfactual predictions of what would happen to one variable given an intervention on another variable. These models consequently are at best only implicitly causal. The aim of my research is to understand various biological systems from ecology, epidemiology, evolutionary genetics, evolutionary game theory, conservation biology, and cultural evolution through the mathematical framework of causal modeling. In these models, causal relationships are represented by directed edges from one variable to another and represented by structural equations. As I use these representational tools, my main objective is to understand the methodological benefits (and costs) that emerge when explicitly representing the causal structure of systems. From the point of view of philosophy, I use explicitly causal representations of systems to shed light on conceptual issues from the philosophy of science such as what is an adaptation and what is group selection. From the point of view of biology, I use the framework to develop methodological theses assuming we are dealing certain underlying causal structures.

Evolution, Dysfunction, and Disease: A Response to Griffiths and Matthewson

Zachary Ardern *(University of Auckland)*

In a paper forthcoming in the British Journal for Philosophy of Science, Paul Griffiths and John Matthewson argue that selected effects play the key role in determining whether a state is pathological. In response, it is argued that a selected effects account faces unrecognised difficulties, particularly in light of modern genomic research. I explain four difficulties posed by evolutionary human genetics for a selective effects account of dysfunction. Firstly, a modern history approach to selection is shown to be a poor basis for assigning function to human traits in light of small population sizes in the hominin lineage. The average effective population size of humans has been under 10,000 for most of the duration of the lineage; this implies that selection has had limited effect on human-specific features of the human genome. The ENCODE project's attribution of function to large areas of the genome, including many disease-associated variants, consequently calls into question a selected effects account of dysfunction for these genetic elements. ENCODE discovered repeatable biochemical signatures associated with approximately 80% of the human genome, and argued that these regions were therefore functional. Only a limited portion of the genome however, approximately 10%, shows signatures of selection. The ENCODE project's claim was highly controversial, but the consequence for the functions debate is that either many functional elements are not selected, and so a selected effects account is inadequate for biological function, or otherwise many non-functional elements are relevant to disease. In either case, a selected effects account of dysfunction in the context of health faces a serious challenge from non-selected disease-causing variants. Secondly, determining the genetic basis of disease, let alone the selective histories of the various alleles involved, is often experimentally intractable. The genetic factors contributing to disease are generally complex, involving different combinations of multiple alleles. The selective histories stretching deep into the past of these complex interactions are beyond the reach of the modern experimenter, making inferences here generally tentative at best. If only recent evolutionary history is deemed relevant, a selected effects account will not be explanatory for most genetic variants contributing to disease,

given the limited proportion of the genome that is under selective pressure in the human lineage. Thirdly, the existence of ‘selected disorders’ is well established. Some genetic elements underlying disorders are demonstrably also evolutionarily advantageous, but in modern social contexts the overall result of these alleles is detrimental to health. Fourthly, many important diseases do not plausibly reduce evolutionary fitness. This is just one instance where the end promoted by natural selection, maximal reproductive success of a lineage, does not align exactly with the aims of healthcare. Because of this mismatch, there are situations where evolution and medicine may be antagonistic forces. Finally, in light of these arguments, a normative account of dysfunction is suggested to have advantages in the context of health over attempts to naturalize this property. Difficulties with the alternative biostatistical account have been highlighted by Griffiths and Matthewson, leaving naturalistic explanations in a quandary over how to deal with the insufficiencies of the main contenders.

The Function of Dysfunction in Mental Disorder

Jennifer Asselin *(Ohio State University)*

When it comes to analyzing the concepts surrounding health and illness, normativists argue that such concepts are impossible to define in non-evaluative terms. In “On the Distinction between Disease and Illness,” Christopher Boorse argues that all versions of normativism are objectionable and that all attempts to analyze health concepts in normative terms are misguided. Assuming that Boorse is correct, we are now in the position of needing to analyze health and illness concepts in purely non-normative terms. In this paper I argue that our MENTAL DISORDER concept cannot be analyzed in purely non-normative terms. If this is the case, then there are at least some health related concepts that cannot be analyzed as Boorse intends and Boorse’s argument that all health and illness concepts must be analyzed purely non-normatively fails.

I argue in this paper that there is no non-normative necessary condition for being a mental disorder. Since analyses of concepts give necessary and sufficient conditions, if there is no unifying non-normative necessary condition for being a mental disorder, then there is no unifying non-normative analysis. A unifying analysis asks for an analysis of the concept that does not present several sufficient conditions, the disjunction of which, is necessary for the concept. I argue in this paper that such a disjunctive analysis of being a mental disorder fails to provide us with a successful account of our concept MENTAL DISORDER. Ultimately I conclude that since there is no non-normative unifying necessary condition for being a mental disorder, then there is no non-normative account of being a mental disorder.

If there is a unifying non-normative necessary condition for being a mental disorder, then our best candidate is likely some variant on being a dysfunction as mental disorders are commonly thought to occur as the result of something having gone wrong internally. The paper thus proceeds by arguing that the most popular non-normative accounts of being a dysfunction fail as necessary conditions for being a mental disorder. The etiological account of being a dysfunction (as presented by Larry Wright and Jerome Wakefield) is trivially true and therefore fails to be an informative condition for

being a mental disorder. A Boorsian account that claims that being a statistical account of function is necessary for being a mental disorder is not extensionally adequate as it fails to deliver the correct verdict for paradigm cases of mental disorder. Finally, the Robert Cummins style account that claims that being a system theoretic dysfunction is necessary for being a mental disorder also fails to be extensionally adequate. With no successful account, I conclude that currently no account of being a dysfunction is necessary for being a mental disorder. Since being a dysfunction was our best candidate for a non-normative necessary condition for being a mental disorder, we are left with no non-normative necessary condition for being a mental disorder. As there is no non-normative necessary condition for being a mental disorder, there is no non-normative account of being a mental disorder.

Functions, Norms, and the Moral Emotions

Riana Betzler *(University of Cambridge & KLI Klosterneuburg)*

Genealogical approaches, broadly construed, trace the origins of a particular biological capacity or cultural concept through (1) functional speculation and (2) detailed history. These types of genealogical approaches have become popular for things that have characteristics of both social and natural kinds (e.g. Haslanger, 2006; Prinz, 2007). They also often have interesting, and potentially problematic, normative implications. In this paper, I will consider what, if anything, such genealogical approaches may contribute to normative pursuits. I will do so by looking specifically at Jesse Prinz' (2007) exposition of genealogical approaches to morals and his analysis of where norms enter the picture in his book, The Emotional Construction of Morals. Prinz (2007) argues that our values should undergo genealogical critique, or in other words, that our investigation of their history may lead us to reassess their role within our contemporary society. The historical details themselves cannot, on Prinz' view, carry normative force. Rather, to evaluate our values, Prinz suggests that we need to consider their function. He does not, however, provide us with a detailed account of function or explain how function might carry normative force. In this paper, I will consider whether contemporary accounts of function may fit in with Prinz' project and provide room for normativity—or whether Prinz has placed too much of a burden on the notion of "function."

The Roles of “Function-talk” in Evolutionary Reconstructions

Thomas Bonnin *(Egenis, Exeter)*

Functions are often perceived to be the reason a trait is selected for. This understanding assumes an evolutionary grounding to the emergence to present-day biological structure. This presentation aims at understanding the discursive roles of functions of present-day structures in the discussion of their emergence. To do this, I will focus on attempts at explaining the emergence of eukaryotes, and the relation to the emergence of mitochondria, the “power plant” of the cell. How does our functional understanding of mitochondria influence the elaboration of evolutionary scenario for its emergence? What happens when this understanding changes? Does understanding of mitochondrial evolution influence present-day understanding of these organisms?
Tom Cavalier-Smith and William Martin have very different views on these matters, can this disagreement be partially explained by differing views on the functions of these entities? This talk hopefully will highlight the diversity of “function-talk” in the practices of evolutionary biologists.

The Role Of Functions In Data-Intensive Research

Stefano Canali *(University of Hannover)*

The main theme of my PhD research project regards the practices and epistemological consequences of the use of big data in the life sciences. It is often argued that big data brings about an epistemological revolution in the sciences, in the sense that the availability of increasing amount of data allows for a new kind of science, possibly driven by data only in an unbiased and objective way. At the same time, however, a number of authors in the philosophy of science have raised concerns about the application of these views, especially regarding the domain of the life sciences. Therefore, in my project, I study whether and how big data changes scientific epistemology and I aim at developing a comprehensive account of big data science. In particular, this means that I review the practices involved in each main stage of research (data curation, exploration and hypotheses generation, statistical analysis, etc.) from the perspective of philosophical and methodological discussion. In this way, I hope to build a broad understanding of methodological choices in the use of big data use in the life sciences and develop possible improvements to big data methodology.

Thus, significant part of my research is based on the use of established philosophical concepts to understand novelties and continuities of big data science. From this perspective, I would argue that the concept of function plays an important role. On the face of it, one might say that big data does not help with the discovery of functions or malfunctions: the value of having big datasets is usually presented as a consequence of the large amount of correlations which can be found; correlations might help with the development of predictions, but it unlikely proves to be useful for the discovery and study of functions. In other words, one might say that the study of functions cannot be carried out with a data-driven methodology, but only in a hypothesis-driven one. In my research, I investigate a number of big data projects to try and assess this kind of claims. One of these cases is EXPOsOMICS (www.exposomicsproject.eu), a EU-funded biomedical project which studies the relation between exposure and chronic disease: interestingly, researchers aim at improving our understanding of disease

mechanism, causal paths and functions as well. Consequently, I think that a number of important questions arises. One might say that EXPOsOMICS suggests that disease function can also be studied in a data-driven way, or that the projects' methodology is not really data-driven. At the same time, one may also raise questions about which concept of function is employed as part of EXPOsOMICS and what the big data research developed in the project say in connection with the philosophical debate on function. One may also say that the project's methodology, based on the study of causal path linking exposure to disease, suggests causal theories of function and that we do not need new theories of functions for big data science.

The Organizational Theory of Functions and Higher-Level Functions

Mark Canciani *(Universidad del Pais Vasco, San Sebastian)*

The philosophical debate on biological functions has historically mainly focused on functions at the level of organisms and below; for instance, the function of flying, the function of wings, the function of hearts, the function of DNA, etc. But in biology functional explanations are also used at levels higher than organisms, for example in ecology, as in the function of a particular component of biodiversity on nutrient recycling within an ecosystem. But is this higher-level functional explanation justified? Are there actually genuine higher-level functions? In this presentation I will examine whether higher-level functions exist by using a case study of a eusocial colony of termites, showing the complex feeding process of the colony as a whole and the roles of the different termite workers in this process. I will begin by outlining the organizational theory of functions developed by Mossio, Saborido and Moreno (2009) and show the advantages this theory has over selected effects and dispositional theories; i.e. because it gives a clear definition of what a function is and can account for the teleological and normative aspects of functions better than the latter theories. I will then use the organizational theory of functions to assess whether objective higher-level functions exist. To do this I will outline the colony feeding process of *Macrotermes bellicosus*, a fungus-cultivating termite. Showing how colonies in this species forage for food, process the food for the fungus comb and how the workers then distribute the older fungus comb as food amongst the colony via trophallaxis. With this case study I will assess whether the behaviour of the two types of workers, major and minor, are just cooperation amongst a group of individuals or genuine higher-level colony functions. Using the organizational theory's criteria of functions I will assess whether the colony is the right type of system that can possess functions and whether the behaviour of the workers fulfils the function criteria.

Putting Dysfunction to Work in Functional Analysis

Brandon Conley *(Cornell University, New York)*

Robert Cummins' classic account of functional analysis is often perceived, even by its most staunch defenders, to have trouble accounting for dysfunction. The problem is that the capacity to φ is a necessary condition, on Cummins' account, for the possession of the function of φ-ing, but being dysfunctional is a matter of lacking the capacity to perform a function. Extant accounts of dysfunction avoid this difficulty by defining dysfunction as deviation from the Cummins-function of some ideal token of a given type. Selected effect accounts, in which the ideal is defined by the Cummins-functions of past tokens which were naturally selected, and Christopher Boorse's biostatistical view, according to which the ideal is defined by statistically normal contribution to a "goal," are the most popular. This strategy has an important drawback: Since the causal profile of some ideal token of a type is causally irrelevant to the workings of other tokens, it is unclear how dysfunction could do explanatory work in functional analysis. From the perspective of functional analysis, dysfunction is equivalent to lack of function. I provide an account of how dysfunction contributes to explanation in functional analysis.

The apparent incoherence of causal role dysfunction arises from insufficient attention to the structure of the dispositional properties which are invoked in, and are the target of, explanations in functional analysis. The standard argument against causal role dysfunction implicitly assumes a fixed set of "appropriate circumstances" for the performance of the function, but I argue that this is a mistake. I define a notion of equivalence classes of dispositional properties and argue that a given functional analysis applies to items with dispositions or capacities falling anywhere within a given equivalence class. Two items can thus possess the same Cummins-function despite considerable variation in the circumstances under which the disposition to perform that function would be manifested. I then argue that considerations about the natural kinds to which we attribute functions constrain the equivalence classes, so we avoid trivial or uninteresting equivalence classes in practice.

Finally, I argue that the notion of dysfunction allows us to locate items within partially ordered equivalence classes, thereby allowing for the expression of more fine grained causal information than function attributions alone can capture. Dysfunction attributions therefore contribute to explanation by providing information about an item's causal role in a system. Extant views are correct to characterize dysfunction as departure from an ideal, but the most useful ideals will be defined, often imprecisely, by the mechanisms uniting the relevant natural kind and by our explanatory goals. In addition to putting dysfunction to work my updated definition undermines a strong form of pluralism, since other accounts of function, including selected effect functions, emerge as special cases. However, a more modest pluralism arises from variation in the factors relevant to selecting an ideal.

Function as a Goal Oriented Behaviour: The Case of Cancer

Anna Maria Dieli *(University of Rome Tor Vergata)*

Cancer, also known as malignant tumour, is a disease involving aberrant proliferation of cells and the ability to invade other tissues. In cancer, cells grow out of control and become invasive: therefore, it is described as a cell disease (Weinberg 2007). The aim of the talk is to analyze from which point of view it can be stated that a cancer cell has lost its physiological functions and why.

The starting point is Boorse's definition of function. According to him, a function is a contribution to a goal, a goal oriented behavior (Boorse 1977). The goal is given by natural selection: a trait has a certain function because it has been selected for that. Weinberg approach to cancer follows this scheme. Cancer cell has a certain activity, which means that it has some functions: cancer cells become independent of external growth signals, insensitive to external anti-growth signals, able to avoid apoptosis, capable of unlimited replication, capable of sustained angiogenesis, and capable of tissue invasion and metastasis. Mutation, competition and natural selection between cells are thus the main components of the phenomenon of cancer (Nowell 1976). Therefore, applying the Darwinian model to cancer could explain why tumours usually originate from a single cell. Among cells, some of them have a selective advantage because of genetic mutations. These mutations could allow the cell to escape the mechanism of apoptosis as well as proliferating quicker than their normal counterparts. Thanks to these characteristics, mutated cells have a selective advantage and become tumorigenic.

However, cancer phenomenon cannot be described merely from a cellular point of view when considering the possibility of reverting the neoplastic phenotype of a tumour cell. For instance, it has been proved that transplanting a cancer cell in a normal tissue not always gives rise to a tumour (Mintz & Illmensee, 1975). A tumour arises as a disruption of the the interaction among cells, within a tissue and an organ and within the whole organism. The microenvironment has, therefore, a role in the development of cancer, which must not be underestimated. Cancer cannot be considered just as a genetic or

cell disease: the systemic perspective has to be taken into account. Therefore, it will be maintained that the failure of Weinberg perspective in explaining cancer has consequences on Boorse's account of function. It is correct to say that a function is a goal directed behavior: what has to be clarified is how the goal is identified and what is considered to be a behavior. The normal functioning of a cell in an organism is commonly considered a coherent activity that maintains the organism alive. On the contrary, as in the case of cancer, the activity of the cell is no more directed toward the survival of the organism. This opens the discussion to other authors who have defined cancer as an aberrant growth and a formless phenomenon, a morphogenesis that goes awry (Aranda-Anzaldo 2002). The study of function and malfunction in cancer cells may help to discuss and adjust Boorse's conception of function.

ARANDA-ANZALDO, A. (2002) "Understanding cancer as a formless phenomenon" in Medical Hypothesis; 59 (1): pp. 68-75.

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MINTZ, B., ILLMENSEE, K. (1975) "Normal genetically mosaic mice produced from malignant teratocarcinoma cells", in Proc Natl Acad Sci USA 72: pp. 3585-3589.

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Functional Explanation and Functional Equivalents

James DiFrisco *(KLI Klosterneuburg)*

This paper examines the problem that functional equivalents pose for functional explanations in biology. In a functional explanation, the existence and/or properties of a trait are explained by its having a given function. For example, hemoglobin has the function of transporting oxygen in the blood, and it exists in some sense because it transports oxygen in the blood. Many philosophers think this form of explanation is legitimate (Nagel 1961; Wright 1973; McLaughlin 2001). But any defense of functional explanation needs to contend with a problem that was originally raised by Hempel (1959) and that motivated his skepticism about biological functions – namely, the problem of functional equivalents.

It seems to be an empirical fact that distinct traits can have the same biological function: hemoglobin transports oxygen in the blood in vertebrates, whereas hemocyanin plays this role in invertebrates such as mollusks. This generates a problem for functional explanations: one cannot explain why a given organism has hemoglobin simply from the fact that it has to transport oxygen in the blood, since it might have had hemocyanin instead. Because the mere function of oxygen transport does not specify how it is realized structurally, the function cannot explain the presence and/or properties of its specific realization.

The problem of functional equivalents featured prominently in Hempel's (1959) and Nagel's (1961) early analyses of functional explanation in terms of the deductive-nomological (DN) model of scientific explanation. A consensus subsequently emerged that functional explanations could not be conformed to the DN model due to functional equivalents, and instead should be interpreted along the lines of the etiological account of functions.

Against the prevailing view, I argue that functional equivalents pose a problem for etiological functional explanations as well as for DN functional explanations. The way in which biologists construct functional explanations is more complex than assigning an

etiological function to a trait, and requires looking at more than just sufficient conditions for function performance. There are also functional explanations in fields like ecology and physiology that resemble the DN model more than the etiological one. In view of these difficulties, I propose an alternative dual model of adaptive explanation and design explanation, parallel to etiological and DN explanation, respectively. I show that this alternative model has greater explanatory scope and depth than the etiological model, and performs better overall on the problem of functional equivalents than either etiological or DN models.

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A Place for Normativity in Functional Analysis

Daniel Hanson Dzah *(Central European University, Budapest)*

Formulating a satisfying functional analysis of organisms and artefacts has proven to be a very difficult task. The failure of conceptual analysis becomes obvious in a thorough study of the various attempts to provide an analysis of malfunction. The systemic capacity account of Cummins (1975) and the goal contribution account of Boorse (1976) have not been concerned with accounting for malfunctions. The etiological account of Wright (1973) remains the only account that attempts an explication of malfunctions. Whereas normativity in functional talk is largely unproblematic for the analysis of artefacts, in the case of organisms teleological notions remain suspect. Still the etiological account in its various revisions remains the only solid claim to an inherently normative account of function/malfunction.

The normativity in question here is one that would pick out an entity that has a function but is at the same time incapable of performing that function. When we ascribe functions to entities, we are inclined to point out instances when they fail to perform these functions that they otherwise ought to or are supposed to perform. Recently, the claim to inherent normativity and hence malfunction explication has been undermined by Davies (2000, 2001). Davies convincingly argues that the normativity ascribed to the etiological accounts is ill-founded.

In response, some have argued that normativity in functional analysis in general (McLaughlin 2009, Franssen 2009) and in the etiological account in particular (Bisset 2016) is not doomed to failure. Franssen's (2009) work is particularly informative as he assesses Davies' charges and clarifies major issues on the supposed normativity of the notion of function in technology and biology. Franssen prescribes a non-literal understanding of normative statements such as "ought to" or "supposed to" and ties the normativity of concepts of function to human intentionality. Significantly, both Franssen and Davies give a rendering of a notion of "expectation" which they place at the heart of our ascriptions of malfunctions. In the realm of biology, Davies believes that human expectations about the performance of types and tokens explains our inclination to

speak of malfunctions. He flags the illegitimacy of this inclination, and suggests a restrictive application of these expectations. Moreover, Franssen provides a rational, epistemic justification for our expectations about biological entities and concludes that Davies' account is too restrictive.

I critically assess the concept of expectation Franssen posits as an epistemic justification and contrast it from a prescriptive expectation for explaining biological malfunctions. I highlight and assess a principle of "responsibility transfer" (Bourgeois 2014) which shows how our epistemic expectations are transferred unto the functional entity itself. I further press the point that in light of this human expectation, as well as its source in our relation to artefacts and the engineering domain, we should be extremely reticent in our ascriptions of malfunction, especially in biology. Our mental projections of functional obligations should be flagged for their strictly epistemic role and thus should be duly separated from ontological conclusions about obligations of functional entities.

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On the Biological Organization of Pathologies: Functions, Relations and the “Normal–Broken” View

Arantza Etxeberria *(University of the Basque Country, San Sebastian)*

Medical knowledge aims to identify different diseases as wrong conditions of biological organization. One main issue within the field of the philosophy of medicine is the question of just how confident we can be that what we know about biological organization will help us to identify diseases and propose cures or treatments for them. The concept of biological organization is a complex abstraction which requires the coexistence of constitutive, interactive and experiential aspects; while the main attempts at naturalist descriptions of the concept (functional, mechanistic and systemic) fail to be fully comprehensive. Different arguments have supported a naturalist normativity in medicine; the strongest such perspective contrasts the normal or typical state of organizational elements with their “broken” versions. However, the complexity of biological organization suggests that there are multiple ways of being healthy or diseased. Thus, the normative goal of medicine of identifying diseases encounters two fundamental questions: 1) Is biology itself normative and can it define the “natural” state? 2) Can medicine rely on knowledge other than biological knowledge to identify what goes wrong? As a normative discipline, medicine comes into conflict with the multiplicity in the very ontology of diseases, which needs to be complemented with epistemic pluralism. Philosophy of medicine therefore needs to explore the sources of that normativity.

Homeostasis and Disease: Analyzing a Systemic Alternative to Functional Accounts

Eva Fernández-Labandera Tejado *(University of the Basque Country, San Sebastián)*

Although medical knowledge often conceptualizes disease as failure in homeostasis, this notion has not been enough analyzed philosophically and it was left aside by authors like Boorse (1977: 550) because it cannot "profitably be viewed as a general model of biological function". In this paper I intend to start with an examination of the relation between homeostasis and health: health has been widely considered as a state of equilibrium, and this notion has inspired classical medical knowledge as well as developments related to the notion of homeostasis, in the tradition starting with Claude Bernard (1865), continuing with Cannon (1932) and Cybernetics (1950+) and, more recently, Systems Biology (see Ahn et al., 2006a and 2006b). The goal is to analyze the framework where diseases can be understood as failures in homeostasis, and to compare it with the functional account.

In order to achieve this, I will attempt to accomplish three goals here, divided in three sections. In the first one I will briefly reconstruct the historical landmarks of the concept of homeostasis in the tradition already mentioned.

In the second section I will specifically examine how pathological states can be conceptualized as failures of homeostasis. Several authors since Cannon have defended that homeostasis is not some fixity, as Bernard said, but an oscillation within an acceptable range. Any account on disease needs to acknowledge which range of homeostasis oscillation is acceptable for an organism to be considered healthy, so to establish where to place the frontier beyond which we are allowed to talk about pathological states or disease. I will take into account some of the ideas brought around with the coming on stage of cybernetics and their notions of feedback loops, as well as more recent ideas of Systems Biology, as means of explanation of the working homeostasis and the processes that make it possible.

The third section intends to reveal what I think is a simplification of homeostasis, which confines it to a mere euphemism of equilibrium of some variables in the organic body (temperature, blood pH, etc.) and brought some problems relative to its connection with disease. Here I will present two contemporary debates about that problematic relation. One is based on Cannon's perception about the link between homeostasis, disease and stress, but broadened to psychosomatic influences. According to this, homeostasis decays with age, leading ultimately to death (Caccioppo and Berntson, 2007). The other perspective is related to problems arising from homeostasis being dependent on environmental conditions. For instance, Dussault and Gagné-Julien (2015) base their proposal on a conception of health as homeostatic maintenance of design (as teleological concept related to function).

Finally, in the last section of this paper I will compare the homeostatic approach to health and disease with the classical functional approach, considering whether disease can be explained in terms of homeostatic oscillations falling out of the allowed range.

Mad Disease and Martian Disease

Shane Glackin *(Egenis, Exeter)*

In David Lewis' classic paper, "Mad Pain and Martian Pain", he investigates the functional correlates of pain with a pair of thought experiments concerning, respectively, a "madman" who is physiologically like us, yet does not display pain behaviour in the same physical states, and a "Martian" who displays pain behaviour in the same states, despite possessing radically different physiology.

I use several variant thought experiments here to interrogate our intuitions concerning the functional role of disease, in order to suggest a new way of looking at the debate about the disease concept, and a consequent "rebranding" of social constructivism about disease. A Lewisian "madman" may possess the same physiology as us, yet differ from us in his judgements about whether or not his physical state counts as diseased; the "classical" debate between etiological and causal accounts of function primarily aims to resolve questions of this sort.

Two other well-known sorts of problem case in the literature, I argue, concern rather different kinds of debate. I present this through the prism of two different sorts of "Martian" found in Phillip K. Dick's *Martian Time-Slip*; an indigenous population which inhabits time in a radically different way from us, raising Davidsonian "swampman"-type objections to the etiological account, and an artificially-selected settler population, corresponding to reference-class problems faced by the causal account.

I go on to argue that a third sort of "Martian" case, suggested by Michael Bérubé's recent analysis of Dick's narrative technique in the novel, helps us to understand the main alternative theory of disease – social constructivism – as a species of modernism, concerned with showing the contingency of the modes of existence, of cognition, and of physical functioning privileged and favoured in a given society. On this view, the facts on which judgements of disease and health supervene are not concerned with a patient's past (as in the etiological function account) or present (the causal function account) states, but with the possibilities available to her for the future.

Cognitive Dysfunctions in Neuroscience: Stuck in Old Biology?

Paola Hernández Chávez *(Centro Lombardo Toledano, Mexico City)*

The aim of this work is to contribute to clarify the classificatory framework for cognitive dysfunctions in neuroscience. From the cognitive stance, it is important to determine if the difficulties we currently face in our understanding of the brain and its malfunctioning arise from poor experimental design, a technology limitation, or an interfering background idea that is biasing our interpretations. I claim this last is the case in cognitive neuroscience, since it is based on the following notions: subtraction method, reverse engineering, residual normality, modularity, and double dissociation. These notions are somehow biased by a closed causality and an agent-centered logic.

I will be showing that illness and dysfunctions are key concepts emerging from a basic notion of causality coming from biology, where the same atomistic logic pattern prevails: a single agent X causes infection X. For example, bacterium *Vibrio cholera* causes cholera; *Treponema pallidum* causes syphilis; H1N1 causes swine flu. The problem is that similar explanations are pursued when accounting for cognitive neuroscience dysfunctions.

Old biology assumptions have been challenged mainly by two fronts. The first one displays evidence showing that few genetic variants, which used to be biology's favorite explanatory framework, in fact, cannot account for a lot of illness risks. Diseases like diabetes, heart disease, and most cancers have no clear genetic, closed causality story that can be traced. These facts have demonstrated that genetic information is not enough to explain prevailing conditions in families/populations.

In the second front, we have authors noticing that the 'organism' disappeared as a fundamental explanatory concept in biology (Webster & Goodwin 1982; Laubichler 2000; Gutmann et al. 2000; Huneman and Wolfe 2010; Cornish-Bowden 2006; Nicholson 2014). So, the need to bring systems biology to the center is becoming imminent. Two ideas are strengthened: there is no privileged scale at which biological

functions are determined (Noble 2012), and we should focus on the organizing principles underlying living systems (Mesarovic, Sreenath, Keene, 2004).

Once it was obvious that old reductionistic paradigm was not successful in determining a vast number of dysfunctions, and old biological basic ideas having been modified; one would expect cascading effects penetrating into cognitive neuroscience. Notwithstanding, such ideological improvements have not been updated in our conceptualizations of dysfunction.

Henceforth, my proposal consists in showing that systems biology would be a way out of that old linearly causal and reductionistic logic, which has mistakenly permeated our ideas of function and dysfunction in cognitive neuroscience. Doing so includes taking into consideration the following facts: organisms, not genes, are the agents of evolution; phenotypic plasticity (when genotype generates different phenotypes depending on the environmental circumstance); that adaptation includes phenotypic innovations that can be genetically inherited; organisms' heterogeneity and dynamics and variability of living organisms (they are in constant change); niche construction (organisms do not just adapt, they also help to construct their environment in a constant feedback).

An interesting case incorporating an organism-based biology, or a systematic approach, is Noble's modeling of the heart cells (Noble 2012, 58). As he notices, there is not a program for cardiac rhythm in the genome. This applies not only to circadian rhythm but to all functions that require cellular structural inheritance as well as genome inheritance. From there he concludes, "we cannot yet characterize all the relevant concentrations of transcription factors and epigenetic influences. It is ignorance of all those forms of downward causation that is impeding progress."
(Noble 2012: 60)

I will conclude by quoting Cornish-Bowden since he summarizes a recommendation I would like to extend to dysfunctions in cognitive neuroscience: "As long as an organism is treated as no more than a collection of components, one cannot ask the right questions, and certainly cannot answer them" (Cornish Bowden 2006: 475).

Functions in Biology and in Technology: A Systemic Account

Michal Hladky *(University of Geneva)*

The notion of function plays a central role in biological explanations. In engineering, it is used in design and in analysis of technologies. The different uses of the term in these disciplines would suggest that there are different kinds of functions. Furthermore, even in the domain of biology, different analyses can attribute different functions to the same feature of the same organism.

For instance, Aristotle, contrary to Plato, thought that the heart was the seat of sensations and intelligence and the role of the brain was important but limited to the cooling of the heart (Gross 1995). After a basic course of biology, one would dissociate the sensations from functions of the heart and rather say that the function of a heart and even an artificial one is to pump blood. But is it? A study of heart–brain system in a patient with an artificial heart suggests that the heart affects the emotional and cognitive processes (Couto et al. 2014).

Aristotelian teleological explanations had the advantage of being applicable to both biological organisms and artefacts. However, the teleological notions are not satisfactory for empirical science, such as biology. Darwin's theory of evolution and natural selection removed teleology and a designer from the explanations concerning biological systems, but did not affect functional attributions based on intentions in artificially created systems.

The distinction between functions in biology and in technology would be acceptable if one believed that there is a fundamental difference between living systems and artificial (man-made) systems. A consequence of such a distinction is the multiplicity of the meanings of 'function' – one for biology, one for artefacts. Even if it seems that taking intentions into account for functional ascriptions in man-made systems is not problematic, it is not clear whose intentions should count. An obvious choice would be the designer's intentions. However, there are cases of failed design but successful use. The problem with user's intentions is that there may be several users of the same

system. To avoid these problems, I would suggest to follow the example of biology and to remove intentions from functional ascriptions.

A further argument for a unified account of functions in biology and in technology is that the biological-artificial system distinction is not tenable. A simple conceptual analysis is sufficient to show that these two notions are independent and their extensions may overlap. Therefore, it is possible for a system to be both biological and man-made. Furthermore, developments in synthetic biology provide concrete examples of such systems (Hutchison et al. 2016).

The systemic account proposed by Cummins (1975) provides an analysis of functions applicable to both biological and artificial systems (and even combinations of those). The seeming shortcomings of this analysis are surmountable with suitable definition of the studied system and its activities.

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Function Pluralism Explained

Maximilian Huber *(University of Geneva)*

For a (token) trait X, functions $F1$, $F2$ and theories of function $T1$, $T2$ where $F1 \neq F2$ and $T1 \neq T2$, compare two cases:

According to $T1$, X has function $F1$; according to $T2$, X has function $F2$ (1)

According to $T1$, X has functions $F1$, $F2$ (2)

In (1), X gets attributed two distinct functions by two distinct theories of function. By contrast, in (2), X gets attributed two distinct functions by one and the same theory of function. Let us call cases like (1) `function pluralism' and cases like (2) `functional pluralism'. The notion of function pluralism is popular (e.g., Bouchard 2013) yet notoriously underdefined. In this paper, I try will remedy this situation by
1. distinguishing the logically available variants of pluralism, 2. mapping them to existing theories of function, and 3. spelling out some challenges for each variant. To give a taste, let me briefly outline 1. and 2.:

1. Global function monism is the claim that with respect to all objects (that is, biological or not), there is at most one adequate theory of function. Biological function monism states that with respect to all biological objects, there is at most one adequate theory of function. Local function monism is the claim that with respect to a certain subclass of biological objects (for simplicity correlated with a certain domain of biology), there is at most one adequate theory of function. Global, biological and local function pluralism replace the quantifier `at most' with `at least'. This yields four consistent variants:

	global	biological	local
super monism	monism	monism	monism
not consistent	monism	monism	pluralism
not consistent	monism	pluralism	monism
not consistent	monism	pluralism	pluralism
weak pluralism	pluralism	monism	monism
not consistent	pluralism	monism	pluralism
strong pluralism	pluralism	pluralism	monism
super pluralism	pluralism	pluralism	pluralism

2. (a)

Super monism is not a version of function pluralism but the only consistent version of function monism. Note that it is consistent with functional pluralism. Super monism has been the prevalent position in pre-Darwinian biology (Godfrey-Smith 1993: 203) and has still contemporary proponents. For example, the etiological theory à la Wright (1973) “applies univocally to organisms and artifacts" (Boorse 1976: 71); Griffiths (1993: 419) more generally claims that “the etiological account can be extended to artifacts because human selection does for artifacts what natural selection does for organisms"; Longy (2013) has recently proposed a new etiological theory applying to both biological objects and artifacts. Other examples include Kitcher (1993), Mossio et al. (2009) and Nanay (2010).

(b)

Weak pluralism is the least permissive version of function pluralism. It is not a popular notion; most philosophers of biology seem to ascribe to super monism or a more permissive version of pluralism, at least since the 1990s (Wouters 2005: 146). There are exceptions, for example, Walsh (1996), Buller (1998) and more recently Graur et al. (2013).

(c)

In contrast to weak pluralism, strong pluralism allows more than one theory of function to be adequate for biological objects. However, in contrast to super pluralism, it maintains that specific domains of biological research of require specific theories of function. For example, while the systemic theory of function might be adequate for molecular biology, it might not be adequate for evolutionary biology; and while the etiological theory of function might be adequate for evolutionary biology, it might not be adequate for molecular biology.

(d)

Super pluralism is the most permissive version of function pluralism. For example, Amundson and Lauder (1994: 444) argue against the “generally accepted [position that the etiological theory of] function is the concept uniquely appropriate to evolutionary biology."

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Productive Functions and the Selected Dispositions Theory

Fabian Hundertmark *(University of Bielefeld)*

Biological traits like hearts or coloring mechanisms, social institutions like conventions or laws, and technical artifacts like engines and thermometers have functions. According to the etiological theory a trait has the function to φ if it was selected for φ-ing. The standard etiological theory is a selected effects theory that presupposes that a trait was selected for φ-ing if and only if its existence can be explained by the φ-ing of its predecessors (cf. Wright 1973; Millikan 1984; Neander 1991 among others).

In the first part of my presentation I will argue that the selected effects theory has a problem with explaining productive functions. A trait has a productive function to φ if and only if the trait has the function to φ although nothing has ever φ-ed before. Simplifying somewhat, we can say that, when we see a grass green chameleon sitting in front of chartreuse yellow background, something in the chameleon is malfunctioning, although no chameleon ever sat on something chartreuse yellow before. While the selected effects theory of functions requires ancestors of a trait to have φ-ed in order for the trait to have the function to φ, a function to φ is only productive, if nothing has φ-ed before. Consequently the selected effects theory is unable to explain the existence of productive functions.

In the second, positive part of my presentation, I will argue that this problem arises only if we assume that explanations always track actual causal histories. However, Sterelny and Griffiths (1999) have suggested that selectional explanations are so called robust process explanations. These kinds of explanations reveal the insensitivity of a particular outcome to some feature of its actual history. This suggests a selected dispositions theory as an alternative to the selected effects theory of functions. According to this theory, not effects but dispositions are selected. Dispositions can be complex such as the disposition of the chameleon to produce a skin pattern matching the background, which includes but is not limited to the two simple dispositions of producing a grass green skin color in front of a grass green background, and producing a chartreuse yellow in front of a chartreuse yellow background. A robust process explanation for the

existence of a trait can make reference to a complex disposition, if and only if some of its simple dispositions actually have been manifested and contributed to fitness of the trait in question, and every of its unmanifested simple dispositions would have contributed to the fitness of the trait in question, if it would have been manifested. As a consequence complex dispositions with unmanifested simple dispositions as constituents can be selected, which in turn makes them function according to the etiological theory. This allows for productive functions which are nothing but the unmanifested constituents of selected complex dispositions.

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Functional Explanation, Reciprocal Causes, and Self-organization

Caglar Karaca *(Egenis, Exeter)*

In my paper, I argue that Wright's functional explanation can serve as a basis of self-organization in biology and social sciences, and that reciprocal causal relations are the key to understanding both the biological functions and self-organized processes. According to Wright (1973), "Saying that the function of *X* is *Z* is saying that *X* is there because it does *Z*, or doing *Z* is the reason *X* is there, or that *X* does *Z* is why *X* is there" (p. 157). About the natural functions, Wright argued that the causal statement that oxygen is found in human bloodstreams because it is carried by hemoglobin and the causal statement that oxygen must be there because of its function to provide energy in oxidation reactions point out different sorts of etiologies.

Hitchcock's (1996) interpretation can reconcile the mentioned sorts of etiologies. Hitchcock set forth that Wright's functional explanation described a second order causal relation. According to his interpretation: "(1) *A* causes *B*; (2) *A* is present at time *t* because *A* causes *B*" (p. 370). Second order causal relations define the very relation between *A* and *B*. Thus, Hitchcock suggested that the previous statement could be read as: "*A*'s causing *B* causes *A* to be present at time *t*" (p. 370). This represents "the causal connection between *A* and *B* that sustains *A*" (p. 371).

Hitchcock's (1996) description of the causal relations based on Wright's functional explanation can also help us understand the fixation of biological functions with reciprocal relations. One of the figures given in Hitchcock's paper is consistent with this reciprocity. According to this, "*A* causes *B*, which in turn causes *A* at later times" (p. 372). I think the repetitive and regulatory characteristics of these relations are essential to understand the causal relations in biological functions. For example, let us take the feedback mechanism regulating the body's sugar consumption and storage. Cells break down sugar and this generates the end product of ATP, in which chemical energy is stored. The accumulation of ATP leads to a negative feedback and this inhibits the enzyme activity to produce ATP, and the process self-regulates itself in this way. This kind of causal relation is everywhere in biological processes, and recent

studies show that even evolution and ecological relations can be dealt with reciprocal relations.

Laland et al. (2013) argues that reciprocal causation is a better concept than the unidirectional causation in contemporary evolutionary and developmental biology. They stated that niche construction, human cooperation, and cultural evolution are the phenomena in which reciprocal causes appear based on the analyses in the areas such as coevolution and habitat selection, in which selective feedback codirects evolutionary outcomes.

Self-organization is a general phenomenon in which reciprocal relations in a process transform themselves into an ordered system. In biology, recent studies show that self-organization is not limited to spatial dimension of reciprocal relations, instead, it enforces its own rules on the evolutionary outcomes even to a degree that resist the effects of natural selection (Wilson, 2005). Hogeweg (2005) suggests addressing the issue in an eco-evolutionary context by stating that “spatial self-organization generates new levels of selection, and thereby directs evolution of the basic replicating entities into unexpected directions” (p. 170).

In light of these works about self-organized processes, I believe that the concept of function can be redefined with an evolutionary perspective.

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Human Genome and Human Identity: Non-coding Matters

Aleksandra Kornienko *(Medical University of Vienna)*

Exponential progress of the genome and transcriptome research in the beginning of the 21st century, starting with the completion of the Human Genome Project, has revolutionized our understanding of human identity and opened a lot of new questions. One of the biggest surprises of the last decade was the realization that what previously was considered as “junk” DNA in fact comprises a myriad of regulatory non-protein-coding genes and genomic elements, such as tissue-specific enhancers, insulators, small and long noncoding RNAs.

This paper reviews the philosophical discussion on human identity in the light of human genome and introduces the terms such as identity-over-possible-worlds, identity-over-time, sameness and others. It then discusses how the view on human identity can be developed with regards to the most up-to-date knowledge on the biology and evolution of the human genome, particularly focusing on its non-coding (non-protein-coding) part. I will argue that with the realization of the importance of the non-coding genome and the multilayer information it contains crucial for regulation of gene expression, development and differentiation, as well as the ongoing studies comparing the regulatory systems of different organisms, provides new insights into identity and the paper discusses these insights.

The paper then discusses both inter-organismal (human vs. other animals) and intra-organismal (human vs. other humans) identity issues, touching the twin cases in the latter discussion as a crucial topic for distinguishing genome vs. environment factors influencing human identity. In all the above I will give special attention to the non-coding regulatory part of our genome. The paper argues that the most variation occurs in this part of the genome and that it is very important for both intra-organismal and inter-organismal identity. In conjunction with the above I will then discuss the notion of complexity that is important for our perception of human identity as a species and argue that it is bound to regulatory systems and preliminary to non-protein-coding genome.

I will then focus on human variation and will discuss the notion of function and malfunction in the light of our current understanding of how small genetic and epigenetic changes in coding and non-coding parts of human genome cause variation in human traits, starting from “harmless” hair color, going through personality traits, disease predispositions and coming to disease. Where is the border between variation in function and malfunction and can we reduce the explanation today when our knowledge of the underlying genome changes is so (relatively) profound? How does the discovery of unexpectedly high non-coding variation affect this discussion?

Last point the paper touches in the discussion on human identity is the notion of “minimal genome”, most known from the work of Craig Venter. With the discovery of high variation in the non-coding genome and some non-coding genes switched off in healthy humans the question rises if the “minimal human genome” can hypothetically exist and how such discussion contributes to the understanding of human identity.

Genome research will continue to provide novel insights into what humans are and what of/how we are made, and we and our philosophical concepts will need to be up to date with it.

To be announced

Ulrich Krohs *(University of Muenster)*

Abstract to follow

On Defense of the Organizational Account of Ecological Functions

Victor Lefèvre *(IHPST Paris Sorbonne)*

Ecologists ascribe functions to parts of ecosystems. This practice raises at least two problems:

1) the problem of normativity – ascribing a function to an entity seems to be saying what this entity should do in violation of the dichotomy between facts and values.

2) the problem of teleology – ascribing a function to an entity seems to be explaining its existence by its activity, an inversion of the classical direction of causal explanations.

To solve these problems, philosophers of biology have developed different approaches which face specific difficulties when we try to apply them to ecological functions. Odenbaugh (2010) adopts the systemic approach of Cummins (1975) and faces the difficulty of the underdetermination of the ecosystem functions – for example he attributes ecological functions to volcanoes and lightning what ecologists don't do. The application of the selective-etiological approach of Neander (1991) faces the difficulty to endorse the controversial hypothesis of ecosystem selection.

I subscribe to the alternative approach of Nunes Nunes-Neto, et al (2014) which consider ecological functions as constraints under closure in a given ecosystem. I propose to precise this approach by using the recent characterization of closure of constraints elaborated by Montévil and Mossio (2015). I suggest that this new formulation generate a class of ecological functions closer to the ascriptions of ecologists than the those generated by the original one and the systemic one, and that the underlying assumption of ecosystems closure of constraints is no more unacceptable than the assumption of ecosystems selection. In particular, this approach allows to ascribe functions to “abiotic” parts of ecosystems – like beaver dams or soil – and answers to an objection of Bouchard and Dussault (under publication) against the organizational approach.

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Can the Biological Accounts of Function Be Applied to Human Morality?

Parisa Moosavi *(University of Toronto)*

The notion of function in biology and natural sciences is generally taken to be irrelevant to moral philosophy. But there is an Aristotelian approach to naturalizing ethics, which appeals to the 'natural good' of living organisms to show that moral goodness is continuous with this idea of natural goodness (Foot 2001, Thompson 2008, Hursthouse 1999). Neo-Aristotelian naturalists claim that moral virtue for humans is akin to deep roots for oak trees. In the same way that deep roots are good for an oak insofar as they allow it to flourish as an oak, virtue is good for a human insofar as it helps her to flourish as a human. This form of teleological naturalism is committed to a notion of function that is ascribed based on the notion of flourishing of an organism.

One of the major challenges for neo-Aristotelian naturalists is showing that their flourishing-based notion of function captures something real about living things, e.g., showing that deep roots really are good for oak trees. Critics have argued that, on the one hand, the flourishing-based notion of function has no place in biology (Fitzpatrick 2000), and on the other hand, replacing it with the biological notion of function leads to implausible moral judgments (Odenbaugh 2015). However, this critique is made based on the assumption that the only account of biological function that can capture the normativity of functional ascriptions is the etiological account going back to Wright (1973) and Millikan (1984, 1989). What is missing is an appreciation of recent accounts of biological function such as the organizational theory (Mossio et al. 2009, Saborido et al. 2011) or Krohs' (2011) post-adaptationist theory, which locate the normativity of functional ascriptions in other sources, like biological organization or the type-token relation.

Another thing missing from this discussion is a clear account of what makes the flourishing-based notion of function suitable for naturalizing ethics, and whether it is the only notion of function that can do the job. It's generally understood that the relevant notion of function has to capture normativity. But the kind of normativity that is particular

to moral judgments requires more than a mere distinction between function and malfunction. Moral judgments are action-guiding and speak to reason. This is why the etiological theory of function cannot capture the relevant kind of normativity: our natural selection history does not issue the right sorts of reasons for us to act in a certain way. The flourishing-based account of function issues the right sorts of reasons, but it is based on a metaphysically heavy account of flourishing that evades empirical investigation. So the question is whether the requirements of moral normativity can be met by a notion of function that is genuinely naturalistic.

In this paper, I articulate the requirements of moral normativity and discuss some of the constraints that they put on the relevant account of function. I argue that the accounts of function that locate the normativity of functional ascriptions in sources that can be traced back to our own interests (cognitive or otherwise) cannot meet the requirements of moral normativity.

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What are Biological Malfunctions?

Alvaro Moreno *(University of the Basque Country, San Sebastian)*

The organisational view on biological functions has a major challenge in the characterization of malfunctions. The reason is that claiming that a trait can function "well" or "poorly" implies a reference to an explicit norm, which may or may not be fulfilled. Yet, the norms grounding functions and malfunctions are not the same, and an independent justification must be provided for each. Since the norms generated by closure are blind with respect to the distinction between these two types of effects, (because both of them contribute to the maintenance of the organisation, albeit in some cases poorly), both are therefore *functional*. Hence, malfunctions require an additional set of norms, on the basis of which it might be possible to discriminate between different ways of contributing to the maintenance of a closed organisation.

In this talk I will develop an account of malfunctions on the basis on a theory of regulation, understood as the creation in an autonomous system of a domain of "second-order" functions, dynamically decoupled from the basic ones (or "first-order" functions). In this account a malfunction is any functional activity with respect to which there has been a failure of regulation. In other terms, malfunctions are a subset of functions that fit first-order norms (of the first-order on-going organization in which they match functional presuppositions), but not second-order ones (since they do not obey to second-order regulatory functions, and prevent the shift to another first-order organisation). In this respect, the degree of malfunction of a trait could be assessed in terms of the set of first-order organisations of which it prevents the realisation. The degree of malfunction is, therefore, inversely proportional to the degree of adaptivity of the organism.

The Organizational Account of Biological Functions

Matteo Mossio *(IHPST Paris Sorbonne)*

In this talk I offer a detailed presentation of the organizational account (OA) of biological functions. According to the OA, functions are those effects produced by organized biological parts, which collectively contribute to maintaining the conditions of existence of the whole system and, thereby, of the parts themselves. I first develop the characterization of ‘organization” on which the OA relies on, and I then examine its connections with a set of closely related notions as causal circularity, teleology, aetiology, normativity, and functions. Finally, I argue that the OA integrates the qualities of existing “selected effects” and “dispositional” accounts, while avoiding (most of) their drawbacks. In particular, I contend that organizational functions avoid both epiphenomenalism and overinclusiveness.

Structure and Function: A Process-Centred View

Daniel J. Nicholson *(Egenis, Exeter)*

The understanding of function has been a prominent topic in the philosophy of biology for at least forty years. Various philosophers have advocated some version of the causal role account (Cummins 1975), which treat functions as the contribution of parts to the overall behaviour of a larger system, or the etiological account (Wright 1973), directed more specifically at evolutionary biology, that see the function of a trait or an organ as the selective advantage it confers, and which explains its current presence in members of a species.

Both these approaches assume a fairly similar background ontology: entities are composed of smaller entities, and the fixed properties of the latter combine somehow, to generate the properties or dispositions of larger entities. The function of an entity—whether or not tied to the explanation of its selective advantage—is seen as its contribution to the behaviour of a system of which it is part, and this contribution is seen as made possible by its structural properties. This picture has gained support from its congruence with the new mechanism discourse, the rising influence of which has been a prominent feature of recent philosophy of science. Indeed, a mechanism is often characterized as “a structure performing a function” (Bechtel 2006: 26), or as “a complex system that produces [a] behavior by the interaction of a number of parts” (Glennan 2002: S344).

In this paper I argue that this picture is seriously misleading when viewed against a more nuanced attention to biological phenomena. Biological systems, for example organisms, do not consist of a fixed set of component parts with fixed properties. The persistence of an organism is achieved by constant adaptive changes of the whole, and by countless internal processes in which constituent entities at different levels of the organizational hierarchy are continuously produced, transformed, and destroyed at varying turnover rates. The delimitation of fixed structures in an organism is at best an abstraction, implicitly assuming an appropriate time scale over which the structures in question are sufficiently well-stabilized to be treated as static things for the purposes of

their investigation. Functions are attributed to entities on the basis of structural properties; hence they are at least as much abstractions from the dynamic reality as the structures on which they are based. In this way, structure-talk explains the possibility of a certain kind of activity, whereas function-talk locates an activity in a wider context to which it contributes.

The thesis I shall defend is that function and structure in biology are, in fact, best regarded as alternative ways of abstracting a permanently changing reality. While these abstractions are unquestionably useful in helping us understand and interact with biological systems, there are serious dangers in interpreting either too realistically.

This view converges with the position advocated by a number of early twentieth-century theoretical biologists, who also interpreted structural and functional characterizations of living systems as complementary means of representing an underlying processual reality. The implication they drew from this is that structure and function are interdependent and interdefining concepts. In the words of J. S. Haldane (1931: 22), “Structure and functional relation to environment cannot be separated in the serious scientific study of life, since structure expresses the maintenance of function, and function expresses the maintenance of structure”. Similarly, L. v. Bertalanffy (1941: 251) remarked that “The old contrast between ‘structure’ and ‘function’ is to be reduced to the relative speed of processes within the organism. Structures are extended, slow processes; functions are transitory, rapid processes”.

My paper will explore the advantages of this process-centred perspective on structure and function in biology. I will illustrate our argument by drawing on a number of examples from various areas of biological enquiry, including cell biology, plant morphology, and evolutionary biology.

Functions and Health

Lennart Nordenfelt *(University of Stockholm)*

In order to characterize the modern Western concept of disease the American philosopher Christopher Boorse has proposed and defended the idea that health is conformity to *species design*. Boorse says: "[S]pecies design is the internal functional organization typical of species members, which (as regards somatic medicine) forms the subject matter of physiology: the interlocking hierarchy of functional processes, at every level from organelle to cell to tissue to organ to gross behaviour, by which organisms of a given species maintain and renew their life. All conditions which are called pathological by ordinary medicine constitute disrupted part-function at some level of the hierarchy." (Boorse 1997, p. 7)

There are two central definitions that form the basis of Boorse's characterization of health. There is first the definition of disease: "A *disease* is a type of internal state which either is or causes an impairment of normal functional ability, i.e. a reduction of one or more functional abilities below typical efficiency." Then there is the definition of health based on this characterization that says laconically: health is identical with the absence of disease.

In contradistinction to Boorse's analysis I will take as my starting-point *the concept of health* and *not* the concept of disease. Methodically I will start, not in the language of biological function, but indeed in ordinary linguistic usage. I shall ask the question: What do we normally mean when we speak of a healthy person? What are those people like that we call healthy persons? And subsequently, what do we mean when we say that a person has an illness or a disease?

So we can now see the contrast: we have on the one hand a functional analysis of disease, and indirectly of health, and we have on the other hand a praxis-oriented analysis of health, and indirectly of disease.. (I use here for the first time the term "praxis-oriented". I have earlier talked about a holistic concept of health. I think "praxis-oriented" may be better suited for my particular version of a holistic theory, since it

stresses the place of the concept of ability in health.) We have thus been introduced to two conceptual worlds.

But how should a praxis-oriented concept of health be further characterized? We cannot just rely on the core concepts of suffering and disability as unanalysed concepts. That would not make us credible in the clinical and scientific discussion of the matter. There is a lot of further conceptual work to be done. In some of my earlier work I have, in particular, focused on the concept of ability as the core concept in health. I have asked the questions: what should a healthy person — according to the concept of health — be able to do? And should the healthy person A necessarily be able to do the same things as the healthy person B? Or, is there a minimal set of acts that all healthy persons should be able to perform?

In my presentation during the workshop I shall proceed by considering some earlier attempts to characterize a praxis-oriented analysis of health. The authors whose works I will scrutinize are Ingmar Pörn, with his equilibrium theory of health, and Bill Fulford, with his focus on the ordinary actions in life.

I will conclude by defending my own proposal for a praxis-oriented theory of health, which can be summarized in the following formula: *health (or optimal health) is the bodily and mental state of a person which is such that he or she has a second-order ability to realize his or her vital goals, given standard or otherwise accepted circumstances.*

My proposal also involves the specification of a set of goals — that is, goals of the actions to be performed by the healthy person. However, a goal in my sense does not mean a goal consciously set by the agent him- or herself. The latter idea led to counterintuitive results in Pörn's theory. But there is a further crucial reason. Some people don't — or even *can't* — consciously set any goals for themselves. Babies clearly cannot set any goals; nor can people with severe dementia. And we certainly wish to be able to attribute health to babies and wish to be able to characterize the health of persons with dementia.

For my purposes I have introduced the notion of a *vital goal*. Vital goals are goals that are inherent in all humans, and indeed, I have argued, in all sentient organisms. The vital goals include survival but they encompass much more. I propose that *a person's vital goals are the states of affairs which are necessary and jointly sufficient for his or her minimal long-term happiness*.

Two Varieties of Dysfunction in Mental Disorder

Christopher Parker *(University of Cincinnati)*

Since the 1970s, it has been customary for philosophers to argue that mental disorders are instances of systemic dysfunction in much the same way that physiological diseases are, and that the analogy between disorder and disease is sufficient to establish the legitimacy of psychiatric medicine (e.g. Boorse 1975; 1976a; 1977, Wakefield 1992; 2007, Murphy 2006). I argue that there are two distinct ways in which we should think of mental disorders as dysfunctions and that only one of these categories is coextensive with the type of dysfunction characteristic of disease. Even so, the other category of dysfunction which I highlight is analogous to some physiological conditions targeted for medical intervention which are not diseases. Recognition of this distinction stands to clarify the criteria which must be satisfied for a condition to merit psychiatric intervention.

Determining the sense in which diseases are instances of dysfunction requires a general conception of what constitutes a natural function. This is a controversial matter (cf. Wright 1973, Cummins 1975, Boorse 1976b), but I argue it is nonetheless possible to formulate a minimal set of necessary conditions which must be met by any explanation of a phenomenon as an instance of disease. Once such criteria have been established, current successful explanations of various mental disorders can be matched against them to determine whether they conform to a disease model. I offer a set of such criteria and argue that it shows that in order for any state to count as a disease, the system in which it is instantiated must be identical to one that typically produces or sustains a norm of operation, with what constitutes such a norm to be determined by one's specific theory of function. It is deviation from that norm that constitutes disease, and treatment must either return the operations of the system to that norm or mitigate any harmful effects of deviation. On this model, some mental disorders clearly qualify as diseases, for example, psychosis, whose symptoms have been explained as the outcome of hyperactivity of the dopaminergic system (Kapur 2003; Howes and Kapur 2009). The efficacy of antipsychotic medications which suppress dopamine production supports this account. Explanations which invoke

dysfunctional cognitive systems – for example, Baron-Cohen's (2009) account of autism spectrum disorder – can also be subsumed under this model.

Still, there is a large segment of mental disorders which do not conform to this model. This can be seen by considering disorders effectively treated with cognitive-behavioral therapies – these include many instances of depression and anxiety disorder, panic disorder, social phobia, obsessive-compulsive disorder, post-traumatic stress disorder, and bulimia nervosa (e.g. Butler et al. 2006). I argue that it is improper to think of these therapies as repairing systemic dysfunctions. Nevertheless, I propose that these disorders belong to a different species of dysfunction; that is, despite being the product of functional systems, they tend to defeat the desires and aims of the persons in whom they are instantiated. Physiological medicine has historically intervened on such dysfunctions, and recognition of this variety of dysfunction does not threaten the analogy between physiological and psychiatric medicine. Even so, failure to recognize this distinction has led many theorists (e.g. Wakefield 1997; Horwitz 2002) to offer criteria for entitlement to psychiatric care which are far too restrictive.

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Development at the Edge of Dysfunction: Accounting for Developmental Change in the Light of the Organizational Approach of Functions (AOF)

Alessandra Passariello *(Sapienza University, Rome)*

The aim of this intervention is to test the organizational approach of functions (AOF) (Mossio;Saborido; Moreno 2010) with respect to its capacity to explain developmental change. I will argue that developmental change is the capacity of an organism to overcome dysfunction by restoring a functional organization, though different from the preceding one. In this perspective, developing organisms are organisms at the edge of dysfunction because, at every developmental step, they face the possibility of loosing their previous organization without being able to establish a new one. Developmental reliability – the regular transition from one previous functional state to the next one becomes thus a major explanandum.

AOF defines organisms as autonomous systems. This basically means that organisms are different from other organized systems (such as artifacts) because their functionality is not accounted in terms of their capacity to carry out a specific global task (to move, to perceive, to sense) but in terms of their autonomy. No matter which are organisms' performances[6], autonomy makes reference to their capacity to selectively exploit external resources so as to maintain a set of mutual dependencies between its processes. The particular layout of this set of mutual dependencies is not at issue here.

So, while in artifacts the set of mutual dependencies between their parts accounts for the realization of a specific task and dysfunction can only be defined with respect to the loss of the capacity to accomplish it; differently, in organisms, the set of mutual dependencies between their processes account for processes' self-maintenance and dysfunction can be defined with respect to the loss of the capacity to self-maintain.

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[6] I use the term “performance” as a synonymous of “global capacity,” basically to express the same “dispositional” concept of functions that we find in Cummins (1975). I will use the word “function” to designate biological “autonomy.”

I think that the first step to frame development within the organizational approach of function is to hypothesize that major phenotype stages occurring in unicells' (transition to colonial species) and multicells' life cycle can be accounted for in terms of “functional organizations”: this means to identify (many) different autonomous systems in a life cycle[7]. What I argue is that, besides unicells and adult multicellular organisms, transitory developmental stages can be also explained through a set of processes whose mutual interactions set the conditions for their self-maintenance: the only difference between traditionally admitted autonomous systems (unicells and adult multicells) and developmental ones would be one of duration, that is time of self-maintenance. In this perspective[8], autonomy is not only the result of a developmental process (multicellular adult autonomous systems) but also a “developmental invariant”: development is therefore a succession of autonomous systems.

Two main questions must be further addressed:

1) the first deals with the logic of developmental change, in other words how can an autonomous system convert non buffered variation into a new stable (autonomous) organization;
2) the second concerns developmental reliability: how can the transition from one ontogenetic (autonomous) stage to another be a regular phenomenon, a reliable one?

As far as the first issue is concerned 1), I think it is useful to distinguish “developmental change” into three different steps:

– in the first step we have a functional autonomous system (eventually a developmental stage), whose processes are able to self-maintain through a set of mutual dependencies.

[7] A different perspective has been adopted by [Nuno de la Rosa 2010]. The author suggests that development is a set of processes (and stages) through which an unicellular autonomous system (ex. egg cell) becomes a multicellular autonomous one (an adult organism). In this case, development explains the existence (the generation) of an autonomous organization, but it is not itself explained by it. Saying it differently, development is an explanans of organization (the production of multicellular adult one) but it not an explanandum of the organizational theory, because the theory cannot account for it.

[8] We need empirical proofs that embryonic stages can act like autonomous system.

– The second stage corresponds to the origin of variation (either a change in the environmental – biotic and abiotic – conditions or embryonic threshold phenomena) which seriously prevents the organismic processes from regenerating the conditions for their own maintenance. This stage is what I call “dysfunction” and it can be triggered by whatever variation the system is not able to cope with[9].
– The last stage is the accomplishment of a developmental change, which means the exploitation of (initially dysfunctioning) variation so as to reestablish an autonomous system, though different from the preceding one.

This last stage is different from mere “regulation” which is nothing but the re-establishment of a previous autonomous state. Differently developmental change gives rise to a new autonomous regime in which a new self-maintaining set of mutual dependencies is established. Developmental change is thus a risky process which can either end up in successfully converting deleterious variation into a functional one or persist in a dysfunctioning state, likely amplifying its effects. This risk is what I refer to when I use the expression “being at the edge of dysfunction.”

However, contemporary developing organisms seem to be exempt from this uncertain fate: developmental reliability is a matter of evidence for most of them. In order to deal with developmental reliability 2) we need to explain why development is most likely to preserve “autonomy” instead of surrendering to dysfunction and why, specifically, this rearrangement of deleterious variation into a functional one, ends up in the same[10] developmental phenotype.

[9] “To cope with” is a general expression standing for “to regulate”, “to absorb”, “to buffer”, “to adapt”, depending on the particular model we use to explain a system's capacity to neutralize the effect of variation by restoring its actual functional organization.

[10] In organisms pertaining to the same species, we have reliable transition from one developmental stage to the next one.

I argue that in order to answer those two questions we need to make use of the notion of “recurrent variation” and “developmental constraints”:

- “recurrent variations” are those variations which are likely to affect a particular instance of autonomous organization.
  If we consider an autonomous organization made out of an aggregate of cells and we attribute to those cells the capacity to divide mitotically, “recurrent variations” will be, for example, (mitotically driven) variations in the composition of the inter-cellular environment or in the mechanical stresses affecting the cells. Those variations are “recurrent” in so far as they are reliably provoked by cellular mitosis. They are also potential sources of dysfunction in so far as changes in the inter-cellular environment can yield a change in the type of environmental resources and mechanical stresses can affect, among other things, cells' access to those same resources. Many other parameters can play the role of “recurrent variations” such as parasites invasion or embryonically induced micro-environmental changes.
- “developmental constraints” are those characteristics of a particular instance of an autonomous system which “constrain” its change toward the next functional stage, provided that “recurrent variations” have occurred. Hence, “developmental constraints” are coupled with “recurrent variations”: their constraining capacity act onto one specific dysfunctioning state of a system.

From an evolutionary point of view, “developmental constraints” could be organisms' adaptive strategy to cope with the destroying action of “recurrent variations”. They could be an evolved[11] strategy to avoid dysfunction, dis-integration (processes' decoupling) and finally death. In this perspective, developmental reliability is an evolutionary acquired trait but it can mechanistically be explained by making reference to the occurrence of a specific kind of dysfunctioning organization (triggered by “recurrent variations”) and the organism capability to direct the organisms toward a new autonomous state.

[11] I make no reference to the hereditary mechanism (genetic, epigenetic etc..) which permit this evolution.

Some final remarks concern some future research targets likely to provide an empirical ground to these theoretical (conceptual) arguments:

- firstly, if developmental reliability is grounded on the coupling between "recurrent (and dysfunctioning) variation" and "developmental constraints," we should be able to loosen the constraints on developmental change by modifying the kind of dysfunctioning variation. As for engineering designed systems, organisms could be highly robust to "expected" (recurrent) variations but only poorly robust with respect to unattended (rare) ones. (Artificially) induced dysfunctioning states should trigger a wider (poorly constrained) repertoire of developmental changes and possibly some functional–innovative–ones.
- secondly, research on complex life cycle is likely to provide an empirical application of the notion of autonomy with respect to developmental (transitory) phenotype.
- thirdly, regenerating phenomena (where development can be partially repeated once the autonomous multicellular system has been injured) as much as life cycle lacking either a unicell (egg) stage or an adult multicellular stage could be preferential research models in order to understand this organizationally driven account of development.
- Finally, research of unicells environmentally triggered colonies, phenotypic plasticity (Minelli 2010), or facultative symbiosis can provide examples (on the ontogenetic time scale) of "recurrent variations" (environmental lack of resources as a trigger of colonial aggregates, environmental parameters as a trigger for phenotype change, or parasite presence as a trigger to symbiosis establishment) though still poorly "constrained" (facultative) developmental changes.

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The Concept of Function in Christopher Boorse´s Bio-statistical Theory of Health

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Functionalist theories of health and disease are prominent in the philosophy of medicine. Among those, Jerome Wakefield's Harmful Dysfunction Analysis (HDA) and Christopher Boorse's bio-statistical theory (BST) are certainly two of the most cited and the most debated. Both theories seem, at first sight, to rely on completely distinct concepts of functions: an etiological concept, based on a selected effects analysis in the case of the HDA, and a causal role (C-R) approach based on a goal contribution analysis in the case of the BST. In this paper, I argue that Boorse's theoretical concept of health integrates a two-fold account of the concept biological function. On the one hand, I maintain that Boorse theory indeed accepts typically C-Rs functional ascription, but on the other hand I also contend that the BST displays some purely etiological features. My main argument draws on the distinction between two kinds of functional ascriptions, namely: typical functional statements and individual functional statements. Whereas "typical" functional statements are attributed to classes or collections of entities, "individual" functional statements are attributed to singular entities. While the former points towards an etiological account of function, the latter (at least in general) implies a C-R based analysis. Since Boorse's theory in its formal structure implicitly refers to those two kinds of functional ascriptions, it entails that the BST draws upon both etiological and C-R based account of the concept of biological function. On many occasions, however, Boorse has claimed that his theoretical concept of health is function-neutral and that it could well be adapted to functional theories other than his general goal-contribution analysis. My analysis imposes certain constraints to this claim as both the etiological and C-R approaches to the concept of biological function seem inseparable in the context of the BST.

Kind-Formation for Functionally Defined Groups

Thomas Reydon *(University of Hannover)*

On a traditional model of scientific classification, the kinds that feature in a particular area of science aim to "carve nature at her joints" – that is, they aim to classify the entities within the subject domain of that science in a way that adequately represents groupings of entities as they exist in nature. This has led to a view of scientific kinds as "special" in the sense that they uniquely represent aspects of order in nature. For quite some time now this traditional model has been rejected in the philosophy of the special sciences in favor of models that are supposed to better fit the variety of kinds that feature in the various sciences. However, recent accounts of kinds still tend to be unwarrantedly narrow in that they fail to cover the entire spectrum of kinds that play epistemic and other roles in the sciences (Ereshefsky & Reydon, 2015).

In this talk I want to address the question what recent accounts of natural kinds (e.g., Magnus, 2012; Khalidi, 2013; Slater, 2015) make of functionally defined kinds, and to propose a way of thinking about functional kinds as "good" scientific kinds. In the life and social sciences functionally defined kinds play important roles in explanation, prediction, generalization, heuristics, and so on. Thus, functional kinds should fall within the scope of any account of scientific kinds. Extending recent work on kinds in the sciences that focuses on practices of kind-formation rather than on the finished products (Kendig, 2016) I want to argue for a more dynamic model of classification than is found in available accounts, according to which practices of kind-formation and classification are inescapably characterized by a constructivist as well as a realist element (Reydon, 2016). Accordingly, the products (kinds and classifications) have what – inspired on recent work in the philosophy of technology (Houkes et al., 2011; Reydon, 2014) – may be called a "dual nature": they are in part natural, in part artificial. I will use the classification of genes to illustrate my account.

(* = recommended reading for commentator & participants)

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Functional Explanation Requires an Intentional Choice Constrained by Computational Factors

Luca Rivelli *(IHPST Paris Sorbonne)*

Current views of mechanistic explanation consider mechanistic explanations as requiring the functional characterization of the mechanisms' parts and activities. But does this require intelligibility of the explanation so produced? There are two general positions on mechanisms: the ontic view (embraced among others by Carl Craver) and the epistemic view (represented especially by Cory D., Wright and William Bechtel). According to the ontic position, which stems from Wesley Salmon's view of causal explanation, a mechanistic explanation could amount the mere exhibition of a mechanism. On the contrary, for the epistemic position, an explanation is essentially an epistemic and communicative task, and as such it requires the conveying of information about the system explained, and presupposes that such information can be understood by the receiver of the explanation. But, in large enough complex systems, their lowest-level description in terms of their elementary entities and activities could, due to its sheer complexity, overcome human cognitive resources, resulting unintelligible and thus explanatory null. Understandability requires thus higher-level redescriptions of the system, in other words, a hierarchical, multi-level description: the higher-level descriptions convey a more understandable (even if less informative) explanation of the system, while the lower-level ones provide a progressively higher amount of information to the observer, at the cost of being less graspable as a whole. The possibility to traverse this hierarchy up and down allows the observer to optimally grasp the functional structure of the system.

Now, even if the ontic conception of mechanistic explanations could admit of single-level (that is, lowest-level-only) explanations in the form of the exhibition of the fine structure of the mechanism, when this low-level description is too large and complex to be grasped by human comprehension the epistemic position of mechanisms would not admit it as an explanation proper.

I argue that the same holds for purely functional (for example, computational) explanations: in systems of a sufficiently intricate and complex structure, composed by a sufficiently high number of interrelated elementary non-linear functions, the lowest-level functional description, could result unintelligible due to its sheer size and complexity.

In Cummins-style functional analysis functions are understood as explanatory roles fulfilled inside the explanation of the overall system's function, and if explanations in general require to be human-understandable, as per the epistemic standpoint, then it seems that also Cummins-style functional descriptions would necessarily require to be understandable in order to manifest explanatory power. And understandability is conferred to functional explanations by their multi-level, hierarchical descriptions.

The problem is that a hierarchic multi-level reconstruction of the functional structure of a system is inherently "intentional": the relation between higher-level and lower-level functions is the relation between "specification" (understood, as in computer science, as the function a program must perform) and the "implementation" of this specification (understood as the way lower-level functions realize, in their orchestrated functioning, the higher level function/specification). And, I argue, the attribution of a specification is not a "natural" fact, but requires an intentional act on the part of the observer, in the form of the pragmatic choice of a specific mapping of the input and output configurations of the program, or the mechanism, performing the function, a choice limited by certain mathematical constraints related to computational power and computational complexity. Thus, a pragmatic, intentional, normative decision underpins functional attribution.

Normality, Function, Dysfunction in Biology: Function as a Variable

Étienne Roux *(IHPST Paris Sorbonne)*

In natural sciences, ascription of functions is specific to biological systems, compared to physical ones. A biological function can be broadly defined as some specific property of a trait correlated to a given state of a higher level biological system, the "functional norms" of the system. As the analysis of life science practice shows, normativity has a specific heuristic value in biological systems. Ascription of functions requires norms, which criteria are beyond the mere observation of the behavior of the system. If we consider that the behavior of the system is sufficient to define its normativity, function as a regulatory concept and function/dysfunction distinction do not make sense, since both imply that the behavior of the system can escape its own normality. Function requires normativity, and hence cannot define it, which leaves open the question of how the criteria of normality are objectivized.

Function is usually considered as a qualitative property, but distinction between function and dysfunction as quantitative. However, if function is a qualitative concept, it cannot be quantitatively differentiated from dysfunction. Discrimination between function and dysfunction can be clarified by considering a function as a particular quality of a trait, and this quality as a variable. For example, pumping the blood is a quality of the heart. A function is, in this sense, a qualitative notion. However, this quality should be considered as a variable, that is, a quality that can take different values, e.g. blood flow rate. A function is hence a quality to which is associated a definite set of values. Though the formulation of function usually refers only to a given quality of a trait, analysis of past and present scientific literature shows that attribution of function is empirically grounded not only on the identification of a quality of a trait but also on an associated set of values, determined in reference to the normality of the system. The different theories of biological functions also share the fact that functions are variables, though this is usually implicit. In this view, function and dysfunction are the same quality, or variable, of the same trait, and differ in the values associated to this variable. Whereas function is defined by a set of values that match the normal state of the system, dysfunction occurs when the associated set of values does not match this normal state. Additionally,

variables can be either quantitative (e.g. size) of qualitative (e.g. color), so that distinction between function and dysfunction is either quantitative or qualitative depending on the nature of the variable.

In conclusion, (i) function/dysfunction concept requires normativity which criteria are grounded beyond the mere behavior of the biological system. (ii) A function can be conceptualized as a given variable which values are defined relatively to the norms of the system. (iii) Dysfunction discriminates from function when the set of values of the given variable does not match the normality of the system.

Plasticity of Functions: The Plasticity–Pathology Continuum of the Nervous System

Isabella Sarto-Jackson *(KLI Klosterneuburg)*

Can psychopathologies be defined objectively based on biological factors? The biostatistical account of pathologies claims that disorders are failures of normal species functions with respect to a given reference class. According to this theory, organisms show means-end hierarchy requiring that every physiological process and every body part at every level must function in its typical way in order to contribute to the organism´s goals (survival and reproduction). To comply with this theory, six distinct requirements (some of them explicated by Boorse) must be met: (1) An unambiguous reference class is necessary. (2) A mean value must be established based on the *continuous* distribution of values within a population. (3) A maximum distance from the group´s mean must be defined to determine what lies still within or already out (*below)* of the boundaries of normality. (4) To calculate the population mean, quantitative measurements of valid parameters (not proxies or surrogate markers) are necessary. (5) The contribution of an organism´s part or process to the overall organism´s goal must be defined. (6) To be meaningful at all for health science, clearly distinct biological functions must be defined and attributed to separate entities of the organism. In this paper, I will claim that pathologies of the nervous system seriously challenge the biostatistical account, because these six requirements cannot be fulfilled in most cases.

Natural complex systems, such as the brain, have evolved the adaptive property of robustness to withstand manifold environmental perturbations and return to an unperturbed state. To compensate for injury and disease, the brain uses mechanisms of neural plasticity, the nervous system´s capacity to reorganize itself throughout life by forming new connections on synaptic, cellular, and network levels. Similar mechanisms are used during learning and memory formation, when long-lasting enhancements or attenuations of signal transmission between neurons or neural circuits attuned to environmental feedback are generated. Such highly plastic mechanisms, however, entail risks of maladaptation. In fact, there is good evidence for such claims, like in the case of epilepsy, chronic pain or tinnitus. Thus, the molecular, cellular, and network

processes that underlie the system´s robustness, learning capacities, and pathologies all seem to represent sections on a given plasticity–pathology continuum.

Assuming a continuum rather than a relatively strictly defined functional ascription to certain brain processes makes it difficult to meet the requirements mentioned above. For example, neuroplastic changes preclude any strict modularity, any functional conservation over time, and thus, depending on the environment, result in varying contributions to the organism´s overall goal. As a consequence, the necessary statistical parameters remain–in the best case–ill-defined, or are–in the worst case–simply wrong.

The biostatistical account relies on the assumption that optimal “brain states” as well as levels of neurotransmitters and neuromodulators, etc. can be unambiguously determined allowing for targeted clinical and therapeutic interventions. In contrast, an alternative view based on a plasticity–pathology continuum argues that crucial neurobiological parameters depend on an organism´s life history and idiosyncratic factors that are in continuous cross-talk with the organism´s niche. Coerced alterations intervening in this continuum will override the “canonical” mode of action of given brain processes to sense the organism´s own physiological state and to evaluate external, environmental information thereby altering a given function rather than restoring a “normal” function.

Function and Value: Calibrating Philosophical Theories of Function Through Their Evaluative Compromises

David Suarez Pascal *(UNAM Mexico City)*

Through the history of the contemporary debate on functions, prima facie evaluative notions have frequently been linked to, or have figured as core elements in, philosophical analyses of functional attributions and functional explanations. Yet the role that such concepts play in achieving the characteristic explanatory task that functions usually perform has not been so thoroughly examined as would be desirable given the refractoriness of some problems and the apparent insufficiency of some analysis to be on par with the different uses of functions in scientific disciplines. This problem has been recognized by some authors (for instance, Godfrey-Smith, 1993; Roszkowski, 2010) and has motivated certain despair regarding the possibilities of offering a unified analysis of functions.

Before Wright's analysis became dominant, there was a much greater diversity of views regarding the correct analysis of functional attributions than after this view reached the status of received view. These views included those of Hempel, Ruse, Nagel, Achinstein, Lehman, Canfield, Wright and Cummins, among others. While both Wright and Cummins distance themselves of welfare- or good-consequence-centered views, some of their contemporaries seem more sympathetic to the employment of seemingly evaluative notions, although most of they think that there is some way to correctly operationalize such concepts or that it is possible to link them with seemingly unproblematic systemic goals.

Today's panorama is not less complex than it was before the short consensus around etiological accounts. Indeed, some recent volumes (Krohs & Kroes, 2009; Gayon & Ricqlès, 2010; Huneman, 2013) evince a striking diversity of views regarding the nature of functions as employed in a not less diverse set of scientific and technological areas. From this outlook is however possible to discern a couple of problems which are specially acute in philosophical literature: the unity of the analyses of natural and

artificial functions, and the question about the normativity of functions as used in various contexts.

Explicit analysis of value-considerations has frequently been disregarded, or plainly rejected, in recent debates on the scientific concept of function. However, recent discussions regarding the tenability and usefulness of the fact-value distinction, as well as the development of mixed etiological-evaluative, or barely evaluative, analyses of function, motivate a reexamination of the links between function and value. Although some defenders of the so-called good-consequence or welfare view have considered that the evaluative character of the concepts they employ is unproblematic, some of them also coincide in their rejection of the Hempelian thesis about the topic of functional explanations, which, paired with their preference for ostensibly evaluative concepts, is an important sign of unresolved issues.

More recently, some authors (Bedau, 1992; Kitcher, 1993; McLaughlin, 2001, 2002; Franssen, 2009) have adopted views which can be aligned with previous evaluative analyses, either because they ground their analysis of functions in typically evaluative concepts or processes, or because they center their research in the question of what is good for the systems to which we attribute functions. Even if these proposals are more concerned with evaluative issues, most of them can be subsumed under etiological analyses, since they suggest either to assign a causal role to valuation or they are said to constitute naturalizations of valuation.

In this paper I propose to analyze the depth and the nature of the evaluative compromises made by different analyses of function, from the decade of 1960 to the present day. Given the importance, recognized or unrecognized, that evaluative concepts have had in conveying the semantics of functional attributions in many analyses of functions in science and technology, a careful examination of the degree in which the evaluative overtones of concepts employed in such analyses can be very useful to give us a good measure of the progress in the field.

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How Objective Are Biological Functions?

Marcel Weber *(University of Geneva)*

John Searle has argued that functions owe their existence to the value that we put into life and survival. In this paper, I will provide a critique of Searle's argument concerning the ontology of functions. I rely on a standard analysis of functional predicates as relating not only a biological entity (e.g., the heart), an activity that constitutes the function of this entity (e.g., pumping blood) and a type of system but also a goal state (e.g., survival or evolutionary fitness). A functional attribution without specification of such a goal state has no truth-value. But if completed with a goal state, functional attributions understood as four-place relations attain a truth-value. The truth conditions of all attributions of function involve a dependence claim of the goal state on the function bearer's activity. The nature of this dependence may differ; I consider five different possibilities: causality, mechanistic constitution, mereology, supervenience and metaphysical grounding. If these dependency relations are objective, Searle's central ontological thesis fails. What he ought to have said is that our valuing survival or other goal states may be the reason why biology seeks functional knowledge, but this has nothing to do with ontology. I will show further that Searle also raised an interesting challenge concerning the relationship of functional and causal truths, but it does not threaten the objectivity of functions either. At best, it could show that functional vocabulary is eliminable. However, I will show that functional vocabulary is not so eliminable.

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Moderation of Round Table Discussion

"Philosophical Theories of Biological Function"

Laurent Loison *(IHPST Paris Sorbonne)*

Closing Remarks & Moderation of General Discussion

Mark Perlman *(Western Oregon University)*

Guest Discussant in the Round Table Discussion

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Notes

Notes

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