# A Contextualized Approach to Biological Explanation

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## 1. Introduction

Most contemporary literature on the concept of scientific explanation concerns approaches that, more or less directly, derive from, or measure their strength against, the first model proposed by C. G. Hempel and P. Oppenheim in 1948.[1] By suggesting the so-called nomological-deductive model, they opened the way to the explications, exactly in the sense clarified by R. Carnap two years later,[2] of the notion of 'scientific explanation'. All of us know the subsequent history, also thanks to the critical reconstruction given by W. Salmon.[3] It was characterized by a) attempts to show the flaws of that first model by using counter-examples; b) attempts to modify it, or to propose a new one, to overcome the problems arising from those counter-examples; c) attempts to construct different explanatory models in the scientific fields where the existing one did not work well.

After almost seventy years of philosophical efforts in 'scientific explanation' we have a lot of good analyses and explanatory models. Now we could work in a different way. That is, instead of working on the proposed models, or on the proposal of new models, that should be capable of overcoming the weaknesses of the previous ones, I suggest starting from the scientific event to be explained and ask ourselves: 'What is the best explanatory model available nowadays that allows us to offer a good explanation of that scientific

[1] G. C. Hempel and P. Oppenheim, 'Studies in the Logic of Explanation', *Philosophy of Science* **15**, 1948, 135–75; now in C. G. Hempel, *Aspects of Scientific Explanation* (New York: The Free Press, MacMillan Company, 1965).

[2] R. Carnap, *Logical Foundation of Probability* (Chicago: Chicago University Press, 1950); cf. G. Boniolo, 'Kant's Explication and Carnap's Explication: the *redde rationem*', *International Philosophical Quarterly* **43**, 2003, 289–98.

[3] W. C. Salmon, *Four Decades of Scientific Explanation* (Minneapolis: University of Minnesota Press, 1990).

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event?'. In particular, since I focus my attention on biological events, 'What is the best explanatory model available nowadays which allows us to offer a good explanation of that biological event?'. To answer such a question, I propose to construct a meta-model of the explanatory models which has to be capable of organizing them, and, by resorting to a given biological context, capable of allowing us to find the right one as to that particular *explanandum*.

It is worth noting that the seventy-years long history of explanation has not only been characterized by the search for a good explication of the locution 'scientific explanation', and therefore for the construction of powerful explanatory models. Actually, this history was strongly influenced by what happened in the physical sciences and by what a physical explanation should be. Physics was the reference science for most analyses and counter-analyses, and physics was the gym where the proposed models exercised and tested their strength. In comparison with this great attention to physics, less consideration was given to biology. Besides the pioneering works by, for example, F. Ayala, D. Hull, E. Mayr, and M. Ruse, who tried to shift the focus of the discussion on to the peculiarity of biological sciences, the debate on teleology and functions should be recalled. Nevertheless, it should be said that these attempts were on the sidelines of a mainstream which was mainly physicalistic.

Needless to say, biology is extremely different from physics and not only trivially since it deals with different issues. The two sciences are structured in a totally different way. Therefore if we want a good account of the biological explanation we must consider such a diversity. This means that instead of beginning by asking what a good physical explanatory model is and then transferring it to biology, we must direct our attention to the latter. In so doing we easily realize that biology is not something unitary but something made up of many different domains: evolutionary biology, developmental biology, population genetics, molecular genetics, molecular biology, neurobiology, comparative biology, systematics, ethology, ecology, etc. Each of these domains demands a different way of explaining. The biological matter is much more complex than what Mayr thought with his distinction between 'biology of remote causes' and 'biology of proximate causes'.[4]

To realize a meta-model capable of ruling the explanation in all

[4] E. Mayr, 'Cause and Effect in Biology', *Science* **134**, 1961, 1501–6; cf. E. Mayr, *The Growth of Biological Thought. Diversity, Evolution, and Inheritance* (Cambridge (Mass.): The Belknap Press, 1982).



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these biological domains seems an extremely ambitious project. May be it is. However we should embark on it, that is, on the construction of what I call *a contextualized approach to biological explanation*. It will be a *synthetic attempt* which will oblige us to neglect some details of the extremely accurate analyses produced in the seventy-years long history of philosophy of scientific explanation. Nevertheless, this is a price that we must pay to now attempt this unavoidable task. To travel safely through the world we need both extremely precise and detailed topographical maps of the region we want to visit, and general maps which allow us to understand where that region is. What follows is an attempt to design a general map which, nevertheless, has been made possible precisely because topographers have already designed the particular maps.

I believe the best way to successfully pursue this aim is to start from the pragmatical explanatory model offered by B. van Fraassen.[5] In what follows, it will be considered as a powerful meta-model capable of ruling the many explanatory models that can be used in the biological domains. However, first I will emphasise some necessary assumptions. Then I will focus on the meta-model. Eventually I will show how it works in some cases.

## 2. Preliminary assumptions

Before starting with the meta-model, some assumptions, that cannot be dwelt upon here, must be made explicit. The first one concerns biological laws. As is known, there is a great debate on whether there are scientific laws, how they are characterized, and what their epistemological status is. Besides this debate, there is a sometimes overlapping one on whether there are biological laws, how they are characterized, what their epistemological status is, and how they differ from the other scientific laws, especially from the physical laws.

As just stated, I cannot face these problems here, and for the scope of the following discussion it suffices to assume that in the biological field there are *nomological generalizations*. Are they laws in a real sense? Are they ontologically grounded in nature, or Kantianly constructed by the knowing subject, or mere descriptions of biological regularities? It does not matter here, and I may suspend judgment.

However, I do not commit myself in a given way just because I use the term 'nomological'. In the ancient Greek, *nomos* indicated

[5] B. C. van Fraassen, *The Scientific Image* (Oxford: Oxford University Press, 1980).



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neither a scientific law, nor a law of nature (if there were differences between laws of nature and scientific laws), nor, in particular, a deterministic scientific law. *Nomos* was used in the political domain more than in the philosophy of nature domain and it designated the rule to behave by. In this paper by *nomological generalizations* I mean generalizations 'describing' (without any epistemological commitment) natural events and processes, in particular biological events and processes.[6]

The second assumption concerns a characteristic of such nomological generalizations. Are they deterministic or probabilistic? It is widely recognized that biology is a probabilistic field: sometimes a causal probabilistic field, sometimes a non-causal probabilistic field. Everything that happens in the biological domain happens with a certain degree of probability (this means, of course that something may happen with certainty, that is, with probability equal to 1). Therefore I assume that each nomological generalization in the biological field can be interpreted as a probabilistic rule on the occurrence of certain events.

At this point a question could be posed: is this probabilistic rule in nature? That is, should we give to biological probability a strong ontological foundation? Along the line of metaphysical *epoché* here assumed, I leave these problems aside. However, on the one hand, the problems connected with a frequentist interpretation of probability are well known in the discussion on explanation, as I will briefly recall later. On the other hand, the propensity interpretation of probability, which was suggested for many probabilistic explanatory models, involves a too realistic commitment which I want to avoid. Nevertheless, if I should denounce my faction, I would confess to prefer a subjectivist interpretation *à la* B. de Finetti of the calculus of probability.[7] But this is not the right place to discuss it, or argue for it.

From what has been said above, it should be clear that beyond the pragmatical interpretation of the meta-model, I am proposing also a particular interpretation of the biological explanation. It is not considered as connected with the search for the ontological (causal or non causal) mechanisms of the world. Instead, more modestly, *it is seen as the search for the plausible set of statements which make up a*

[6] With reference to the discussion on the relations between biological explanations and biological laws, even if arriving at a different conclusion, cf. A. Rosenberg, 'How is Biological Explanation Possible?', *The British Journal for the Philosophy of Science* **52**, 2001, 735–60.

[7] Cf. M. C. Galavotti and R. C. Jeffrey (eds.), *Bruno de Finetti's Philosophy of Probability*, *Erkenntnis* **31**, 1989.



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*particular kind of context-dependent answers to a particular kind of context-dependent questions*.

## 3. The pragmatical meta-model

In van Fraassen's proposal explanation is considered in the light of the triad context-theory-facts, rather than in the light of the couple theory-facts. It follows that the formulation of the demand for explanation—that is, the why-question—and the reply—that is, the because-answer - depend both on who formulates them and on the context in which they are formulated.

From this point of view, the logical structure of the explanation is no longer the pivotal issue, even if it remains necessary. Instead the communicative conditions that motivate and support it turn out to be the focus of the matter. This involves realizing that 1) certain why-questions linked to a given context cannot be posed in a different context; 2) in different contexts the same why-questions do not necessarily refer to the same problems and therefore do not require the same because-answers; 3) in the same disciplinary domain the same why-question can be posed at different levels, giving rise to different because-answers.

As C. L. Hamblin already pointed out,[8] this leads to the idea that a question is identifiable by its answer. But an answer is given by an answerer, who provides it starting from a given context. Therefore, more correctly, a question is identifiable by the context from which the answer is given. This puts us in the uneasy, but necessary, situation of defining for every explanatory situation the correct context, and therefore the set of acceptable and unacceptable answers. Each science, and each scientific domain, that is, each context brings with itself a certain structure of questions and answers, i.e., of acceptable and unacceptable explanations. Then referring to a well-defined context is the equivalent of marking the boundary of the admitted questions and admissible answers. This is exactly what was emphasized by S. Bromberger, when he claimed that to provide an adequate explanation we should give more than a nomological generalization under which the *explanandum* can be subsumed.[9] We need to show that such a nomological generalization is the right one for the explanation we are proposing.

[8] C. L. Hamblin, 'Questions', *Australian Journal of Philosophy* **36**, 1958, 159–68.

[9] S. Bromberger, 'Why-Questions', in R. G. Colodny (ed.), *Mind and Cosmos* (Pittsburgh: University of Pittsburgh Press, 1966), 86–111.



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To sum up, to conceive explanation as an answer to a question requires particular attention to the communicative activity concretely carried out by speakers inside describable enunciative contexts containing beliefs and knowledge. However, of course, it does not preclude the possibility of setting logical structures or ontological commitments in those contexts.

Without any doubt van Fraassen's proposal provides us the most satisfying framework for such a choice. Given a why-question, Q, it is constituted of three elements: the topic of the question, $P_k$; the contrast class, $X=\{P_1, \ldots, P_k, \ldots\}$, which includes the topic; and the relevance relation, R. Hence, for van Frassen,

> *a why-question Q is an ordered triple* $\langle P_k, X, R\rangle$,

and

> *because A is an answer to* $Q = \langle P_k, X, R\rangle$ *if 1) only* $P_k$ *is true among the elements of X; 2) A is true; 3) A bears the relation R with* $\langle P_k, X\rangle$.

Such an approach is quite powerful, since it is capable of justifying the use of why-questions as much in science as in history, and in everyday life. However, precisely this applicative extent represents, for some critics, a weakness due to the difficulty of specifying what characterizes the scientific explanations and their respective relevance relations.[10] This is a real problem that cannot be solved, as for example Salmon suggests,[11] by tying the relevance relation to a given idea of science and turning it into a well-defined objective and causal relation. If it is too vague to render all the relevant relations acceptable, it is too committing and limiting to choose a given one on the basis of a particular ontological assumption about the structure of the word. Instead, we can individuate the right relevance relations by tying them to *the context in question*.[12] Said differently,

[10] Cf. Ph. Kitcher and W. C. Salmon, 'Van Fraassen on Explanation', *Journal of Philosophy* **84**, 1987, 315–30.

[11] Op. cit. note 3.

[12] With reference to this point, both P. Railton, in 'Probability, Explanation and Information', *Synthese* **48**, 1981, 233–56, and W. C. Salmon, in *Four Decades of Scientific Explanation* (Minneapolis: University of Minnesota Press, 1990), seem to agree that we should distinguish between *relevance relations* and *salient relation*s. Even if the correct Latin etymon of 'relevance' and 'salience' does not support such a distinction, they claim that the former has an ontological and objective value while the latter is only subjective. It means that a 'relevant relation' is a relation really existing in nature, while a 'salient relation' is a relation having value only with reference to the subject. Beyond the fact that for a



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the alleged lack of restriction on the relevance relation R lies in the fact that if we start from the pragmatic model *qua* pragmatic model, R is, of course, not specified.[13] But let us consider the matter from the other side.

Given a biologist b, let $K_b$ be its entire background knowledge, with $K_b \subseteq K_B$, where $K_B$ is the entire biological knowledge at disposal of the biologists at the time in which the biologist b is living. Finally, let $C_b \subseteq K_b$ be the particular context from which the given biologist b answers the question Q. If we start considering what the relevance relation is from $C_b$, we find that R is no longer vague, since it is precisely individuated and fixed by $C_b$. Of course there may be an audience that do not accept that answer, precisely because it does not accept the R indicated. But this is another problem completely. It concerns the choice and the acceptability of the context $C_b$ from which the answer is offered. Some may be disposed to ask for a different R, since they prefer a different context $C_b$ from which the answerer should start. In this case, a debate between the opponents and the answerer b can arise. Nevertheless, this dialectical situation has nothing to do with the fact that choosing a given $C_b$ means indicating and fixing R, *at least for the answerer, that is, the explanainer* b.

It should be pointed out that, according to this way of thinking, R is no longer necessarily a causal (even if a probabilistic causal) relation. Its nature is fixed by $C_b$, as I will exemplify in what follows.

Moreover, by introducing $C_b$, another problem met by van Fraassen vanishes. Van Fraassen spoke in terms of background knowledge K which was intended as constituted by a given theory

[13] I agree that to invoke the context is not enough to epistemologically deal with it. An example of this rather widespread ingenuity may be found in a book by K. Lambert and G. Gordon Brittain jr., *An Introduction to The Philosophy of Science* (Reseda: Ridgeview Publishing Co., 1979). Here they observe that in the classic model of explanation proposed by Hempel and Oppenheim a two-place-relation between an event and a set of assertions is posited. Instead they emphasize the role of the context and a five-place-relation: phenomena, *explanans*, theories, circumstances, and people. Unfortunately, in this way the context becomes totally ungovernable and referring to it is pointless for operative aims.

pragmaticist the meaning of two terms collide in one, particularly in the second one, it is worth noting that *each relation is theory-laden*, or, rather, *context-laden*. Therefore to determine if a relation is really ontologically relevant or simply subjective salient seems extremely difficult, if not impossible.



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*plus* the data in relation to which the question Q is valued. In this case, given K, the why-question Q can be posed only if K implies $P_k$. In addition, the evaluation of a possible because-answer to the given why-question Q would be possible only by referring to a certain part of it, K(Q), which is not easily identifiable. Nevertheless it seems quite clear that K cannot be identified only by the background *plus* a certain set of adjunctive information. We must identify it in a better way. This problem is overcome if we start from $C_b$. As said, $C_b$ is a subset of K relativised to b, but given b also $C_b$ is given.

Let us move to another weakness. According to some critics, not all the explanations are why-questions. Some, for instance, are how-is-it-possible-questions, or how-does-it-happen-questions, etc. The difference, for example for Salmon,[14] should lie in the fact that only the why-questions are real scientific explanations, since they demand a true causal explanation of the occurrence of the *explanandum*, while the other questions demand simply a plausible explanation.

In a pragmatical approach, as the one here supported, the explanations are requested to furnish plausible, as to a given context, answers, and not ontologically grounded answers. From this point of view, each kind of explanation can be translated, or rewritten, into a why-question where the 'why' is made explicit by resorting to the context. Sometimes the context may require that the why-question is a how-is-it-possible-question, sometimes a how-does-it-happen-question, sometimes other kinds of questions. It depends. In particular it depends on $C_b$.[15]

At this point, it is worth noting that, in a context-dependent approach, the first two conditions posed by van Fraassen (1. only $P_k$ is true among the elements of X; 2. A is true) can be reformulated in a weaker way: among the elements of X, *$P_k$ is the more valid and*

[14] Op. cit. note 3.

[15] A similar objection was formulated by C. B. Cross who sustained that the how-questions are different from the why-questions. Cf. C. B. Cross, 'Explanation and the Theory of Questions', *Erkenntnis* **34**, 1991, 237–60. According to Cross, we should construct a pragmatic model for the why-questions and a pragmatic model for the how-questions. Only the latter is furnished by van Fraassen. Actually each question can be translated into, and therefore stenographed by, a why-question. It is the context that allows us to interpret that why-question as a how-question. For example, "How are force and mass related?" (a case discussed by Cross) is easily translatable into "Why force and mass?", and the context will allow us to understand the real meaning of that 'why'.



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*plausible relatively to a given context*; *A is the more valid and plausible relatively to a given context*. In a context-dependent approach, *truth turns into validity and plausibility relatively to a given context*.

Now we have all we need to rethink van Fraassen's proposal from the point of view of $C_b$ and therefore to state the meta-model:

*because* $A_b$ *is an answer given by the biologist b, with reference to its context* $C_b \subseteq K_b$, to $Q = \langle P_k, X, R \rangle$, *if*

1) *X is the contrast class enucleated by* $C_b$*;*
2) $P_k$ *is the more valid and plausible topic, with reference to* $C_b$*, of the question*;
3) *there exists at least one* $A_b$*, valid and plausible with reference to* $C_b$*, which bears the R-connection with* $\langle P_k, X \rangle$*;*
4) *the R-connection is probabilistic and it is individualized and fixed by* b *by resorting to its* $C_b$ *(the probabilistic interpretation is to be intended in a subjectivist way* à la *de Finetti)*.

## 4. Which probabilistic model?

In section 2, I have assumed that each biological science, and therefore each $C_b$, demands a probabilistic explanation. Of course, this does not mean that some biological events can never occur with certainty. But, as said, they can be considered in a probabilistic framework as events to whose occurrence we attribute subjectivist probability equal to 1.

If any $C_b$ is made up of probabilistic generalisations of what happens in nature, we should both interpret the why-questions as probabilistic why-questions and the because-answers as probabilistic because-answers. But, how to cope with this probability framework?

Seventy years of explanatory models have provided us with many probabilistic models. Some of them are worn-out models, some others work better. Here it is not necessary to trace the history, but some elements must be recalled. It suffices to say that Hempel's proposal of a statistical-inductive model was wrecked by the ambiguity problem, which could be solved neither by resorting to the total evidence requirement, nor to the maximal specificity requirement.[16] Also Salmon's statistical relevance model bumped

[16] Cf. G. C. Hempel, 'Deductive-Nomological vs. Statistical Explanation', *Minnesota Studies in the Philosophy of Science*, III, H. Feigl and G. Maxwell (eds.) (Minneapolis: University of Minnesota Press,



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into two not yet solved problems: 1) the non-ambiguous discrimination between statistical relevance and causal relevance; 2) the extremely difficult definition of homogeneous partitions.[17] Moreover J. A. Coffa and J. H. Fetzer's idea to put propensities inside a probabilistic explanation came up against the wall of grasping what propensity means.[18] Etc.

Nevertheless, it seems that what P. Railton proposed has less serious problems.[19] He argued that a probabilistic explanatory framework must contain 1) a probabilistic nomological generalization (derived from a superior generalization giving the real mechanisms occurring in the world); 2) a deductive inference of the *explanandum* from that probabilistic nomological generalization; 3) a statement claiming that the *explanandum* has (or has not) really occurred (the so-called 'parenthetic addendum'). As is well-known, the third requirement allows us to have a probabilistic explanation that does not fall into Hempel's ambiguity, nor is it deductively trivial. Actually Railton's idea was addressed to providing a model permitting the unveiling of the mechanism by means of which the world works. Yet, here I am not interested in the real (in a strong metaphysical sense) mechanisms by means of which the world works, but in the representations of both how the world is and how it works. From this point of view, a slightly modified version of Railton's proposal seems still less problematic, because the derivations of the probabilistic nomological generalization in the *explanans* from a relevant theoretical account of the real mechanisms occurring in the world is not required.[20] In particular, the

[17] Cf. W. C. Salmon *et al*., *Statistical Explanation and Statistical Relevance* (Pittsburgh: Pittsburgh University Press, 1971).

[18] Cf. J. A. Coffa, *The Foundation of Inductive Explanation*, Ph.D. Thesis, University of Pittsburgh, 1973; J. H. Fetzer, 'A World of Dispositions', *Synthese* **34**, 1977, 397–421.

[19] P. Railton, 'A Deductive-Nomological Model of Probabilistic Explanation', *Philosophy of Science* **45**, 1978, 604–13; P. Railton, 'Probability, Explanation and Information', *Synthese* **48**, 1981, 233–56.

[20] With reference to a tentative explication of the notion of 'mechanism', cf. S. Glennan, 'Rethinking Mechanistic Explanation', *Philosophy of Science* **69**, Suppl., 2002, 342–53. Note that Railton's approach was inserted into a framework where the calculus of probability was interpreted propensionally. It is worth pointing out that also Railton's model has a

1962); C. G. Hempel, *Aspects of Scientific Explanation* (New York: The Free Press, MacMillan Company, 1965); J. A. Coffa, *The Foundation of Inductive Explanation*, Ph.D. Thesis, University of Pittsburgh, 1973; J. A. Coffa, 'Hempel's Ambiguity', *Synthese* **28**, 1974, 19–26.



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following scheme is sufficient:

Explanatory deduction:

- All the biological events of the type T are R-connected with a probability p with the biological events of the type $T^1$.
- The event e is a biological event of the type T.

*Therefore*

- e has the probability p to be R-connected with biological events of the type $T^1$.

Parenthetic addendum:

- A biological event of the type $T^1$ really occurred.

Of course the just mentioned R-connection is precisely the R-connection discussed in the previous section, that is, the one identified and fixed by the biologist-answerer b with reference to its particular $C_b$.

## 5. Applying the contextualized approach to biological explanation

At this point we have the pragmatic meta-model to rule the different biological explanatory models, that is, any biological why-question in function of b's $C_b$. For the reference to b's $C_b$ allows us 1) to identify and fix the probabilistic R-connection and therefore to make explicit the particular meaning of the 'why' at issue; 2) to individuate the right contrast class X.

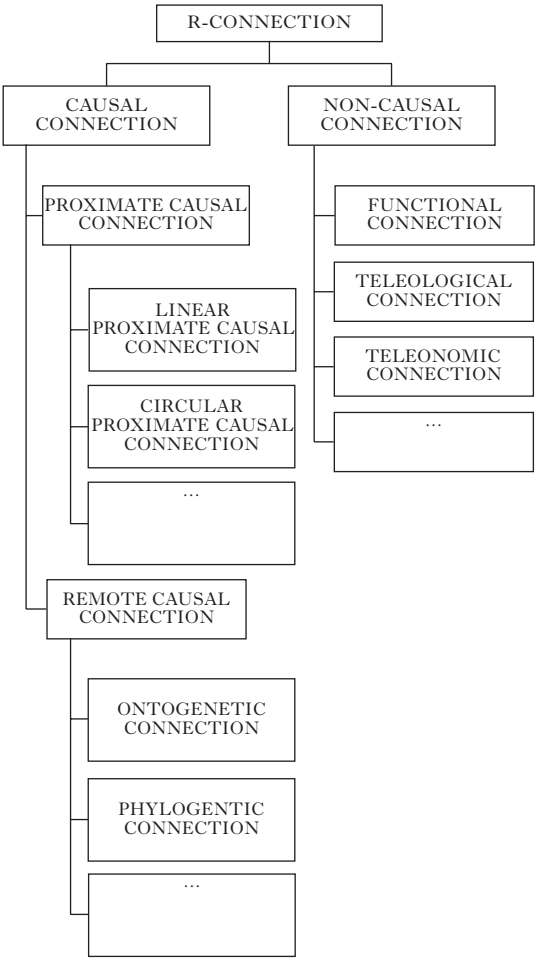
Therefore any time we have a biological why-question, the first step concerns understanding who the biologist-answerer (the explainer) b is, and which the $C_b$ from which b answers (explains) is. Then, in a natural way, we have the identification of the R-connection and so the meaning of the 'why'. It follows that we have as many kinds of R-connection (and therefore of why) as requested by the different $C_b$ and the different b.

Now it is time to sketch some of the possible R-connections (and therefore of why), that is, some of the possible explanatory models. I will do this by following the scheme below, which proposes a plausible taxonomy of the R-connections:

counterexample, cf. S. Gluck and S. Gimbel, 'An Intervening Cause Counterexample to Railton's DNP Model of Explanation', *Philosophy of Science* **64**, 1997, 692–7.



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### 5.1. The causal R
### 5.1.1. The linear proximate causal R

Let us begin exemplifying questions like Q = 'Why does a molecule M bind with a molecule $M^1$ in the cell nucleus?'. Let us suppose that the answerer b is a molecular biologist with its particular $C_b$. In this case $C_b$ demands a linear proximate causal explanation, that is, it indicates and fixes a linear proximate causal connection. Therefore we have



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*because* $A_b$ = *'In the nucleus M and* $M^1$ *have probabilistic causal interactions, whose probabilistic effect is their bond' is an answer given by the molecular biologist b, with reference to its context* $C_b \subseteq K_b$, to $Q = \langle P_k, X, R \rangle$, *where*

1) $X$ *is enucleated by* $C_b$;
2) $P_k$ = *'the molecule* M *binds with the molecule* $M^1$*' is valid and plausible in b's* $C_b$ *among the elements of* $X$ = {'M *binds with* $M^1$', 'M *binds with* $M^2$', 'M *binds with* $M^3$', …};
3) $A_b$ *is valid and plausible with reference to* $C_b$, *and it bears a probabilistic linear causal proximate connection with* $\langle P_k, X \rangle$.

In this case, the explanatory structure is

Explanatory deduction:

- Inside the cell nucleus, there is a probability p of having a bond between molecules of the type T and molecules of type $T^1$, and the bond is the probabilistic effect of their probabilistic causal interactions.
- M is a molecule of the type T and $M^1$ is a molecule of the type $T^1$, and they are probabilistically interacting in the cell nucleus.

*Therefore*

- The bond between M and $M^1$ has the probability p to be realized.

Parenthetic addendum:

- A bond between the molecules M and $M^1$ has really occurred.

Let us see the explanation in detail. In the cell nucleus, two molecules, say M and $M^1$, bind, if they interact, in particular if they interact correctly. So that M and $M^1$ can reach such a distance that the electromagnetic forces between them are effective (and therefore they can bind), they must be pushed one against the other. Inside the cell, there is, beyond the osmotic pressure from the outside, a random thermal motion 'kept alive' by the thermal energy. The latter has to be considered the first cause of the probabilistic causal chains occurring inside the cell.

As said, not only do the two molecules have to interact, they have to interact correctly. What does this mean? We know that the molecular chains are kept together by covalent bonds and that two parts of the same molecule, or two different molecules, can be bound by non covalent bonds. However, we know that a non covalent bond is not sufficient to keep the two sites of the two molecules



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bound.[21] Not only does the thermal motion allow the interactions among the molecules, but it also causes difficulties in the bonds due to both the thermal agitation of the molecules considered in themselves and the possible pushes caused by other molecules.[22] It follows that between two molecules the force to keep them stuck is sufficient if between the two respective binding sites a sufficient number of non covalent bonds are realized.

This happens if the two binding sites have suitable three-dimensional atomic configurations. In other words, the non covalent bonds between two molecules are sufficient, if the stereospecificity of the two binding sites is complementary, that is, if they match in a good way.

Let us try to be more formal, and let us consider a generic intracellular molecular interaction. Let T a type of molecules, and let us suppose that inside the cell we have also molecules of types $T^1$, …, $T^n$. In such a case, $p_{T/T^i}$ is the probability that a molecule of the type T interacts with an arbitrary molecule of the type $T^i$. Of course, the probability strongly depends on the concentration of the two molecules and on the strength of their thermal motion.[23]

We said that it is not sufficient the occurrence of an interaction; it must be a good one, that is, it must produce a bond. For this to happen an event must take place: the two interacting molecules must have sufficient kinetic energy, in particular a kinetic energy capable of overcoming the two repulsive potential barriers of their interacting sites. Of course this kinetic energy depends on the strength of the thermal motion, that is, on the temperature t. Let $p_k$ be this probability.

There is a last step. This concerns the fact that the two molecules, which have interacted and whose kinetic energy allows the overcoming of the potential barriers, actually have to have the two binding sites 3-dimensionally conformed in such a way as to permit a correct and stable sticking. Let $p_c$ be the property that the two interacting molecules have the correct 3-dimensional configuration.

[21] The non covalent bond is from 10 to $10^2$ weaker than a covalent bond, especially in aqueous solution as there is inside the cell. In the case of non covalent bonds, the binding energy is 1÷7 Kcal/mole; in the case of covalent bonds, the binding energy is 50÷100 Kcal/mole.

[22] At room temperature, the thermal energy of a molecule is about 0·6 Kcal/mole.

[23] To have an idea of the enormous number of intracellular collisions, let us recall that if there is the standard concentration of ATP (about 1mM), each site of a given protein will interact more or less $10^6$ times per second with ATP molecules.



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It follows that the probability $p_{bond}$ that a molecule T generates a bond with a certain other molecule $T^i$ is

$$P_{bond}=p_{T/T^i} \cdot p_k \cdot p_c$$

It may happen, and it actually happens, that the interaction between two molecules is mediated by other molecules: the enzymes. In this case the situation changes and we have to take into account both the probability of the interaction between the enzyme and the first substrate, $p_{e/s1}$, and the probability that this is a good (in the sense just considered) interaction, $p_{e+s1}$. Moreover we have to consider the probability of the interaction between the complex (enzyme + first substrate) and the second substrate, $p_{(e+s1)/s2}$. Finally the probability that this is a good interaction must added. In conclusion, the probability of the bond between the two molecules is

$$P_{bond}= p_{e/s1} \cdot p_{e+s1} \cdot p_{(e+s1)/s2} \cdot p_{(e+s1)+s2}$$

At this point our molecular biologist b, starting from its $C_b$, has arrived at its explanation of the nuclear bonds by resorting to a particular probabilistic connection individualized and fixed exactly by $C_b$.

### 5.1.2. The circular proximate causal R

Sometimes to answer certain questions, the molecular biologist b cannot use a linear probabilistic causal connection, but it must introduce a probabilistic circular causal connection. For example, let us think about questions like Q = 'How is the right density of proteins P, expressed by a gene G, realized?'. Given b's $C_b$, we have

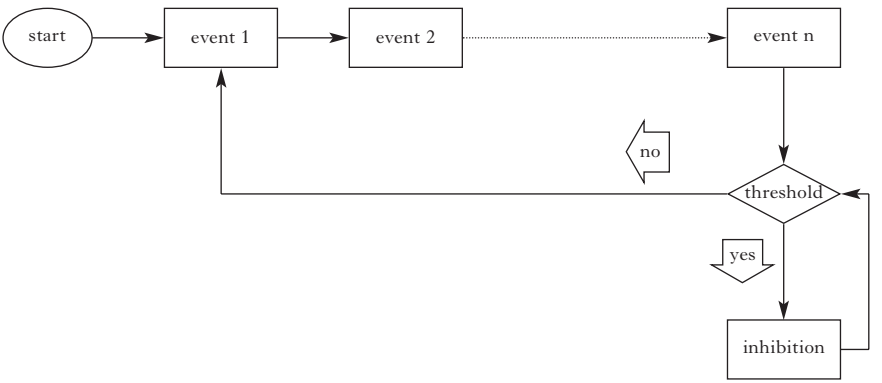
*because $A_b$ = 'There is a genetic homeostatic process allowing the regulation of the right density of proteins P, expressed by the gene G' is an answer given by the molecular biologist b, with reference to its context* $C_b \subseteq K_b$, to $Q = \langle P_k, X, R\rangle$, *where*

1) *X is enucleated by* $C_b$;
2) $P_k$ = *'the right density of proteins P, expressed by a gene G, is realized' is valid and plausible in b's* $C_b$ *among the elements of* $X$ = {*'the right density of proteins P, expressed by a gene G, is realized', 'the right density of proteins P, expressed by a gene G, is accidental', 'the right density of proteins P, expressed by a gene G, is possible', …*};
3) $A_b$ *is valid and plausible with reference to* $C_b$, *and it bears a probabilistic circular causal proximate connection with* $\langle P_k, X\rangle$.



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In this case we no longer have a linear probabilistic causal chain, but a circular probabilistic causal chain schematisable as follows:




Of course, in this circular causal chain, each step from an event to another one is probabilistic linear proximate causal, and it can be explained by means of the model shown in the previous section.

Let us exemplify such circular causal connection by recalling a well known case: the regulation of the tryptophan expression in *Escherichia coli*. In this bacterium there is a gene cluster transcribed from only one promoter into a mRNA molecule by the RNA-polymerase. The gene cluster codes for a set of enzymes which allow the biosynthesis of an amino acid: the tryptophan.

To begin the expression of the gene cluster, a molecule of RNA-polymerase must bind with the promoter. If this succeeds, the enzymes are coded and the tryptophan synthesis begins. Nevertheless, it can occur that the tryptophan produced is sufficient, or that it enters *Escherichia coli* since it is in the medium where the latter is living. In this case there is no need of other tryptophan, and its expression must stop. This happens because within the promoter, there is a short nucleotide sequence, called *operator*, which is recognized by a gene regulatory protein called *tryptophan repressor*. If the tryptophan repressor binds the operator, then the RNA-polymerase cannot bind to the promoter and the expression of the enzymes cannot begin. This is the case when the tryptophan level is high and a molecule of tryptophan binds to the tryptophan repressor, which in turn binds to the operator and the expression is blocked. As soon as the tryptophan level drops, the tryptophan repressor releases the tryptophan, and it leaves the



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operator. Now the promoter is free to accept the RNA-polymerase and the enzymes expression starts again.

In this way, the molecular biologist b has provided a because-answer to the why question at issue by starting from its $C_b$.[24]

### 5.1.3. The ontogenetic and phylogenetic Rs

Asking for the remote causes of a given biological event means asking for the historical reconstruction of the causal path which started with an initial cause and have finished with that event. This is exactly what both the ontogenetic why and the phylogenetic why demand. Therefore if we want to grasp which kind of answer they request, first we should grasp what a historical explanation is. Here I cannot enter into the details,[25] nevertheless it seems that there is an agreement on some fundamental points highlighted by W. Dray in his seminal *Laws and Explanation in History*.[26]

[24] Note that this is a paradigmatic explanation of all the homeostatic processes, or feedback processes. As is well known, this circular causal model of explanation has been indirectly considered with the introduction of the term 'homeostatic' by W. Cannon, and it has been deeply analysed within the cybernetics and within the theory of systems since the beginning of the 1950's. Cf. W. Cannon, *The Wisdom of the Body* (New York: Norton, 1932); N. Wiener, *Cybernetics, or Control and Communication in the Animal and the Machine* (Cambridge (Mass.): MIT Press, 1948–1965[2]); W. B. Ashby, 'Self Regulation and Requisite Variety', in W. B. Ashby (ed.), *Introduction to Cybernetics* (London: A. P. Watt & Sons, 1956); P. Weiss, 'Animal Behaviour and System Reaction', *Yearbook Society of General System Research* **4**, 1959, 1–44; L. von Bertalanffy, *General System Theory* (New York: Braziller, 1969); H. Maturana and F. Varela, *Autopoiesis and Cognition. The Realization of Living* (Dordrecht: Reidel, 1980); H. von Foerster, *Observing Systems* (Seaside (Ca): Intersystems Publications, 1981); P. Watzlawick (ed.), *Die erfundene Wirklichkeit* (München: R. Piper & Co. Verlag, 1981); P. Dumouchel and J.-P. Dupuy (eds.), *L'auto-organisation. De la physique au politique* (Paris: Seuil, 1983); N. Luhmann, *Soziale Systeme. Grundriss einer allgemeinen Theorie* (Frankfurt am Main: Suhrkamp Verlag, 1984).

[25] Cf. P. Gardiner (ed.), *The Philosophy of History* (Oxford: Oxford University Press, 1974); F.R. Ankersmit, 'The Dilemma of Contemporary Anglo-Saxon Philosophy of History', *History and Theory* **25**, 1986, 1–27; M. G. Murphey, *Philosophical Foundations of Historical Knowledge* (New York: State University of New York, 1994); F. R. Ankersmit and H. Kellner (eds.), *A New Philosophy of History* (London: Reaktin Book, 1995).

[26] W. Dray, *Laws and Explanation in History* (Oxford: Oxford University Press, 1957).



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First of all, Dray did not deny the causal explanation of historical events; instead he denied the relevance (but not the logical necessity) of the nomological generalizations in the historical explanations.[27] The nomological generalizations are logically necessary, but they are not so historiographically relevant as the causal conditions that have led to that event. Therefore, the historian's job does not concern the search for the nomological generalizations, but for the right succession of the conditions that have caused the event to be explained. From this point of view, the objection brought up, for example, by C. B. Joynt and N. Rescher,[28] according to which history is made up of unique events, loses its strength. In a historical explanation we emphasize the particular conditions occurring in that particular moment precisely because we want to explain an event which is unique.

If we must focus our historiographical attention on the succession of causal conditions that have led to the event to be explained, we need a good technique to find out such a causal imputation.[29] Once this point has been taken into account, it is easy to grasp what Dray's *continuous series model* is: the reconstruction of the continuous succession of causal conditions that from an initial event, occurring at time $t_0$, have led to the final event, occurring at time $t_f$.

Thus if we want to explain from a phylogenetic point of view, why a certain living being possesses a certain phenotypic trait T at a time $t_f$, we must reconstruct the phylogenetic history that has led to T by individualizing the continuous series of sufficient causal conditions characterizing each moment of the period $[t_0, t_f]$. *Mutatis mutandis*, the same happens if we want an ontogenetic explanation of T.

[27] With reference to the seminal debate on the nomological generalisations in the historical explanations, cf. G. C. Hempel, 'The Function of General Laws in History', *Journal of Philosophy* **39**, 1942, 35–48; M. Mandelbaum, 'Historical Explanation: the Problem of Covering Laws', *History and Theory* **1**, 1961, 229–42.

[28] C. B. Joynt and N. Rescher, 'The Problem of Uniqueness in History', *History and Theory* **1**, 1961, 150–62.

[29] To enter into this problem is not possible here. Note that its contemporary discussion may be dated back to M. Weber, 'Kritische Studien auf dem Gebiet der kulturwissenschaftlichen Logik', (1906); now in M. Weber, *Gesammelte Aufsätze zur Wissenschaftslehre* (Tübingen: Mohr, 1922). Cf. G. Boniolo, 'Fictions, Judgments Of Possibility And Counterfactuals: Is There Room For Hermeneutics?', in R. Dottori (ed.), *Normativity and Legitimacy, Yearbook for Philosophical Hermeneutics*, (Münster: LIT, 2001), 55–173.



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Nevertheless, to correctly grasp the question of the phylogenetic history of T, Dray's continuous series model is not sufficient. We need to integrate it with L. Wright's etiological approach, which allows us to insert what is typically biological into a general historical reconstruction of the past.[30] From a biological point of view, reconstructing the past of a trait, that is, offering a phylogenetic answer to the question Q = 'Why the trait T of the living being B?', means understanding T 'in the light of evolution'.[31] In other words the existence of T in the living being B at a time $t_f$ must be explained also by taking into account that some of B's ancestors had T and T was positively selected by the environment E in which they lived. And Wright's indications permit us to grasp this aspect.

However, it is not so easy to individuate all the steps of the phylogenetic history of B (and of its trait T): this is the core problem of the evolutionary reconstruction of the past of B's T; or, said differently, the reconstructions of Dry's *continuous probabilistic series of causal conditions*; or M. Weber's possible causal scenarios from the T of B's ancestors to B's T.[32] And this is exactly the problem tackled by E. Sober in his *Reconstructing the Past*.[33]

When, at the end of its job, the evolutionary biologist has reconstructed the phylogenetic history of that trait, or of that living being, he has also offered a phylogenetic-answer to the question at issue, that is, an answer based on a phylogenetic connection. Let us consider, for example, an evolutionary because-answer of Q = 'Why the trait T of the living being B at the time $t_f$?'. Let us suppose that our evolutionary biologist b starts from a $C_b$ that takes into consideration only genetic mutations and natural selection. In this case, we may state that the nomological generalization we need is

> N = 'Part of the mutations of the genotype G of a living being X leads to variations of its phenotype P which allow a positive selection by the environment E in which X lives'.

Suppose that

[30] L. Wright, 'Functions', *The Philosophical Review* **82**, 1973, 39–168; L. Wright, *Teleological Explanation* (Berkeley: University of California Press, 1976).

[31] Cf. Th. Dobzhansky, 'Nothing in Biology Makes Sense Except in the Light of Evolution', *American Biology Teacher* **35**, 1973, 125–9.

[32] Op. cit. note 29.

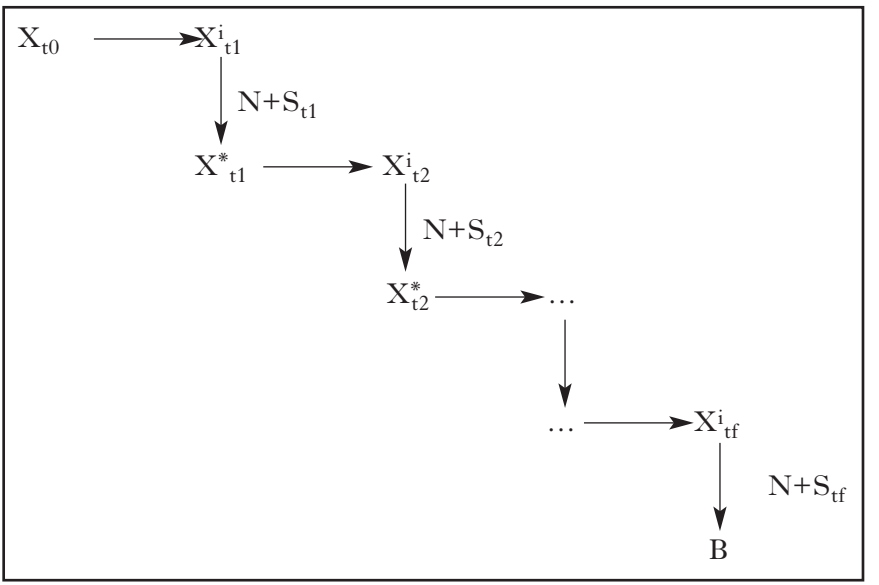
[33] E. Sober, *Reconstructing the Past: Parsimony, Evolution, and Inference* (Cambridge (Mass.): MIT Press, 1988).



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- $X_{t0}$ is the starting living being at time $t_0$ possessing a genotype $G_{t0}$ and a phenotypic trait $T_{to}$ in an environment $E_{t0}$ (note that we are interested only in a part of such environment, i.e., in $E_{t0}(X_{t0})$).
- $X^i_{tl}$ is one of the possible offspring of the $X^i_{tl-1}$ characterized by a variation of the genotype $G_{tl-1}$.
- $X^*_{tl}$ is the living being positively selected at the time $t_l$ among the $X^i_{tl}$.
- B is the living being possessing T whose phylogenetic history is to be reconstructed.

In this case we can apply the continuous series model integrated with the etiological indications and we obtain the following scheme:




In this model, the nomological generalization N is not so historiographically (in our case, also phylogenetically) important. The real core is given by the $S_{ti} = <G_{ti}(X^i_{ti}), T_{ti}(X^i_{ti}), E_{ti}(X^i_{ti})>$, that is, the set of the tentative causal conditions which allows us to explain the trait $T_{ti}$ of the being $X_{ti}$ living in an environment $E_{ti}$, and therefore, step by step, to arrive at the explanation of the B's T at $t_f$.

The same explanatory framework can be applied in the case of the ontogenetic explanation. Also in this case we need Dry's model plus Wright's integration. The only difference concerns the fact that now the biologist is no longer searching for the phylogenetic continuous series of causal conditions, but for the ontogenetic



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continuous series of causal conditions. Moreover, also the nomological generalization will be different. Now, it will resort to the genetically, epigenetically, and environmentally governed properties which drive the ontogenesis.[34] Nevertheless, also in this case the main problem concerns the individuation of the succession of the causal scenarios which have led a living being from an ontogenetic stage to a subsequent one.

However any time we have questions like 'How has the phenotypic trait T been reached by the being B living in an environment E?', if the answerer is an evolutionary (or developmental) biologist b, we have

*because* $A_b$ = *'The being B living in an environment E has the trait T since it is the result of its evolution (of its development)' is an answer given by the evolutionary (developmental) biologist b, with reference to its context* $C_b \subseteq K_b$, to $Q = \langle P_k, X, R \rangle$, *where*

1) $X$ *is enucleated by* $C_b$;
2) $P_k$ = *'The being B living in an environment E has the trait T' is valid and plausible in b's* $C_b$ *among the elements of* $X$ = {*'The being B living in an environment E has the trait T', 'The being B living in an environment E has the trait* $T^1$*', 'The being B living in an environment E has the trait* $T^2$*', …*};
3) $A_b$ *is valid and plausible with reference to* $C_b$, *and it bears a probabilistic evolutionary (developmental) connection with* $\langle P_k, X \rangle$.

[34] Here by *genetically governed properties* I mean those phenotypic properties which appear only thanks to the genetic expression. Of course a genetically governed property is totally different from the *property of having a particular genome*. The former concerns a possible phenotypic result of the latter. By *epigenetically governed properties* I mean those phenotypic properties which appear thanks to the expression of certain genes belonging to certain cells activated by their interactions with surrounding cell populations and with their peptidic products. However, there is a discussion on the definition of 'epigenetic process' and 'epigenetic property'. Cf. the seminal C. H. Waddington, *The Strategy of the Genes* (London: Allen & Unwin, 1957); also E. Jablonka and M. J. Lamb, *Epigenetics*, in M. Pagel (ed.), *Encyclopaedia of Evolution* (Oxford: Oxford University Press, 2002), 310–11; M. J. West-Eberhard, *Developmental Plasticity and Evolution* (Oxford: Oxford University Press, 2003); A. Minelli, *The Development of Animal Form* (Cambridge: Cambridge University Press, 2003). By *environmentally governed properties* I mean those phenotypic properties which appear thanks to the interactions between cell populations, or even the entire human being, and the environment in which the latter lives.



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## 5.2. The non causal R

### 5.2.1. The functional R

Even if, at the beginning many scholars considered the functional explanation as 'something strange' that had to be reduced to the causal nomological deductive explanation, soon it became clear that it had its own identity and therefore it should not be studied as a particular case of other 'more standard' explanations. In particular since the '70s, it was realized that a functional attribution ('the function of T within the system S is F') is a functional explanation, that is, an explanation which accounts for a trait T of a system S in terms of its function F.[35] Moreover it was also realized that any functional attribution, and thus any functional explanation, depends on the particular meaning of the concept of 'function' there used. This is particularly important for our contextualized approach to explanation. For many of the problems arising from different meanings of the term 'function', and therefore of the functional explanation, vanish thanks to our approach, as it will be shown. Let us recall the three main meanings.

Given a biologist b, asking, from the point of view of the context $C_b$, which is the function F of a phenotypic trait T of a system S of a living being B means asking

1) or the reason why the function F is the aim linked with the phenotypic trait T, of a system S of a living being B (*aim-emphasising interpretation of the function* F);
2) or the reason why F is the future event induced by T of S of B; in this case, T is intended as having certain dispositions, or propensities to cause F (*dispositional interpretation of the function* F);[36]
3) or the historical reason why F is what allows (S of) B to exist (*historical, or etiological, interpretation of the function* F).

[35] Cf. E. Nagel, 'Teleology Revisited', *Journal of Philosophy* **74**, 1977, 261–301; B. Enc, 'Functional Attributions and Functional Explanations', *Philosophy of Science* **46**, 1979, 343–65.

[36] As is well known, there is a wide debate on which the nature of a dispositional explanation is. Cf., for example, D. M. Armstrong, 'Dispositions are Causes', *Analysis* **30**, 1969, 23–6; J. H. Fetzer, *Scientific Knowledge* (Dordrecht: Reidel, 1981); L. Addis, 'Dispositions, Explanation, and Behaviour', *Inquiry* **24**, 1981, 205–27; J. E. Tiles, 'A "Rationalist" Approach to Dispositional Concepts', *Theoria* **51**, 1985, 1–15; J. I. Thompson, 'Real Dispositions in the Physical World', *The British Journal for the Philosophy of Science* **39**, 1988, 67–79; A. Hüttemann, 'Laws and Dispositions', *Philosophy of Science* **65**, 1988, 121–35.



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Let us recall that there has been an extremely lively debate concerning both whether a functional explanation is a teleological explanation,[37] and whether a dispositional account of function could be unified with an etiological account of function.[38] As it should be clear, I am suggesting that a plausible solution to all the problems on the real nature of the functional explanations may be found within a pragmatical approach. From this point o view, we do not ask any longer whether a functional explanation is always an aim-emphasising explanation (or a teleological explanation), or whether a dispositional explanation can be unified with an etiological one, or something like that. Instead, starting from the particular b's $C_b$, we can individuate and fix which of the possible meanings of the term 'function' best satisfies our explanatory investigation. *It is* b's $C_b$ *that allows us to grasp the meaning of the functional explanatory connection, and not an abstract analysis of the notion of 'function'.*

Therefore, the kind of functional connection b uses will depend on b's $C_b$: sometimes it will be an aim-emphasising connection, sometimes a dispositional connection, and sometimes an etiological connection. There will be cases in which only one of these interpretations is possible; others in which two of them are possible; and still others in which all of three are possible. It depends on what b must explain and from which $C_b$ b starts.

[37] Cf. B. Enc, 'Functions and Goal Directedness', *Philosophy of Science* **59**, 1992, 635–54; D. M. Walsh and A. Ariew, 'A Taxonomy of Functions', *Canadian Journal of Philosophy* **26**, 1996, 493–514; R. G. Millikan, *Language, Thought, and Other Biological Categories* (Cambridge (Mass.): MIT Press, 1984); K. Neander, 'The Teleological Notion of "Function"', *Australasian Journal of Philosophy* **69**, 1991, 454–68; P. E. Griffiths, 'Function Analysis and Proper Functions', *The British Journal for the Philosophy of Science* **44**, 1993, 409–22; Ph. Kitcker, 'Function and Design', *Midwest Studies in Philosophy*, 1993, 379-397; P. Godfrey-Smith, 'A Modern History Theory of Functions', *Nous* **28**, 1994, 344–62; P. S. Davis, 'The Nature of Natural Norms: Why Selected Functions Are System Capacity Functions', *Nous* **34**, 2000, 85–107; P. S. Davis, *Norms of Nature. Naturalism and the Nature of Functions* (Cambridge (Mass.): MIT Press, 2001).

[38] Cf. Ph. Kitcker, 'Function and Design', *Midwest Studies in Philosophy*, 1993, 379–97; P. Godfrey-Smith, 'A Modern History Theory of Functions', *Nous* **28**, 1994, 344–62; M. Walsh and A. Ariew, 'A Taxonomy of Functions', *Canadian Journal of Philosophy* **26**, 1996, 493–514; R. Amundson and G. V. Lauder, 'Functions Without Purposes. The Uses of Causal Role Function in Evolutionary Biology', *Biology and Philosophy* **9**, 1994, 443–69; C. Allen and M. Bekoff, 'Biological Function, Adaptation and Natural Design', *Philosophy of Science* **62**, 1995, 215–54.



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I must now point out that all the possible kinds of functional explanations seem to have to satisfy two requirements. The first, highlighted by D. M. Walsh,[39] concerns the relevance in a biological explanation of the relation to the environment E. The second, pointed out by J. Bigelow and R. Pargetter in a different context,[40] deals with the possibility that a phenotypic trait has no function at all, or no function as to a given environment E.

However, any time we have a why-question, and it has been clarified (by resorting to b's $C_b$) that it can be answered by using a functional connection, we must understand (always by resorting to b's $C_b$) which is the meaning at issue.

To conclude, given a question Q = 'Why is T?', it is the b's $C_b$ that allows us to understand that the question can be functionally reformulated as Q = 'Which is the function of the phenotypic trait T of the system S, belonging to a being B living in an environment E?'. In this case, we would have

*because $A_b$ = 'the phenotypic trait T of the system S of a being B living in an environment E has the function F' is an answer given by the biologist b, with reference to its context* $C_b \subseteq K_b$, to $Q = \langle P_k, X, R \rangle$, *where*

1) *X is enucleated by* $C_b$;
2) $P_k$ = *'The being B living in an environment E has the trait T of S with the function F' is valid and plausible in b's* $C_b$ *among the elements of* $X = \{$*'The being B living in an environment E has the trait T of S with the function F', 'The being B living in an environment E has the trait T of S with the function* $F^1$*', 'The being B living in an environment E has the trait T of S with the function* $F^2$*', …*$\}$;
3) $A_b$ *is valid and plausible with reference to* $C_b$, *and it bears a probabilistic functional connection with* $\langle P_k, X \rangle$.

Moreover, it is always the b's $C_b$ that allows us to grasp which is the right interpretation of the functional connection: an aim-emphasising interpretation,[41] a dispositional interpretation;[42] a historical, or etiological, interpretation.[43]

[39] D. M. Walsh, 'Fitness and Function', *The British Journal for the Philosophy of Science* **47**, 1996, 553–74.

[40] J. Bigelow and R. Pargetter, 'Functions', *Journal of Philosophy* **84**, 1987, 181–96.

[41] Cf. the seminal C. Boorse, 'Wright on Functions', *Philosophical Review* **85**, 1976, 70–86.

[42] It was pioneered by R. Cummings, 'Functional Analysis', *Journal of Philosophy* **72**, 1975, 741–56.



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### 5.2.2. The teleological and teleonomic Rs

We have seen that one of the three possible interpretations of the functional why is in terms of aims. Now we must dwell upon it. In biology, as Mayr pointed out,[44] there could be teleological why, but the aims there referred should not be interpreted in an incorrect way. In particular, they should not be considered as final causes (in an Aristotelian sense), nor as cosmic aims (i.e., as aims to which the entire cosmos should direct); nor as aims due to a transcendent design. Nevertheless, there are aims which may be correctly considered; more specifically those dealing with certain processes or certain kinds of behaviours.[45]

Let us begin considering living beings endowed with intentionality. In this case the living being H intentionally can behave in a way W to reach the aim A. It seems wise, at this point, to presuppose that there are living beings H endowed with intentionality, and that only the species endowed with intentionality can admit a teleological explanation of certain behaviours of theirs.[46]

It follows that we should be extremely careful any time we offer a teleological explanation of behaviour. We must not attribute intentionality to species which do not have it, or it is extremely dubious if they really have it. From this point of view, a sort of

[43] It is rooted in L. Wright, 'Functions', *The Philosophical Review* **82**, 1973, 39–168; L. Wright, *Teleological Explanation* (Berkeley: University of California Press, 1976). But it was pioneered by J. Canfield, 'Teleological Explanation in Biology', *The British Journal for the Philosophy of Science* **14**, 1964, 285–95; and M. Ruse, *The Philosophy of Biology* (London: Hutchinson University Library, 1973). Now is discussed by R. G. Millikan, *Language, Thought, and Other Biological Categories* (Cambridge (Mass.): MIT Press, 1984); R. G. Millikan, *White Queen Psychology and Other Essays for Alice* (Cambridge (Mass.): MIT Press, 1993); K. Neander, 'The Teleological Notion of '"Function"', *Australasian Journal of Philosophy* **69**, 1991, 454–68; K. Neander, 'Functions as Selected Effects: The Conceptual Analyst's Defence', *Philosophy of Science* **58**, 1991, 168–84.

[44] E. Mayr, 'Teleological and Teleonomic: A New Analysis', *Boston Studies in the Philosophy of Science* **14**, 1974, 91–117.

[45] Note that any behaviour is a process, but, of course, not any process is a behaviour.

[46] Cf. the pioneering works by R. Brown, 'Dispositional and Teleological Statements', *Philosophical Studies* **3**, 1952, 73–80; R. D. Braithwaite, *Scientific Explanation* (Cambridge: Cambridge University Press, 1953).



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epistemological wisdom should suggest us to teleologically explain only certain human behaviours.

There are two other aspects to be recalled. On the one hand, even if *Homo sapiens* H intentionally behaves in the way W to reach the aim A, it is not said that A is really reached. There is a probability to reach A, and this probability is greater or lesser in function of the aim A itself, of the context in which the behaviour W is made, of who are (for example, from a social point of view) the H behaving in the W way, etc. On the other hand, A can be reached also by behaving in a way W' different from W.[47] Nevertheless, this second possibility is not so important for what we are saying, since we are considering the behaviour W and not if A could be obtained differently.

Now we may state that teleonomy can be used correctly in a biological explanation, in particular in an ethological explanation, if it concerns situations dealing with an intentional behaviour of a member of the species *Homo sapiens*.

Let us consider unintentional behaviours, or, more generally, unintentional processes. Could they be interpreted teleologically? If we do not want to introduce Aristotelian final causes, nor cosmic aims, nor transcendent designs, and if we do not want to fall into the fallacy of attributing intentional capabilities to living beings which do not have them, or it is extremely dubious if they have them, we are forced to answer negatively. Nevertheless, how to explain the unintentional biological processes? Since C. S. Pittendrigh's 'Adaptation, Natural Selection and Behaviour', we have had a good solution:[48] to explain them in a teleonomic way,[49] that is, by considering them as processes which are genetically, and/or epigenetically, and/or environmentally governed. Of course there could be biological processes, as in the ontogenesis, which are

[47] Of course, this is the well known problem of the functional equivalents. Cf. A. Rosenblueth, N. Wiener and J. Bigelow, 'Behaviour, Purpose, and Teleology', *Philosophy of Science* **10**, 1943, 18–24; E. Nagel, *The Structure of Science* (New York: Harcourt, Brace & World Inc., 1961).

[48] C. S. Pittendrigh, 'Adaptation, Natural Selection and Behaviour', in A. Roe and G. Simpson (eds.), *Behaviour and Evolution* (New Haven: Yale University Press, 1958).

[49] Cf. also J. Huxley, 'The Openbill's Open Bill: a Teleonomic Enquiry', *Zool. Jb. Abt. Anat. U. Ontog. Tiere* **88**, 1960, 9–30; E. Mayr, 'Cause and Effect in Biology', *Science* **134**, 1961; G. De Laguna, 'The Role of Teleonomy in Evolution', *Philosophy of Science* **29**, 1962, 117–31; F. M. Wuketis, 'On the Notion of Teleology in Contemporary Life Sciences', *Dialectica* **34**, 1980, 277–90.



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genetically, epigenetically, and environmentally governed at the same time.

There is another aspect to be noted. In chapter 7 of *The Origin*,[50] Ch. Darwin, even if he writes that he does not intend to define 'instinct', he actually does it some lines below:

> 'An action, which we ourselves should require experience to enable us to perform, when performed by an animal, more especially by a very young one, without experience, and when performed by many individuals in the same way, without their knowing for what purpose, is usually said to be instinctive'.[51]

After this definition, Darwin distinguishes *instinctual behaviour* and *habitual behaviour*. Both of them are unintentional, even if the former is an inherited trait and the latter is a will-independent behaviour which is not a trait of an entire population, but only of some individuals. More precisely the habitual behaviour is the result of a conscious and continuous repetition of a certain non-instinctual behaviour. It is a kind of behaviour that one acquires by means of training and, from a certain point on, it results so natural that it seems to have one of the characteristics pointed out by Darwin for instinctual behaviour: it is 'performed […] without their knowing for what purpose'. Besides the fact that in many cases the instinctual behaviour and the habitual behaviour cannot be easily differentiated (which is due to nature and which to nurture?), both of them are unintentional and may be explained teleonomically.

By taking all of this into account, we may state that 'teleonomy' can be used correctly in a biological explanation, if it concerns a situation dealing with either 1) genetically, and/or epigenetically, and/or environmentally governed processes (or behaviours) directed to an aim; or 2) habitual behaviours directed to an aim.

Note that in this way we avoid also explicating the notion of 'teleonomy' by resorting to the notion of 'program' which is extremely ambiguous, especially when we speak of 'genetic program'.[52]

There is another relevant remark. As seen the question 'Why the process W?' can be interpreted as 'Which is the physiological teleo-

[50] It is Chapter 8 in the 1872 edition.

[51] Ch. Darwin, *The Origins of Species* (1859–1872) (New York: Gramercy Books, 1976), 234.

[52] For a critique to this way of speaking, cf. E. F. Keller, *The Century of the Gene* (Cambridge (Mass.): Harvard University Press, 2000); G. Boniolo, 'Biology Without Information', *History and Philosophy of the Life Sciences* **25**, 2003, 257–75.



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nomic explanation of the process W?'. But it can be interpreted also as 'Which is the evolutionary teleonomic explanation of the process W?'. This second question is different from the first, and if the former can be answered by resorting to the model just proposed, the latter has to resort to the phylogenetic model discussed in the section 5.1.3. Of course, not all the teleonomic processes may be explained evolutionarily, for example neither the habitual behaviours, nor the ontogenetic processes can.[53]

In conclusion, if we have a question Q = 'Why the process W?', and if b's $C_b$ has allowed us to interpreted it as Q = 'Which is the aim A of the process W performed by a being B (or by a system S of B) in an environment E?', we have

*because* $A_b$ = *'The being B (or a system S of B) in an environment E performs the process W to reach the aim* A*' is an answer given by the biologist b, with reference to its context* $C_b \subseteq K_b$, *to* $Q = \langle P_k, X, R \rangle$, *where*

1) $X$ *is enucleated by* $C_b$;
2) $P_k$ = *'The being B (or a system S of B) in an environment E performs the process W' is valid and plausible in b's* $C_b$ *among the elements of* $X$ = {*'The being B (or a system S of B) in an environment E performs the process W', 'The being B (or a system S of B) in an environment E performs the process* $W^1$*', 'The being B (or a system S of B) in an environment E performs the process* $W^2$*', …*};
3) $A_b$ *is valid and plausible with reference to* $C_b$, *and it bears a probabilistic aim-directed connection with* $\langle P_k, X \rangle$, *where the aim-directed connection can be a) a teleological connection if the process is an intentional behaviour performed by a human; b) a teleonomic connection if the process is genetically, and/or epigenetically, and/or environmentally governed, in particular if it is an unintentional (instinctual or habitual) behaviour.*

[53] For the debate between teleonomy and evolution, cf. F. Ayala, 'Teleological Explanations in Evolutionary Biology', *Philosophy of Science* **37**, 1970, 1–15; K. Lorenz, *Die Rückseite des Spiegels* (München: R. Piper & Co. Verlag, 1973); E. Mayr, 'Teleological and Teleonomic: A New Analysis', *Boston Studies in the Philosophy of Science* **14**, 1974, 91–117; F. M. Wuketis, 'On the Notion of Teleology in Contemporary Life Sciences', *Dialectica* **34**, 1980, 277–90. There has been also a debate concerning whether a teleonomic why is an homeostatic why, i.e., a circular causal why (cf. the just quoted works by F. Ayala, E. Mayr, and F. M. Wuketis). Of course, it depends on the context $C_b$.



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## 6. For a contextualized approach to biological explanation

Above I have constructed a general map of possible explanatory models adoptable when we work with biological explanations. Out of metaphor, that general map is the pragmatical meta-model allowed by our *contextualized approach to biological explanation*. As shown, it is really capable of ruling the different biological explanatory models by emphasizing the role of the particular context $C_b$ from which the given biologist b begins answering. From this point of view, the R-connection, which is the real core of any explanation, is determined and fixed in a plain way, without uncertainty or vagueness. The $C_b$ chosen may be discussed, but once this is done the R-connection follows.

It has been shown that many discussions on the nature of certain R-connections (for example, if they are real functions; what the real meaning of 'function' is; if functional connections can be reduced to causal connection; if we have real teleological o teleonomic connections; etc.) vanish. Which meaning of 'function' is to be used, if we can reduce it to a causal connection, if we can use a teleological o teleonomic connection, etc., depends on the context $C_b$ from which the particular biologist-answerer (*the explainer*) b decides to provide his because-answer.

In this way we pay more attention to what the particular biologist does when it explains than to how the biological world really is. My aim in proposing a *contextualized approach* was not to ontologically describe the real nature of the world but to offer a good account of the many possibilities that we have when we must give a biological explanation. And this, pace the metaphysicians, depends on the context from which we furnish it.

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