MSR SESSION ON PHILOSOPHY OF BIOLOGY:

SIZE, DEVELOPMENT AND EVOLUTION

METHODS OF SCIENTIFIC

REPRESENTATION (MSR) SEMINAR

THURSDAY 20 JUNE 2019

Seminar Room A-25, Faculty of Philosophy, Complutense University

11.00 – 12.15: Daniel J. Nicholson (Konrad Lorenz Institute): On Being the Right Size, Revisited

12.15 – 12.30: Coffee Break

12.30 – 13.45: Miguel Brun-Usan (University of Southampton): Unburdening Evolutionary Thought through Development

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ABSTRACTS:

DANIEL J. NICHOLSON: “On Being the Right Size, Revisited”

In 1926, Haldane published an essay titled ‘On Being the Right Size’ in which he argued that the size of an organism fundamentally shapes its particular way of life. Size constrains the kind of structure an organism can have, as well as its mode of behaviour. Many of Haldane’s examples are based on the square-cube law, which states that the volume of a body increases much faster than its surface area. The shape of warm-blooded animals, the form of the leaves and roots of plants, and the structure of the pulmonary alveoli and gastrointestinal tract of mammals can all be explained by appealing to this remarkably simple geometric relation. Haldane also showed that the functional capacities of organisms are conditioned by the physical forces that exert the greatest effect at the scale at which they exist. For example, gravity poses no danger to small animals, but the surface tension of water can be a very serious threat to them. The exact opposite is true for large animals.

This talk revives Haldane’s ninety-year-old argument and puts it to work in the context of contemporary molecular biology. Specifically, my claim is that this field would benefit greatly if it took seriously Haldane’s insight that differences in the scale at which entities exist are of overwhelming importance in determining their structure and behaviour. Owing to their minuscule size, cells and their macromolecular components are subject to very different forces than macroscopic organisms. In a sense, macroscopic and microscopic entities inhabit different ‘worlds’: the former is ruled by gravity and inertia, whereas the latter is governed by Brownian motion. This has serious implications for the explanations we formulate. Most crucially, our intuitions—based as they are on our everyday experience of the macroscopic world—fail us when estimating the adaptive problems that cells and molecules have to overcome. The implication is that we should be extremely sceptical of models and analogies that seek to explain properties of microscopic entities by appealing to the properties of macroscopic ones.

Unfortunately, this is precisely what the appeal to mechanical and electronic engineering metaphors in molecular biology attempts to do. Researchers routinely resort to machines because they are familiar and intuitively intelligible. But if our machines were the size of molecules they would not be able to function the way they do, as their physical environment would be completely different. It follows that we should avoid distorting biological reality by construing it in engineering terms. I will illustrate this by examining four key metaphors in molecular biology—‘genetic program’, ‘cellular circuitry’, ‘molecular machine’, and ‘molecular motor’—and showing that their various deficiencies ultimately derive from their neglect of scale.

Finally, I will explain why late twentieth-century biology came to forget the lessons concerning the importance of size and scale that early-twentieth century biologists like Haldane (D’Arcy Thompson is another example) repeatedly drew. I will suggest that the reason has to do with the influence of Schrödinger’s argument in What is Life? (1944) regarding the stability of the gene.

MIGUEL BRUN-USAN: “Unburdening Evolutionary Thought through Development”

Phenotypic plasticity (phenotypic sensitivity to the environment), genetic evolvability (phenotypic sensitivity to mutations) and epigenetic inheritance (phenotypic effects of maternally inherited non-genetic elements) are evolutionary phenomena which play crucial roles in modern evolutionary thought. However, it is still unclear how these concepts relate to each other at deeper levels, or how can they be adaptively evolved in order to produce adaptive variation.

In this work, we identify connections between these three phenomena and explore the evolutionary consequences of such connections. By means of computational modelling, we show that the phenotypic variation arising from genetic evolvability, phenotypic plasticity or epigenetic variation are correlated.

We show that these correlations arise because all these sources of phenotypic variation affect the same dynamical system. This dynamical system is development.

In addition, we show that because of these correlations, any specific phenotypic variation can be evolved from genetic, environmental or epigenetic perturbations alike. That is, from a developmental perspective, the evolution of these three processes is equivalent. However, from a selective perspective, it is not equivalent: natural selection is much more efficient in evolving phenotypic variation using environmental variation, strongly supporting the suggestion that plasticity is a leader in evolutionary change (plasticity-first).

We conclude that a mechanical understanding of development may 1) greatly simplify the evolutionary thought (by revealing that some concepts are just partial descriptions of a more fundamental entity) and 2) offer new approaches to old evolutionary puzzles (plasticity vs genetic-first scenarios) that remain largely unsolved.