



# The role of regulation in the origin and synthetic modelling of minimal cognition



Leonardo Bich<sup>a,b,\*</sup>, Alvaro Moreno<sup>a</sup>

<sup>a</sup> IAS-Research Center for Life, Mind and Society, Department of Logic and Philosophy of Science, University of the Basque Country (EHU/UPV), Avenida de Tolosa 70, 20018 Donostia-San Sebastián, Spain

<sup>b</sup> Laboratorio de Neurobiología y Biología del Conocer (Biology of Cognition Lab), Facultad de Ciencia, Universidad de Chile, Las Encinas 3370, Ñuñoa, Santiago, Chile

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## ABSTRACT

In this paper we address the question of minimal cognition by investigating the origin of some crucial cognitive properties from the very basic organisation of biological systems. More specifically, we propose a theoretical model of how a system can distinguish between specific features of its interaction with the environment, which is a fundamental requirement for the emergence of minimal forms of cognition. We argue that the appearance of this capacity is grounded in the molecular domain, and originates from basic mechanisms of biological regulation. In doing so, our aim is to provide a theoretical account that can also work as a possible conceptual bridge between Synthetic Biology and Artificial Intelligence. In fact, we argue, Synthetic Biology can contribute to the study of minimal cognition (and therefore to a minimal AI), by providing a privileged approach to the study of these mechanisms by means of artificial systems.

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## 1. Introduction

This paper explores the deep connection between biological organisation and cognition at its very roots in basic living systems. The argument that we propose in the following pages pursues two main objectives. In the first place, starting from the framework of biological autonomy, we aim at developing a theoretical account of the origin of some minimal cognitive properties, by analysing the organisational requirements for their realisation: basically, the instantiation of regulatory mechanisms.

One of the essential aspects of cognition, which can be analysed at the basic level, is that cognitive agents should be able to distinguish between some specific features of their interaction with the environment and to act accordingly, in such a way as to maintain their viability.<sup>1</sup> As we shall argue here by developing some of

the insights coming from the tradition of research based on the notion of biological autonomy, this implies that they should be able to associate environmental perturbations with internal patterns of self-regulation (Bich and Damiano, 2012a).<sup>2</sup> All present day living beings have this capacity, let us just think of how bacteria respond adaptively to the composition (and variation in composition) of the environment by means of internal changes, such as the synthesis of different sets of enzymes necessary for metabolising different substances, or by modulating their movement in the environment according to gradients of concentrations, like in the case of chemotaxis (Bich et al., 2015). And it is plausible that more primitive prebiotic self-maintaining systems had also been capable

\* Corresponding author at: IAS-Research Center for Life, Mind and Society, Department of Logic and Philosophy of Science, University of the Basque Country (EHU/UPV), Avenida de Tolosa 70, 20018 Donostia-San Sebastián, Spain.

E-mail address: [leonardo.bich@ehu.es](mailto:leonardo.bich@ehu.es) (L. Bich).

<sup>1</sup> By relying on this property, some theoretical approaches to the study of life and cognition have developed the L = C thesis according to which cognition is co-extensive with life or coincides with the interactive dimension of life (Piaget, 1967; Maturana and Varela, 1973, 1980; Heschl, 1990; Varela et al., 1991; Bitbol and Luisi, 2004; Bourguine and Stewart, 2004). According to other authors, instead, even

though those exhibited by minimal living systems are important aspects of cognition, they are not sufficient to define it. According to these latter approaches, it is increased behavioural capacities (Christensen and Hooker, 2000) or a higher degree of organisational complexity, namely a nervous system with its own distinctive norms (Barandiaran and Moreno, 2006; Moreno and Mossio, 2015), which are the primary discriminating dimensions of cognition. We will not address these issues here, as we will limit ourselves to analyse how some specific features related to cognition emerge in biological systems, and not whether or not they can be considered sufficient for full-fledged cognition.

<sup>2</sup> For a discussion of the relation between cognition and (homeostatic) regulation in higher organisms with nervous system see, for example Damasio (2003), Ziemke and Lowe (2009).

of maintaining their organisation against environmental perturbations; even though this capacity might have been based on simpler mechanisms.<sup>3</sup>

In the second place, by developing a theoretical account of minimal cognitive properties we aim at providing a bridge between the concepts and languages of Synthetic Biology (SB) and Artificial Intelligence (AI). While AI usually aims at studying and modelling high-level cognitive properties (e.g. at the human level), surprisingly interesting properties for understanding the origin of cognition can be found also in simpler biological systems such as bacteria and invertebrates, and they can provide insights into the functioning of more complex forms of cognition (Bechtel, 2014). Hence, an AI focused on the investigation of cognition at the minimal level requires an approach that is directly linked to biological processes, and this is what SB can provide.<sup>4</sup> The connection between the two disciplines has not been explored exhaustively yet, and the two disciplines still use distinct languages: cognitive for AI and biochemical for SB. Historically, in fact, SB has played an important role in Artificial Life, but not as much with respect to AI, apart from some pioneering approaches in bio-chem-ICT (see, e.g. Amos et al., 2011; Rampioni et al., 2014). The aims, scope and conceptual foundations of this enterprise are still in course of definition and, we argue, a theoretical account of those minimal cognitive properties that can be studied at the level on which SB operates can provide both the missing connections between SB and AI, and a theoretical support for the empirical study of minimal cognition by means of artificial biochemical systems.

On the basis of theoretical considerations on what minimal cognitive properties are and how they originated, we aim at providing a framework for SB-based AI that is distinct in target and goals from existing ones. Focused on developing a dimension of autonomy in artificial systems analogous to that exhibited by living organisms, it aims at a deeper understanding of the origin of minimal cognition in terms of instantiation of regulatory capabilities in artificial systems. In such a scenario the dimension of normativity (Bickhard, 2009; Mossio and Bich, 2014) – how an autonomous system can generate its intrinsic goals and norms, that coincide with its own self-maintenance, rather than having them imposed from outside by the designer – plays a crucial role. Other more engineering-oriented approaches such as those reviewed in Amos et al. (2011), instead, are mainly focused input–output relations, and on computing through biochemical systems. The research proposed by Rampioni et al. (2014), on the other hand, shares with our approach a common theoretical framework, that of autonomy, and similar general goals: a better understanding of minimal cognition rather than the development of biochemical tools only. Then it focuses on different issues, even though closely related to those addressed in this paper: the study of bacterial communication and, specifically, of signal transmission between synthetic and living cells – namely synthetic cells sending signals to bacterial ones – while we will focus here on the biochemical requirements for the emergence of cognition and for the implementation of cognitive-like properties in synthetic systems.

Our goals, as stated above, are primarily theoretical. In this paper we will mainly focus on how some essential requirements for cognition have appeared. In particular, we will argue that specific mechanisms of internal compensation for perturbations are those responsible for the emergence of a capacity to distinguish between specific features of the interactions with the environment which, otherwise, would constitute only a mere source of noise for the system. In Sections 2 and 3, we will distinguish between two forms of compensation for perturbations: respectively, dynamic stability and adaptive regulation. And we will show how only the second – based on a decoupling between constitutive metabolism and regulatory mechanisms, and on the capacity to produce endogenous interpretations and evaluations of environmental stimuli – enables more complex interactions between an organism and its environment, in which a world of ‘meaningful’ (i.e. functional for the system) specificities emerges for the system. In Section 4 we will present a comparative case study in which to confront the role of these different compensatory mechanisms in distinct instances of chemotactic behaviour. In Section 5, we will argue in favour of a privileged role for Synthetic Biology in the study of these properties at the very roots of agency and cognition.

## 2. Basic self-maintaining metabolic networks: structural stability against environmental noise

According to the framework based on the notion of biological autonomy (Varela, 1979; Kauffman, 2000; Ruiz-Mirazo and Moreno, 2004) living systems can be characterised as far from equilibrium self-maintaining chemical systems capable of producing their own functional components and physical boundary. In doing so, they maintain themselves as organised unities by promoting the conditions of their own existence through interactions with a changing environment. The idea of biological autonomy (Fig. 1) emphasises: (1) the self-referential character of living systems as self-producing and self-maintaining systems – understood through the notion of *organisational closure* (Piaget, 1967; Rosen, 1972, 1991; Maturana and Varela, 1973, 1980; Ganti, 1975, 2003; Kauffman, 2000; Mossio and Moreno, 2010; Montévil and Mossio, 2015)<sup>5</sup> – and (2) the intrinsically interactive dimension of their organisation: the autonomous organisation cannot exist unless it maintains a continuous coupling with its environment. In such a scenario compensatory mechanisms, by modulating internal processes in relation to environmental changes, constitute a crucial factor in characterising living systems from their most basic instances.

Self-maintaining metabolic systems can implement a variety of qualitatively different response mechanisms, in such a way as to ensure their viability: from simple buffering to the synthesis *on-demand* of specific sets of enzymes. Let us consider basic responses first. Autonomous systems can respond to environmental changes in the simplest way by means of changes transmitted through the actual network of processes of production of components.<sup>6</sup> This

<sup>3</sup> It can be argued that proto-mechanisms of regulation, in addition to molecular stability, could have played a role in prebiotic evolution (Bich and Damiano, 2012b).

<sup>4</sup> A clarification is necessary in this respect. We are interested here in that branch of synthetic biology which aims at a better understanding of how living systems work, especially their minimal instances, rather than at engineering organisms that perform specific tasks (for the latter approach see Silver and Way, 2014; Arnold and Meyerowitz, 2014). We refer to that practice of knowledge that, instead of studying living systems by analysing their parts or by formulating predictive models of their behaviours, intends to understand their functioning by actually constructing the object of study (Pfeifer and Scheier, 1999; Damiano et al., 2011; Ruiz-Mirazo and Moreno, 2013), an alternative biological or proto-biological system, and study the properties and behaviours it exhibits.

<sup>5</sup> There are important differences in how these authors conceive the concept of organisational closure. For a detailed analysis of this question, see Moreno and Mossio (2015).

<sup>6</sup> Let us think of Ganti’s chemoton, a model of minimal living system organised as a biochemical clockwork (Ganti, 2003) in which three autocatalytic subsystems – respectively, a metabolic cycle, a template subsystem and a compartment – are directly coupled like chemical cogwheels. In such a system any change in one subsystem affects directly the others through supply and demand of metabolites, and can be compensated through changes transmitted through the network. For example, an increase in the amount of nutrients entering the system, after a first rise in metabolic activity, causes an accumulation of the products of the metabolic cycle, thus slowing again the whole dynamics. Yet, other responses are possible, involving also the two other subsystems. For example, after the products of metabolism reach a certain threshold of concentration, determined by the structure and length of the template,

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