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Comment

Deterministic versus probabilistic causality in the brain: To cut or not to cut Comment on "Foundational perspectives on causality in large-scale brain networks" by M. Mannino and S.L. Bressler

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In recent decades the rapid growth of new imaging technologies and measurement tools has dramatically changed how neuroscientists explore the function of the brain. A careful examination of the conceptual basis of causal inference using such methods is long overdue. Mannino and Bressler (M&B) [1] provide an informative review on the notion of causality from the perspectives of philosophy, physics, complex systems and brain sciences.

M&B assert that causality in the brain is probabilistic, not deterministic in nature. Later on, they say that they have not tried to answer the question of whether the brain is inherently deterministic or stochastic. The two statements cannot be consistent unless "causality" in M&B's definition speaks not to how the brain actually works but instead to statistical relations between measurements. This is akin to the Born interpretation of quantum mechanics which gives not the density of the stuff but rather the density of probability of finding the stuff [2]. Beyond general (unfortunately unsubstantiated) claims that the brain is self-organizing (cf. [6]) M&B seem to be telling us not how the brain *is*, but how we happen to measure it.

We wish to draw a distinction between probabilistic relations (statistical relations) and probabilistic causal relations (stochastic processes). Probabilistic relations are what we can calculate statistically from some measurement. However, when we say *causal* relations, we are talking about whether one event truly influences a second—which is to say that if we are able to manipulate the first event, we will see corresponding changes in the second, no matter if the change is stochastic or deterministic. It is not guaranteed that all statistical relations are also causal relations, since we cannot know if we have recorded all causal events and/or for a sufficiently long time to screen off spurious (non-causal) statistical relations. Even if a statistical relation is found to reflect a causal relation, it is not guaranteed that this underlying causal relation has to be stochastic. One can also make statistical measurements on deterministic processes. In practice, the experimental verification of the most deterministic theories still requires doing statistics. In statistical mechanics, for example, one can assume the microstates follow deterministic laws, while still studying their statistical properties [3].

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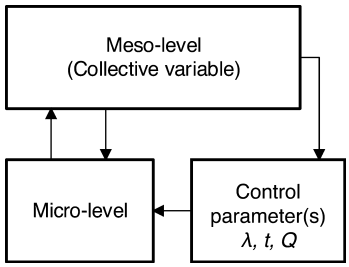

Fig. 1. The causal loops of coordination dynamics. Control parameters, such as λ, which can be specific or non-specific, t (time), and Q (stochastic noise) lawfully influence the micro-level. Resulting component interactions may produce collective effects at the meso-level (upward causation) that in turn may modify component behavior (downward causation). In certain situations, such as the human dynamic clamp [12] collective variables act back on control parameters invoking circular causality.

With the above clarification in mind, let us consider the reasons M&B use for the abandonment of determinism in terms of inference methods. First, M&B argue that deterministic causal inference leads to spurious conclusions. They use the following example: suppose an event X (barometric pressure drop) causes both event Y (mercury drop in a column) and event Z (storm). M&B argue that the spurious regularity $Y \rightarrow Z$ can be screened off statistically by the probability of $X \rightarrow Z$ but "*a deterministic interpretation of causality would mistakenly infer that the drop in mercury causes the storm*." However, a meteorologist with a deterministic view of the system could falsify this hypothesis by heating up the mercury to see if a height increase can stop the storm. Rather than looking for more variables, she is applying systematic inductive inference by devising a crucial experiment that discriminates alternative hypotheses of interest, the path of strong inference [4]. Thus, one would not want to confound the problem in the inference methods with that of the causal assumptions.

M&B further argue that the ubiquitous mutual causality in the brain, and complex systems in general (e.g. bidirectional influence between two nodes in a neural network), renders determinism logically impossible. We do not see how this conclusion follows. Staying within the classical world, Newton's 3rd law says if A exerts a force on B, then B must also exert a force on A: if the earth is pulling the moon, the moon is also pulling the earth, which is being mutually causal. Nonetheless, most would view the system composed of earth and moon as deterministic, meaning that given an initial state of the system, the future trajectory is uniquely determined. Without that, it would be difficult to send people to the moon on a rocket. So this is logically possible and practically useful. In complex systems, mutual causality is a key to self-organization in sociological, physical, chemical, and biological systems. With their corresponding mathematical formalisms, both deterministic and stochastic views of causality contribute to the understanding of the dynamics of such systems [5].

M&B also argue that due to the high dimensionality of the brain, "even if the brain does employ deterministic influences," it would be indistinguishable from a stochastic one. However, we should point out that converging evidence shows that some interactions can lead to low-dimensional collective dynamics [5–9]. Essentially, while interacting components (say at a micro-level) give rise to the *emergence* of collective patterns (at meso-level), the collective patterns can in turn *enslave* the behavior of those very same components (at micro-level). These two opposing forces result in so-called circular causality (Fig. 1). Collective patterns are temporally assembled in order to accommodate certain functional demands and their low-dimensional dynamics is invariant to the different specific membership configurations of the components. This nonlinear property of biological complex systems is called degeneracy [9], which is essential for the system to resist micro-level fluctuations and perturbations [10]. In the meantime, fluctuations or stochasticity can assist in the switching of patterns in order to adapt to new functional needs [11]. Now maybe we can ask: what is the nature of the causality that makes a pattern persist and what is the nature of the causality that makes a pattern change? Wouldn't it be nice to let them complement each other with regard to the function of the brain?

We humbly suggest that probabilistic and deterministic notions of causality each hold half of the puzzle of the function of the brain, rather than being mutually exclusive. How then can we make inferences about them? Experimentally, we can create tasks (functional demands) and apply perturbations that carry the system through different behavioral repertoires, the dynamics of which can be observed by measures of stability [5,6,9,13,14]. For modeling, as M&B agree, Granger causality and dynamic causal modeling are complementary [15] and can be used in tandem to understand the brain. One would also want to supplement these with techniques that treat essentially nonlinear complex systems [16]. Phase space [17] and projection methods with appropriate basis functions for nonlinear stochastic

systems [18] may illuminate more of the features of the system and yield better predictions than linear statistical inference.

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