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Sociality and Timing: Correlation or Causation? Comment on 'The evolution of social timing' by Verga L., Kotz S. & Ravignani A.

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Verga et al. [1] put forward an intriguing hypothesis: timing and sociality might have evolved together. They hypothesize that these capacities have become functionally and mechanistically linked throughout animal evolution. The linkage between these capacities leads to the definition of "social timing", specifically referring to mutual adaptations in time, between two or more agents, that support communicative or collaborative behavior. If timing and sociality share a phylogenetic trajectory, the authors predict that social (group living) species should outperform solitary (non-group living) species in their timing capacities. Further, assuming that such correlational evidence is provided, the authors highlight another key question: Which mechanisms are behind social timing?

The above hypothesis and research question set the stage for an ambitious and interesting research program aiming to compare the co-occurrence of timing and sociality across different species. Despite the topicality of such a program, we wish to highlight that such proposed research is mostly correlational. When studying the relationship between two variables or phenomena, such as timing and sociality, correlational evidence implies that timing and sociality co-occur. However, we suggest that co-occurrence alone is not sufficient to prove the functional and mechanistic linkage hypothesized by Verga et al. [1]. Testing this would also require taking a causal approach, i.e. testing whether one of these variables causes changes in the other, as opposed to the possibility that they are both the result of a third nested variable.

Take the example of a barometer predicting thunderstorms. Because the behavior of the barometer and the likelihood of observing a thunderstorm are correlated, one must infer a relationship between them. But a relationship does not imply that one is causing the other. Even if a barometer anticipates the occurrence of a thunderstorm in time, it is not causing it. In fact, both the barometer and the thunderstorm are caused by a third variable: changes in air pressure. To test this, one might take a causal approach and manipulate (rather than simply measure) variables. Manipulating a barometer will not cause a thunderstorm. Instead, manipulating air pressure will impact both the barometer and the likelihood of observing a thunderstorm, hence shedding light upon the causal relationship between these example variables.

What can causal methodologies reveal about the relationship between timing and sociality? Causal approaches might reveal whether manipulating sociality in a given species or exemplar (i.e. enhancing or reducing the aptitude for social interaction with conspecifics) is sufficient to influence timing or, vice versa, whether manipulating timing (i.e. the capacity to encode and adapt to temporal patterns) impacts sociality. Assessing this would be pivotal to exclude the possibility that timing and sociality are correlated because they depend on a third variable impacting both. Examples of such putative variables might include brain structures and functions, cognitive abilities (e.g. memory constraints), physical constraints (e.g. impacting upon mobility), environmental niches or

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even genetic background [2].

How could this new research program incorporate a causal approach? One possibility is to use genetic manipulations. Researchers could alter the function of genes prevalently involved in sociality and then test if this also influences timing [3–5]. Possible targets could be genes relevant to autism or schizophrenia, which have been implicated in social dysfunctions [6–8]. Vice versa, manipulations of genes involved in timing [9–11] might lead to social dysfunctions. A general advantage of within-species genetic manipulations, in addition to cross-species comparisons, is that this approach permits researchers to selectively manipulate specific mechanisms while focusing on the same animal model, the same developmental period, and the same environmental niche. However, a disadvantage of this approach is that it is very difficult to target genes that are specifically linked to either timing or sociality.

Other causal approaches could be exploited to test Verga et al. [1]'s hypothesis. For instance, one could manipulate neural processes or circuits instead of genes. Verga et al. [1] highlight some technical challenges associated with measuring neural activity across species, but they do not address the possibility of manipulating (rather than simply measuring) neural signals. Brain stimulation techniques can be used to manipulate brain circuits relevant to either timing or sociality [12]. This can be done non-invasively in several species (including humans). Alternatively, other invasive techniques exist to manipulate specific circuits in several non-human species such as drosophilae, zebrafishes, rodents, and non-human primates [13–15]. Dysfunctional sociality and timing, following the manipulation of either sociality or timing, would support Verga et al. [1]'s hypothesis. Additionally, pharmacological manipulations such as the administration of oxytocin could be used to augment sociality [16] and, possibly, timing.

We now turn to a second issue, highlighted by Verga et al. [1], that could benefit from adopting causal approaches. We specifically refer to the endeavor of research investigating the mechanistic basis of social timing. Verga et al. [1] list several levels of description for such a mechanism: a neural, a physiological, and a cognitive (also addressed as behavioral and emotional) level. Verga et al. [1] suggest that these distinct levels likely reinforce each other, having additive effects, and that the causal relationship between them is unclear. How could such a causal relationship be tested?

To address this question, Verga et al. [1] propose to record neural activity, physiological rhythms, and behavior simultaneously from multiple individuals. We suggest that simply measuring these variables is necessary but not sufficient to neatly tease apart neural, physiological, and behavioral contributions to establishing social timing. For instance, recent human work shows how infant-caregiver dyads engaged in a social interaction synchronize both neural (hemodynamic changes) and physiological (heart rate variability) processes from early on [17]. By adulthood, dyads simply looking at each other spontaneously synchronize a number of behaviors (body kinematics, smiling) and neural activities (envelopes of neural oscillations) ([18,19]). These different levels of interpersonal synchronization occur simultaneously and constitute plausible indices of social timing. Are they all manifestations of a common timing mechanism [20]? Do they all influence each other reciprocally [19], or do some synchronies cause others [21]?

One causal approach to address these questions, and to shed light upon the mechanisms underlying social timing, is to manipulate synchrony at one (e.g. neural) level and monitor the effect of such manipulation on the other (e.g. behavioral or physiological) levels. One such empirical approach is multi-brain stimulation (MBS) [21]. This permits researchers to test whether exogenously establishing synchronization between multiple brains is sufficient to promote synchronization of behavior, physiological processes, or generally social functioning. Work in humans demonstrated that synchronizing two individuals' motor cortices (using non-invasive electrical stimulation) is sufficient to enhance their capacity to establish interpersonal coordination [22]. Following MBS studies have extended this approach by targeting different brain regions and different social tasks. Inducing interpersonal neural synchrony using MBS has been shown to augment social learning [23], communication capacities [24], and mutual understanding [25].

MBS can also be tested in other species besides humans. This could be achieved using either non-invasive [26–28] or invasive protocols that could complement Verga et al. [1]'s research program [21]. For instance, invasive optogenetic approaches have the potential to improve spatial specificity concerning the targeted neural network and cellular type, thus addressing a limitation of non-invasive stimulation protocols. A study developing wireless devices for optogenetics has demonstrated that synchronizing the prefrontal cortex of mice dyads results in augmented spontaneous social behavior [29]. This particular result reinforces the above evidence from human studies and advocates the use of MBS across different species.

Finally, while MBS entails neural-specific manipulations of interpersonal synchrony (see also [30,31]), it is conceivable that other approaches might be developed to manipulate synchrony at different levels. This could be accomplished through exogenous manipulation of physiological processes that are associated with social behavior e.g. vagal stimulation [32], or through tasks that might indirectly modulate e.g. cardiorespiratory physiology [33,34]. While embracing the research agenda proposed by Verga et al. [1], we wish to witness the emergence of these and other causal approaches contributing to this exciting scientific endeavor.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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