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Review

Can neurofeedback provide evidence of direct brain-behavior causality?

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A B S T R A C T

Neurofeedback is a procedure that measures brain activity in real-time and presents it as feedback to an individual, thus allowing them to self-regulate brain activity with effects on cognitive processes inferred from behavior. One common argument is that neurofeedback studies can reveal how the measured brain activity *causes* a particular cognitive process. The causal claim is often made regarding the measured brain activity being manipulated as an independent variable, similar to brain stimulation studies. However, this causal inference is vulnerable to the argument that other upstream brain activities change concurrently and cause changes in the brain activity from which feedback is derived. In this paper, we outline the inference that neurofeedback may causally affect cognition by indirect means. We further argue that researchers should remain open to the idea that the trained brain activity could be part of a "causal network" that collectively affects cognition rather than being necessarily causally primary. This particular inference may provide a better translation of evidence from neurofeedback studies to the rest of neuroscience. We argue that the recent advent of multivariate pattern analysis, when combined with implicit neurofeedback, currently comprises the strongest case for causality. Our perspective is that although the burden of inferring direct causality is difficult, it may be triangulated using a collection of various methods in neuroscience. Finally, we argue that the neurofeedback methodology provides unique advantages compared to other methods for revealing changes in the brain and cognitive processes but that researchers should remain mindful of indirect causal effects.

1. Introduction

In a neurofeedback experiment, the analysis of brain activity keeps pace with data acquisition allowing it to be fed back to the participant as a visual, auditory, or another representation of the brain activity. Continuation of this process in iterative loops enables an individual to self-regulate brain activity linked with a specific behavior or pathology (Cox et al., 1995; Ros et al., 2014; Sitaram et al., 2017; J. Sulzer et al., 2013). Several neuroscientists argue that since these techniques intervene on brain activation as an independent variable, it provides evidence for a *causal* link between brain activity and cognition as opposed to a mere correlation, thus making neurofeedback comparable to brain stimulation approaches (Birbaumer et al., 2013; Caria et al., 2010; Sitaram et al., 2017; Sulzer et al., 2013; Weiskopf et al., 2004, 2012). While many researchers have the intuition that neurofeedback research seems to provide something more than a mere correlation, there is no consensus on what this "more" is. In other words, the question of whether one can genuinely infer causality using evidence from neurofeedback studies has remained unanswered. Despite this, several authors of rt-fMRI and M/EEG neurofeedback papers make causal claims based on results showing changes in a cognitive function following a neurofeedback intervention (Bagherzadeh et al., 2020; Brickwedde et al., 2019; Okazaki et al., 2015; Scharnowski et al., 2015, 2012; Shibata et al., 2011; Yoo et al., 2012; Yun et al., 2020) (excerpts presented in Box 1). In what sense are the causal inferences made in neurofeedback studies justified? –The question is not whether the neurofeedback training intervention causes a collective change in both brain activity and cognitive processes, which control groups can confirm (Ros et al., 2020; Sorger et al., 2019; Thibault et al., 2016). Instead, the present question is whether it can be inferred that neurofeedback training affected a specific brain activity which in turn caused a specific cognitive function to change.

Box 1. Examples of Causal Inferences in Neurofeedback Studies

"The present decoded fMRI neurofeedback method allowed us to induce specific neural activity patterns in V1/V2, which *caused* visual perceptual learning" (Shibata et al., 2011).

"We found that learned voluntary control over these functionally distinct brain areas *caused* functionally specific behavioral effects" (Scharnowski et al., 2015)

"Thus, neurofeedback training alters alpha lateralization, which in turn decreases performances in the untrained hemifield. Our findings suggest that alpha oscillations play a *causal* role for the allocation of attention" (Okazaki et al., 2015).

"We used MEG neurofeedback to train subjects to manipulate the ratio of alpha power over the left versus right parietal cortex. The results support the proposal that alpha synchrony plays a *causal* role in modulating attention and visual processing" (Bagherzadeh et al., 2020).

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Dominant causal theories hold that to claim causality; it is required that a variable "X" is intervened on and varied systematically while measuring a change in the outcome "Y" while controlling for confounding causes of the change in Y (Pearl and Mackenzie, 2018; Peters et al., 2017; Woodward, 2005). The causal theory of interventionism (Woodward, 2005) emphasizes interventions for discovering causality and claims that for the definition of causality to make sense, we need to ask if the intervention appropriately affects X and not confounds Z which may also cause Y.

This has particular relevance for neurofeedback, because an alternatively "indirect" causal interpretation could be that training subjects with feedback from brain activity X might also affect other types of brain activity "Z." If this latter brain activity Z is genuinely causal for the cognitive process "Y", then results from neurofeedback studies provides no way to distinguish between a direct and indirect causal interpretation. In fact, some authors interpret their findings without necessarily postulating that the trained brain correlate is causal for the change in the cognitive process. Instead, they presume that neurofeedback training caused a change in brain activity which *correlated* with a change in cognition (Bauer et al., 2020; deBettencourt et al., 2015; DeCharms, 2007; Gundlach and Forschack, 2020; Kvamme et al., 2022; Micoulaud-Franchi et al., 2014; Ros et al., 2013; Rota et al., 2009). This paper aims to explore the reasons for this non-causal interpretation. In some sense, the present issue of causality is unimportant for delivering meaningful therapeutic effects. There is no worry about which brain dynamic was responsible for a therapeutic effect, as long as it is reliable. However, we will argue that a clearer understanding of causality in the brain will inevitably lead to more successful therapeutic interventions.

One issue that often arises when discussing the relation between brain activity and cognitive functions is the mind-body problem (Chambliss, 2018; Dijkstra and de Bruin, 2016; Fell et al., 2004; Kurthen, 2010; Nagel, 1993). For the present purposes, we employ the common assumption in neuroscience that all cognitive functions depend causally on a specific neural substrate (i.e., its structure) and the temporospatial patterns of neuronal activity it produces (i.e., its dynamics). Hence, "cognitive functions" thus do not necessarily refer to phenomenal experience but instead pragmatically to the mechanisms of information processing that give rise to behaviors such as perception, higher-order thoughts, and motor functions (Bergmann and Hartwigsen, 2020). An important but separate issue is that a satisfying explanation of the relationship between the brain and cognition is predicated on carefully explaining how cognitive functions can be inferred from behavior (Krakauer et al., 2017).

2. Upgrading correlation to causation

Inferring causality between brain activity and cognitive processes is a fundamental and challenging goal in neuroscience. For instance, there is a longstanding debate about whether causality can be inferred from neuroimaging data using causal models such as Granger causality, dynamic causal models, and causal Bayesian nets (Friston et al., 2013, 2003; Koller and Friedman, 2009; Lohmann et al., 2012; Marinescu et al., 2018). There is a similar debate about whether estimates of connectivity between neural entities are sufficient for a causal interpretation (Reid et al., 2019). It has also recently been discussed whether interbrain synchronization (similar brain activity recorded from multiple brains) can be considered causal for social interactions or if brain stimulation approaches are required to confirm causality (Novembre and Iannetti, 2021a, 2021b).

Interestingly, neurofeedback has been claimed to provide causal evidence for causal models of neuroimaging and interbrain synchronization (Grosse-Wentrup et al., 2016; Gvirts Provolovski and Perlmutter, 2021; Moreau and Dumas, 2021). Similarly, neurofeedback is often advocated to be similar to brain stimulation in terms of being able to derive causal evidence (Birbaumer et al., 2013; Caria et al., 2010; Sitaram et al., 2017; Sulzer et al., 2013; Weiskopf et al., 2004, 2012). However, neurofeedback is also different from brain stimulation. Unlike traditional brain stimulation, where an exogenous current is applied to the brain, neurofeedback can be considered an endogenous version of brain stimulation where the individual self-stimulates certain brain activities (Ros et al., 2010; Sitaram et al., 2017). This, we will argue, has a consequence for causal inference.

At its core, causal inference assigns a direction of the relationship between two variables and assumes that active manipulation of the cause (experimentally or counterfactually) while holding everything else constant produces the effect. The relationship is said to be “asymmetric” because it is always the cause that produces the effect and not vice versa. This can be formalized via the do operator, where P(effect|cause) = P(effect|do(cause)) while P(cause|effect) ≠ P(cause|do(effect) = P(cause) (Pearl and Mackenzie, 2018). A particular emphasis is placed on the ability of classic experiments to randomize the allocation of levels of independent variables to observational units while observing changes in the dependent variable.

Importantly, when using these criteria for causal inference, brain stimulation studies can provide causal evidence (Bergmann and Hartwigsen, 2020; Dijkstra and de Bruin, 2016; Silvanto and Pascual-Leone, 2012). Using a simple example in the context of brain stimulation would be a TMS (transcranial magnetic stimulation) (with sufficient intensity) of the primary motor cortex (M1) of the hand area, which causes or increases the likelihood of a finger movement (P(contraction|do(TMS), but not the reverse the (i.e. P(TMS|do(contraction)). The causal inference is possible because the asymmetry holds for every cause-effect pair throughout the causal chain, which mediates the effect from a TMS pulse to the finger movement (Bergmann and Hartwigsen, 2020).

However, several factors make causal inference for cognitive neuroscience studies using brain stimulation more complex (Beliaeva et al., 2021; Bergmann and Hartwigsen, 2020; Hobot et al., 2020). For example, the effects on cognition may depend on co-occurring brain activity at the time of brain stimulation. Moreover, the induction of specific brain activity may also have downstream effects on other brain activities, which could be causally primary for the cognitive process (see Bergmann and Hartwigsen, 2020 for a review). The complications are hard to formulate using the causal frameworks because they often assume that we can change one mechanism without affecting others (Peters et al., 2017), i.e. we can place "X" under the influence of a mechanism (the intervention) while keeping all other mechanisms undisturbed (Pearl, 2009). This is a strong assumption in neuroscience because of the many interacting mechanisms in the brain.

Here, the causal framework of interventionism is relevant because it explicitly states that to speak of the causal effect of X on Y when intervening on X, we need a clear notion of how to appropriately intervene on X without affecting confounding Z. One of the most established interventionist definitions of causation comes from Woodward (2005) (see Box 2).

Box 2. Interventionist Principles of Causation

Interventionism originate from (Woodward, 2003):

(M) A necessary and sufficient condition for X to be a *direct cause* of Y with respect to a variable set V is that there is a possible intervention on X that will change Y or the probability distribution of Y when one holds fixed at some value all other variables Z_i in V.

A necessary and sufficient condition for X to be a *contributing cause* of Y with respect to variable set V is that (i) there be a directed path from X to Y such that each link in this path is a direct causal relationship…

And that (ii) there is some intervention on X that will change Y when all other variables in V that are not on this path are fixed at some value.

Critically for the above definition to make sense, we need to ask what an appropriate intervention of X is. Woodward defines an Intervention variable as follows:

(IV) I is an intervention variable for X with respect to Y if:

1. I causes X;

2. I acts as a switch for all the other variables that cause X. That is, certain values of I are such that when I attains those values, X ceases to depend on the values of other variables that cause X and instead depends only on the value taken by I;

3. Any directed path from I to Y goes through X. That is, I does not directly cause Y and is not a cause of any causes of Y that are distinct from X except, of course, for those causes of Y, if any, that are built into the I–X–Y connection itself; that is, except for (a) any causes of Y that are effects of X (i.e., variables that are causally between X and Y) and (b) any causes of Y that are between I and X and have no effect on Y independently of X.

4. I is (statistically) independent of any variable Z that causes Y and is on a directed path that does not go through X.

An *intervention* can be understood in terms of an intervention variable I for X with respect to Y. When I takes on a specific value, it causes X to take on some determinate value. From the perspective of experimental science, an intervention can be anything that changes the variable X with an effect on Y. However, to claim that the intervention revealed a causal effect of X on Y, the intervention must meet the requirements in (IV).

Critically, the interventionist definition of causality lists several criteria for an appropriate intervention on X. With the interventionist causal framework in mind; the issue for neurofeedback addressed in the present paper is mentioned in IV4, e.g. that we cannot be sure that the intervention "I" is statistically independent of any variable Z that causes Y and is on a directed path that does not go through X. In neuroscientific terms, we cannot be certain that co-occurring brain activity does not affect the target brain activity we are attempting to induce, nor that the intervention affects confounding brain activity involved in the cognitive function.

One separate issue is the issue of confounding variables intersecting the relationship between X and Y. One everyday example is the causal effect of "pain killer medication" as X on "headache relief" as Y, which one could consider directly causal. However, suppose one would consider the biochemical processes Z that mediate the effect of the medication on headache relief. In that case, one could say that the causal relation between X and Y becomes indirect because Z is a mediator (X → Z → Y). However, according to interventionism (IV3 exception a), X can still be considered as *contributing cause* of Y because the biochemical processes are built into the I–X–Y connection itself. In other words, the cascade of activity that occurs after the induced or trained activity which ultimately affects the cognitive process, is a separate issue for causal inference, one that is also shared between brain stimulation and neurofeedback.

The complications in causal inference for brain stimulation are arguably lesser than in neurofeedback studies because we know that there is an asymmetric relationship between the TMS pulse and the electrical field. We can be certain that it was not the participant's brain that produced the TMS pulse. The main issues for causal inference in brain stimulation studies arise after a known electrical field strength is induced in a known region (Bergmann and Hartwigsen, 2020). For neurofeedback studies, the added issue is that the initial induction of brain activity may be caused by a cascade of intricate brain activity patterns that have downstream effects on the brain activity that is being trained.

Neurofeedback is fundamentally different from traditional exogenous brain stimulation because it works using operant learning principles rather than an externally applied electric current. Transient neural activities are ideally reinforced immediately – i.e., speed of reinforcement is crucial. Most neurofeedback protocols, therefore, focus on providing feedback as soon as possible (Belinskaia et al., 2020; Jackson et al., 2006; LaConte et al., 2007; Sherlin et al., 2011; Smetanin et al., 2019, 2018; Stoeckel et al., 2014; Sudre et al., 2011). However, delayed timing of reinforcement does not make neurofeedback impossible. Instead, it merely leads to the requirement of more conditioning trials (Cox et al., 1995; Grice, 1948; Posse et al., 2003; Sherlin et al., 2011; James Sulzer et al., 2013; Yoo and Jolesz, 2002).

This has a critical yet often overlooked consequence for causal inference of neurofeedback results. Suppose the targeted brain activity X in a region of interest or frequency component is itself caused by an upstream (e.g., supervisor) dynamic "Z," which also occurs close enough in time to the resulting reward signal. The brain dynamic Z could potentially cause the brain activity X to change within the same time window for reinforcement (Lattal, 2010). In that case, Z might get conditioned as well. Formalistically it may be that the probability of P(brain activity X |do(regulate brain activity Z)) and P(cognitive function "Y" |do(regulate brain activity Z)) is greater than 0. In other words, we cannot appropriately intervene on X. For instance, in a study by deCharms et al., participants were trained to down-regulate activity in the rostral anterior cingulate cortex (rACC) using rt-fMRI to reduce pain perception (deCharms et al., 2005). Although successful down-regulation of rACC activity was correlated with decreases in pain perception, the authors caution that top-down connections from an upstream region might drive changes in rACC activity (deCharms et al., 2005; Sulzer et al., 2013). In other words, neurofeedback might intend to regulate a specific brain activity but might inadvertently only be able to do so by regulating its prior causes.

This is not to say that the issue of causal inference in exogenous brain stimulation studies is non-trivial. In fact, there are similar "costimulation" issues, such as the "click" sound and mechanical vibration affecting the peripheral nervous system generated by the discharging TMS coil, which may also affect brain activity in targeted regions (Bergmann and Hartwigsen, 2020). However, with the endogenous stimulation employed in neurofeedback, it is a bigger question what brought about the initial induction of brain activity, making an indirect causal explaination a possibility researchers should not ignore.

3. Indirect causality

A neurofeedback study is typically motivated by a series of prior neuroimaging studies that are themselves unable to test a hypothetical causal relation between variations in specific brain activity and a correlated cognitive function. For instance, neurofeedback studies that aim to regulate the alpha activity, rACC activity, and amygdala activity are all based on prior studies correlating these brain variables with cognitive functions such as attention, pain perception, and emotional processing, respectively (Bagherzadeh et al., 2020; deCharms et al., 2005; Herwig et al., 2019). Because traditional correlative neuroimaging studies cannot rule out that the measured brain activity is epiphenomenal to the cognitive function, the nature of the correlation is unconfirmed (Keizer et al., 2010). In other words, the specific brain activity and specific cognitive function might be spuriously or non-spuriously correlated. We use the term spurious correlation in the general neuroimaging sense, to denote an actual correlation that is not due to an underlying causal relation (commonly occurring because of ignorance of confounds (Lazic, 2010)) i.e. not spurious due to the lack of a normalization term (Pearson, 1897), to circularity in the analysis (Vul et al., 2009) or low sample size and improper outlier correction (Rousselet and Pernet, 2012).

To explain spurious correlations, an often-used analogy is the known spurious correlation between ice cream sales and shark attacks. No amount of regulation of ice cream sales (e.g., a ban or decreasing the cost of ice cream) will ever affect shark attacks. In other words, ice-cream sales are epiphenomenal to shark attacks as it is a mere byproduct of another co-occurring process. Instead, it is warmer temperatures that ultimately cause an increase in ice cream sales and an increase in shark attacks. Alternatively, regulating something like beach attendance is suf-

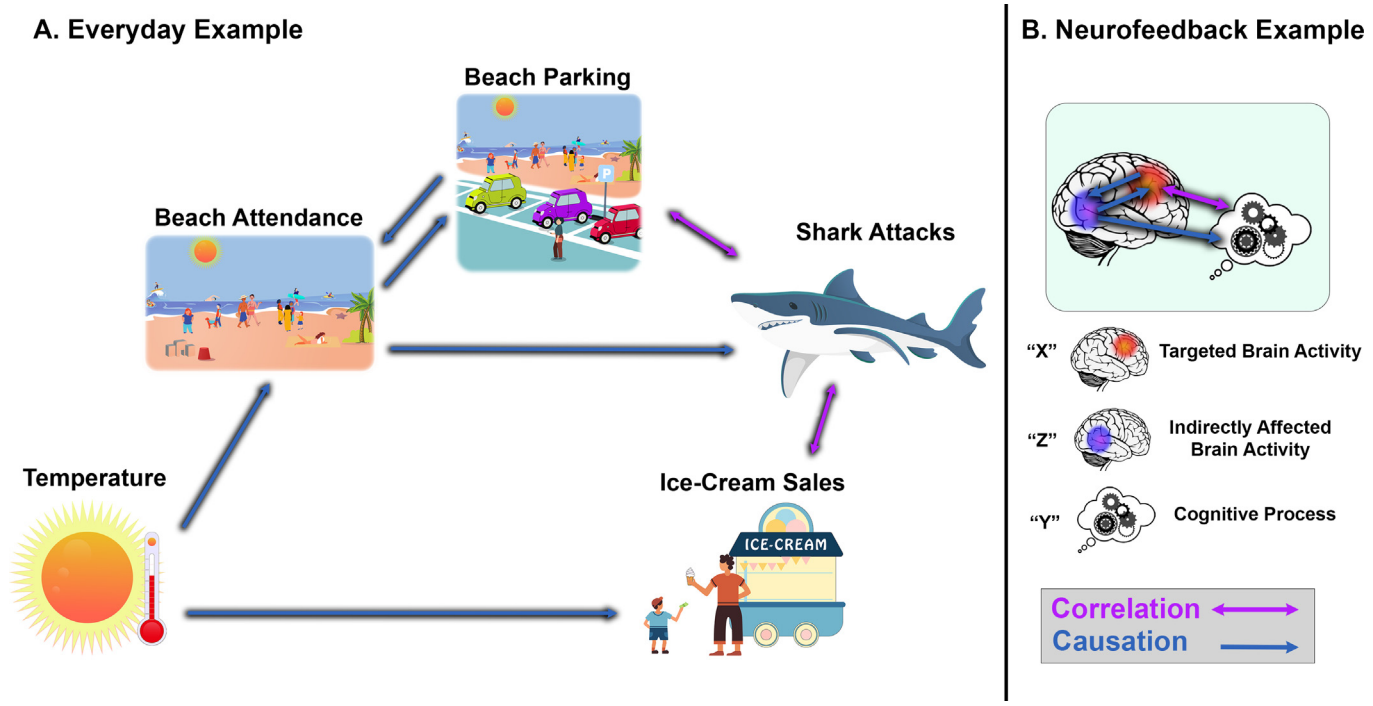

Fig. 1. A. Everyday Example: The ice-cream sales and shark attacks analogy. A spurious correlation rather than a causal relation exists between ice-cream sales and shark attacks as they are independently affected by temperature. Regulating beach attendance or beach parking will affect shark attacks because they are non-spurious correlates of shark attacks. However, only beach attendance is directly causing changes in the probability distribution of shark attacks, whereas beach parking is only indirectly causing shark attacks. Manipulating beach parking and seeing changes in shark attacks reveals that beach parking is part of the causal network which affects shark attacks while not showing beach parking is causally primary for shark attacks. B. Neurofeedback Example: an example of a causal network of brain areas involved in changing the cognitive process. The targeted brain activity, "X" is affected by another brain activity, "Z" which is inadvertently affected by the neurofeedback training; this latter brain activity could be truly causal for the cognitive process "Y".

ficient to cause changes in the probability of shark attacks (see Fig. 1: A. Everday Example).

Concerning neurofeedback, if researchers repeatedly attempt to regulate a specific brain activity and find that successful conditioning of the brain activity does not lead to changes in a correlated cognitive function. For example, suppose that repeated neurofeedback studies consistently demonstrate strong evidence for the null hypothesis indicated by Bayes factors (Dienes, 2014). In this case, we can conclude that training individuals to self-regulate using neurofeedback using the specific brain activity is insufficient for influencing the cognitive function. This provides evidence that the correlation between the brain activity and the cognitive function is likely spurious (i.e., the correlation is in some sense similar to the correlation between ice cream sales and shark attacks).

On the other hand, if the neurofeedback study is successful, meaning that researchers can demonstrate changes in the cognitive function occurring following the trained specific brain activity. Ideally, the specificity of the effect is confirmed by various control groups (such as inverse feedback group, yoked feedback, feedback from unrelated brain activity, or placebo control) (Lubianiker et al., 2019; Ros et al., 2020; Sorger et al., 2019; Thibault et al., 2016). In that case, we can categorically confirm that the trained brain activity is non-spuriously correlated with the cognitive function. We can conclude that the trained brain activity must be part of the "causal network" which causes the cognitive function. However, we cannot disentangle whether we have affected brain activity that directly affects the cognitive process or affected brain activity that is only indirectly causal (see Fig. 1: B. Neurofeedback Example). In the ice-cream sales and shark attacks analogy, this is similar to not knowing whether we have affected beach attendance directly (direct causality) or something like beach parking (indirect causality), which only indirectly affects the prospect of beach attendance and thereby affects shark attacks. The central claim of the present paper is that preliminary neurofeedback studies cannot provide evidence for direct versus indirect causality but rather only for non-spurious versus spurious correlates.

Even in the case of a null-finding, neurofeedback cannot completely confirm that the trained brain area is non-causally related or spuriously correlated with the cognitive function because neurofeedback may also inadvertently activate compensatory brain activations, which may cancel the effect (Mehler and Kording, 2018). I.e. neurofeedback researchers should be wary of potentially "false negative" non-causal inferences as well (a possible partial solution to this issue is provided by implicit neurofeedback, which we will explain in Section 6).

To further illustrate how neurofeedback may work through an indirect causal route, imagine a researcher who performs a biofeedback study using electromyography (EMG) in a clinical ADHD population. By training individuals to down-regulate muscle activity, the researcher observes similar beneficial effects on ADHD symptoms as EEG-based neurofeedback (Barth et al., 2017; Maurizio et al., 2014). Should the researcher conclude that the muscles are *causally* involved in ADHD? Although that interpretation is not disproved, the researcher should consider that muscular hyperactivity is a *correlate* in a causal chain that leads all the way up to the brain. Hence, there are brain circuits that control motor neurons that control hyperactive muscles. When reversing along the causal chain, EMG-biofeedback can indirectly train the brain circuits related to ADHD symptoms (Barth et al., 2017). This is not to dismiss the importance of muscle activity in ADHD symptoms; in fact, the findings clearly underlie their usefulness in bringing about therapeutic effects. However, when inferring causality, it cannot be excluded that EMG-biofeedback evokes systematic activations in brain activity that is causally involved in ADHD, even though direct feedback from said brain activity was not provided (see Fig. 2).

In the case of neurofeedback, things become less clear, as the brain activity that feedback is provided from might also be indirectly affected by other types of brain activity. This makes both the indirect causal and

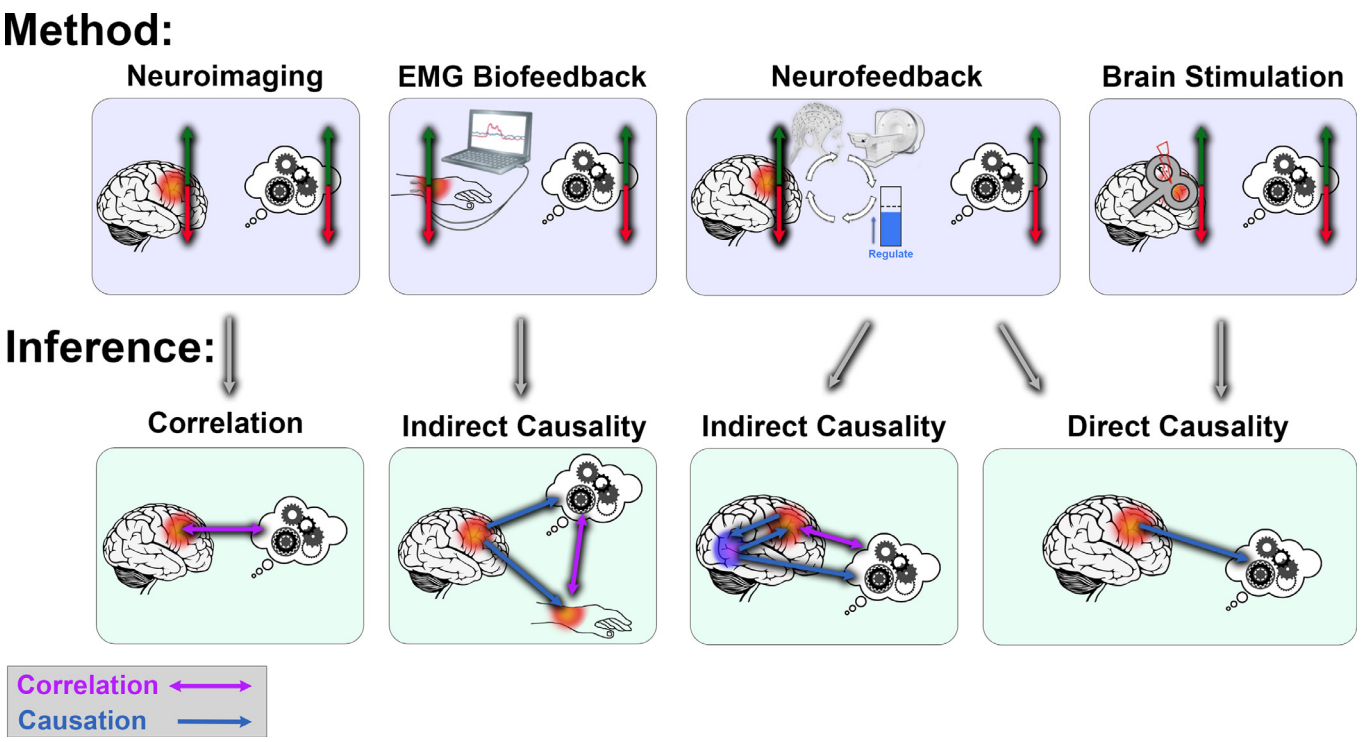

Fig. 2. Top row: Methods used to investigate the brain and cognition. Going from left to right, neuroimaging (typically single-sample) measures variability in specific brain activity and relates it with variability in a cognitive phenomenon. EMG biofeedback decreases hyperactive muscles, and changes in cognition (such as ADHD symptoms) are observed. Neurofeedback is a process where brain activity is measured using (fMRI or M/EEG) and fed back to the individual in real-time to influence the activity (up-regulation or down-regulation), which leads to changes in cognition. Lastly, brain stimulation is the induction of current, which causes direct changes in brain activity and a given cognitive phenomenon. Bottom row: Predominant inferences follow the top row's specific method (grey arrows). Neuroimaging studies using a single sample are only correlative (purple arrows). Effects of EMG biofeedback studies can arise from the indirect training of brain activity (not used to provide feedback from). However, this activity is causing (blue arrows) a change in muscle activity and the cognitive phenomenon. Neurofeedback has two possible inferences. An indirect causal inference (similar to EMG biofeedback), where a certain brain activity (blue activity) not used to provide neurofeedback causes (potentially bidirectionally) changes in both the brain activity that is used to provide feedback (red activity) and in the cognitive phenomenon. Another option for neurofeedback is the direct causal inference, where the trained brain activity and the brain activity causing a change in the cognitive phenomenon are the same. Lastly, brain stimulation studies can dismiss the indirect causal inference because the brain activity is induced directly using an electro/magnetic current (although several other causal inference issues arise for brain stimulation; see (Beliaeva et al. 2021, Bergmann and Hartwigsen 2020, Hobot et al. 2020)).

the direct causal interpretation able to explain results from neurofeedback studies (see Fig. 2). This does not mean that the neurofeedback methodology cannot provide evidence. We argue that evidence from neurofeedback research can constrain the likelihood of causal hypotheses by showing that correlations between specific brain activity and cognitive phenomena are either spuriously or non-spuriously correlated.

4. Alpha band activity and attention

One research topic where we will argue that neurofeedback evidence has been misinterpreted as revealing direct causality is the relationship between oscillatory alpha-band activity and attention. Here, two MEG-neurofeedback studies trained participants to lateralize alpha-band activity while visuospatial attention was measured (Bagherzadeh et al., 2020; Okazaki et al., 2015). Critically, because measures of attention changed following neurofeedback, both studies claim that alpha-band activity plays a causal role in allocating attention (see Box 1). Unlike prior studies, (Bagherzadeh et al., 2020) did not cue participants with stimuli that directed their attention to the trained hemifield during neurofeedback. Instead, participants fixated on a centrally presented grating stimulus and were instructed to use "mental effort" to enhance its visibility during neurofeedback. Thus, unknown to the participants, the real-time measure of the asymmetry of alpha activity from their parietal cortex (meaning more alpha activity in one parietal hemisphere than the other) was directly related to the visibility of the grating stimulus. An inverse control group was used, where the alpha asymmetry training was reversed (i.e., alpha was increased in the opposite hemisphere). As decreased alpha activity is hypothesized as an attentional mechanism that enhances visual processing, the strength of visual evoked responses was assessed with a task-irrelevant probe stimulus (small gray dot) during neurofeedback (Jensen and Mazaheri, 2010; Noesselt et al., 2002). To test if alpha neurofeedback produced changes in attention, the authors employed a Posner-paradigm to assess reaction time changes to visual probes in either hemifield (Posner, 1980). Consistent with the study's hypotheses, the results showed group-specific changes in the asymmetry of alpha activity that correlated with enhanced probe-related evoked responses during neurofeedback and with reaction time changes after neurofeedback training.

The authors claim that their results support the proposal that alpha synchrony plays a causal role in modulating attention and visual processing. They claim that since the participants were only instructed to alter their alpha activity using the feedback display, they can conclude that participants did not covertly shift their attention during neurofeedback. The authors further provide eye-tracking measures to substantiate this claim; however, such overt measures of attention cannot be used to disprove that covert attentional strategies were employed (Gundlach and Forschack, 2020; Jones and Sliva, 2020). Covertly shifting attention is a well-documented strategy to modulate alpha-band activity often used in BCIs (Jensen et al., 2011; Schneider et al., 2020; Treder et al., 2011).

Although the study by Bagherzadeh et al. (2020) methodologically represents a state-of-the-art neurofeedback investigation, the claim that

the results are sufficient to show that alpha activity causes visuospatial attention is an overreached inference. It is plausible that participants simply employed attentional strategies caused by other brain mechanisms underlying attention. A recent line of research investigating how spatial attention is related to alpha activity and steady-state visual evoked responses (SSVEPs) found evidence that the two change independently of each other (Antonov et al., 2020; Gundlach et al., 2020; Gutteling et al., 2022; Keitel et al., 2019; Zhigalov and Jensen, 2020). In one study, changes in alpha-band activity appeared after behavioral and neurophysiological measures of attentional selection, such as hit rates and SSVEPs (Antonov et al., 2020). Hence, these findings are consistent with the notion that alpha-band activity may not be a singular cause of increased sensory responses and may instead be one of several contributing mechanisms subserving the attentional process (Gundlach et al., 2020). A more likely interpretation is that alpha activity is part of the causal network involved in attention or alpha activity is rather a consistent product of attention, such that operant conditioning of alpha using neurofeedback leads indirectly to correlated changes in attention (Ros et al., 2013).

It's important to stress that the relationship between alpha and attention is an active area of research. It is thus still debatable whether alpha indeed fulfils a causal role for attention. Brain stimulation-induced alpha-band activity (e.g. (Romei et al., 2010) establishes a clear causal relationship between alpha oscillations and visual perception; however, alpha oscillations might still only exert their influence at a later point in visual processing and thus remain a product of multiple stages of attention (Schneider et al., 2021; Van Diepen et al., 2019; Zhigalov and Jensen, 2020). The objective of the present paper is not to argue whether or not alpha causes attention or not (see (Peylo et al., 2021) for a review). Instead, our goal is merely to state that preliminary neurofeedback studies cannot conclusively confirm direct causal evidence when the underlying neurophysiological mechanisms of the brain state being regulated remain unclear (Gundlach and Forschack, 2020; Jones and Sliva, 2020). We argue that a more parsimonious interpretation is that alpha neurofeedback studies that demonstrate behavioral effects provide evidence that alpha is part of the "causal network" involved in attention (although potentially only indirectly) while not demonstrating that alpha is causally primary for attention.

5. rt-fMRI regulation of brain metabolism

To further explain the concept of causal network, it is instructive to describe studies that aim to down-regulate amygdala activity using rt-fMRI neurofeedback. Emerging evidence points to the down-regulation of amygdala activity as a potential treatment option for anxiety and affective disorders (Herwig et al., 2019). Here, neurofeedback studies generally find that down-regulation of amygdala activity is accompanied by changes in other brain dynamics such as increased prefrontal activity and the increased connectivity between the prefrontal cortex and the amygdala (Brühl et al., 2014; Herwig et al., 2019; Nicholson et al., 2018, 2017; Paret et al., 2018, 2016, 2014). Moreover, when prefrontal cortex activity is up-regulated, it is accompanied by amygdala down-regulation (Sarkheil et al., 2015), and neurofeedback aimed at increasing the connectivity between the two areas also reduces measures of anxiety (Zhao et al., 2019). Consequently, what characterizes the causal interpretations of these studies is that none of the authors claim that any particular brain activity or dynamic is the primary cause of anxiety, but rather that each makes up individual nodes in a network that collectively causes anxiety. This should be seen in addition to the brain areas involved in neurofeedback self-regulation per se (Emmert et al., 2015; Ninaus et al., 2013; Sitaram et al., 2017). The inclination to interpret the effects of neurofeedback in this manner may derive from the well-established neurobiological model of affective disorders involving a collection of brain regions (Etkin et al., 2015; Herwig et al., 2019; LeDoux, 2000). We will argue that the inclination to infer that the trained brain variable only indirectly affected the target cognitive function should be the default causal interpretation following neurofeedback studies. This is in contrast to authors employing neurofeedback approaches which do make direct causal claims from the trained brain variable to the cognitive process (Caria et al., 2010; Kawato, 2017; Orlov et al., 2018; Scharnowski et al., 2012; Shibata et al., 2019; Tang et al., 2021).

Another question that arises is whether rt-fMRI neurofeedback can ever uncover causal relations since it relies on the blood oxygenation level-dependent (BOLD) signal, representing indirect vascular coupling to neuronal activity (Kim and Ogawa, 2012; Logothetis et al., 2001). Because of the immense complexity of biophysical, physiological, and engineering procedures which generate the BOLD, it has been discussed whether there are brain changes that ultimately are not captured by fMRI (Hillman, 2014; Kim and Ogawa, 2012; Logothetis, 2008; Magistretti and Allaman, 2015; Poldrack and Farah, 2015). Consequently, our appeal to the indirect causal interpretation should be seen in the light of both potential changes in upstream areas, the changes in brain regions involved in self-regulation per se, and the undetected neurophysiological changes which are not captured by the employed neuroimaging modality.

6. Implicit MVPA approaches may hold an advantage

A new trend in fMRI neurofeedback that might resolve causality is afforded by employing multivariate pattern analysis (MVPA) or decoded brain responses (Haxby et al., 2001; Haynes and Rees, 2005; LaConte et al., 2007; Shibata et al., 2011). The technique is also being developed in the electrophysiological domain (Rana et al., 2020; Ray et al., 2015; Tuckute et al., 2021); however, rt-fMRI neurofeedback is currently the predominant form of MVPA neurofeedback (Sitaram et al., 2017). Whereas most conventional rt-fMRI neurofeedback studies aim to increase or decrease the average amplitudes of the fMRI signal in a region of interest (ROI), a key feature of MVPA neurofeedback is that a fine-grained multivariate and distributed pattern of brain activations is used for training (Shibata et al., 2019; Watanabe et al., 2017). The MVPA neurofeedback approach is thus capable of finding, in a data-driven way, the multivariate brain activations most related to a given cognitive process.

A further characteristic of investigations employing MVPA neurofeedback is that the participants are often not provided with an explicit strategy to regulate their brain activity. Instead, the MVPA approach often employs the neurofeedback training "implicitly," e.g., without providing participants with a strategy or informing them of the purpose of the experiment (Amano et al., 2016; Cortese et al., 2016; Koizumi et al., 2017; Oblak et al., 2017; Ramot and Martin, 2022; Shibata et al., 2016, 2011; Taschereau-Dumouchel et al., 2018). In contrast to prescribing an explicit strategy to participants, the implicit approach attempts to avoid the possibility of the targeted neural activity being influenced by neural activity related to meta-cognitive aspects like intention, explicit strategy, and conscious effort (Ninaus et al., 2013; Ros et al., 2014).

Concerning the present issue of causal inference, the argument has been made that implicit MVPA neurofeedback provides the ability to induce specific activities at the neuronal level, which are empirically derived rather than defined *a priori* (Shibata et al., 2019). The implicit MVPA approach thus attempts to limit the possibility that various cognitive factors or physiological artefacts may also influence and change the cognitive process (Ramot and Martin, 2022). The implicit MVPA neurofeedback approach thus attempts to finds the *sufficient* neural dynamic that, when trained, leads to a change in the desired behavior, i.e., training *this neural dynamic (X)* and ensuring that another neural dynamic (Z) associated with conscious effort is not altered, thereby increasing the confidence that intervening on X was sufficient for a change in the behavior, and not a change in Z (Shibata et al., 2019). By limiting the amount of cognitive processes involved, the argument is that the implicit neurofeedback approach also attempts to circumvent the issue of potential compensatory regions being activated by explicit strategies.

Implicit neurofeedback may also circumvent compensatory effects that would have been present and would have produced a false negative null finding.

The causal claim from implicit MVPA neurofeedback can become stronger by showing that activity elsewhere in the brain is unrelated to changes in the induced activity. This can be done formally with an information transmission analysis that ideally complements MVPA neurofeedback studies (Taschereau-Dumouchel et al., 2020b). An information transmission analysis can be used to conduct a whole-brain searchlight analysis that quantifies the degree to which activation patterns in other brain areas can predict the likelihood of neurofeedback-induced activity. In a seminal paper, it was shown that induced activity was confined to the trained area (e.g., V1/V2), except for brain responses involved in reward processing (e.g., the striatum) (Koizumi et al., 2017). Moreover, it has been demonstrated that information transmitted from other areas to the target area drops significantly when implicit neurofeedback is performed (unconscious occurrence of target stimuli-induced brain activity), compared with target presentation during decoder construction (conscious occurrence of target stimuli-induced brain activity) (Taschereau-Dumouchel et al., 2018). This provides evidence that implicit neurofeedback (e.g., neurofeedback without strategies) remains less affected by upstream regions than explicit neurofeedback. However, it has also been recently found that implicit MVPA neurofeedback depends on similar "network" effects among a collection of brain areas for training fear processing similar to conventional explicit neurofeedback approaches (Taschereau-Dumouchel et al., 2020a).

Implicit MVPA neurofeedback is an exciting new frontier that currently provides the strongest evidence for specific causal relationships between brain activations and cognitive functions. However, the technique is not resistant to the argument that there are brain responses that fMRI does not capture. Moreover, the issue of conscious awareness of the targeted cognitive function being trained during neurofeedback is arguable only an issue if said cognitive function can be assumed to be influenced by the awareness of the participant (e.g. similar to placebo effects in clinical neurofeedback studies). There is also debate about the extent to which implicit neurofeedback truly works in a completely implicit manner (Kvamme et al., under review; Muñoz-Moldes and Cleeremans, 2020; Ros et al., 2014).

However, the larger issue facing MVPA and conventional neurofeedback is the finding that the patterns of brain activity under control changes dynamically during training. Therefore, the latest advances within MVPA-neurofeedback make use of decoding methods that dynamically adapt to the changing brain (deBettencourt et al., 2015; Taschereau-Dumouchel et al., 2020a; Taschereau-Dumouchel and Roy, 2020; Zhang et al., 2020). One consequence for causal inference is that it also makes it difficult to say with absolute confidence which brain dynamic(s), at any given time, is being regulated and its precise neurophysiological underpinnings (Sitaram et al., 2017).

7. Overall perspectives

This paper aimed to ask whether neurofeedback studies can derive direct causal evidence from brain activity to cognitive processes. We believe that the opening argument to this discussion is that neurofeedback can clarify whether a given brain dynamic is non-spuriously correlated with a cognitive function and provide evidence only for indirect causality. In contrast, the assumption that neurofeedback can reveal direct causality is inherently problematic. In other words, we question whether, through successful manipulation of the targeted behavior, neurofeedback is only ever capable of revealing that the targeted brain dynamic is merely part of a "causal network" causing the behavior without it being causally primary for the behavior. The idea of a causal network is also similarly appropriate for lesion studies, where a lesion to a particular area may have affected a hub region, which although it may define the network it may not be causally primary (Siddiqi et al., 2022). It could be the tacitly intended meaning in many of the papers we highlight as performing a direct causal interpretation of causality. However, we believe that raising this issue is important for other neuroscientific researchers who are not necessarily well-versed in this limitation.

A question and perspective that arises from the line of inquiry in this paper is; how simple or complex do our causal inferences need to be in neuroscience? (Dijkstra and de Bruin, 2016; Gundlach and Forschack, 2020). We suggest that the issue facing neurofeedback is similar to other interventional techniques, such as TMS, where unequivocal and direct causal evidence has also recently been questioned (Beliaeva et al., 2021; Bergmann and Hartwigsen, 2020; Hobot et al., 2020).

One might argue that given the vast complexity of brain dynamics, which includes self-organization, non-linearity, degeneracy, redundancy, and closed-feedback loops, it is theoretically impossible to establish clear-cut unidirectional causal relationships (Kelso, 1995; Ros et al., 2014; Varela et al., 2001). We believe that although it is appealing to cast the issue of causal inference off as too complex, it is nevertheless possible to create a framework for discussing and considering causality using the current variables available in neuroscience (Dijkstra and de Bruin, 2016; Weichwald et al., 2015; Weichwald and Peters, 2021). In other words, we argue that it is possible to infer that emergent entities such as regional BOLD activity and oscillatory activity act as causal entities without making sense of the causality of the individual 86 billion neurons in the brain (Herculano-Houzel, 2012). Moreover, although the brain is a self-organizing system involving complex feedback loops and circular causality, we still have to make sense of circular causality using notions of linear and "unidirectional" causation (Dijkstra and de Bruin, 2016; Von Bertalanffy, 1967). To this end, we propose that evidence from neurofeedback studies cannot stand alone in deriving causation but instead require multiple neuroscientific methods. For instance, (Grosse-Wentrup et al., 2016) provide an example of inferring from TMS causality (Chen et al., 2013) to neurofeedback causality (Grosse-Wentrup and Schölkopf, 2014) and finally to causal modeling. In short, no cognitive neuroscience method alone is perfect for deriving causality. A combination of methods is thus required such that the limitations of one method are compensated by the strengths of another (D'Esposito, 2013; Mehler and Kording, 2018; Siddiqi et al., 2022). The burden of causality is thus present for all methods and causal frameworks. The central issues in neuroscience is mainly the assumption of causal sufficiency, whether all causally relevant variables have been accounted for, and the issue of meaningfully constructing causal entities from lower-level entities (Bergmann and Hartwigsen, 2020; Dijkstra and de Bruin, 2016; Grosse-Wentrup et al., 2016; Weichwald et al., 2015; Weichwald and Peters, 2021).

Our central argument is that preliminary evidence from neurofeedback studies has an additional issue regarding potential upstream brain activities being concurrently affected along with the intended-to-be-trained brain activity. Consequently, the value for causal inference that preliminary neurofeedback research brings to neuroscience is that it can be employed to arbitrate between spurious versus non-spurious correlates. In this sense neurofeedback can provide stronger causal inferences than traditional task-based neuroimaging thus transcending mere correlations (Siddiqi et al., 2022). When neurofeedback is used in addition to exogenous brain stimulation, it can be used to confirm that a causal effect is also achievable using endogenous brain stimulation. This paper is not meant to discourage researchers from pursuing investigations that employ the neurofeedback methodology. We argue that while it is often unclear how neurofeedback causally brings about the brain and cognitive state changes, it still holds particular advantages compared to other methods.

For instance, neurofeedback holds an unparalleled capacity to induce endogenous brain activity. This is a particular disadvantage with other invasive and non-invasive brain stimulation methods because they may potentially induce exogenous brain activity at artificial and unnatural levels than what is found under normal physiological conditions (Bagherzadeh et al., 2020; Krakauer et al., 2017). The development of

MVPA-neurofeedback also holds a unique advantage compared to brain stimulation methods in being capable of affecting multiple brain areas at once as well as the network connectivity between them (Bauer et al., 2020; Koush et al., 2013; Scheinost et al., 2020; Watanabe est al., 2017). We predict that the next decade of neurofeedback research will see a greater emphasis on the training of multiple brain activation patterns and their concurrent connectivity (e.g., a greater multivariate focus). For example, the recent advance in dual-modality EEG and fMRI neurofeedback holds an additional advantage in being able to target subcortical neural substrates in a non-invasive manner (Keynan et al., 2019, 2016; Mano et al., 2017; Perronnet et al., 2017; Zotev et al., 2018).

8. Conclusion

In sum, we argue that researchers only employing neurofeedback as a methodology should reserve judgment about whether their results can reveal direct causality from brain activity to a particular cognitive state. Plausible causal inferences in neuroscience arise from the use of multiple methods of inquiry, and preliminary neurofeedback methods should at least entertain the possibility of indirect causality. Neurofeedback holds several other advantages than providing causal evidence, and an overemphasis on causal inference may detract from the true potential of neurofeedback.

Data and code availability statement

The paper is a review and features no Data or Code.

Declaration of Competing Interest

The authors have no conflict of interest to delcare

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