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Neural correlates without reduction: the case of the critical period

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Abstract Researchers in the cognitive sciences often seek neural correlates of psychological constructs. In this paper, I argue that even when these correlates are discovered, they do not always lead to reductive outcomes. To this end, I examine the psychological construct of a critical period and briefly describe research identifying its neural correlates. Although the critical period is correlated with certain neural mechanisms, this does not imply that there is a reductionist relationship between this psychological construct and its neural correlates. Instead, this case study suggests that there may be many-to-many psychological-neural mappings, not just one-to-one or even one-to-many relations between psychological kinds and types of neural mechanisms.

Keywords Reductionism · Natural kinds · Neural correlates · Multiple realizability · Taxonomy

1 Introduction

Recent developments in the philosophy of mind and cognitive science include a resurgence of work on the topic of reductionism (Kim 1998; Bickle 2003; Shapiro 2004) and a growing interest in mechanisms and mechanistic explanation, especially in neuroscience (Craver 2008; Bechtel 2009; Piccinini and Craver 2011). These two developments raise the question as to whether the discovery of mechanisms and mechanistic explanations is conducive to reductionism and whether there is a one-to-one relationship between psychological kinds and neural mechanisms. In this paper, I will

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argue that despite the fact that neural mechanisms are often correlated with psychological processes, states, and functions, these correlations do not always yield reductive outcomes. In Sect. 2, I will introduce the psychological construct of a *critical period*, a stage during which an organism is particularly sensitive to inputs of certain types, and will briefly describe research identifying some of its neural correlates. Then, in Sect. 3, I will argue that despite the fact that the critical period is correlated with certain neural mechanisms, there may be a many-to-many relationship between this psychological construct and its neural correlates. Finally, in Sect. 4, I will bring up various objections and will respond to them. The overall aim will be to argue that there can be many-to-many psychological-neural mappings, not just one-to-one or even one-to-many relations between psychological kinds and types of neural mechanisms.

2 Neural correlates and the critical period

Neural correlates are not a philosophical pipe dream; they are widely sought in recent work in cognitive neuroscience. Researchers attempt to find neural correlates of: *fear*, *envy*, *memory consolidation*, and *consciousness*, among many others.[1] In recent years, widespread connections have been discovered between cognitive-behavioral categories and neuroscientific constructs. The search for neural correlates is often tied to a "localization strategy," a research program that posits that activation in a neural structure is "maximally sensitive" or "selective" for a specific psychological function. This posit is what enables the practice of "reverse inference" in neuroscience, which concludes that a particular psychological function is engaged on the grounds that a certain neural structure is activated (Poldrack 2006). To be sure, localization can allow that there can be more than one neural correlate for any given psychological function, in other words, that co-activation of multiple structures is necessary for the implementation of a certain psychological function. But what localization does not appear to allow is the possibility that activation in a particular neural structure may correspond to an entirely distinct psychological function on different occasions or in different contexts. Thus, the search for neural correlates is typically associated with a reductionist strategy, according to which neural structures and mechanisms are specialized for specific psychological functions, either singly or in conjunction with other neural structures. Opposed to this research strategy is one that is sometimes labeled "contextualism" or "constructionism" (Lindquist et al. 2012), which holds that activation in a neural structure may correlate with one psychological function in one context while being correlated with a completely different function in another. The context in question is usually thought to be the neural context, so that activation in some neural structure N_1 may correspond to psychological function P_1 in conjunction with activation in another neural structure N_2, but to some entirely distinct psychological function P_2 in conjunction with activation in a third neural structure N_3. Moreover, there need be no distinct psychological function that corresponds to the contribution of N_1 in particular. For example, according to what Anderson (2010, p. 246) terms the "neural

[1] For *fear*, see LeDoux (1998); for *envy*, see Takahashi et al. (2009); for *memory consolidation*, see Shadmehr and Holcomb (1997); for *consciousness*, see Rees et al. (2002).

reuse" hypothesis, "low-level neural circuits are used and reused for various purposes in different cognitive and task domains." However, a variant that is not sufficiently explored is the possibility that the *non-neural* context also plays a role in determining which psychological function is performed by a particular neural structure or mechanism. In this paper, I will try to make the case that a particular neural mechanism can be correlated with different psychological functions in different non-neural contexts. Not only can there be distinct neural correlates for some psychological functions in different systems (as in multiple realization), the same neural correlate can correspond to different psychological functions depending on its relationship to the broader (neural and) non-neural context. Hence, I will conclude that this situation leads to a many-to-one mapping between psychological functions and neural structures, not just a one-to-many mapping (as in multiple realization), and that such a mapping blocks familiar forms of reductionism such as the localization strategy.

To make this case, I will look at research on the psychological category of the *critical period*. Critical periods have been the focus of work by neuroscientists, developmental psychologists, cognitive ethologists, psycholinguists, and others for well over half a century.[2] There is widespread agreement that a critical period is a relatively brief, well-defined phase in the early part of an organism's lifespan, in which a particular type of input has a strong influence and is vital for the acquisition of a certain perceptual or cognitive capacity. Outside of this critical period, acquisition is either impossible or more difficult and prolonged. To distinguish the former case from the latter, sometimes a distinction is made between a *critical period* and a *sensitive period*, respectively, but I will be using *critical period* as a blanket term for both phenomena. Critical periods have been observed for a range of perceptual-cognitive capacities, including song learning in birds (George et al. 1995), auditory localization in barn owls (Knudsen and Knudsen 1990), whisker representations in the mouse barrel cortex (Erzurumlu and Gaspar 2012), and human language acquisition (Grimshaw et al. 1998). In animal models, evidence for the critical period is often obtained from experiments in which individual organisms are deprived of a certain type of stimulus during a developmental phase. They are then compared with controls that have not been subject to deprivation during that phase. If the controls have acquired a capacity that the deprived individuals cannot acquire (or cannot acquire without considerable additional effort), this is taken as evidence that the capacity is subject to a critical period (e.g. Knudsen and Knudsen 1990).

Critical periods can vary along several dimensions. First, some critical periods begin at or directly after birth while others take place later, even in adolescence. Second, some perceptual and cognitive capacities feature a single critical period while others are characterized by a series of interdependent periods. Third, in some cases there is a sharp cutoff, while in others there is a gradual decrease in the ability to acquire the cognitive capacity (this distinction is not the same as the one drawn between a critical period and a sensitive period, mentioned above). Fourth, the critical period is sometimes dependent on a very specific type of stimulus while in others it is more generic.

[2] Lorenz (1937) seems to have borrowed the term "critical period" from embryology to describe the phenomenon of imprinting. See also Michel and Tyler (2005) and Bateson and Hinde (1987) for brief historical surveys of empirical work.

One of the earliest research programs on critical periods concerned the visual system in various species of mammals, including mice, rats, cats, and monkeys. Cats with one eye surgically covered from birth up to several weeks after birth never develop normal vision in the covered eye once it is reopened, even after many years have passed. By contrast, when the same experiment is repeated with adult cats, even when one eye is covered for a year, normal vision in that eye is restored after it is reopened (Hubel and Wiesel 1970). As one researcher puts it, "the seemingly innocuous act of covering an eye can profoundly alter the physical structure of the brain during the critical period only" (Hensch 2005, p. 877). This simple act affects the development of ocular dominance (OD) columns, patterns of neurons in the primary visual cortex (area V1). As numerous studies have demonstrated, the neural wiring of the OD columns is altered in these experiments such that the columns serving the open eye expand and acquire greater connectivity at the expense of the columns that normally respond to the covered eye.

Recent research has looked more closely at the cellular and molecular processes involved in closing the "window" that marks the ending of the critical period. One common mechanism for achieving this in several species involves the secretion of proteoglycan molecules (sticky sugar-protein hybrids), which condense into a tight net around the dendrites and cell bodies of some of the neurons in the OD columns, impeding axons from moving (Hensch 2003, p. 20). Once these molecules condense around neurons in the extra-cellular matrix (ECM), neuronal connectivity is impaired. After the net of proteoglycan molecules tightens around the neurons that normally serve the covered eye, the neurons can no longer form new connections or develop in response to sensory input. The timing of the production of the proteoglycan molecules is under the control of the expression of certain genes, and this process is sensitive to developmental phases in ontogeny. It is also known that these proteoglycan molecules can be dissolved with chondroitinase-ABC enzyme, which has been used to extend the critical period in some animal models (Pizzorusso et al. 2002).

This explanation of the closure of the critical period would seem to conform closely to the ideals of mechanistic explanation. Although there are untold complexities in the process sketched in the previous paragraph, the explanation clearly refers to "entities and activities organized such that they are productive of regular changes from start or set-up to finish or termination conditions" (Machamer et al. 2000, p. 3). The entities include molecules, cells, and parts of cells (e.g. axons, dendrites), and the activities can be described (albeit in a simplified fashion) in terms of verbs like *secrete*, *condense*, *adhere*, *impede*, *dissolve*, and so on. An account of this mechanism provides a satisfying explanation for the fact that (for example) cats that have one eye covered from birth to several weeks of age never fully recover from this impairment and that the covered eye is unable to regain normal vision even after it is uncovered. It would seem as though we have supplied a neural correlate for the closure of the critical period and that the cognitive or psychological construct of a critical period is a good candidate for reduction to a neural process.

One complication is that there is also evidence to suggest that in some instances, different neural processes can achieve the same result in different species or for different cognitive and perceptual capacities. As a recent review article states: "The mechanisms underlying the control of critical periods are most likely complex and diverse"

(Levelt and Hübener 2012, p. 311). Indeed, they cite evidence that another mechanism for closure of the critical period has to do with synaptic changes in certain inhibitory neurons, specifically functional changes in the speed of inhibitory synapses (Levelt and Hübener 2012, p. 318). Similarly, Hensch (2004, p. 552), refers to "a diversity of molecular mechanisms across systems or even at various stages along the same pathway." Although research on this issue is ongoing, it is not difficult to envision different types of cellular and molecular mechanisms that might lead to a decline in neural plasticity under the control of genetic and epigenetic processes that are sensitive to ontogeny. Before concluding that there is a single neural mechanism responsible for the closure of all critical periods, we will need to look at a range of critical periods in different species and across cognitive and perceptual capacities to determine whether they are all served by the same neural mechanism. To put it in familiar philosophical terms, we need to ascertain whether the critical period is *multiply realized* relative to neural mechanisms. Rather than a one-to-one reduction of the closure of the critical period to a neural mechanism, preliminary evidence indicates that the relationship is one-to-many.

If it turns out that there are indeed a variety of cellular-molecular mechanisms that subserve the closure of the critical period, there are at least three responses that reductionists can make to such cases of putative multiple realization. The first should be familiar from the philosophical literature on reductionism (e.g. Kim 1998). It states that the existence of multiple neural mechanisms corresponding to critical periods does not threaten reductionism, since we can still achieve a limited or local reduction, say to different species or different capacities within species. What we know about natural selection suggests that we will not typically find different neural correlates when it comes to a single capacity within a single species (though polymorphisms do exist within species), but it is not as implausible across species or lineages. However, if we discovered a local reduction of this sort, we would still need to understand why different reducing bases all correspond to critical periods. Assuming that lumping them together is justified, we would still need the psychological characterization of critical periods in order to recognize that we have different neural mechanisms for accomplishing the *same* cognitive and developmental function. Hence, a local reduction does not negate the need for a psychological kind and does not render it dispensable. It just equates a unitary psychological kind with a disjunction of neural ones (which is not, in general, a unitary neural kind, since the class of natural kinds is not closed under disjunction). A local reduction of this kind is sometimes also characterized as a *functional* reduction, since the same psychological role or function is performed by different neural occupants. But that presupposes that there is a single psychological function that these diverse neural states are performing (cf. Khalidi 2005).

The second response goes further than the first in that it denies that the posited multiple mechanisms that correspond to the closure of the critical period are instances of the very same phenomenon even *at the psychological level*. (Notice that it would be misleading to say that these neural mechanisms correspond to subkinds of a superordinate psychological kind, *critical period*, since the existence of such a superordinate kind is just what is being denied by this response.) The reductionist could argue that despite certain superficial similarities the neural differences do not result in significant psychological commonalities, and that lumping them together is not legitimate.

Rather, the psychological construct may be a folk category that will be superseded by scientific research (cf. Kim 1998; Shapiro 2004). The success of this move can only be settled by determining whether scientists who investigate the perceptual and cognitive capacities of organisms are justified in viewing all critical periods as members of a single kind, despite the multiplicity of neural mechanisms that underlie them. It is certainly conceivable that these mechanisms will eventually be regarded as being grouped together without warrant. However, though important differences obtain in terms of molecular and cellular implementation, one can abstract away from them for many cognitive and behavioral purposes, yielding one psychological kind that corresponds to various neural kinds. That is because there are weighty scientific considerations from cognitive ethology and evolutionary biology that justify lumping together different instances of the *critical period*. Many perceptual and cognitive capacities are subject to such a developmental phase, particularly those that must be acquired rapidly and early in life for the sake of the organism's survival, and the similarities among them are explanatorily significant. For example, some researchers have pursued parallels and investigated commonalities among the critical period for language learning in humans and the critical periods for other psychological capacities in other species (see e.g. Newport et al. 2001). The *critical period* cannot be dismissed as a folk category that picks out certain phenomenological or semantic features of interest for certain practical purposes but lacking in scientific credentials (I will return to this reductionist response in Sect. 4).

The third reductionist response is the opposite of the second. Instead of saying that the psychological kind ought to be split, it suggests that the neural kinds should be lumped.[3] It consists in saying that the allegedly diverse neural correlates of a unitary psychological kind must have something in common at the neural level, after all. Even if we were to discover different neural mechanisms for a critical period in species S_1 for capacity C_1 and in species S_2 for capacity C_2, a reductionist could insist that there is a commonality at the neural level that abstracts away from the messy details of molecular and cellular implementation. In this case, it may be maintained that they share something like a decline in neural plasticity or a decrease in neural connectivity. However, against this response, it may be conceded that there is a sense in which there is a common neural denominator in these cases, but it could also be maintained that the phenomenon of a decline in neural plasticity is underspecified. Perhaps it could be made more precise and be of use in neuroscientific theorizing. However, as I will argue in the next section, any attempt to add further precision to this construct will encounter two problems that make it doubtful that the central features of a critical period could be captured in neuroscientific terms.

[3] Much of the recent debate concerning multiple realization has revolved around the issue of whether the neural or biological realizers of allegedly multiply realized psychological kinds are indeed "relevantly" different, or whether the psychological kinds themselves are "relevantly" the same. Opponents of multiple realization cast doubt on the claim that the psychological kinds have relevantly different realizers at the neural level (e.g. Bechtel and Mundale 1999; Shapiro 2004; Polger 2009). Meanwhile, proponents of multiple realization reply by producing cases in which the realizers are indeed relevantly different (e.g. Aizawa and Gillett 2009; Weiskopf 2011).

3 One-to-many or many-to-many?

There are two reasons for thinking that there are inherent obstacles to characterizing the phenomenon of the critical period entirely in neural terms. The first obstacle arises from the fact that the neural mechanism described in the previous section corresponds not to the critical period as such but to the closure of the critical period. While the critical period is a developmental phase in the life of the organism, the closure of the critical period marks the endpoint of that phase. Discovering a neural correlate for the closure of the critical period is not the same as finding one for the critical period itself. Still, it may be said that if one also finds the neural correlate for the *onset* of the critical period (the search for which is an active and productive research program in its own right), then one could identify the critical period as the interval of time between these two types of neural process. But characterizing the endpoints of a process in neural terms is different from characterizing the process itself in neural terms. These two neural correlates would still not correspond to the critical period itself and we would not thereby have given that developmental phase a neural description.[4] It may be objected here that a developmental phase is not a likely candidate for reduction, since it denotes a period in the life of an organism (or rather, a *type* of period in a *type* of organism), rather than an entity, mechanism, or even a process. But that is part of the point: some types of psychological construct do not seem to correspond neatly to the types of things commonly individuated in the neurosciences.

But there is a second, more serious, problem with the effort to capture all and only critical periods in neural terms. The neural mechanism described in the previous section would only correspond to the closure of a critical period if it were to be active relatively early in ontogeny. The critical period is defined relative to the organism's lifespan. If the decline in neural plasticity occurs towards the end of life, it would not be considered the closure of a critical period, but perhaps the onset of dementia or some other psychological phenomenon. Indeed, a search of the literature indicates that there is some evidence of a connection between Alzheimer's disease and the build-up of proteoglycans in the extracellular matrix: "PGs [proteoglycans] possess diverse physiological roles, particularly in neural development, and are also implicated in the pathogenesis of neurodegenerative diseases such as Alzheimer's disease (AD)" (Cui et al. 2013). Moreover, not only is the critical period defined in relation to the lifespan of the individual, it may also be individuated in relation to the history of the species. A developmental change corresponding to the closure of a critical period may not be considered such unless it has the right evolutionary history, that is unless it has evolved to fulfill a particular purpose. Hence, the critical period, its onset, and closure, are at least partly a matter of the environmental and etiological context.

This second obstacle to reduction implies that rather than a one-to-many psycho-neural relation, we have a many-to-many relationship. The very same type of neural

[4] A similar situation seems to arise in other instances of purported psycho-neural reductions, whereby what is reduced to neuroscience is a limiting case or boundary condition of a certain psychological phenomenon rather than the phenomenon itself. Compare Sullivan (2008, p. 509) on the alleged reduction of memory consolidation to the neural mechanism of long-term potentiation (LTP): memory consolidation *initiation* has been reduced to neural level, not memory consolidation.

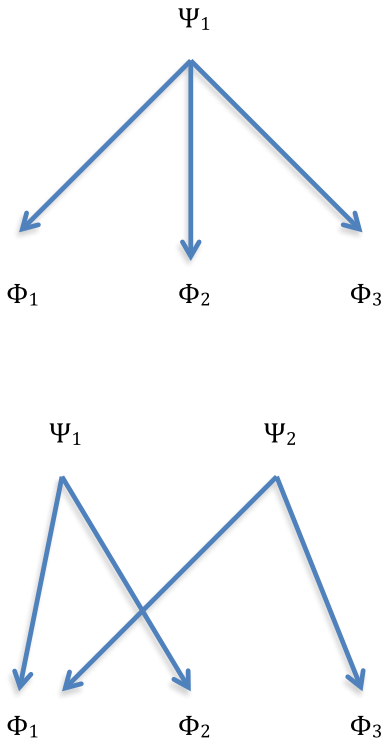

Fig. 1 In the *top diagram*, there is a one-to-many relationship (multiple realization) between a psychological kind (Ψ_1) and a number of neural kinds (Φ_1, Φ_2, Φ_3), whereas in the *bottom diagram*, there is also a many-to-many relationship (crosscutting) between psychological kinds (Ψ_1, Ψ_2) and neural kinds (Φ_1, Φ_2, Φ_3)

mechanism that corresponds to the closure of the critical period relative to the lifespan of one type of organism and in one evolutionary setting may correspond to a different type of psychological phenomenon in different circumstances. Not only is there empirical evidence that the closure of the critical period is subserved by a diverse set of neural mechanisms, the neural mechanism that is correlated with the closure of the critical period in one ontogenetic and phylogenetic context may be correlated with another psychological process (e.g. the onset of dementia) in a different context. Instead of a one-to-many relationship between the psychological and the neural, there is a many-to-many relationship. In other words, the relationship is not just one of multiple realization but of crosscutting (see Fig. 1). This seems to preclude even weak varieties of reductionism, since a many-to-many relationship prevents a local or functional reduction of the type discussed in the previous section. It also shows why the third response to the apparent multiple realization of the closure of the critical period is unsatisfactory. That response tries to equate the closure of the critical period with a decline in neural plasticity or connectivity, but I have argued that such a decline may not correspond to the closure of the critical period in other ontogenetic contexts.

The claim that there can be a many-to-many psycho-neural relationship may be met with the response that neuroscientists can surely make distinctions between neural mechanisms active early and late in ontogeny. If they are able to distinguish between mechanisms that subserve the closure of the critical period and those that underwrite the onset of dementia, they can restore a one-to-one (or at least a one-to-many) rela-

tionship between the psychological and the neural. To be sure, neuroscientists can easily distinguish different manifestations of this process, but if they do so on the basis of ontogenetic or phylogenetic criteria they are no longer investigating neural mechanisms as such, but their relation to the organism's lifespan or their evolutionary history. When it comes to the properties of the neural mechanisms themselves, assuming that they do not differ in terms of the types of entities that constitute them and the types of activities that they undergo, there is no reason to make a distinction. Neuroscientists are surely not oblivious of the lifespan of the individual organism or of the distal causes of the phenomena that they study, but the question is whether they should consider mechanisms to belong to different kinds if they differ only in their place in ontogenetic development or phylogenetic origin. There seem to be no grounds for them to individuate neural mechanisms differently or classify them into different kinds based on such factors if they are otherwise identical. The claim is not that neuroscience is incapable of making such distinctions, but that the scientific division of labor is such that different sciences attend to different causal processes and that the distal origins of neural mechanisms and their extra-organismic environment is not ordinarily a subject of investigation for neuroscience.

4 Objections and replies

The claim that the psycho-neural relationship can be many-to-many (rather one-to-many or one-to-one) has not been sufficiently explored (much less advocated) by philosophers and cognitive scientists, and there are a number of objections that can be raised against it. In this section, I will consider a few of these objections, arguing that none of them is persuasive.

Metaphysically speaking, there may appear to be a fundamental problem with the claim that there can be psychological difference without neural difference, since it seems to violate the principle of supervenience. If we understand this principle as saying (roughly) that there can be no psychological difference without neural difference, then it seems to be breached by the example developed in the previous section, because I have claimed that different types of psychological phenomena can be correlated with the same type of neural mechanism. But the psychological phenomena are distinguished on the basis of other non-neural (and non-psychological facts), such as developmental facts or facts about evolutionary history. The psychological differences supervene upon non-psychological facts, not all of which are neural. Even though a certain type of local psycho-neural supervenience is contravened, global psychophysical supervenience is preserved. Hence, there is no mystery as to why the very same type of neural mechanism could correspond to different types of psychological phenomena in different contexts.

The idea that the psychological or cognitive kind *critical period* is individuated in part relationally or extrinsically may lead to a second objection, to the effect that this discussion assumes that all neural kinds are intrinsic and all psychological or cognitive kinds are extrinsic. But no such claim is being made, just that some neural kinds are identified without reference to a broader environmental and etiological context, while some (correlated) cognitive kinds are at least partly identified in this manner,

and that is what blocks a type correspondence between them. The critical period is interesting because it shows how this can obtain in the case of a category central to research in cognitive science. A follow-up objection could be raised here, to the effect that intrinsic individuation (or individualism) is a basic principle of individuation in the sciences and that scientists never appeal to extrinsic or relational properties, which are causally inert.[5] But this principle, which has been endorsed by some philosophers, is not widely accepted in the sciences, especially the special sciences where individuation according to relational or extrinsic properties, including historical or etiological properties, is commonplace. Phylogenetic taxonomy is largely etiological and biological categories are often characterized by "homology thinking," which is based on historical descent or etiology (Ereshefsky 2007; Brigandt 2007). Moreover, there are other bona fide scientific kinds outside biology that are distinguished, at least in part, on the basis of relational or etiological properties (e.g. *meteor*, *igneous rock*). In the case of psychological taxonomy, as in these other cases, there are often good grounds for individuating kinds on the basis of relational properties, since whether or not a cognitive capacity is subject to a critical period influences survival and adaptive advantage, and this makes a causal difference to the continued existence of the organism and the species.[6]

A third objection to the claim that there can be a many-to-many psycho-neural relationship would deny that there is a unitary psychological kind, *critical period*. As already mentioned in Sect. 2 (see the second response to multiple realization), a reductionist might insist that there is no reason to gather together these diverse neural phenomena into a single category. Strictly speaking, such a response is eliminativist rather than reductionist, since it maintains that there is no single psychological kind, just a set of distinct neural kinds, which should not be lumped together. The objection might add that the construct of a critical period is a vestige of a pre-scientific account of the mind and will wither away as research advances. In response, it is impossible to anticipate which scientific categories are liable to endure and which will be abandoned as more is known about the mind-brain. Suffice it to say that the category *critical period* continues to play a prominent role in the cognitive sciences and research into it is conducted by a number of sub-disciplines. Moreover, it plays an important role in explanations in evolutionary psychology, cognitive ethology, and related fields, where researchers address questions such as why birdsong is subject to a critical period in some species and not in others, or why there should be a critical period for language in humans and song in some bird species. For these purposes, it is largely irrelevant whether the neural underpinnings of a critical period are the same. What matters is the selective advantage (or lack thereof) of having a perceptual or cognitive capacity subject to a critical period. Clearly, it makes a difference to selection whether a psychological capacity is acquired early or late in development, since early acquisition can make the difference between survival and non-survival. Similar selection

[5] Fodor thinks that individualism "is a constitutive principle of science" (1987, p. 45) and claims that *planet* is not a scientific kind since it is individuated relationally. But planets have properties (e.g. life-supporting features) that they would not have if they were just rocks hurtling through space.

[6] For a discussion of some of the selection pressures that would favor the emergence of critical periods in different species and for different capacities, see Immelmann and Suomi (1981).

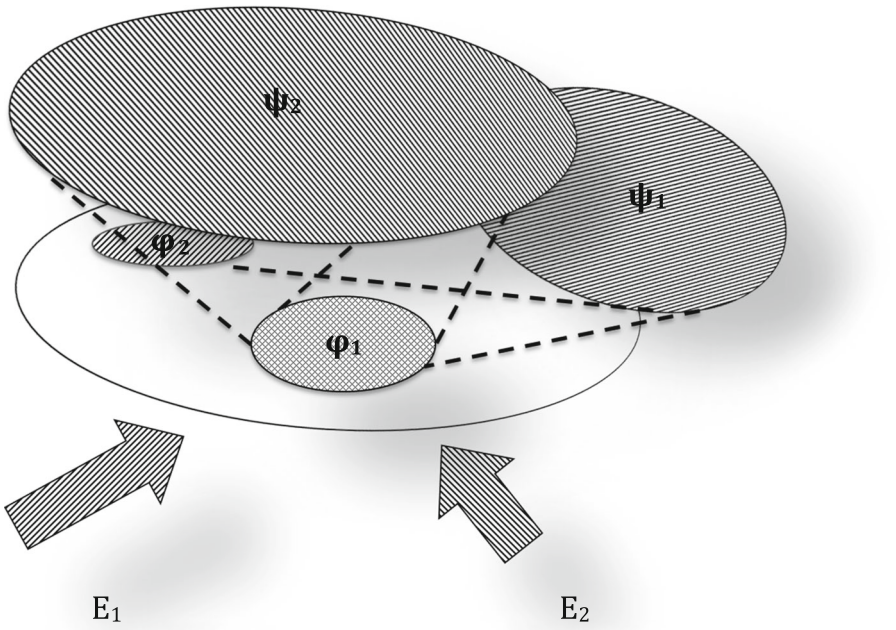

Fig. 2 The same psychological phenomenon, Ψ_1, can be multiply realized in different neural mechanisms, φ_1 and φ_2. But additionally, the same neural mechanism φ_1 can correspond to different psychological phenomena, Ψ_1 and Ψ_2, depending on different extrinsic factors, E_1 and E_2

pressures may give rise to the instantiation of critical periods for different psychological capacities and in different species, and these may be underwritten by different neural mechanisms. The resultant picture of the relationship between psychological capacities and neural mechanisms is one in which the very same type of psychological process may be underpinned by different types of neural mechanism (multiple realization) *and* the same type of neural mechanism may also correlate with different types of psychological process. This yields a crosscutting relationship between neural and psychological kinds (see Fig. 2).

5 Conclusion

I have argued that when it comes to at least one psychological construct, the *critical period*, instead of a one-to-one or even a one-to-many relationship between psychological and neural kinds, we have a many-to-many relationship. Not only does the critical period lend itself conceptually to such a relationship, there is also some empirical evidence that there are different neural mechanisms involved in the closure of the critical period, and also that some of these same mechanisms subserve entirely different psychological constructs. If the closure of the critical period bears a many-to-many relationship to its neural correlates, this would block a type reduction of this psychological construct. Hence, the existence of neural correlates does not necessar-

ily imply a relationship of reduction between the psychological and neural domains. Reduction is blocked primarily because of the fact that this psychological kind is partly individuated based on etiology and the broader environmental context, whereas the neural mechanisms correlated with it are not so individuated. As long as such a relationship obtains, there will not just be multiple realization among psychological and neural kinds, but crosscutting among these kinds. It is an open question as to whether this outcome is commonly attested for other psychological and neural kinds, but it is likely that there are other psychological kinds that are (partly) individuated with reference to etiology and broader contextual factors, whereas neural mechanisms are typically not individuated in this manner.[7] More work needs to be done to ascertain whether a similar relationship obtains between other psychological kinds and their neural correlates.[8]

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[7] In Khalidi (2017), I make a similar case for the psychological capacity of *episodic memory*.

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