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Tinbergen on mirror neurons

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Fifty years ago, Niko Tinbergen defined the scope of behavioural biology with his four problems: causation, ontogeny, survival value and evolution. About 20 years ago, there was another highly significant development in behavioural biology—the discovery of mirror neurons (MNs). Here, I use Tinbergen's original four problems (rather than the list that appears in textbooks) to highlight the differences between two prominent accounts of MNs, the genetic and associative accounts; to suggest that the latter provides the defeasible 'best explanation' for current data on the causation and ontogeny of MNs; and to argue that functional analysis, of the kind that Tinbergen identified somewhat misleadingly with studies of 'survival value', should be a high priority for future research. In this kind of functional analysis, system-level theories would assign MNs a small, but potentially important, role in the achievement of action understanding—or another social cognitive function—by a production line of interacting component processes. These theories would be tested by experimental intervention in human and non-human animal samples with carefully documented and controlled developmental histories.

1. Introduction

Fifty years ago, Tinbergen [1] published an article entitled 'On the aims and methods of ethology', that has had a huge impact on the biological study of behaviour. He argued that four problems define the scope of this field: *causation*, *survival value*, *ontogeny* and *evolution*. To this day, many textbooks use Tinbergen's four problems, or 'four whys', to introduce students to behavioural biology [2,3]. Tinbergen did not, of course, write about mirror neurons (MNs). However, in this article, I use his four problems to draw out the differences between two well-developed accounts of MNs—the 'genetic view' and the 'associative account'—and to explain why these differences matter. This seems appropriate given that the discovery of MNs was another highly significant development in behavioural biology; MNs also had a birthday recently (mere striplings at 20 years old) and the genetic view of MNs has been shaped by ethological principles. I hope to show that, although the associative account did not emerge from ethology, it is entirely compatible with that tradition. Indeed, I argue that a careful reading of Tinbergen's four problems [4] reveals that he was advocating a type of functional analysis which, according to the associative view, should be a high priority for future research on MNs.

In a recent article [5], my colleagues and I briefly recommended some new directions for MN research. Drawing on work in the philosophy of biology and cognitive science, as well as empirical literatures in psychology and neuroscience, this article explicates and extends those recommendations. For example, it explains exactly what is meant by 'system-level' or 'functional' analysis, why it is important, and how it could be applied in research on MNs.

In the first section, I give a brief overview of the genetic and associative accounts of MNs. In the second section, I discuss each of Tinbergen's four problems, reviewing data on the *causation* and *ontogeny* of MNs that distinguish the genetic and associative accounts, and noting that the *evolution* and *survival value* of MNs have not been studied in a systematic way. In the final section, I argue that, in combination, Tinbergen's discussion of *survival value* and the associative account motivate important new directions for MN research.

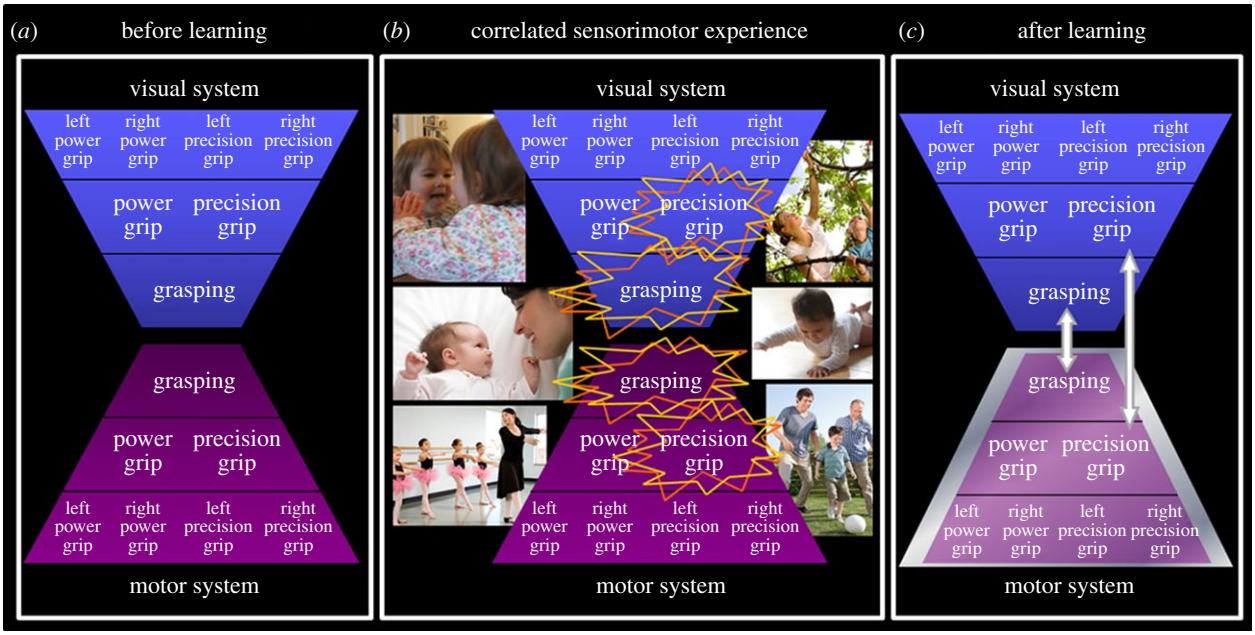

Figure 1. Mirror neurons from associative learning. (*a*) Before learning, sensory neurons in the superior temporal sulcus, encoding visual descriptions of observed action, are not systematically connected to motor neurons in premotor and parietal areas involved in the production of similar actions. (*b*) Through self-observation and social interaction (e.g. being imitated, synchronous activity) in the course of typical development, agents receive correlated sensorimotor experience; they see and do the same action at about the same time (contiguity), with one event predicting the other (contingency). This experience produces correlated activation of sensory and motor neurons coding similar actions, and, through associative learning, strengthens connections between these neurons (*c*). Owing to these connections, neurons that were once involved only in the execution of action will also discharge during observation of a similar action; motor neurons become MNs. Because the visual system and motor system are organized hierarchically, some types of sensorimotor experience produce correlated activation of sensory and motor neurons coding relatively low-level features of action (e.g. left or right hand, power or precision grip), and thereby generate strictly congruent, hand- and direction-sensitive MNs. Other types produce correlated activation of neurons coding relatively high-level features (e.g. grasping), and generate broadly congruent MNs. Reprinted with permission from Cook *et al.* [5]. (Online version in colour.)

2. Two accounts of mirror neurons

I use the term 'genetic account' (or, elsewhere, 'adaptation account' [6]) to refer to a coherent hypothesis about the origin and function of MNs which, although it has not been formulated explicitly by other researchers, is consistent with what has been claimed about the evolution of MNs [7], the likelihood that they are present at or shortly after birth [8], the role of experience in their development [9] and the importance of MNs with respect to 'action understanding' [10]. The genetic account suggests: (i) among common ancestors of extant monkeys and humans, some individuals had a stronger genetic predisposition to develop MNs. (ii) These individuals were more reproductively successful than those with a weaker genetic predisposition, because the presence of MNs enhanced their capacity for 'action understanding'. (The term 'action understanding' was introduced specifically to characterize the function of MNs.) (iii) Consequently, through natural selection, a genetic predisposition to develop MNs became universal, or nearly universal, in monkeys and humans. (iv) Motor experience (the performance of actions) and/or sensory experience (the observation of actions) plays a tuning or facilitative [11] role in the development of MNs, but the 'mirrorness' of MNs—their cardinal capacity to match observed with executed actions—is due primarily to this genetic predisposition.

The associative account is schematically represented in figure 1. It suggests: (i) neither monkeys nor humans have a specific genetic predisposition to develop MNs; they do not genetically inherit a set of MNs, or even a special-purpose learning mechanism that promotes the development of MNs. (ii) Rather, both monkeys and humans have genetic predispositions (a) to develop connections between particular sensory and motor areas of the cortex, which evolved because it promotes precise visual control of action, and (b) to develop a domain-general capacity for associative learning. (iii) When individuals with these predispositions receive correlated experience of observing and executing the same actions, they develop MNs for those actions. (iv) MNs may contribute to behaviour in a number of important ways, and their development may have been favoured by cultural evolution [12], but they are not a genetic adaptation for action understanding or any other social cognitive function.

The genetic and associative hypotheses both acknowledge that the development of MNs depends on the interaction of nature *and* nurture, genes *and* the environment, evolution *and* learning. The two accounts differ in the specific roles they assign to genetic evolution and to learning, and in the types of experience they take to be important. The genetic hypothesis says that genetic evolution has played a specific and decisive role—that MNs are a genetic adaptation for action understanding—and learning, based on sensory and/or motor experience, plays a merely facilitative role in their development. By contrast, the associative hypothesis says that genetic evolution has played a non-specific background role—that associative learning and connectivity between sensory and motor cortex, but not MNs, are genetic adaptations—and that the characteristic matching properties of MNs are forged by sensorimotor learning.

3. Tinbergen's four problems

Table 1 lists Tinbergen's four problems [1]. I leave discussion of *survival value* until last because it is the contentious problem;

Table 1. A summary of the differences between the genetic and associative accounts of mirror neurons, and of priorities for future research, in the light of Tinbergen's four problems.

Tinbergen's problem	foci	genetic account	associative account	future research
causation	internal and external 'preceding events'	goal-directed action	high- and low-level properties of body movement	developmental history
ontogeny	internal and external influences on development	facilitation by sensory and/or motor experience via dedicated learning processes	induction by sensorimotor experience via general learning processes	experimental control of sensory, motor and sensorimotor experience in monkeys
evolution	phylogeny	mirror neurons in the primate lineage	older and more diverse	
	selection	for action understanding	for visuomotor control and associative learning	
survival value	functional analysis	action understanding	unknown	developmental history, system-level theory, 'small' role intervention, animal models

the problem that has been remodelled by textbook writers. Tinbergen presented each problem as a question or set of questions that ethologists should ask about behaviour. Most of his examples related to the behaviour of whole animals—for example, egg-fanning in sticklebacks and crowding behaviour in caterpillars. However, with considerable foresight, Tinbergen saw ethology as 'moving towards a fusion with the fields conventionally covered by Neurophysiology and Physiological Psychology' (p. 416). It is therefore true to the spirit of Tinbergen's analysis to apply it to the behaviour of MNs; their defining tendency to discharge during the observation and execution of similar actions.

(a) Causation

The problem of *causation* is to identify internal and external events that trigger a focal behaviour. In the case of MNs, causation has been studied extensively through single-unit recording in monkeys. This research investigates the 'field properties' of MNs; the characteristics of executed actions (internal events) and observed actions (external events) that correlate with MN firing. The genetic view takes these data to show that MNs are selectively responsive to high-level features of action—to 'motor acts' or action 'goals'—and infers from this that MNs 'allow the observer to understand directly the goal of the actions of others' [10, p. 268].

The associative view suggests that MNs are not selectively responsive to high-level features of action, to action 'goals' [13]. It acknowledges that early research seemed to indicate this kind of selectivity; for example, to show robust MN responses to object-directed actions, and little or no responding to pantomimed and object-free actions [14,15]. However, subsequent studies have indicated that a sizable proportion of MNs respond during observation of object-free body movement. For example, recording from pyramidal tract neurons, Kraskov *et al*. [16] found that a significant proportion of MN responses modulated by observation of object-directed grasping showed similar modulation during observation of pantomimed grasping. In addition, it is now known that substantial proportions of MNs respond to facial gestures such as lip-smacking, lip-protrusion and tongue-protrusion [17]. There is also evidence of selectivity with respect to relatively low-level features of action such as the hand used for grasping (left or right), direction of movement (left-to-right or right-to-left) [15], distance from the observer [18] and the observer's viewing angle (first person or third person) [19].

The associative account assumes that motor neurons acquire mirror properties whenever there is a contingency (or predictive relationship) between the firing of motor neurons and of sensory neurons coding properties of similar actions. Therefore, the associative account is compatible with there being a mixture of strictly congruent MNs, which are sensitive to the low-level features of observed actions (effector used, type of grip, direction of motion, viewing angle, proximity to the observer), and broadly congruent MNs, which are responsive to a range of related actions, regardless of the manner of their execution. Hierarchical organization is characteristic of both visual and motor systems [20–23]; they comprise different neural populations encoding relatively low-level and more abstract representations. Therefore, contingencies can be experienced between both low-level (e.g. descriptions of particular 'precision' or 'power' grips) and high-level (e.g. descriptions of 'grasping') sensory and motor representations. When a monkey observes itself performing a precision grip, the excitation of sensory and motor populations encoding a specific grip are correlated. However, when feeding in a group, a monkey might observe and perform a range of grasping actions, thereby causing correlated excitation of higher-level visual and motoric descriptions of grasping.

By emphasizing the power of contingency, the associative account also explains the existence of logically related, audiovisual [24,25] and tool-use MNs [26,27]. According to the associative hypothesis, MNs acquire sensorimotor properties whenever individuals experience a contingency between 'seeing' and 'doing'. It is not necessary for the actions that

are seen and done to be the same, or indeed for the 'seen' component to be a natural, action-related stimulus, such as the sight of animate motion or a sound that could have been heard by ancestors of contemporary monkeys. Both monkeys and humans often experience non-matching sensorimotor contingencies, where the observation of one action predicts the execution of another; for example, you push and I pull [28,29]. The associative account therefore explains in a very straightforward way why logically related MNs respond to different actions in observe and execute conditions. Equally, it suggests that tool-use MNs develop when action performance is reliably predicted by the sight of actions performed with tools (e.g. food items being gripped with pliers), and that audiovisual MNs develop when action performance predicts characteristic action sounds (e.g. paper tearing or plastic crumpling; [30]).

To summarize: studies of *causation* suggest that MNs do not selectively encode such high-level properties of body movement as early research seemed to indicate, and the associative account can explain in a straightforward way the full range of MN properties revealed by these studies.

(b) Ontogeny

According to Tinbergen, research on *ontogeny* should first describe 'change of behaviour machinery during development', and then investigate internal ('innate') and external ('learned' or 'environmentally induced') factors controlling this change. To investigate these factors, he recommended experiments manipulating the developmental environment, and noted that 'One receives the first indications of *internal* control from demonstrations of the ineffectiveness of certain environmental properties' [1, p. 426]. Thus, in common with many behavioural biologists today [31–33], Tinbergen assumed that success in changing a characteristic by environmental intervention indicates the importance of learning in development, and that failure indicates the importance of internal factors or 'genetic instructions'.

Experiments of the kind recommended by Tinbergen have not been used to study the ontogeny of MNs in monkeys. However, my group and others have used a logic similar to Tinbergen's to test the associative account using indices of MN activity in human subjects. These experiments have isolated the effects of environmental manipulations that give adults novel sensorimotor experience—in which observation and execution of similar actions is systematically correlated or anti-correlated—from the effects of purely sensory (observation) and purely motor (execution) experience. Using all of the behavioural and neurophysiological measures of mirror mechanism activity commonly applied to humans, these experiments indicate that relatively brief periods of sensorimotor experience can enhance [34,35], abolish [36–40], reverse [41–43] or induce [44–46] mirror mechanism activity. These findings suggest that MN development shows exactly the kind of plasticity predicted by the associative account.

Of particular interest, given Tinbergen's assumption that resistance to environmental intervention would indicate internal or genetic control, these experiments have found no evidence that MNs are resistant to coding relations that are irrelevant or potentially antagonistic to 'action understanding'. Specifically, they have encountered no resistance to coding of inanimate stimuli, rather than observed body movements, or to coding of dissimilar, rather than similar, observed and executed actions.

Evidence that MNs are not resistant to coding inanimate stimuli comes from studies showing that arbitrary sound, colour and shape stimuli can induce mirror motor-evoked potential (MEP) [44,47], functional magnetic resonance imaging (fMRI) [45,46,48] and behavioural effects [35] following sensorimotor training [49]. For example, Press *et al*. [46] found using fMRI repetition suppression that, after a brief period of training in which participants made distinctive hand gestures (e.g. point, fist) in response to arbitrary geometric shapes (e.g. triangle, hexagon), shape presentation was sufficient to activate a mirror representation of the action with which it had been paired during training.

Evidence that MNs are not resistant to coding dissimilar actions comes from studies showing that non-matching (or 'counter-mirror') sensorimotor training abolishes automatic imitation [36–40], and reverses both fMRI [43] and MEP mirror responses [50]. For example, Catmur *et al.* [50] gave participants approximately 90 min of non-matching sensorimotor training in which they repeatedly made an index finger movement while observing a little finger movement, and vice versa. Before this training, they showed mirror MEP responses, for example, observation of index finger movement elicited more activity in an index finger muscle than observation of little finger movement, and vice versa for the little finger muscle. After training, this pattern was reversed, for example, observation of index finger movement elicited more activity in the little finger muscle than observation of little finger movement.

As predicted by the associative account, these induction and reversal effects indicate that sensorimotor experience perturbs but does not damage MNs: it prevents MNs from selectively encoding similar observed and executed actions, but does not stop them from encoding systematic relations among stimuli and responses. This is hard for the genetic account to accommodate because (i) as Tinbergen implied, if a trait is a gene-based adaptation, then its development tends not to be perturbed by environmental variations that were present when the trait evolved, and (ii) sensorimotor experience of the kind that induces and reverses MN activity is likely to have been present in the period when, according to the genetic account, MNs evolved. Specifically, it is likely that the common ancestors of extant monkeys and humans experienced contingencies between objects and actions (e.g. when distinctive actions were made on distinctive objects), and between observation and execution of non-matching actions (e.g. when one individual countered a blow from another).

It has been suggested that some of our training effects reflect changes in dorsal premotor cortex (PMC) rather than in ventral PMC, where most MNs have been found in monkeys, or in control mechanisms that override MNs rather than in MNs themselves [51]. We have responded by showing that the effects occur in both dorsal and ventral PMC [41], and that they are present too early in response preparation to be due to effects of training on control mechanisms [52].

Several hybrid models have been developed in the wake of the genetic and associative accounts. These suggest that associative learning plays an important role in the ontogeny of MNs, but that it has been 'canalized' or 'exapted' to allow MNs to fulfil one or more social functions. For example, the canalization hypothesis suggests that MNs are acquired through associative or 'Hebbian' learning [53], but their development is supported by certain features of the

perceptual-motor system, including the tendency of infants to look at their own hands in motion [54]. This view differs from the associative account if it assumes that these canalizing features were favoured by natural selection specifically because they support the development of MNs. Similarly, one exaptation hypothesis assumes that domain-general mechanisms of associative learning are necessary for the development of MNs, but emphasizes that the inputs to these mechanisms are constrained to represent hand–object relationships [55]. Like the canalization hypothesis, this view differs substantively from the associative account only if it assumes that the connectivity providing these constraints evolved, at least in part, for the development of MNs, rather than the visual control of hand actions. Another exaptation hypothesis suggests that associative learning or a special form of sensorimotor learning underwrites the development of hand-related MNs, but the development of facial MNs is minimally dependent on experience [56,57]. Finally, the latest hybrid model draws attention to the role of 'epigenetics', and suggests that associative learning mediates MN plasticity in adulthood, whereas the early development of MNs is mediated by other processes [58].

Each of these hybrid models is plausible, consistent with the observed distribution of MN types (strictly congruent, broadly congruent, etc.), and has distinctive and significant strengths. For example, compared with the associative account, the Hebbian canalization hypothesis provides a more specific neural characterization, and the first canalization hypothesis provides a more computationally explicit characterization, of the development of MNs through associative learning. Consequently, they may provide superior guides for future research on *causation*, and for studies of *ontogeny* using single cell recording in monkeys. Similarly, the epigenetic hypothesis reminds us that, whether or not MNs constitute a genetic adaptation for action understanding, their development must involve, not necessarily epigenetic *inheritance*, but complex mechanisms of gene expression. These contributions are valuable and make hybrid modelling a very promising direction for future research. However, this article focuses on the associative account because, as my colleagues and I have argued elsewhere [5,59], insofar as the hybrid models make predictions distinct from those of the associative account, these predictions either have not been tested, or the current data favour the associative view. For example, the idea that hand and face MNs have different origins would be supported by evidence that face MNs are less susceptible than hand MNs to modification by sensorimotor experience. However, as far as I am aware, this novel prediction of the hand/face canalization model has not been explicitly tested, and a recent study of improvement in facial imitation suggests that face MNs are as susceptible to modification by sensorimotor experience as hand MNs [60]. Similarly, it is difficult to test the hypothesis that associative learning mediates MN plasticity in adulthood, whereas the early development of MNs is mediated by other processes [58], because it is hard to manipulate the early developmental environment in the way that Tinbergen recommended, and there is currently no widely accepted index of MN activity in infants [5]. However, in accord with the associative account, studies using electroencephalographic measures suggest that experience [61], and specifically sensorimotor experience [62], plays a key role in the early development of MNs.

To summarize: studies investigating the ontogeny of MNs, of the kind recommended by Tinbergen, have been conducted in humans but not in monkeys. By Tinbergen's lights, the human studies have provided evidence that sensorimotor experience is crucial; they have shown that the development of MNs can be readily and radically changed by environmental manipulations providing novel sensorimotor experience. By contrast, they have provided no evidence of internal, genetic factors specifically promoting or canalizing the development of MNs (coding similar observed and executed actions), rather than visuomotor neurons more generally (coding executed actions and the animate or inanimate stimuli with which the performance of those actions has been correlated).

(c) Evolution

Tinbergen's *evolution* problem has two components, one relating to phylogeny—at what point(s) in the tree of life the behaviour evolved—and the other to natural selection—the features of the focal behaviour, and of ancestral environments, that led the behaviour to enhance reproductive fitness. The genetic account suggests that the MNs found in monkeys and humans evolved in the primate lineage, and that they were favoured by natural selection because they enhanced 'action understanding'. By contrast, the associative account suggests that MNs do not have a specific, genetic evolutionary source. However, their development is made possible by connectivity between sensory and motor areas which evolved earlier than the primate lineage, for visuomotor control, and by mechanisms of associative learning, enabling event prediction, which are widespread in the animal kingdom.

In Tinbergen's time and today, systematic studies of phylogeny compare groups of closely related species, and hypotheses about selection are tested by selective breeding experiments. Evolution is a central focus of the genetic account and of hybrid models. However, possibly owing to the major methodological challenges involved, neither the phylogeny nor the selection of MNs has yet been investigated systematically.

(d) Survival value

Tinbergen's remaining problem, *survival value*, has not survived [4,63]. Textbook writers have relabelled this category 'function' [2] or 'adaptive advantage' [3], filled it with the selection component of Tinbergen's *evolution* problem and identified *evolution* solely with the study of 'evolutionary history' or 'phylogeny'. In some ways, this was a legitimate and sensible thing to do. Tinbergen certainly thought that *survival value* can inform hypotheses about selection, and it was less than elegant to subsume two major questions, about phylogeny and selection, under 'one' problem. However, Tinbergen was forceful in asserting that the study of *survival value* should not be equated with the study of selection. He stressed that whereas selection is a historical matter, and therefore necessarily involves a degree of 'guesswork', *survival value* can and should be 'established experimentally' (p. 418). He also underlined the independence of questions about *survival value* by insisting that they would be important even if animals were products of special creation rather than evolution: 'To those. . . who argue that the only function of studies of survival value is to strengthen the theory of natural selection I should like to say: even if the present-day

animals were created the way they are now, the fact that they manage to survive would pose the problem of how they do this' (p. 423).

Tinbergen's purpose in laying out 'the four problems of Biology' was 'pragmatic rather than logical' (p. 426). It is therefore unsurprising that he did not provide a crisp, general definition of the problem of *survival value*. However, Tinbergen's discussion of *survival value*—and particularly the studies he presented as good examples of this kind of research (see below)—suggest to me that he was recommending (i) functional analysis, (ii) of small components, (iii) using experimental intervention. Furthermore, Tinbergen's examples suggest that, in spite of the label 'survival value', he was recommending functional analysis at multiple levels, not specifically in relation to survival. Most of his examples examine the role of a trait in specific feeding or predator avoidance systems; they do not attempt to trace its effects all the way up to longevity or reproductive fitness.

Functional analysis views a structure or process (e.g. eggshell removal in black-headed gulls) as a component in a system, characterizes the capacity of the system (e.g. predator avoidance) and identifies the function of the component with those of its effects (e.g. reducing nest detection by carrion crows) that contribute to the capacity of the system [64]. It is the strategy typically used to identify the function of each component in an industrial production line. If the capacity of the line as a whole is to produce jackets, the function of any one person in the line is the contribution she makes to jacket production. She may be doing, or capable of doing, many things—cutting, sewing, suffering, making bread—but her function is whatever she contributes to jacket production—for example, making button holes. A commitment to this kind of functional analysis is suggested not only by Tinbergen's discussion of eggshell removal, but also by his comments on the blackbird's bill: 'one wants to know whether a bill of this size and shape is best suited to feeding in the environment in which the blackbird lives; similarly, one needs to understand in detail the suitability of every aspect of its feeding behaviour' (p. 419).

Turning to small components, most of Tinbergen's examples cast the focus of an investigation of *survival value* as a small cog in a big wheel. Egg-fanning in male sticklebacks renews the water around the eggs, reducing the risk that the eggs will die, but many other processes and structures are required to keep the eggs alive, and egg survival is just one of many stages in the survival of the individuals inside the eggs. Thus, egg-fanning is a relatively simple process that is ascribed a small job in a big enterprise. Philosophers have subsequently stressed the importance of this aspect of functional analysis, suggesting that the explanatory value of assigning a function to a system component increases with the gap in sophistication [64], or 'stupidity' [65], between the component and the system as a whole.

Finally, Tinbergen was very clear about the importance of experimental intervention. He commended studies in which the function of eye spots on moth wings, and spines on sticklebacks, was established by measuring predation rate after researchers had removed these structures, and stated plainly that 'the method to demonstrate survival value of any attribute of an animal is to try whether or not the animal would be worse off if deprived of this attribute' (p. 419).

A great deal has been written in the past 20 years about the function of MNs, but it has not been based on the kind of functional analysis that Tinbergen recommended in his discussion of *survival value*—on functional analysis of small components using experimental intervention. Many claims about the function of MNs have been purely speculative—for example, that they are 'cells that read minds' [66], and 'the neurons that shaped civilization' [67]. Others have drawn on evidence from single-unit recording in monkeys (see §3*a*), and fMRI in humans, but this evidence is correlational. To date, single-unit and brain imaging studies have correlated MN or mirror mechanism activity with external conditions—the kind of action being observed, or the kind of judgement a human subject has been asked to make about an observed action. They have not done the equivalent of removing spines from sticklebacks; intervened to prevent (or enhance) MN activity, and examined the effect of this intervention on the performance of behavioural tasks.

A few studies have attempted this kind of intervention by applying disruptive transcranial magnetic stimulation (TMS) to areas of the human PMC where MNs have been found in monkeys. Their results indicate that disruptive TMS of the PMC can interfere with, for example, detection or discrimination of actions [68–70], judgement of body aesthetics [71], initiation of predictions about ongoing actions [72] and imitation of simple finger and hand actions [73–75]. These studies are of independent interest, and disruptive TMS is the right kind of experimental intervention to use in conjunction with functional analysis. However, for two reasons, as yet these studies fall short of implementing the kind of functional analysis recommended by Tinbergen and endorsed by philosophers as a core biological method. First, there is a major localization problem. Monkey studies suggest that only a small proportion of neurons in PMC are MNs, and MNs have been found in many other areas of monkey and human brains. Therefore, we cannot assume that the effects of TMS to the PMC are due specifically to disruption of MN activity. If our TMS studies were investigating the function of spines in sticklebacks, we would be removing some fraction of the spines along with a fair sized chunk of the fins.

Second, and yet more important, research on MN function has not been guided by the sort of theory that functional analysis requires. It requires theories in which MNs (or a subset of MNs, circumscribed anatomically or by their field properties [58]) are located on a 'production line'; viewed as one, small component of a system defined by its outcome or typical effect, and in which the role of MNs is clearly distinguished from, and related to, the roles of other components. System-level theories of this kind have been used with considerable success in the cognitive neuroscience of reading [76]. For example, they have allowed researchers to postulate that, in relation to reading (the system-level outcome), certain neurons in the fusiform gyrus (a small component) have the function of detecting visual word forms, and to explain how the function of these neurons differs from the functions of neurons that precede (low-level visual analysis) and follow (phonological and semantic processing) them in the 'production line' [77]. By contrast, most research on the function of MNs has been guided by an idea that does not lend itself to functional analysis because it ascribes to the focal component, MNs, a function so big, 'action understanding', that it could easily describe the outcome of the whole social cognitive system—from low-level visual analysis of body movements, through language comprehension, to advanced theory of mind. The term 'action

understanding' implies an encompassing achievement, more like 'reading' than 'detecting visual word forms'; something that whole, complex animals—rather than one type of neuron—is able to do.

Recent discussions of the action understanding hypothesis suggest that MNs mediate one kind of action understanding—'understanding from the inside as a motor possibility'—and acknowledge other 'visual' and 'semantic' types of action understanding [10]. This is certainly an advance, but functional analysis requires theories that postulate pathways of interacting processes, rather than classifications of fixed types. For the kind of analysis Tinbergen recommended, at minimum, we would need system-level theories explaining the causal relations between visual action understanding, semantic action understanding and understanding 'from the inside', and how they together produce the action understanding manifest in effective social behaviour. However, even this may not be enough. As long as the putative function of MNs is described in a 'big' way—in terms of 'action understanding'—rather than a 'small' way—for example, as visual activation of correlated motor programmes—it is likely to be hard to get beyond explanations like: 'the monkey was able to understand the human's action because it has MNs that understand action'. As Sober has pointed out, this kind of 'Chinese box' or 'Russian doll' explanation can have some value in science; it is not inevitably as empty as the suggestion, in Moliere's joke, that a sleeping potion engenders sleep because it has 'dormative virtue' [78]. For example, if we want to know why a particular animal is able to digest certain nutrients, it can be informative to learn that the creature is host to parasites that can digest these nutrients. However, if we want to know about the nature of digestion, about the kind of processes that constitute digestion, it is of little value to be told that the locus of digestion is, strictly speaking, the parasite rather than the host [78]. Similarly, if we want to know about the nature of action understanding, about the cognitive and neural processes that constitute action understanding, it is not very helpful to be told simply that the job is being done by MNs.

4. Two accounts and four problems: what next?

In the foregoing discussion of Tinbergen's four problems, I pointed out there has been plenty of research on the *causation* of MNs and, in humans, but not in monkeys, on their *ontogeny*. By contrast, although the *evolution* of MNs has been a topic of lively discussion, their phylogeny and selection have not been investigated in a systematic way. Similarly, although there have been many studies seeking to cast light on the function of MNs, these studies have not involved the kind of functional analysis that, I argue, Tinbergen was recommending in his discussion of *survival value*.

What does this summary imply about priorities for future research on MNs? It might be taken to indicate that we should simply fill the empty boxes, prioritizing research on *ontogeny* in monkeys, on *evolution*—both phylogeny and selection—and on the *survival value* of MNs. But that would, I think, miss the point of the contrast between the genetic and associative accounts. *Ontogeny* is an important problem from all perspectives—genetic, associative and hybrid—and therefore all would suggest that studies distinguishing the impact of sensory, motor and sensorimotor experience on the development of MNs in monkeys are overdue. However, if the associative account is correct, then functional analysis of MNs is likely to prove much more rewarding than research on the *evolution* of MNs. The associative account acknowledges that, like all phenotypic traits, MNs have an evolutionary history. However, it suggests that if we go looking for that history, using the methods prescribed by Tinbergen for the investigation of phylogeny and selection, we will find only the evolutionary history of visuomotor control in primates, and of associative learning in all vertebrates; that there is no point in the primate lineage, or elsewhere in the tree of life, where MNs evolved by gene-based natural selection. To return to an earlier analogy, the associative account suggests that it would be no more productive to investigate the genetic (rather than cultural) evolution of MNs than to investigate the genetic (rather than the cultural) evolution of neurons that detect visual word forms [79]. We know that reading 'recycles' evolved capacities for object recognition [77], in much the same way as MNs 'recycle' capacities that evolved for visuomotor control and associative learning, but reading is too young—print was invented too recently—to have a distinctive genetic evolutionary history. We do not know the age of MNs—for example, when they first began reliably to develop in the course of human ontogeny—but the associative account suggests that, if MNs have an evolutionary history of their own, it is a history based on cultural rather than genetic inheritance.

Although research on the genetic *evolution* of MNs is unlikely to be fruitful, functional analysis of MNs—of the kind I believe to have been recommended by Tinbergen in his discussion of *survival value*—could be very exciting indeed. It could tell us about the nature of 'action understanding'; about the interacting processes, each of them simple, that together constitute 'action understanding'. A close reading of Tinbergen brings functional analysis—in relation to *survival value* and to more proximate system properties—out from the shadow of *evolution*, and, independently but in a similar way, the associative account distinguishes questions about the function of MNs from questions about their origins [5]. Therefore, in combination, Tinbergen's discussion of *survival value* and the associative account suggest that functional analysis of MNs should be a high priority for future research. They also offer some pointers to the kind of functional analysis required. At the conceptual level, we need system-level theories; theories in which MNs are assigned a small (but potentially very important) role—such as 'visual activation of correlated motor programmes'—in a production line that yields a high-level capacity such as 'action understanding'. (Tinbergen's category label, *survival value*, suggests that the system-level theories need to encompass the even higher-level capacity to stay alive, but his examples do not support this reading.) At the empirical level, we need to be able to test these theories not only with correlational methods, of the kind currently used in research on MN *causation*, but also using experimental interventions.

Intervention studies with human subjects have been obstructed by a localization problem. Techniques such as multivariate pattern analysis, TMS adaptation and fMRI repetition suppression [80–82] hold some promise as means of overcoming this problem. However, alongside the development of these techniques for use with human participants, it would be valuable to conduct intervention studies in non-human animals. These would ask, for example, whether animals with MNs for actions A and B are better than conspecifics who lack these MNs at behavioural discrimination of

A and B, or at imitating A and B? It has been assumed that research of this kind is impractical because it would have to involve monkeys, which are demanding and expensive laboratory animals, and that between-group variation in MN activity would have to be induced via lesions or disruptive TMS. However, the associative account suggests that, in the long term, it may be possible to overcome these problems using a rodent model and sensorimotor training to induce between-group variation in the number and type of MNs present in rodent brains. Rodents are likely to have the potential to develop MNs because they are capable of associative learning. They may or may not receive in the course of typical development the sensorimotor experience necessary to realize this potential, but, in either case, it could be provided by laboratory-based sensorimotor training.

The broadest implication of the associative account, applying to all four of Tinbergen's problems, is that research on MNs should pay more heed to developmental history. If MNs were a genetic adaptation, then there is a fighting chance that their properties would be relatively invariant across developmental environments. Consequently, it would be possible to make valid inferences about species-typical properties of MNs based on a relatively small and developmentally atypical sample of individuals. If MNs are instead a product of associative learning, then this kind of inference is not valid. Whether or not an individual has MNs, which actions are encoded by their MNs, and at what level of abstraction, will all depend on the types of sensorimotor experience received by the individual in the course of their development. Therefore, the associative account suggests that it is crucial for studies of laboratory monkeys to report and control the animals' developmental history; the kinds of sensorimotor experience to which they have been exposed. It also suggests that, if we want to know the species-typical properties of monkey MNs, it will be necessary to test monkeys that have received all and only the types of sensorimotor experience typically available to them under free-living conditions. Similarly, we cannot assume that the mirror mechanisms found in the members of one human culture are representative of the whole human species. With its emphasis on the role of social practices—such as the imitation of infants by adults, sports and dance training, and mirror self-observation—in driving the development of MNs, the associative account provides specific, theory-driven motivation for cross-cultural studies of mirroring.

Finally, I should say something about a phrase I have used more than once in this article: 'if the associative account is correct'. In §3*a*,*b*, I suggested that the associative account provides a better fit with the current data than the genetic account and hybrid models. I argued that it provides a more 'straightforward', or economical, explanation for the range of MN properties found in research on *causation* (e.g. coding both 'goals' and lower-level features of action; logically related, audiovisual and tool-use MNs), and for the impressive degree of plasticity found in research on *ontogeny* (e.g. the speed with which sensorimotor training produces counter-mirror effects). I hope it is clear that these arguments are based not on deductive but on abductive inference—or 'inference to the best explanation'—the kind of inference that many philosophers regard as the cornerstone of explanation in everyday life and in science [83,84].

In abductive inference, the conclusion does not follow logically from the premises/evidence, or, to put it another way, the evidence does not guarantee the truth of the conclusion. If I note a person on the street who is unshaven, wearing very old clothes and drinking cider in the morning, I may infer abductively that he is homeless—the homelessness hypothesis is the best explanation for the data—but in principle, this inference could be wrong. The cider drinker could be an investigative reporter, or a researcher, pretending to be a homeless person, and if he gives me his card—new evidence—the 'best explanation' could change. Similarly, I am not suggesting that the range of MN properties, or their plasticity, *guarantees* that the associative account is correct. It is possible, in principle, that MNs could be highly responsive to change through sensorimotor learning *and* a specific genetic adaptation for action understanding or another social cognitive function [59]. However, like Tinbergen (see §3*b*), many contemporary behavioural biologists believe that this combination is unlikely; that genetic adaptations are typically protected or 'buffered' against environmental perturbations that were present when the trait evolved and could interfere with their adaptive function [31–33]. Therefore, unless or until we find evidence that MNs embody this combination—for example, unambiguous evidence that MNs for a range of actions develop before infants have had the opportunity for sensorimotor learning relating to those actions—the associative account seems to offer, in the light of Tinbergen's four problems, the 'best explanation' of the origin of MNs.

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