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Social plasticity in fish: integrating mechanisms and function

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Social plasticity is a ubiquitous feature of animal behaviour. Animals must adjust the expression of their social behaviour to the nuances of daily social life and to the transitions between life-history stages, and the ability to do so affects their Darwinian fitness. Here, an integrative framework is proposed for understanding the proximate mechanisms and ultimate consequences of social plasticity. According to this framework, social plasticity is achieved by rewiring or by biochemically switching nodes of the neural network underlying social behaviour in response to perceived social information. Therefore, at the molecular level, it depends on the social regulation of gene expression, so that different brain genomic and epigenetic states correspond to different behavioural responses and the switches between states are orchestrated by signalling pathways that interface the social environment and the genotype. At the evolutionary scale, social plasticity can be seen as an adaptive trait that can be under positive selection when changes in the environment outpace the rate of genetic evolutionary change. In cases when social plasticity is too costly or incomplete, behavioural consistency can emerge by directional selection that recruits gene modules corresponding to favoured behavioural states in that environment. As a result of this integrative approach, how knowledge of the proximate mechanisms underlying social plasticity is crucial to understanding its costs, limits and evolutionary consequences is shown, thereby highlighting the fact that proximate mechanisms contribute to the dynamics of selection. The role of teleosts as a premier model to study social plasticity is also highlighted, given the diversity and plasticity that this group exhibits in terms of social behaviour. Finally, the proposed integrative framework to social plasticity also illustrates how reciprocal causation analysis of biological phenomena (*i.e.* considering the interaction between proximate factors and evolutionary explanations) can be a more useful approach than the traditional proximate–ultimate dichotomy, according to which evolutionary processes can be understood without knowledge on proximate causes, thereby black-boxing developmental and physiological mechanisms.

Key words: cognitive appraisal; immediate early genes; social behaviour network; social learning; transcriptome.

INTRODUCTION

Social living organisms have to closely monitor their social environment and fine-tune their behaviour according to previous social experience and social context (available public information), in order to avoid the costs of engaging in social

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interactions or being ejected from their social groups (Oliveira, 2009). This ability of animals to regulate the expression of their social behaviour in order to optimize their social relationships (social plasticity) should be viewed as a performance trait (Arnold, 1983; Irschick *et al*., 2008) that affects the Darwinian fitness of the animal (Oliveira, 2009). In terms of behavioural mechanisms, social competence relies on behavioural plasticity at different temporal scales: (1) life-cycle staging or developmental plasticity, when plasticity is seasonally cyclic, such as changes in behaviour between different life-history stages (*e.g*. breeding *v*. non-breeding) and (2) behavioural flexibility, when behavioural change occurs in the short term (*i.e.* within the same life-history stage) and is reversible (Piersma & Drent, 2003; Kappeler & Kraus, 2010). In this article, the terms behavioural flexibility or social flexibility will be used when referring to short-term labile social behaviour changes, and developmental plasticity when specifically addressing long-term irreversible changes in social behaviour. Whenever the term social plasticity is used, it refers to adaptive changes in the expression of social behaviour at both time scales.

Despite its biological relevance, the study of social plasticity has been neglected and only in recent years has it attracted the attention of behavioural ecologists (Smiseth *et al*., 2008; Dingemanse *et al*., 2010, 2012; Westneat *et al*., 2011). There are important evolutionary implications, however, of social phenotypic plasticity: (1) plasticity can be seen as a constraint that slows down evolution (Pigliucci, 2005), (2) it can give rise to directional selection if plasticity is too costly or incomplete (DeWitt *et al*., 1998; Price *et al*., 2003), (3) plasticity in itself can be seen as a trait that can be under positive selection in heterogeneous environments, where direct genetic control over the phenotype is outpaced by the rate of environmental change (West-Eberhard, 1989; DeWitt *et al*., 1998; Pigliucci & Hayden, 2001; Price *et al*., 2003; Pigliucci, 2005) and (4) given the singular role of the nervous system in orchestrating flexible responses to cues that signal environmental change, the understanding of the ultimate and proximate mechanisms of social plasticity is crucial for understanding behaviour and brain evolution (*e.g*. social brain hypothesis; Dunbar & Shultz, 2007).

Here, the development of an integrative approach is proposed for understanding social plasticity that integrates the study of proximate (gene modules, hormones and neural circuits) and ultimate (evolutionary consequences) mechanisms, and teleosts are presented as a premier vertebrate group to address ‘why’ and ‘how’ questions regarding social plasticity across different levels of biological organization.

AN INTEGRATIVE CONCEPTUAL FRAMEWORK FOR STUDYING SOCIAL PLASTICITY

The integrative approach proposed here is based on the emerging idea that in order to understand behaviour it is necessary to integrate the study of mechanisms (*i.e.* behavioural neuroscience) into the study of function (*i.e*. behavioural ecology), so that functional explanations of behaviour do not assume a phenotypic space with unlimited degrees of freedom and that the evolution of simple rules that must govern adaptive behaviour is understood. This ‘evo-mecho’ framework (McNamara & Houston, 2009) to social plasticity has the following premises (Fig. 1): (1) In order to adjust the expression of behaviour to changes in the social environment, animals have

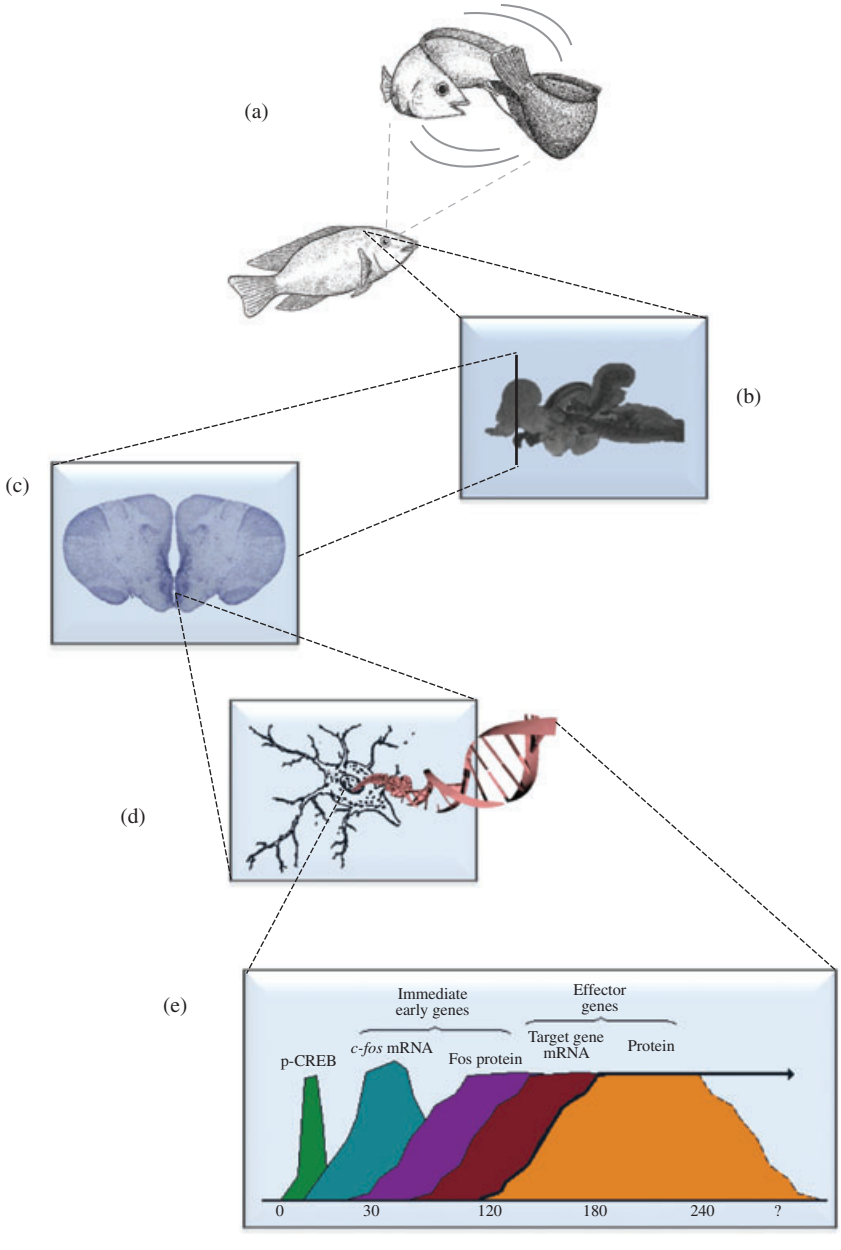

FIG. 1. Mechanisms of social plasticity: (a) social living animals adjust the expression of their behaviour to social information collected in previous social interactions or by observing others; (b) the cognitive appraisal of this information allows them to evaluate the stimulus or event in terms of its valence and salience that will be encoded in a distributed neural network; (c) at each node of this network; (d) neurons will change their neurogenomic state, *i.e.* their gene expression profile in response to the perceived social information; and (e) changes of gene expression are triggered by the activation of neuronal activity-regulated transcription factors (*e.g.* p-cAMP response element-binding) that regulate immediate early genes (*e.g. c-fos*) which can regulate synaptic proteins (e), thereby modulating neural plasticity that underlies behavioural flexibility.

to constantly monitor the environment and collect relevant social cues from direct interactions with other animals or from public sources of information (*i.e.* social learning; Galef & Laland, 2005). Given the wide array of social signals conveyed in multiple sensory modalities, it is postulated that a general appraisal mechanism that assesses the valence and salience of social stimuli across different sensory modalities and functional domains must operate (Paul *et al.*, 2005; Mendl *et al.*, 2010). Thus, the cognitive appraisal of social information collected either directly or indirectly will determine the expression of the appropriate behaviour, given the perceived properties of the social stimulus. (2) Cognitive appraisal of social stimuli triggers neuronal activity-dependent mechanisms at the molecular and cellular level that result in different forms of neural plasticity depending on the duration of the exposure to relevant social signals. In the short term, transient socially driven neuroplasticity can be achieved by three different neuronal activity-dependent mechanisms (Aubin-Horth & Renn, 2009; Wolf & Linden, 2012): (a) activation (*e.g.* phosphorylation) of proteins that act as transcription factors [*e.g.* cyclic adenosine monophosphate (cAMP) response element-binding (CREB)] for immediate early genes (IEG) or for delayed response genes or regulate intracellular signalling pathways [*e.g.* mitogen-activated protein kinases (MAPK) pathway], (b) neuronal activity-dependent transcription factors (*e.g.* pCREB) activate IEGs that can encode other transcription factors (*e.g.* c-fos and egr-1) or synaptic proteins (Arc and Homer1a), hence acting as neuromolecular switches that change the expression of co-regulated gene sets in the brain and (c) transcription of microRNAs that regulate translation of synaptic proteins (*e.g.* miR-134). At the long term, socially driven long-lasting changes in social behaviour or plasticity rely on epigenetic modifications (*e.g.* DNA methylation and histone modifications) of genes involved in social behaviour [*e.g.* oxytocin (OT) and vasopressin (AVP)] or neural plasticity (*e.g.* *bdnf* and *npas4*) (Champagne & Curley, 2005; Szyf *et al.*, 2008; Curley *et al.*, 2011). Together, these neuronal activity-dependent mechanisms change the neurogenomic state of the brain in response to perceived social stimuli. (3) Neural circuits underlying behaviour are composed of a network of brain nuclei with reciprocal connections between each pair (the brain social behaviour network, SBN; Newman, 1999; Goodson, 2005) that encodes information in a distributed and dynamic fashion, such that the expression of a given behaviour is better reflected by the overall profile of activation across the different loci in the network than by the activity of a single node. Different combinations of activation across nodes, and variation in the strength of the connections among them, will generate an almost infinite variation in social behaviour. Therefore, the changes in neurogenomic states mentioned in the previous point can occur differentially at each of the nodes of the SBN, and social plasticity relies both on temporal and spatial changes in gene regulation in the neural networks. (4) Hormones (*i.e.* sex steroids and glucocorticoids) and neuromodulators (*i.e.* neuropeptides and amines) can change the weight of each node of the SBN, and the strength of the connectivity between them, allowing the integration of global organismic state in social decision-making and co-ordinating brain–body responses to changes in the social environment and to transitions between life-history stages (Oliveira, 2009). The actions of hormones and neuromodulators may occur at different frameworks depending on the receptors used to translate the signal. For example, steroids can act as transcription factors by binding to nuclear receptors or have more rapid non-genomic effects on the cell by acting on membrane receptors (McEwen, 1991; Moore & Evans, 1999). (5) The

molecular, neural and hormonal mechanisms mentioned above allow the animal to adjust its behavioural output according to the perceived social environment, resulting in transient (*i.e.* behavioural flexibility) or long-lasting (*i.e.* behavioural consistency, 'animal personalities'; Dingemanse *et al.*, 2010) changes in social behaviour (social plasticity). (6) The organism's ability to switch between social phenotypes in order to optimize its social relationships will increase its Darwinian fitness and can be seen as an organismal performance trait. If there is genetic variation underlying social plasticity, it can come under directional selection and evolve within limits imposed by costs and constraints.

According to this framework, social plasticity is achieved by rewiring or by biochemically switching nodes of the neural network underlying social behaviour in response to perceived social information. Therefore, at the molecular level, it depends on the social regulation of gene expression, so that different neurogenomic states correspond to different behavioural responses and the switches between states are orchestrated by signalling pathways that interface the social environment and the genotype. At the evolutionary scale, social plasticity can be seen as an adaptive trait that can be under positive selection when changes in the environment outpace the rate of genetic evolutionary change. In cases when social plasticity is too costly or incomplete, behavioural consistency can emerge by directional selection that recruits gene modules corresponding to favoured behavioural states in that environment. Therefore, this framework also provides a unifying theory to explain social diversity across different biological levels: social diversity may occur within species, if the weights of each node in the network have a genetic and epigenetic component, giving rise to different social phenotypes, or between species, if the weighting changes with evolution.

TELEOSTS AS VERTEBRATE MODEL SYSTEMS FOR THE STUDY OF SOCIAL PLASTICITY

Krogh (1929) achieved great popularity in the biological literature by highlighting the importance of choosing the right organism in the design of biological experiments. His famous statement that 'for a large number of problems there will be some animal of choice, or a few such animals, on which it can be most conveniently studied' became subsequently known as the Krogh principle (Krebs, 1975). Thus, ideally the choice of the study organism should be based not only on its availability to the researcher (*e.g.* classic model organisms such as mice *Mus musculus*, the fruit fly *Drosophila melanogaster* and nematode *Caenorhabditis elegans*), but rather on its properties that will facilitate the understanding of the biological process of interest. The application of Krogh's principle to the study of social plasticity rapidly leads to the identification of teleosts as the most promising model systems among vertebrates.

Within vertebrates, teleosts are the most diverse and plastic taxa in terms of social behaviour. With over 29 000 species described so far, all the different types of social organization, mating systems and parental care types (Froese & Pauly, 2012) can be found, and it is relatively common to find variation of these characters within closely related groups of species which offers a unique opportunity for comparative studies on the evolution of social behaviour (*e.g.* variation in mating systems and parental care type in African cichlids; Kornfield & Smith, 2000). Fishes are also champions of behavioural and phenotypic plasticity, as can be illustrated by the flexible

patterns of sexual expression, as in the case of protandrous and protogynous sex change, simultaneous hermaphroditism and intra-sexual variation in the form of discrete alternative male phenotypes (Oliveira *et al*., 2005*a*; Taborsky, 2008; Desjardins & Fernald, 2009; Godwin, 2010). Furthermore, highly social species with complex social behaviour that requires advanced cognitive abilities have also evolved in fishes (*e.g.* transitive inference in social interactions; Grosenick *et al*., 2007). A particularly well-studied case is the cleaning mutualism between obligatory bluestreak cleaner wrasse *Labroides dimidiatus* (Valenciennes 1839) and the so-called client fish, in which cleaners present an array of behaviours, including categorization, deception, punishment, reconciliation, partner choice, manipulation and social prestige, that in primates have been seen as indicators of Machiavellian intelligence (Bshary *et al*., 2002; Bshary, 2011). Such cognitive abilities most probably underlie socially driven plasticity in fish social behaviour. Prior experience effects on social behaviour are one of the most studied examples of social plasticity. The outcome of social interactions influences the expression of subsequent social behaviour, so that individuals that win an interaction increase their probability of winning a subsequent interaction and *vice versa* for losers (Oliveira *et al*., 2009). These winner and loser effects are widespread across different animal taxa, including many fish species, with the magnitude of loser effects being, in general, higher than that of winner effects and frequently lasting longer (Hsu *et al*., 2006; Rutte *et al*., 2006). Losing effects can be so profound that in a recent study in *D. melanogaster* a single loss was shown to overcome the effects of artificial selection for aggressiveness (Penn *et al*., 2010). In parallel to winning and losing effects, prior experience can also promote the occurrence of redirected aggression, a phenomenon usually ascribed to primates that has also been described in fishes (Øverli *et al*., 2004). On the other hand, animals also modify their social behaviour depending on social context, such as the presence of an audience, observing third-party interactions (bystander effect) or the familiarity with the opponent (dear enemy effects), and all these contextual effects on behaviour have been shown to be mediated by socially modulated changes in neuroendocrine function (Oliveira *et al*., 2001; Aires *et al*., 2004; Dzieweczynski *et al*., 2006).

In parallel with the above-mentioned behavioural characteristics that make them targets for research on social plasticity, their use for studies that aim to uncover general mechanisms underlying social plasticity is also enhanced by the fact that the neural and gene networks involved in social behaviour seem to be significantly conserved across vertebrates (O'Connell & Hofmann, 2011, 2012). For example, despite the divergent brain development programmes between actinopterygians and other vertebrates, where in the former group the pallial regions undergo an incomplete eversion, contrary to the inversion process observed in those of the latter group (Striedter & Northcutt, 2006), the brains of both groups present a high degree of functional homology (Wullimann & Mueller, 2004; Broglio *et al*., 2005). Similarly, despite the fish-specific genome duplication event observed early in the radiation of actinopterygians (Meyer & Van de Peer, 2005), neurochemical genes relevant for social behaviour [*e.g.* sex steroid receptors, genes of the AVP-like nonapeptides and their receptors and genes of the dopaminergic (DA) system] and their regional expression across brain areas relevant for social behaviour are both well conserved (O'Connell & Hofmann, 2011, 2012).

Finally, a number of social fish species exhibit a set of characteristics that make them interesting model systems: (1) they are in general easier to breed and keep in the

laboratory than other vertebrate animal models, (2) the production of large numbers of offspring, a short inter-generation time and their small size that allows to keep large numbers in captivity and to have many replicates in a research area where the social group, rather than the individuals is often the sampling unit and (3) their social systems can be easily replicated and manipulated in captivity (*e.g.* inducing social status reversals by manipulating group composition), so that more naturalistic social settings are achieved and social behaviour is more readily expressed.

There are, however, still some limitations to the use of fish models in social plasticity research, mainly related to the status of current technology. Namely, neuroanatomy and genetic tools are not readily available for non-model organisms that display relevant patterns of social plasticity (*e.g.* species with alternative behavioural tactics), and the use of brain imaging techniques (*e.g.* calcium imaging and magnetic resonance imaging), although becoming available in fishes, still implies the physical restriction of the focal individuals (Van der Linden *et al.*, 2004; Ahrens *et al.*, 2012). Nevertheless, for a smaller number of teleost model organisms [*e.g.* zebrafish *Danio rerio* (Hamilton 1822) and medaka *Oryzias latipes* (Temminck & Schlegel 1846)] a large number of genetic tools and resources are becoming available, ranging from commercial genome microarrays and chromatin immunoprecipitation (CHIP)-on-chip tiled microarrays to GAL4-UAS (*gal4*-upstream activation sequence) transgenic lines that allow genetic manipulation of specific neural circuits or candidate genes (Muto & Kawakami, 2011), and new comparative genomic tools *e.g.* methods for heterologous microarrays (Machado *et al.*, 2009; RNA sequencing, Wang *et al.*, 2009) are allowing the study of non-classic model systems.

In summary, teleosts offer a singular opportunity to study both proximate mechanisms (*i.e.* genes, neurochemicals and brain circuits) and evolutionary consequences of social plasticity.

COGNITIVE LANDSCAPES OF SOCIAL PLASTICITY: COGNITIVE APPRAISAL AND SOCIAL LEARNING

Social plasticity requires that animals identify and respond to reliable social information that signals changes in the social environment. Social information can be available in multiple sensory channels and can be collected first-hand by directly interacting with other individuals, or by observing other behavioural agents (*i.e.* social learning). Thus, animals have to continuously sense and integrate multiple sensory inputs and extract key characteristics of the social environment from them. Therefore, at the cognitive level, social plasticity relies on some kind of general appraisal mechanism that allows organisms to evaluate the stimuli using a set of stimulus checks to assess its valence and salience in its putative multiple sensory dimensions, in order to determine the appropriate behavioural response (Paul *et al.*, 2005; Mendl *et al.*, 2010). Cognitive appraisal theory proposes that a response to a stimulus is not just a result of direct effects of perceptual information, but rather a function of what that perceptual information means to the organism at that moment of time (Lazarus, 1991; Scherer, 2001). A set of stimulus evaluation checks (SEC) has been proposed (Scherer, 2001) that are also likely to occur in non-humans (Mendl *et al.*, 2010). Examples of these include suddenness, familiarity, predictability, intrinsic pleasantness, discrepancy from expectation and capacity for control (Scherer, 2001; Mendl *et al.*, 2010). Despite the fact that some of these checks have been described

in animals (*e.g*. predictability in fishes; Galhardo *et al*., 2011), a systematic study of SECs and their neural underlying mechanisms in animals is still lacking. According to Lazarus (1991), one of the founders of the appraisal theory, two major types of appraisal occur: (1) primary appraisal, that evaluates the significance of the event to the organism and (2) secondary appraisal, that assesses the ability of the organism for coping with the perceived consequences of the event. These two types of appraisal interact with each other in defining the outcome of appraisal that can be a direct action or a cognitive reappraisal process (Lazarus, 1991). The SECs mentioned above are part of these two processes, with intrinsic valence, novelty (as defined by suddenness, familiarity and predictability) and prediction error related to primary appraisal and controllability to secondary appraisal. In summary, cognitive appraisal is expected to play a key role in the translation of relevant environmental social cues into organismal signals that trigger plasticity.

The study of cognitive appraisal in fishes is still vestigial. One approach that has been used to test the idea that it is the individuals' appraisal of the social event rather than its objective structure that triggers the biological response (Oliveira *et al*., 2005*b*) is to study the response of fishes to mirror-elicited fights. As fishes do not recognize their own image in a mirror, they attack it as if it is an intruder (Rowland, 1999). Mirror fights offer a possibility to study appraisal as there is a decoupling between the expression of behaviour and the behavioural feedback from the interaction that allows the individuals to make an assessment of its outcome. In other words, despite the fact that aggressive behaviour is being expressed at levels similar to those present in real opponent fights (Oliveira *et al*., 2005*b*), the participant does not experience either a win or a defeat in this type of interactions. So, the prediction is that if an assessment of the interaction outcome is needed to trigger a physiological response, then it should not be present in mirror-elicited interactions. On the other hand, if the activation of the biological response is simply triggered by the activation of the behavioural response (*i.e*. expressing fighting behaviour), then it is expected to be present both in real opponent and mirror fights. So far, this hypothesis has been tested in three cichlids and in quail *Coturnix japonica*, with mixed results: hormonal responses to mirror-elicited fights are present in two cichlid species (Desjardins & Fernald, 2010; Dijkstra *et al*., 2012) and absent in the other cichlid (Oliveira *et al*., 2005*b*) as well as in *C. japonica* (Hirschenhauser *et al*., 2008). As there are methodological differences between some of these studies (Oliveira & Canario, 2011), the results are so far inconclusive and further comparative research with standardized methods is needed to clarify this hypothesis. Interestingly, in the anole lizard *Anolis carolinensis*, in which the darkening of postorbital skin eyespots signals sympathetic activation and social dominance, a series of mirror-elicited studies that manipulated eye spot darkening provide evidence that the physiological response of *A. carolinensis* towards mirror images varies with the information content of the image (*i.e*. colour of eyespot; Korzan *et al*., 2000*a*, *b*, 2001). As in these experiments, the painting of the eyespots decoupled the activation of the signal in the focal animal from the observed signal in the mirror image, it allowed the focal animal to assess its fighting ability relative to the different mirror images and to respond accordingly. Therefore, these results can also be interpreted as supporting the role of cognitive appraisal in the activation of physiological responses.

In summary, cognitive appraisal allows the classification of social stimuli regarding their valence, salience and the organism capacity for control, which will reduce the

perception of the complexity of the social environment to key dimensions that will enable the animal to identify ecological opportunities and challenges and to trigger a flexible response by decoupling stimulus and response.

Social environments also offer the possibility to use information that is produced by others (*i.e.* social learning) in order to identify opportunities and challenges that will trigger social plasticity. This allows naive individuals to acquire adaptive information without having to incur in the costs and risks associated with exploring the environment to learn about its contingencies (Dukas, 1999; Galef & Laland, 2005; Burns *et al.*, 2011). Thus, animals living in social groups are expected to have evolved social learning abilities, and these have been widely described in fishes in different evolutionary relevant domains, such as predator evasion (Arai *et al.*, 2007), foraging (Swaney *et al.*, 2001), mate choice (Witte & Nöbel, 2011) or social eavesdropping in territorial systems (Peake & McGregor, 2004; Brown & Laland, 2011). The use of social information can be seen as part of the social plasticity repertoire of social species as there is a trade-off between the use of costly but accurate personal information and the use of cheap but potentially less reliable public information, so that an explanation of social learning should not be invoked indiscriminately. Thus, social learning rules must dictate when to copy others and who to copy (Laland, 2004; Kendal *et al.*, 2009).

Despite the extensive work on ecological and evolutionary aspects of social learning in fishes, its behavioural and neural mechanisms have been much less studied. In an attempt to create a framework for the study of the mechanisms of social learning, a correspondence between different forms of social learning and categories of asocial learning has been proposed: local or stimulus enhancement and single stimulus learning (sensitization or habituation), observational conditioning and Pavlovian conditioning and copying or imitation and operant conditioning, respectively (Heyes, 1994). Unlike asocial learning, however, where the learning signal is the difference between predicted and obtained outcomes (*i.e.* prediction error), social learning cannot be based on directly experienced prediction errors (Burke *et al.*, 2010). Therefore, the mechanisms of observational learning have remained elusive (but a new form of observational prediction error has been proposed recently that would act as a learning signal based on externally observed information; Burke *et al.*, 2010) and are a promising avenue for research.

In summary, cognitive appraisal of social information and social learning are cognitive skills that are expected to play a key role in social plasticity.

NEURAL AND ENDOCRINE LANDSCAPES FOR SOCIAL PLASTICITY: THE SOCIAL DECISION-MAKING NETWORK

At the neural level, social plasticity relies on a social decision-making network that is composed of at least two interconnected neural circuits, that have been conserved across vertebrates as it can be seen from the conserved patterns of expression of developmental genes and neurochemical systems in the telencephalon: (1) the basal forebrain reward system and (2) the SBN (O'Connell & Hofmann, 2011).

A reward system is needed for social decision-making as it enables the evaluation of stimulus valence and salience and the reinforcement of adaptive behaviours through natural rewards that can act either as reinforcers or as hedonic incentives

(Kelley & Berridge, 2002). In mammals, natural rewards are processed by the DA mesolimbic system composed of DA neurons located in the ventral tegmental area (VTA) that project rostrally to the nucleus accumbens, amygdala (AMY) and prefrontal cortex. This mesolimbic reward system has also been subsequently described in birds and reptiles but in anamniotes a midbrain DA population similar to the VTA is missing (Smeets *et al*., 2000). Afterwards, the identification of DA neurons in the ventral diencephalon, in particular in the posterior tuberculum, that project towards the subpallium (Rink & Wullimann, 2001, 2002), raised the hypothesis that this ascending DA pathway might be homologous to the mammalian mesostriatal DA pathway. More recently, however, data on the expression of developmental factors and a more detailed projectome of DA neurons in larval *D. rerio* have made clear that the ventral diencephalic DA groups in *D. rerio* that have ascending projections to the telencephalon (*i.e*. DA groups DC2 and DC4) are specified by a conserved transcriptional network also present in the A11 mammalian diencephalic DA cell group (Lohr *et al*., 2009), but not shared by the midbrain mammalian DA group (*i.e*. A10), and that most subpallial DA inputs originate in a local subpallial DA system that also connects to the ventral telencephalon (Tay *et al*., 2011). Therefore, although the ventral diencephalic DA neurons play a major role in the neuromodulation of behavioural flexibility, they cannot be seen as homologous to the mammalian VTA DA neurons that apparently emerged later in the evolution of DA modulatory systems (Yamamoto & Vernier, 2011).

Newman (1999) originally proposed the existence of the SBN that is involved in the regulation of multiple forms of social behaviour (aggression, affiliation, bonding, parental behaviour and social stress) and includes the extended medial AMY, the lateral septum, the preoptic area, the anterior hypothalamus, the ventromedial hypothalamus and the periaqueductal grey in mammals. The presence of the SBN has been confirmed across all vertebrate classes, using the expression of IEGs as markers of neural activity in relation to the expression of different social behaviours (*e.g*. social communication, aggression and courtship; Goodson, 2005). Other networks for different tasks have also been identified (*e.g*. for stress, for feeding and drinking and for reward) and some neural nodes are shared by more than one core network (Goodson & Kabelik, 2009).

As the expression of a given behaviour is better reflected by the overall profile of activation of the SBN than by the activity of one of its nodes (Crews *et al*., 2009; Goodson & Kabelik, 2009), it is conceivable that different combinations of activation across nodes will be able to produce a wide variation in social behaviour as the weights of each node and of the edges (*i.e*. connections between nodes) in the network may change at different levels: at the individual level, if node or edge weights change temporally; at the intraspecific level, if weights have a genetic and epigenetic component giving rise to different social phenotypes and at the interspecific level, if weighting is changing with evolution (Goodson & Kabelik, 2009). Examples of changes in social behaviour paralleled by changes in the dynamic state of the SBN have started to appear in recent years. In the *A. carolinensis*, repeated exposure to video-playbacks of aggressive displays of conspecific males, that mimic aggressive interactions, led to changes in connectivity within the network (Yang & Wilczynski, 2007), illustrating how social experience can promote temporal changes in the network functionality within the same individual. In estrildid finches, species differences in sociality are also associated with the differential activation of the different nodes

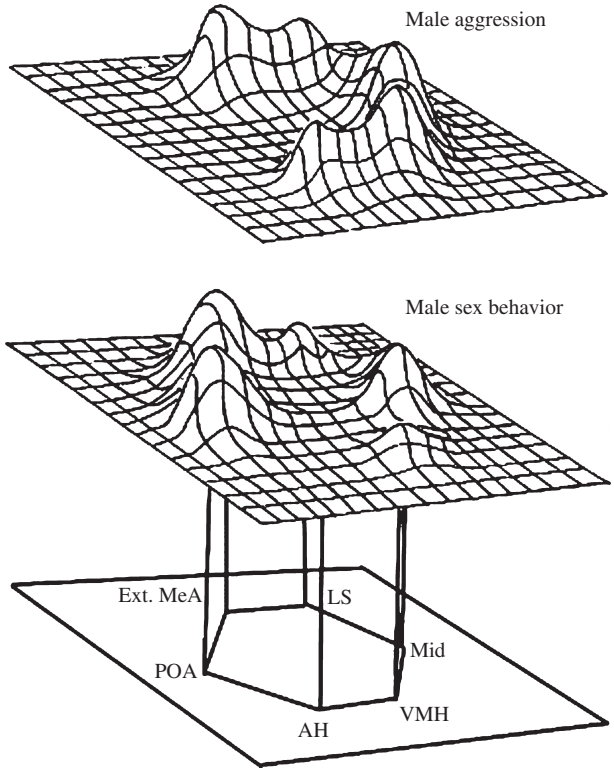

FIG. 2. The social behaviour network is a set of brain areas with reciprocal connections that present different activation states associated with the expression of different social behaviours. Ext. MeA, extended medial amygdala; LS, lateral septum; Mid, central grey in the midbrain; VMH, ventromedial hypothalamus; AH, anterior hypothalamus; POA, preoptic area (adapted with permission from Goodson, 2005).

of the network in response to the presence of a conspecific (Goodson *et al*., 2005; Fig. 2). Therefore, the study of the patterns of activation across the nodes of the SBN is a promising approach for understanding how the nervous system may generate behavioural diversity at different levels, as the almost limitless of combinations generated is a good base for natural and sexual selection to act on.

Hormone and neuromodulator receptors are expressed in all nodes of the SBN (Goodson, 2005; Caldwell & Young, 2006; Skuse & Gallagher, 2009; Munchrath & Hofmann, 2010), which allows the endocrine and neuromodulatory regulation of the activity of the SBN and also of its connectivity by co-regulated expression of receptors across nodes. As a result, peripheral signals that provide information on the internal state of the animal and general brain state signals conveyed by neuromodulators can be integrated in the processes of social decision-making. Thus, hormones and neuromodulators may regulate the expression of behaviour by acting directly on the pattern of activation of the SBN, or by changing the perceptual inputs it receives or the effector outputs it produces (Oliveira, 2005). The major modulators of the SBN are both sex steroids and neuropeptides from the AVP and OT family that have been shown to have extensive effects on social behaviour (Adkins-Regan, 2005). Hormonal effects on the SBN and consequently on behaviour can occur either at

the activational or the organizational level (Arnold, 2009), which reflect two major mechanisms of neural plasticity that mediate changes in network patterns of activity and connectivity across different time scales: structural reorganization of the neural circuits and biochemical switching of neural networks (Zupanc & Lamprecht, 2000). Structural reorganization may be accomplished by different forms of structural modifications that might require adding new cells (neurogenesis) or removing old cells (apoptosis) from the circuit, modifying the connectivity between different components of the network (synaptic plasticity), or changing the responsiveness of the circuit by modifying its molecular components (*e.g.* differential expression of receptors). Biochemical switching mechanisms allow for a variable response of the same neural network under similar stimulation regimes. This is achieved by different neuroactive molecules (neuromodulators and hormones) that interact with the circuit and alter its functional properties, therefore promoting either excitatory or inhibitory states, and resulting in the occurrence of behaviours adapted to a given context (Kravitz, 2000; Libersat & Pflüeger, 2004; Huber, 2005; Balthazart & Ball, 2006). It should also be noted here that constraints and limitations on neural plasticity are also expected. One well-identified trade-off in behavioural decision-making mechanisms is between speed and accuracy, and therefore although neural plasticity allows for increased accuracy, this will limit the speed of social decision-making, especially under noisy conditions (Chittka *et al.*, 2009).

GENOMIC LANDSCAPES FOR SOCIAL PLASTICITY: IMMEDIATE EARLY GENES AND EPIGENOMIC MODIFICATIONS

Information processing in the central nervous system (CNS) occurs at two different time scales. At the scale of milliseconds, excitatory and inhibitory post-synaptic potentials are integrated by neurons to generate (or not) an action potential. Neuronal integration of information at this level mediates immediate behavioural responses to stimuli. The activity of specific neural circuits in response to particular stimuli is followed by a pulse of gene expression, representing a second, much slower level of neural integration, which occurs within minutes [messenger (m)RNA] to hours (protein) of stimulation (Fig. 1), and that is not directed towards an immediate behavioural response but rather towards the modification of the underlying neural circuitry in an experience-dependent fashion (Clayton, 2000). This genomic response is dependent on the activation of intracellular signalling pathways that respond to extracellular signals and change gene expression in an activity-dependent fashion (*e.g.* MAPK cascade; Sweatt, 2004; Thomas & Huganir, 2004). A prominent mechanism that underlies plasticity is the activation of IEG expression by the neuronal activity-dependent phosphorylation of CREB, which acts as an IEG transcription factor (Wolf & Linden, 2012). Therefore, the wave of IEG activation following a stimulus is recruiting temporal correlated associations in neural activity in behavioural significant contexts and promoting the slower alteration of synaptic networks, thereby adjusting the selectivity of long-term information storage and retrieval in neuronal networks (Clayton, 2000; Pinaud & Tremere, 2006). This is achieved by IEG proteins acting either as direct effectors (*e.g.* Arc and Homer) that modify synaptic structure and function, or as transcription factors (*e.g. c-fos, egr-1*, *i.e. ngfI-A*, *zif268*, *krox-24* or *zenk*) that alter the transcription of other target genes encoding downstream

effector proteins (*e.g.* synapsins) (Clayton, 2000; Pinaud & Tremere, 2006). Thus, temporal and spatial variation in gene expression in the brain regulates the remodelling of neural networks that underlie behavioural plasticity. As IEG expression does not require the activation of any preceding gene and because they modify synaptic structure and function, they represent the earliest genomic response to an inducing stimulus that orchestrates integrated genomic responses to social information. Some IEGs have been shown to be activated within minutes of exposure to specific social cues and to vary their activation with the valence (appetitive *v.* aversive) and salience (*e.g.* familiarity and complexity) of the social signal (Mello *et al.*, 1992; Jarvis *et al.*, 1998; Dong & Clayton, 2008). The activation of IEGs has been documented in response to a wide range of social stimuli in different species and sensory modalities (*e.g.* songbirds: Mello & Jarvis, 2008; African cichlids: Burmeister & Fernald, 2005; Burmeister *et al.*, 2005 and frogs: Burmeister *et al.*, 2008), confirming their role as neuromolecular switches for the transduction of social information into changes in brain function and behaviour (Robinson *et al.*, 2008).

In response to IEG expression, co-regulated gene sets (neurogenomic states) are then expected to be co-expressed leading to an association between behaviourally driven gene expression and the expression of social phenotypes. For example, males of an African cichlid *Astatotilapia burtoni* (Günther 1894) can change social status within minutes if an opportunity occurs. Social ascending males exhibit *egr*-1 expression in the gonadotropin-releasing hormone (GnRH)-1 neurons of the preoptic area that controls the reproductive axis (Burmeister *et al.*, 2005). Dominant and subordinate males are also characterized by different brain transcriptome profiles (Renn *et al.*, 2008), suggesting that rapid and transient IEG responses to social cues can lead to sustained long-term changes in neurogenomic states leading to subsequent structural and physiological changes that characterize social phenotypes (*e.g.* subordinate *v.* dominant). Thus, social regulation of gene expression in larger gene networks in the brain can be mediated by initial effects of social experience on IEG activation. The stable maintenance of gene expression profiles induced by social experience can be achieved by epigenomic modifications that regulate, in a more-or-less permanent way, the availability of promoter sites to transcription factors, which is accomplished through the physical modification of DNA (*e.g.* cytosine methylation) or its associated proteins (*e.g.* histone acetylation). Many epigenomic modifications are acquired during development and are thought to be permanent and life-long (Champagne & Curley, 2005; Szyf *et al.*, 2008; Curley *et al.*, 2011). There is increasing evidence, however, that these modifications are plastic and that they respond to events in the external environment being involved in synaptic plasticity and memory in the adult brain (Roth & Sweatt, 2009; Day & Sweatt, 2011*a*,*b*; Nelson & Monteggia, 2011). In particular, DNA methylation is highly responsive to environmental variations and thus is a good candidate to act as a long-term mediator of phenotypic plasticity (Aubin-Horth & Renn, 2009). For example, in a recent study, changes in DNA methylation have been characterized in the brains of worker bees *Apis mellifera* performing differentiated behavioural tasks (nursing *v.* foraging) that illustrate the ability of the neural epigenome to respond to the social environment (Lockett *et al.*, 2012).

The use of high-throughput technologies that make possible measuring the expression of many genes simultaneously, shows that social stimuli induce massive changes (*i.e.* hundreds to thousands of differentially expressed genes) in gene expression in

the CNS (Whitfield *et al*., 2003; Kroes *et al*., 2006; Cummings *et al*., 2008; Dong *et al*., 2009; Sen Sarma *et al*., 2009; Wurm *et al*., 2010). This approach has made it clear that social information has broad effects on gene expression and is not restricted to specific genes, and is unravelling how gene regulation underlies social diversity at different levels (*e.g.* differences in transcriptome profiles between social phenotypes and contexts: hive workers *v*. foragers among *A. mellifera*; Whitfield *et al*., 2003: workers *v*. queens among ants *Lasius niger*; Graff *et al*., 2007; dominant *v*. subordinate fishes and mammals; Kroes *et al*., 2006; Renn *et al*., 2008: monogamous *v*. polygynous African cichlids; Machado *et al*., 2009: response to social *v*. sexual stimuli; Cummings *et al*., 2008: territoriality at different life-history stages; Mukai *et al*., 2009). Differential socially driven changes in transcriptome profile across different brain regions have also been reported and add an extra level of complexity and opportunities for research (*e.g*. songbirds; Lovell *et al*., 2008: bee dance; Sen Sarma *et al*., 2009). All these studies have identified complex network of genes whose expression changes in response to socially relevant information. Gene ontology analysis revealed socially regulated genes involved in a multitude of cellular processes including neuronal plasticity, namely genes from the MAPK signalling cascade that are used to control the cellular responses to external signals across different species (Treisman, 1996) and have been co-opted in mature neurons to function in synaptic plasticity (Sweatt, 2001). The fact that protein kinases are responding to social information is quite relevant for phenotypic plasticity because they mediate phosphorylation changes in other proteins, thereby modifying proteins at a post-translational level, which can lead to the development and maintenance of plastic traits (Aubin-Horth & Renn, 2009). A clear limitation of the above-mentioned transcriptome studies is that whole brain samples have been used, which potentially masks variation between functional brain areas, and even when using specific brain areas these are made up of different neuronal populations. Thus, there is a need to get more detailed information on specific brain areas of interest and inside these on different neuronal populations to better characterize the genomic make-up of plastic traits. This limitation is particularly relevant for fishes as detailed information (*e.g.* homologies with mammalian brain areas that request functional information as well as knowledge of connection patterns between areas) on specific brain areas is still incomplete or missing for most species (Wullimann & Mueller, 2004; Nieuwenhuys, 2009).

EVOLUTIONARY AND ECOLOGICAL LANDSCAPES FOR SOCIAL PLASTICITY: ORGANISMAL PERFORMANCE TRAITS AND PHENOTYPIC PLASTICITY

The ‘performance paradigm’, originally proposed by Arnold (1983), assumes that the variation in the ability of organisms to perform ecologically relevant tasks (performance traits), such as sprint speed, biting force or capacity for endurance, emerges from variation in underlying morphological and physiological traits (lower level traits). On the other hand, as organisms interact with the environment through their functional capacities, natural selection is expected to operate primarily on performance traits and only indirectly on lower level traits (Arnold, 1983; Irschick & Garland, 2001; Lailvaux & Irschick, 2006). Hence, alleles with pleiotropic effects

that facilitate the co-ordinated evolution of components of performance traits are expected to be favoured, and the genetic architecture of the subordinate traits involved is expected to evolve to become more consistent with the prevailing patterns of multivariate selection, leading to further evolution and adaptive radiation (Garland & Kelly, 2006). Therefore, performance should be correlated with the Darwinian fitness of the organism (Irschick *et al*., 2008).

One key performance trait is the ability of the organism to change its phenotype in response to changes in the environment by re-programming the genome (*i.e*. phenotypic plasticity; Pigliucci, 2001; West-Eberhard, 2003). The characterization of the change in a genotype's phenotype across an environmental gradient (reaction norm), has unravelled interindividual differences in the degree of plasticity that rely on genetic variation (Pigliucci, 2005). Hence, phenotypic plasticity can evolve within limits imposed by costs and constraints to plasticity (DeWitt *et al*., 1998), if fitness is increased by the changing phenotype. The possibility that selection acts on variation in plasticity, however, has received little attention to date (Pigliucci, 2005; Nussey *et al*., 2007). On the other hand, despite the considerable attention that phenotypic plasticity has received on evolutionary biology, the emphasis has been on permanent environmental effects on morphological and life-history traits during the development of the phenotype rather than on labile plastic traits in the adult life of the organism, such as behaviour (Pigliucci, 2005; Nussey *et al*., 2007).

At the behavioural level, the social domain, owing to its high variability, is expected to be a major selective force for the evolution of behavioural flexibility. It is important to note here that plasticity is only favoured in predictable variable environments where there are reliable cues available to predict the future state of the environment, but not in stochastic environments where bet-hedging strategies are expected (Olofsson *et al*., 2009; Simons, 2011). Animals vary both in the average level of expression of behavioural traits (*i.e.* personality) and in the responsiveness to social stimuli (plasticity). These two contrasting dimensions, individual behavioural consistency and plasticity, were often approached separately by evolutionary biologists but it is now evident that these aspects are complementary and should be studied simultaneously (Dingemanse *et al*., 2010; Wolf *et al*., 2011). Within-species variation in social plasticity has important evolutionary consequences and evolutionary models that integrate individual consistency and plasticity in order to explain the occurrence of behavioural profiles and variance in behavioural plasticity within populations have now been put forward (Wolf *et al*., 2008). In socially more complex and dynamic environments, selection may act to promote more plastic phenotypes, while in more stable environments selection may favour particular values of the behavioural responses when the costs of maintaining flexibility are high.

A conceptual framework for the study of phenotypic behavioural plasticity based on the notion of behavioural reaction norms has been recently proposed (Dingemanse *et al*., 2010). In this approach, the trait of interest for evolution is the behavioural reaction norm itself, *i.e*. the shape of the behavioural response over an environmental gradient and not the average value of the behaviour (Dingemanse *et al*., 2010). The behavioural response to the environmental gradient integrates both individual behavioural consistency (personality) and plasticity (Conrad *et al*., 2011). Assuming a linear relationship between behaviour and environmental cues, differences in the intercept (elevation) of the fitted regression line represents average differences in behaviour (personality) while different slopes represent variation in behavioural

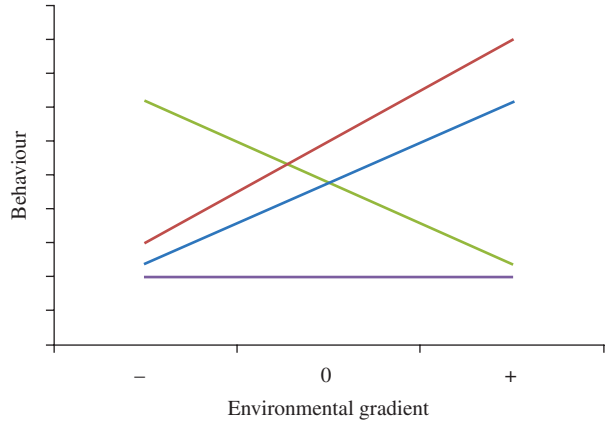

FIG. 3. Hypothetical linear behavioural reaction norms of four different subjects to the same environmental gradient. An example could be the variation in the frequency of aggressive behaviours with social group size. While some animals do not respond to group size increase (subject 4), others increase (subjects 1 and 2) or decrease (subject 3) the frequency of the behaviour. Differences in slopes represent differences in behavioural plasticity. For instance, subjects 1, 2 and 3 are more plastic than subject 4. Differences in the trait mean represent different behavioural profiles. For example, subjects 1, 2 and 3 are, on average, more aggressive than subject 4. ——, subject 1; ——, subject 2; ——, subject 3; ——, subject 4.

plasticity (Fig. 3). Interestingly, certain behavioural types (*i.e.* personalities) are inherently characterized to be more plastic (*e.g.* differences in routine formation and learning between proactiveand reactive coping styles in rodents and fishes; Koolhas *et al.*, 1999; Øverli *et al.*, 2004, 2007), which can be due, for example, to a ceiling effect (*e.g.* bold-aggressive animals are less plastic than shy non-aggressive ones) on the plasticity of their boldness or aggression as they are already closer to the trait maximum and therefore have a lower scope for response (Sih *et al.*, 2004; Sih & Bell, 2008). Importantly, because animals with more plastic behavioural responses in one dimension have been reported to be more plastic in other dimensions (Benus *et al.*, 1990; Branchi, 2009; Curley *et al.*, 2009; Arnold & Taborsky, 2010; Taborsky *et al.*, 2012), molecular mechanisms favouring general behavioural plasticity are predicted. Under the present conceptual framework, interindividual variation in social plasticity would result from variation in the ability to alternate between neurogenomic states in the nodes of the SBN. If this variation has an additive genetic component, social plasticity can be the target of natural selection.

According to the theoretical framework proposed here, intra-individual variation in social plasticity on which selection acts is the outcome of variation in the plasticity of the neurogenomic states that underlie the expression of alternative behavioural states. In that sense, genetic variation affecting the evolution of social plasticity may come: (1) from variation in the coding sequence of structural genes expressed in the gene modules associated with each phenotypic state or (2) from variation in the sequence of regulatory regions (*i.e. cis*-regulatory elements and transcription factors). Interestingly, evidence has been accumulating showing that phenotypic (both morphologic and behavioural) diversification is more dependent on the pleiotropic effects of small regulatory regions than on structural gene modification (Wagner,

2000; Levine & Tjian, 2003; Jovelin *et al*., 2009; Wolf *et al*., 2010). Likewise, regulatory elements are probably fundamental players in orchestrating the switch between gene modules associated with different behaviours, and thus regulatory regions constitute primary candidates for investigating the targets of selection driving phenotypic behavioural diversity.

CONCLUDING REMARKS

The integrative framework for the study of social plasticity presented here is expected to unravel how knowledge of the proximate mechanisms underlying social plasticity is crucial to understanding the costs, limits and evolutionary consequences of social plasticity, thereby highlighting the fact that proximate mechanisms contribute to the dynamics of selection.

The great diversity of social behaviour among fishes that may occur both within and between species and its high plasticity within the same individual place fishes in an outstanding position for the comparative and integrative study of social plasticity. In the near future, careful experimental designs using the right species will certainly allow timely questions in the field to be addressed such as: how do animals perceive social information and how is it translated into a genomic response in the brain? How do perceived changes in social environment trigger adaptive changes in behaviour through changes in gene expression in neural networks underlying social behaviour? Does social plasticity have an additive genetic variation component that allows for its selection?

In a more conceptual view, the integrative framework to social plasticity presented here will provide a comprehensive case study to illustrate how reciprocal causation analysis of biological phenomena (*i.e*. considering the interaction between immediate factors and evolutionary explanations) can be a more useful approach than the traditional proximate–ultimate dichotomy, according to which evolutionary processes can be understood without understanding proximate causes (*i.e*. black-boxing development and physiology).

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