

## Opinion

## Sinking In: The Peripheral Baldwinisation of Human Cognition

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**The Baldwin effect is a hypothetical process in which a learned response to environmental change evolves a genetic basis. Modelling has shown that the Baldwin effect offers a plausible and elegant explanation for the emergence of complex behavioural traits, but there is little direct empirical evidence for its occurrence. We highlight experimental evidence of the Baldwin effect and argue that it acts preferentially on peripheral rather than on central cognitive processes. Careful scrutiny of research on taste-aversion and fear learning, language, and imitation indicates that their efficiency depends on adaptively specialised input and output processes: analogues of scanner and printer interfaces that feed information to core inference processes and structure their behavioural expression.**

## Sinking In

Which features of the human mind, now genetically inherited, were once the products of learning? This question relates to the **Baldwin effect** (see [Glossary](#)) – a process in which an initially learned response to environmental change acquires a genetic basis [1–4]. This process is also known, sometimes in a misleading way [5], as organic selection [1], **genetic assimilation** [6], and experiential canalization [7]. Whatever it is called, Baldwin's hypothesis postulates that, at the population level, learned characteristics can 'sink in'; they can become part of what offspring inherit organically from their parents.

In the following we bring empirical research from cognitive science to bear on the hypothesis that some aspects of human cognition have sunk in – that they were initially learned and are now genetically inherited. We argue that there is good empirical evidence for this hypothesis regarding peripheral cognitive mechanisms (i.e., input or perceptual systems, and output or action systems, both modulated by attention and motivation), but not for the complex and interlocking central processes, involving inference and memory, that have durable effects on the relationships between perception and action [8] ([Figure 1](#), Key Figure).

After introducing the Baldwin effect, we next focus on **preparedness** [9,10]. For 50 years, experimental psychologists have investigated the preparedness of mechanisms involved in learning taste aversions and fears, and how these mechanisms have been specialised by evolution to do their jobs well. We argue that this research not only provides much-needed empirical evidence of Baldwinisation but also illuminates the targets of selection. Specifically, research on preparedness suggests that, across species, Baldwinisation has had a much greater impact on peripheral than on central cognitive mechanisms [11]. There has been genetic assimilation of learning-induced changes in perception, attention, motivation, and motor control – processes that modulate inputs to, and outputs from, the core inference processes of the brain – but little if any change to the structure of central processors themselves. We then extend the analysis to language and imitation, human faculties that are widely thought to have been genetically assimilated. The evidence here

## Highlights

Evolution targets peripheral, not central, aspects of cognition.

Many peripheral mechanisms have evolved via the Baldwin effect.

'Garcia effects' on taste-aversion learning have not withstood the test of time.

Fear learning is 'prepared' by attentional rather than by learning mechanisms.

Specialization for language processing has focussed on perceptuomotor aspects of speech rather than on innate principles of universal grammar.

There is no innate module for imitation but enhanced social motivation consequently leads to greater attention to the behaviour of other agents.

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also suggests that Baldwinisation has targeted peripheral mechanisms – the psychological analogues of keyboards, scanners, speakers, and printers and their associated software – and not the core cognitive processes that make language and imitation possible.

### The Baldwin Effect

Imagine a population of squirrels driven by invaders into terrain where the only edible food is a new type of nut. Although the new nuts are highly nutritious, their shells can be cracked by these squirrels only via a complex, repetitive sequence of biting and hammering. Through luck and desperation some of the squirrels learn this sequence by trial and error. Initially their new nut-cracking behaviour is an **ontogenetic adaptation**. However, if the learning is not too costly, and consumption of the new nuts enhances fitness, there may be enough time for selection to favour any genetic changes that make the learning faster or easier – mutations that enable the nut-cracking behaviour to develop with less environmental input. In that case, and in a range of more complex scenarios, the behaviour would become, at least in part, a **phylogenetic adaptation** – it would be Baldwinised.

The Baldwin hypothesis has been invoked to explain a wide range of behavioural traits across the animal kingdom [12–15], including prey capture in archer fish [16] and the song of zebra finches [17, 18]. In the case of humans, Baldwinisation is thought to have played a key role in the emergence of language [19–24] and imitation [25–28].

A major appeal of the Baldwin hypothesis is that it promises to explain how natural selection finds a ‘good trick’ or needle in a haystack [29] – a highly adaptive phenotype that is too complex to be the product of a single mutation, and too isolated within an **adaptive landscape** to be discovered by an incremental series of mutations (Figure 1) [30]. According to ‘Baldwin boosters’ [31], learning is much more likely than **aplastic evolution** [17] to find isolated spikes in an adaptive landscape because each learner explores throughout its lifetime. The population can search the space of potential solutions more efficiently through learning than through genetic variation. Furthermore, once learning has found a solution, it can help to sustain the adaptive phenotype long enough for it to become progressively fixed, Baldwinised or genetically assimilated, because mutations that reduce the need for environmental input are favoured by natural selection.

Historically, the Baldwin effect was seen by its critics as a Lamarckian threat to the standard, **modern synthesis** view of evolution [32]. It is now clear that Baldwinisation can occur without violation of **Weismann’s doctrine** [33], and the principal problem is empirical. Ironically, the Baldwin ‘effect’ is a hypothesis with few well-validated empirical examples. Verbal reasoning and computational modelling indicate that Baldwinisation offers an elegant and plausible explanation for many morphological and behavioural adaptations [12–15, 19, 20, 23, 24], but in the behavioural case there is very little concrete, empirical evidence of its occurrence [3]. The evolutionary history of most naturally occurring adaptations, ancestral and intermediate forms, cannot be recovered from the mists of time, and artificial selection or **experimental evolution** (e.g., [34, 35]) can be studied only in animals such as *Drosophila* that have very rapid generational turnover.

The explanatory value of the Baldwin hypothesis is also limited by **blackboxing** – failure to enquire about the neurocognitive mechanisms controlling overt behaviour [36, 37]. Baldwin was a psychologist, and in the 125 years since his seminal article [1] experimental psychology and cognitive neuroscience have made significant advances in understanding the computational mechanisms controlling behaviour in human and nonhuman animals. However, very little of this information from cognitive science has penetrated research on Baldwinisation. Modelling suggests that the probability of Baldwinisation depends crucially on ‘details of developmental

### Glossary

**Active intermodal matching (AIM):** a hypothetical mechanism for converting observed actions into a topographically similar motor output via unspecified computations.

**Adaptive landscape:** a space representing potential genotypes, the degree of similarity between them, and their related fitness values.

**Adaptive specialisation:** a trait that is effective in dealing with a problem encountered in the natural environment of a species (e.g., the risk of ingesting toxins).

**Aplastic evolution:** genetic evolution that does not depend on the Baldwin effect.

**Associative learning:** learning based on the establishment of excitatory and inhibitory links between simple, sensorimotor representations of events. Often studied using Pavlovian (classical) and instrumental (operant) conditioning procedures.

**Associative sequence learning (ASL):** a model suggesting that imitation is mediated by learned associations between visual and motor representations of actions (matching vertical associations) and domain-general sequence-learning mechanisms (horizontal processes).

**Baldwin effect:** a process in which an initially learned response to environmental change acquires a genetic basis.

**Blackboxing:** failure to consider underlying mechanisms; in the present context, failure to enquire about neurocognitive mechanisms when investigating behaviour.

**Construction grammar:** an approach to linguistics which proposes that language is built, bottom-up, from layers of local form-meaning mappings, termed constructions, that can be learned independently.

**Evolutionary psychology:** a research programme, founded in the 1990s, that seeks to explain psychological characteristics as the products of gene-based natural selection.

**Experimental evolution:** the use of selective breeding in laboratory and field experiments to investigate how populations adapt to new environments through natural selection.

**Fear-relevant stimuli:** objects and events that were a danger to the evolutionary ancestors of contemporary humans (e.g., snakes and spiders).

processes, such as the psychological mechanisms of learning' [17], but most applications of the Baldwin hypotheses continue to assert that behaviour X has been genetically assimilated without asking what type of learning drove the assimilation of X, or about the neurocognitive processes that were targets of selection.

In short, the Baldwin effect is a promising hypothesis rather than an empirical effect, and, although Baldwin was a psychologist, cognitive science is conspicuously absent from research done in his name. In the next section we address these problems by highlighting experimental evidence that taste-aversion learning has been Baldwinised, and by surveying recent research in cognitive science suggesting that, in both taste-aversion and fear learning, it is changes to peripheral processes that have sunk in.

### Preparedness

In 1966, Garcia and colleagues published experiments suggesting that rats learn some contingencies more readily than others. Relationships between flavours and illness, and between audiovisual stimuli and electric shock, appeared to be learned more readily than relationships between flavours and shock, or audiovisual stimuli and illness [38,39]. Garcia's results were soon dubbed as evidence of 'preparedness' [9], and several psychologists were inspired by them to advance a new **adaptive specialisation** conception of learning [40–42]. Laying the groundwork for human **evolutionary psychology** [43], this new concept challenged the view that all or most learning is mediated by a common set of **associative learning** processes, discovered through conditioning experiments [44]. It suggested instead that animals, including humans, not only have a diverse range of input and output devices but also have many different central mechanisms of learning, each tailored by natural selection to meet specific adaptive challenges. However, a close analysis of the literature on taste aversion and fear reveals that these suggestions were mistaken.

### Taste Aversion

Evidence that taste-aversion learning has been Baldwinised comes from elegant studies of experimental evolution in *Drosophila melanogaster* (see [Figure 1](#) in [Box 1](#)) [34,35]. Female flies first experienced relationships between stimuli (e.g., orange paired with a quinine-adulterated substrate for egg laying; pineapple paired with a safe substrate). They were then allowed to lay eggs on either orange or pineapple substrates without adulteration. Only when subsequent generations were raised exclusively from eggs laid on the previously safe substrate was there an increase in rate of learning flavour–quinine associations. By contrast, control lines of flies, which were treated similarly but without exposure to quinine, did not show changes in learning over generations. After 45 generations, flies from experimentally selected lines associated quinine more rapidly both with orange and pineapple and with the novel flavours, tomato and apple. This is a Baldwin effect: the researchers produced environmental change by adding quinine to substrates; the flies initially responded to this change by learning to avoid flavours encountered with quinine, and over generations of selection this learned response evolved a genetic basis.

However, the *Drosophila* studies do not tell us which part of the learning process acquired a genetic basis: what exactly sunk in. After selection, did the flies learn faster because they were using new computational machinery specifically to encode flavour–quinine relationships (a change in central mechanisms), or were they merely better able to perceive quinine, or to attend to differences between specific types of cue (changes in peripheral mechanisms)? The *Drosophila* experiments did not answer these questions because the flies were exposed to only one type of outcome (quinine) and typically to cues in only one modality (flavour), but studies of taste-aversion learning in rats, using two cue modalities and two outcomes, suggest that the genetic changes altered peripheral rather than central cognitive processes ([Box 1](#)).

**Genetic assimilation:** biologists who do not equate the Baldwin effect with genetic assimilation cast the latter as a process in which environmental change exposes pre-existing genetic variation to selection, in other words a process that is not dependent on learning.

**Horizontal processes:** domain-general psychological mechanisms that encode sequences of body movements and of other inanimate stimuli.

**Matching vertical association:** a bidirectional excitatory link between a visual representation and a motor representation of the same action.

**Modern synthesis:** a reconciliation in the early 20th century of Darwin's theory of evolution and Mendel's observations on heredity.

**Non-matching vertical association:** a bidirectional excitatory link between a visual representation of one action and a motor representation of a different action.

**Ontogenetic adaptation:** a trait promoting survival and reproduction which is acquired through interaction between an individual and its environment in the course of development.

**Phylogenetic adaptation:** a trait promoting survival and reproduction which has been shaped by natural selection acting on genetic variants.

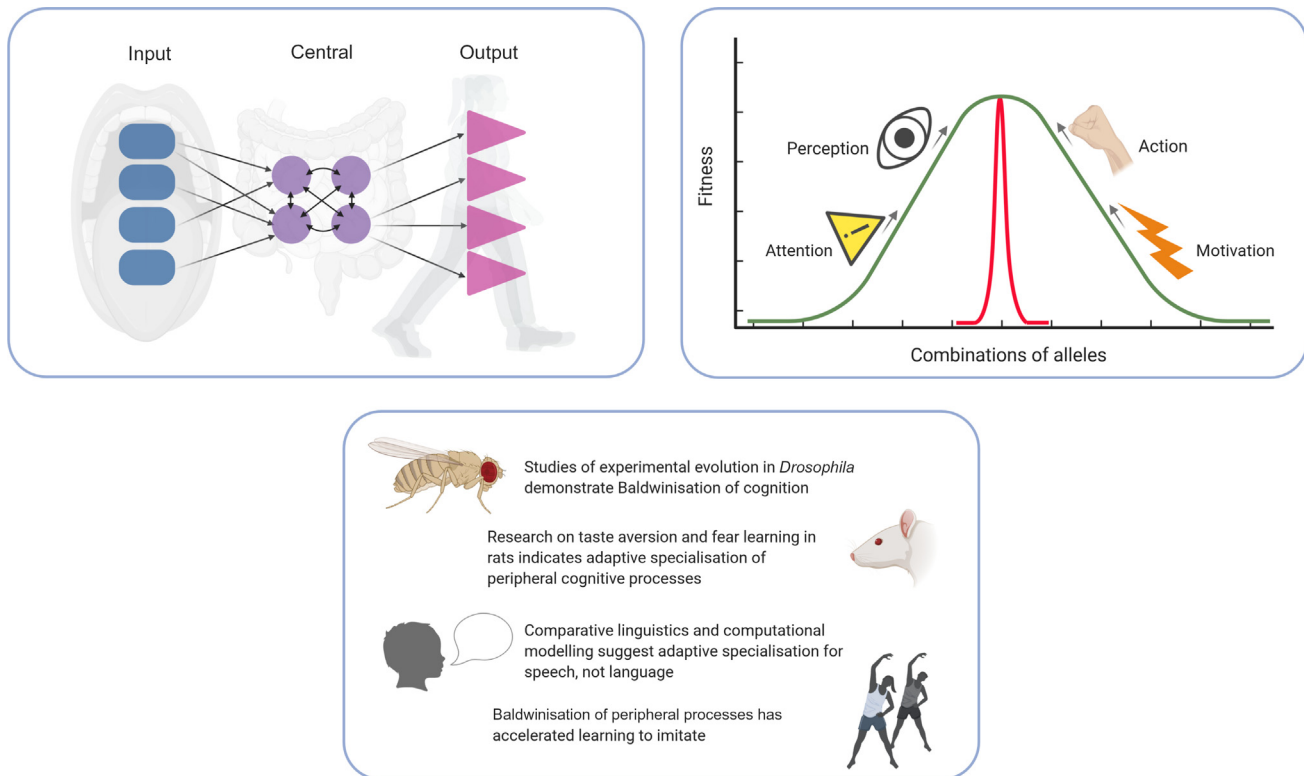
**Preparedness:** a state of a learning process that is thought to indicate that it has been specialised by natural selection to fulfil a particular function.

**Universal grammar (UG):** a hypothetical innate set of abstract grammatical principles that are held to underlie the world's natural languages.

**Weismann's doctrine:** contrary to what is claimed by supporters of Lamarckian inheritance, Weismann's doctrine states that hereditary information does not flow from the soma to the germline.

## Key Figure

## The Peripheral Baldwinisation of Cognition



Trends In Cognitive Sciences

**Figure 1.** (Top left) Generating behaviour involves input, central, and output processes that are analogous to ingestion, digestion, and expenditure of energy. Peripheral processes include the perceptual systems that provide input, the action systems that provide output, and the attentional and motivational processes that modulate both perception and action. Central processes, involving memory and inference, have durable effects on the relationships between inputs and outputs. (Top right) Via the Baldwin effect, learning dramatically reduces the search problem in high dimensional genetic 'fitness landscapes'. Apathetic evolution requires a population to stumble upon good solutions, corresponding to 'spikes' in the landscape (red curve), by chance, for example via mutation. Learning can turn a spike into a gentle hill (green curve); individuals with a wide range of genetic resources, from a variety of genetic starting points, can find progressively better solutions through individual or social learning. The evidence reviewed in this article suggests that peripheral processes (perception, motor, attention, and motivation icons) have evolved via the Baldwin effect. (Bottom) The peripheral Baldwinisation hypothesis is supported by research on experimental evolution in *Drosophila*, studies of taste-aversion and fear learning in rats, and by research on language and imitation in humans.

Garcia and Koelling [38] exposed rats to a compound of a flavour plus audiovisual stimuli before either illness or footshock and reported that the flavour was avoided after illness whereas the audiovisual stimulus was avoided after shock. This result has not withstood the test of time. Subsequent studies have shown that rats avoid flavours paired with shock when the flavour-to-shock interval is lengthened (e.g., [45]), and have no difficulty in learning relationships between non-flavour cues and illness (e.g., [46,47]) (Box 1).

More broadly, recent research suggests that rats learn aversions to flavour and non-flavour stimuli via the same computations, but knowledge that a stimulus is predictive of nausea, and knowledge that it is predictive of pain, is expressed in different behaviours. Pairing palatable flavours with injection of either lithium chloride (LiCl, that induces nausea) or hypertonic saline

(that induces pain) suppresses consumption of flavours at equivalent speeds (e.g., [48]). However, in addition to reduced consumption when the flavours are subsequently presented alone, LiCl-induced nausea results in orofacial reactions indicative of disgust, whereas hypertonic saline-based pain results in freezing indicative of fear. Crucially, these outcome-specific responses are also displayed with non-flavour cues that were previously paired with LiCl or hypertonic saline [49]. Therefore, there is indeed something special about aversion learning. However, the 'specialness' relates, not to connections between tastes and illness, but to the behaviours triggered by knowledge that a stimulus, any stimulus, predicts illness. The motor processes, mechanisms producing a behavioural printout, have been specialised.

### Fear Learning

The concept of preparedness was inspired by Garcia's taste-aversion work, but was first applied by Seligman [9,10] to human learning of fears and phobias. Seligman's account suggested that specialised, central mechanisms of fear learning more readily connect aversive events, such as electric shock, with **fear-relevant stimuli**, such as snakes – which presented genuine threats to our evolutionary ancestors – than with 'fear-irrelevant' stimuli such as geometric shapes or flowers. This account predicts that fear of fear-relevant objects should be learned faster, and be extinguished more slowly when shock no longer occurs, as well as being resistant to top-down modification, for example, by instructions indicating that shocks will not occur.

#### Box 1. Taste-Aversion Learning

Research on rodent taste-aversion learning complements research on the experimental evolution of taste aversion in *Drosophila*. Studies on *Drosophila* provide direct evidence of Baldwinisation, but do not tell us whether central or peripheral processes have sunk in. In a complementary way, rodent studies do not provide direct evidence of Baldwinisation, but indicate that when taste-aversion learning is specialised, via Baldwinian or aplastic evolution, it is peripheral processes that change. Further studies supporting these conclusions are outlined in the following text, together with suggestions about how the two lines of research could be more fully integrated.

Using a procedure similar to that shown in Figure 1, a further study of experimental evolution in *Drosophila* [35] presented colour-flavour compound stimuli and made either the colour or the flavour a reliable predictor of quinine across generations and the basis for egg selection. For example, in a 'colour-reliable, flavour-unreliable' condition, where blue-banana stimuli had been laced with quinine, eggs were allowed to hatch only if they had not been laid on the blue substrate, regardless of the blue substrate's flavour. After 40 generations, colour-reliable/flavour-unreliable lines learned about colour but not flavour cues, whereas colour-unreliable/flavour-reliable lines learned about flavour but not colour cues. This could mean that, through Baldwinisation, successive generations of flies had become better able to associate one type of cue (colour or flavour) with aversive outcomes (a central effect). However, it could instead indicate Baldwinisation of enhanced attention to colour in the colour-reliable lines, and to flavour in the flavour-reliable lines (a peripheral effect).

Turning to rodents, further evidence against the original interpretation of Garcia's results [38,39,119], that posited specialisation of central processes, has come from experiments that do not rely on fluid consumption as the only index of aversion. When suppression of motion, akin to freezing, and avoidance of stimulus location are also used to measure aversion, there is clear evidence of learning when non-flavour cues are paired with lithium-induced nausea (e.g., [46,120]).

These rat studies do not prove a negative; they do not show definitively that central processes have not been specialised. Instead, they show that claims for specialisation of central processes have been based on evidence that is no longer compelling. By contrast, recent work (main text) has provided positive evidence for specialisation of peripheral processes: for both flavour and non-flavour stimuli, illness, and pain elicit very different motor outputs [48,49].

How could these two lines of research, with *Drosophila* and rats, be integrated to provide stronger evidence of the Baldwinisation of central and/or peripheral processes for taste-aversion learning? The rat work suggests that, to find out whether selective breeding leads to Baldwinisation of central or peripheral processes in *Drosophila*, one needs to vary cue type and outcome type within single experiments. For example, one could expose flies to an environment where colour, but not flavour, reliably predicts quinine adulteration at the same time as flavour, but not colour, reliably predicts temperature variation [121]. Only genetic change to central learning mechanisms could explain increased learning across generations about colour when quinine was the outcome and, simultaneously, increased learning about flavour when temperature was the outcome. By contrast, peripheral specialisation would be expected to produce improved learning across generations with both colour and flavour, regardless of which outcome was presented.

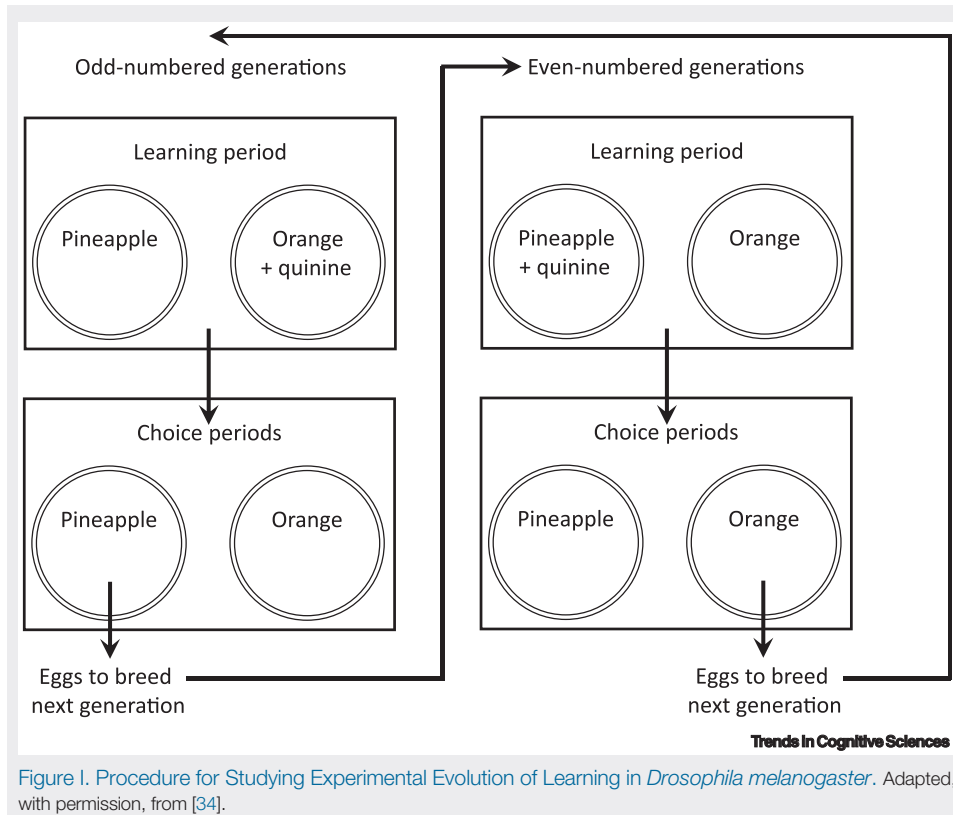


Figure 1. Procedure for Studying Experimental Evolution of Learning in *Drosophila melanogaster*. Adapted, with permission, from [34].

The results of early experiments were consistent with some of these predictions (e.g., [50,51]), but none has withstood extended experimental investigation. Faster or better conditioning with fear-relevant stimuli has rarely been observed, and there is ample evidence that, like most associative learning (e.g., [52]), it can be modified by instruction (reviewed in [53,54]). Initially it seemed that responses to fear-relevant stimuli might extinguish more slowly. However, a recent systematic review [55] found that most positive findings came from a single laboratory, and a large majority of the full set of studies had failed to find differences between fear-relevant and fear-irrelevant stimuli in the rate of extinction.

These results suggest that fear of snakes and other fear-relevant stimuli is learned via the same central mechanisms as fear of arbitrary stimuli. Nevertheless, if that is correct, why do phobias so often relate to objects encountered by our ancestors, such as snakes and spiders, rather than to objects such as guns and electrical sockets that are dangerous now [10]? Because peripheral, attentional mechanisms are tuned to fear-relevant stimuli, all threat stimuli attract attention, but fear-relevant stimuli do so without learning (e.g., [56]). This answer is supported by evidence from conditioning experiments demonstrating enhanced attention to fear-relevant stimuli regardless of learning (Box 2), studies of visual search [57–59], and developmental psychology [60,61]. For example, infants aged 6–9 months show a greater drop in heart rate – indicative of heightened attention rather than fear – when they watch snakes than when they watch elephants [62].

In sum: early research on taste-aversion and fear learning launched the idea that animal minds are populated by adaptively specialised central learning mechanisms – that were later cast by evolutionary psychologists as 'modules'. Over the past 50 years careful experimental work with

### Box 2. Fear-Relevant Stimuli Grab Attention

Early signs that fear-relevant stimuli, such as snakes (Figure 1), are special in the extent to which they grab attention came from human conditioning studies in the 1970s. These showed that, unlike other shapes (excluding faces), which capture attention according to their previously experienced value [122,123], the pulling power of fear-relevant stimuli is experience-independent. Fear-relevant stimuli elicit orienting responses even before they had been paired with electric shock [124], and, after pairing, orienting begins as soon as the fear-relevant stimulus is presented, instead of when the shock is due to occur (e.g., [125,126]).

A classic experiment demonstrated how enhanced attention to fear-relevant cues can give the false impression that these cues are linked to aversive outcomes via a specialised learning mechanism [127]. Lovibond and colleagues compared responses between two pairs of cues that had previously been learned about individually. One pair comprised a fear-relevant and a fear-irrelevant cue (e.g., a snake and a mushroom), the other comprised two fear-irrelevant cues (e.g., a mountain and a flower). In each pair, one cue had been previously linked with shock, and the other had not. Crucially, for half the participants, the fear-relevant cue had been linked with shock, and for the remainder the fear-irrelevant cue had been linked with shock. If fear-relevance produced better learning (i.e., a centrally mediated process), then the response to the test compound of a fear-relevant and a fear-irrelevant cue should be higher than to the test compound of two fear-irrelevant cues only if the fear-relevant cue had been linked with shock. However, if fear-relevant cues are special only in their capacity to capture attention, then the overall response to the compound of a fear-relevant and a fear-irrelevant cue should be higher than to the compound of two fear-irrelevant cues, regardless of whether the fear-relevant cue or the fear-irrelevant cue had been linked with shock. This latter result was observed.

Studies such as this one by Lovibond and colleagues, that tease apart the contributions of attention and learning, use complex experimental designs and are often reported in language that it is difficult for non-specialists to understand (the same is true of experiments on conditioned taste aversion; Box 1). Perhaps that is why many researchers continue to assume that fear-relevant stimuli are processed by specialised central learning mechanisms when the data indicate instead that, owing to specialised peripheral processes, potentially dangerous animals are very good at grabbing our attention [128,129].



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Figure 1. A Western Green Mamba Snake.

rodents, using the original methods, has confirmed the occurrence of adaptive specialisation, and more recent studies on *Drosophila* have shown that it is likely to have occurred via a Baldwinian process. However, the rodent work has also shown that the changes are in peripheral rather than in central cognitive mechanisms.

### Distinctively Human Faculties

Taste-aversion and fear learning occur in a wide range of species and are rarely considered from a Baldwinian perspective. We now turn to two capacities – language and imitation – that are distinctively human, and which are commonly claimed to have evolved through genetic assimilation.

#### Language

Human linguistic behaviour is often viewed as comprising an abstract, modality-independent set of grammatical rules and principles (language), and a complex set of perceptuomotor skills (speech/sign [63]). A Baldwinian account is highly implausible for the former, but highly plausible for the latter.

**Universal grammar (UG)** is a hypothetical innate set of abstract grammatical principles supposed to underlie the world's natural languages [64], variously termed a 'language module' [8], 'organ' [64], or 'instinct' [65]. If some type of UG is part of human biology, how could it evolve? Pinker and Bloom ([24], see also [66]) look to the Baldwin effect: suppose that there are grammatical properties of language that are initially acquired through learning. Such learning will be influenced by genetic bias, and learners with the 'right' bias will be selected. Over many generations, the best learners will be those for whom UG is simply 'built in' to the genes. Learning a specific language then merely involves filling in language-specific details [67], the relevant grammatical parameters, words, idioms, and sounds specific to, for example, English, Hopi, Mandarin, or Xhosa.

The UG picture has, however, collided with several problematic observations: first, close examination of the world's languages suggests that they have few universal features [68]. There are undoubtedly common characteristics that arise from the challenge of robust sequential communication with limited processing resources (e.g., a distinctive level of phonological representation), as well as from the nature of the natural and social worlds (e.g., the ability to refer to people, objects, and actions; to make statements, ask questions, give commands, etc.) [69], but these are implemented in a dazzling variety of ways – diverse phonologies, syntactic and semantic categories, and so on [68,70]. Furthermore, evidence that *FOXP2* is not a 'language gene' [71], that 'specific language impairment' is not specific to language [72], and that Broca's area is not a 'language centre' in the brain – that language processing recruits large areas of the cortex (e.g., [73]) – has undermined the view that there is a dedicated language acquisition device. Many in contemporary linguistics now see language as being built from a **construction grammar**, and regard each language as being assembled, bottom-up, from accumulations of idiosyncratic linguistic patterns that are not built on an underlying Bauplan [74,75], and that is learned piecemeal rather than following universal rules [76].

Even if the UG view was consistent with contemporary evidence from comparative linguistics and cognitive science, the Baldwinian account of UG would be implausible. It predicts that genetically different populations of modern humans should be adapted to their specific linguistic history [77]. For example, speakers of Walpiri, an aboriginal language of Australia, who were genetically separated from speakers of Indo-European languages for at least 40 000 years, should – but do not – have difficulty learning English because they are genetically biased towards the distinctive speech sounds and grammar of Australian aboriginal languages.

A second problem is that linguistic change is far faster than genetic change: hence language provides a 'moving target' that genes cannot successfully follow (Box 3). Computer simulations of gene–language coevolution have indicated that, although the Baldwin effect can operate if the language is artificially held constant, once language change is allowed, the Baldwin effect



**Box 3. Moving Targets: Can Grammar Be Established by the Baldwin Effect?**

A study of the 'moving target' problem [130] simulated the coevolution of grammatical features and the genes of populations of simple learners over 500 generations. The learners evolved by mutation and sexual reproduction to learn a set of binary 'linguistic' features, and their fitness depended only on how quickly they learned these features. 'Genes' could be biased to one or other feature value, or neutral alleles (which learn either feature value more slowly). When the grammatical features do not change over time, the Baldwin effect occurs: neutral alleles are 'driven out' and the genome encodes the 'correct' binary features of the language such that learning is very rapid (dark line in Figure 1). However, if language changes at the same speed as genetic change (light line), or, more realistically, much faster (dotted line), the Baldwin effect vanishes: neutral genes dominate because genes which are biased to 'yesterday's language' are actually harmful. Language change is typically very fast: the entire Indo-European language family, including languages as diverse as Hindi, Danish, Latin, and Welsh, arose in the past 10 000 years [131].

It is tempting to counter that these problems are beside the point; because it is the universal aspects of language that we wish to explain. These, being universal, do not vary when human populations disperse, and are not 'moving targets'; instead, they are putatively completely stable and are hence shared by English, Walpiri, and all other natural languages, thus blocking the possibility of divergent gene–language coevolution [78]. Nevertheless, the Baldwin effect was intended to explain how universal features of language arise; and therefore, on pain of circularity, cannot presuppose universality before Baldwinisation.

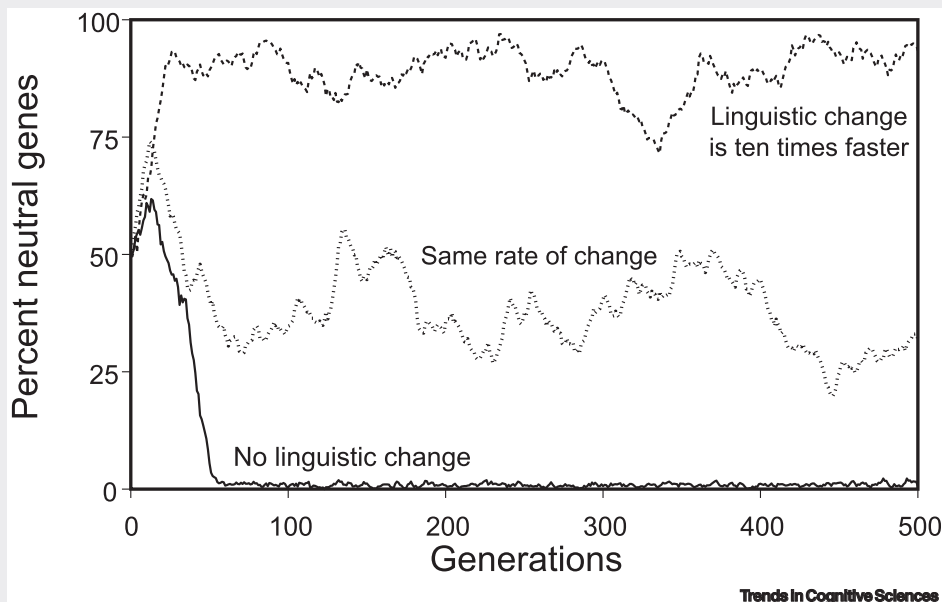


Figure 1. The 'Moving Target' Problem. Adapted, with permission, from [130].

disappears. Indeed, genetic learning biases become disadvantageous because they are typically biased towards yesterday's language, not today's [78].

A third problem for the Baldwinisation of UG viewpoint is that cultural evolution will often favour at best weak, rather than strong, constraints on learning [79–81]. Learners with weak learning biases will, through cultural evolution, create languages that match these biases. However, there is then no selective advantage of having stronger, rigid genetic constraints – and any constraints would be expected to decay in the absence of selection pressure.

By contrast, speech/sign is a good target for Baldwinisation because it presents the same challenges across languages and over long stretches of time (although we focus on the perceptuomotor demands of spoken languages in this article, some scholars have argued that signed rather than spoken languages may be older [82], and hence have also been subject to

Baldwinisation). Speech production involves intricate and rapid manipulation of the tongue, lips, and larynx, combined with breathing control, which is not observed in other primates [83]. Specialised neurocomputational mechanisms controlling these movements [84] and for perceiving rapid streams of subtly distinct sounds [85] are likely to have coevolved with the emergence of speech through Baldwinian evolution [86]. Moreover, regarding speech perception, there is intriguing evidence linking population frequencies of specific genes with speakers of languages with lexical tone (i.e., word identities are partly defined by pitch contours) [87], as well as experimental evidence that one such gene, *ASPM*, correlates with perception of lexical tone at the level of individual speakers [88]. Although causal direction is, of course, difficult to infer from such correlational data, these data fit with a two-way, Baldwinian, interaction of genes and speech perception. Finally, it has been shown [89], by computer simulation, that the Baldwin effect can operate for neural mechanisms for speech processing, in contrast to UG [78,90].

If special-purpose machinery for speech arises through the Baldwin effect, the same should apply for complex vocal behaviours in other species. Indeed, lesion studies show that diverse bird groups (parrots, hummingbirds, and songbirds) all have distinctive neural pathways specific for vocal production/perception, with surprising similarities both to each other, and apparently to speech-specific pathways in humans [91].

Thus, research in comparative linguistics and cognitive science suggest that Baldwinian selection does not underlie a putative UG, but the Baldwin effect may have helped to establish specialised perceptuomotor machinery that underlies speech.

#### Imitation

We are *Homo imitans* [92]. Humans can copy the topography of actions – how parts of the body move relative to one another – more prolifically and with greater precision than any other species, and these imitative skills enable our use of technology and the formation of cooperative social groups.

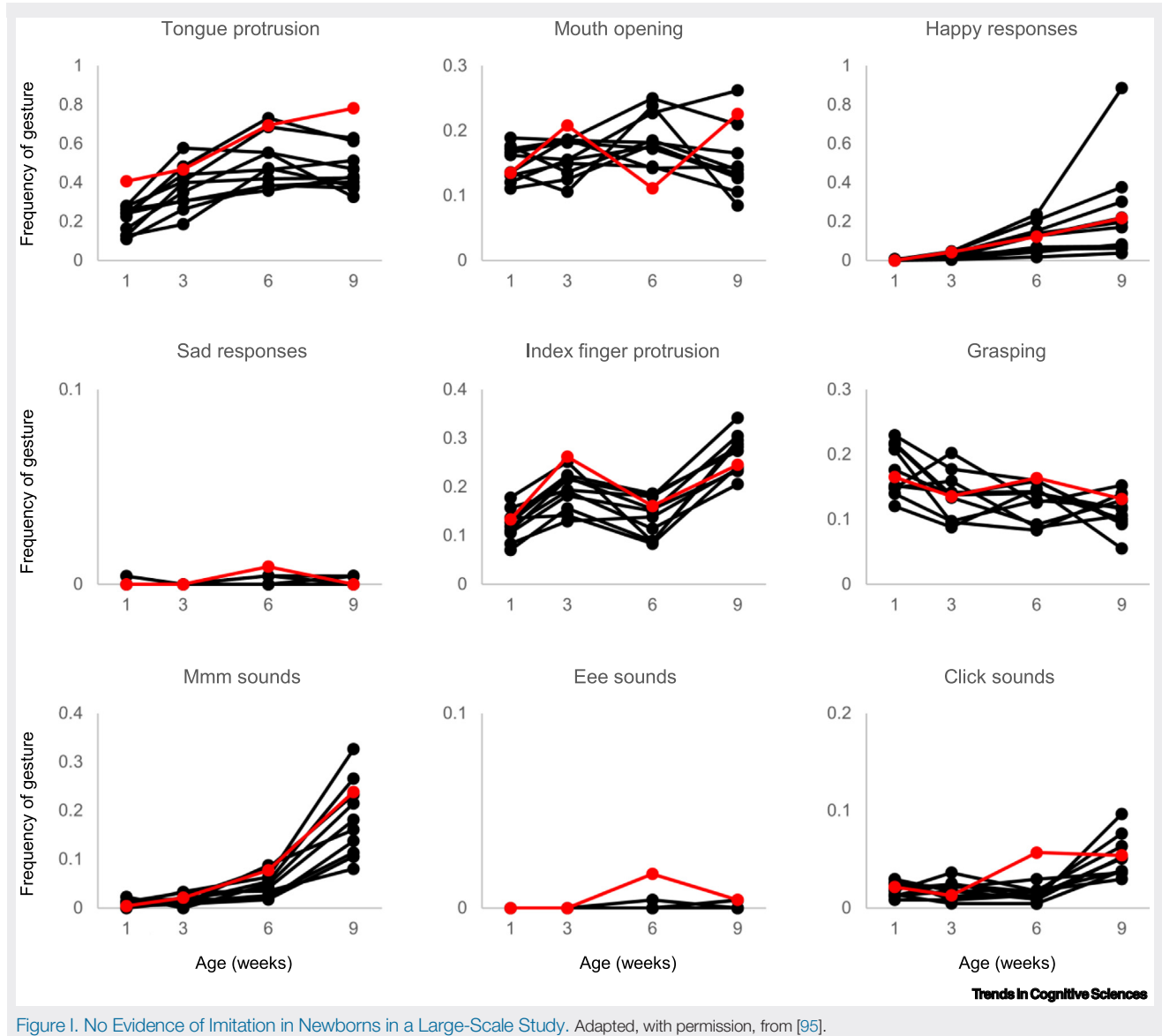
At the cognitive level, the capacity to imitate was until recently thought to depend on a powerful, genetically inherited black box; an **active intermodal matching (AIM)** mechanism for converting observed actions (e.g., the sight of winking) into topographically similar motor output (performance of winking) via unspecified computations [93]. In principle, such a black box could have arisen via Baldwinisation or aplastic evolution [17]. However, AIM is no longer a viable model

#### Box 4. Imitation Is Not in Our Genes

Influential research in the 1970s suggested that newborn human babies can copy a range of facial and manual gestures, implying that a core capacity for imitation is genetically inherited [132]. In 2016, this result was undermined by a large-scale study in Brisbane of more than 100 newborns [95]. The 100 babies were tested at 1, 3, 6, and 9 weeks after birth, for imitation of nine action types, using the gold-standard 'cross-target' method introduced in the 1970s. There was no evidence of imitation for any of the nine action types; in other words, in no case did the infants perform the act more frequently after seeing the same action (red lines in Figure I) than after seeing the alternative actions in the test set (black lines in Figure I). For example, the frequency of mouth opening was no greater after the infants had observed mouth opening than after they had observed tongue protrusion, a sad face, grasping, or the other tested actions.

Previous failures to find neonatal imitation have been attributed to methodological factors, for example the use of an inappropriate model, an inadequate response interval, or suboptimal statistical procedures. A recent meta-analysis of neonatal imitation research, encompassing 336 effect sizes dating back to 1977, sought and did not find a modulating influence of 13 methodological factors that had been previously cited as reasons for replication failure [133]. However, the meta-analysis did find a modulating effect of 'researcher affiliation'; a small number of laboratories were more likely than others to find large positive effects. Furthermore, across the whole dataset there was a relationship between standard error and effect size that was indicative of publication bias, suggesting that smaller studies had been conducted, but they found no evidence of neonatal imitation and had not been published.

In the light of the Brisbane study and subsequent meta-analysis, neonatal imitation can no longer be regarded as a reliable phenomenon. This is consistent with a growing body of evidence that imitation develops slowly in the course of childhood [96] and depends on social experience in which infants and children are exposed to synchronous action, optical mirrors, and imitation by other agents [100,134,135].



of imitation. This model's evidence base has been undermined by studies indicating that newborns do not imitate [94–97], and it has become clear that AIM predicts imitative capacities in infants that are not present even in adults [98,99] (Box 4).

The **associative sequence learning (ASL)** model of imitation [100–102] has provided more fertile ground for Baldwin hypotheses [25–28]. The ASL model suggests that imitation depends on two sets of **horizontal processes** – that encode sequences of observed action components and mediate motor learning, respectively – and on a large repertoire of **matching vertical associations**, bidirectional excitatory links, each connecting a visual representation of an action component with a motor representation of a similar component (e.g., the sight of winking with a motor programme for winking). Two hypotheses suggest that Baldwinisation might have occurred for

horizontal [27] or vertical [28] processes, and two more propose Baldwinisation of attention to body movements [25] or motivation to imitate [28].

The first hypothesis proposes Baldwinisation of horizontal processes that encode sequences of observed actions. The central idea is that, while imitating simple actions using matching vertical associations, possibly in a tool-using context, ancestral humans learned new ways of segmenting and reassembling stimulus sequences [27], and that these new horizontal processes subsequently acquired a genetic basis. This is an intriguing hypothesis with the virtue of testability. However, instead of supporting the hypothesis, current evidence suggests that the same neurocognitive mechanisms encode sequences of observed body movements and of inanimate stimuli, in both imitative [103] and non-imitative tasks [104,105].

The second Baldwin hypothesis is also incompatible with current data. In principle, Baldwinian evolution could have made it easier to learn matching than **non-matching vertical associations** [28], for example it may be easier for agents to associate the sight of a hand opening with performance of hand-opening than with performance of hand-closing. However, training experiments have shown that non-matching vertical associations can be learned with remarkable speed [106–108].

By contrast, there is empirical support for the idea that motivational [28] and attentional processes [25,27] have been Baldwinised in ways that could facilitate imitation. Humans have higher social motivation than chimpanzees – for example, we are more likely to choose social over asocial activities [109] – and, although many species are more attentive to biological than non-biological motion [110], from infancy humans are especially attentive to faces [111–113] and hands [114], particularly hands in motion [115,116]. There is currently no evidence that enhanced social motivation and attentiveness to body movements are adaptations for imitation specifically. Social motivation supports care and cooperation in a wide range of human contexts, and early attention to hands, especially one's own hands in motion, facilitates the development of all hand–eye coordination, for example reaching and grasping movements, not merely the imitation of hand movements [25]. However, increases in social motivation and attention to body movements are likely to have made our ancestors better imitators, and therefore non-specific Baldwinisation of these peripheral mechanisms is a plausible hypothesis.

Another peripheral Baldwinisation hypothesis proposes that evolution has enhanced human motivation to imitate specifically, that it has increased the 'intrinsic rewardingness of imitation' [28]. This is a promising idea for future investigation, but current evidence that children overimitate – imitate more than chimpanzees, and more than is necessary to obtain instrumental rewards [117,118] – is equally compatible with this view and with the idea that general social motivation, rather than imitation-specific motivation, makes humans enthusiastic copiers of body movements [101].

The Baldwinisation of imitation merits further investigation (see [Outstanding Questions](#)) but the current picture – indicating nonspecific, peripheral Baldwinisation – makes evolutionary sense. If matching vertical associations sunk in, or infants genetically inherited a specific motivation to imitate, there is a risk that they would have difficulty learning the non-imitative, complementary actions that are required in many economic and social contexts – to grasp when another agent releases their grip, to push when another pulls.

### Concluding Remarks

The Baldwin effect has seemed promising for a very long time. For more than a century it has been poised to revolutionise our understanding of the evolution of complex behavioural traits,

### Outstanding Questions

How widespread is the Baldwin effect? For example, has it shaped face processing, episodic memory, social exchange reasoning, normative thinking, mathematical cognition, and mentalising?

Baldwinisation is plausible for human fear learning, imitation, and language because there is evidence that traits which now have a genetic basis were learned earlier in the organism's phylogenetic history. Is this a general principle? How confidently can we infer Baldwinisation, rather than aplastic evolution, from evidence that a trait was learned earlier in phylogenetic history?

Are there cases where the Baldwin effect has operated on central cognitive processes?

What sinks in when *Drosophila* are artificially selected for aversion learning? Would studies of experimental evolution using two cue modalities and two types of outcome confirm the evidence from rats that peripheral processes are Baldwinised?

Research using behavioural and physiological measures suggests that young infants are especially attentive to, rather than fearful of, snakes. Can this evidence of peripheral Baldwinisation be confirmed using neurological measures of attention? Are infants better able to associate snakes with positive than with negative outcomes?

Has the motivation to align our thought and behaviour with others, for example in joint action and communication, been Baldwinised?

Many theorists argue that language was originally gestural rather than vocal. If so, are there traces of Baldwinisation of gestural communication, over and above manual dexterity required, for example, in tool use?

Can we find evidence that sequence processing and motivation have been Baldwinised specifically for imitation? For example, are there types of action, or stages in the learning process, where different computations encode action and non-action sequences for imitation and recognition? Is it easier to train infants to copy body movements

but convincing empirical demonstrations have been elusive. We have argued that there is now compelling evidence of the Baldwinisation of cognition from *Drosophila*, and that research in cognitive science indicates that peripheral rather than central cognitive mechanisms have been the primary targets of selection.

Why might selection operate primarily at the cognitive periphery? A parallel with the evolution of other biological mechanisms is suggestive: internal physiological processes and anatomical structures are remarkably well-conserved. The organisation of the digestive, circulatory, and respiratory systems is similar across vertebrate species, and they are so deeply interconnected that modifications beyond changes of size and shape may be difficult without causing substantial collateral damage. Moreover, even such modest changes to central systems will impact on a wide variety of functions and may therefore not be under strong selection from any one function. By contrast, interfaces with the external environment (jaws, teeth, digestive enzymes, bone and muscle structure) can be adapted to local circumstances (e.g., food sources) without interfering with central systems. The central machinery of cognition is less well understood, but may be equally interlocking, with widespread functional ramification, and a consequent resistance to evolutionary change.

Alternatively, it is possible that central cognitive processes are fully evolvable, but, at least in the human case, tend to be adaptively specialised by cultural rather than by genetic selection [101]. In domains such as language, imitation, mathematics, and ethics, changes to central mechanisms can be acquired through cultural learning. Cognitive skills that are taught, and those that are learned from others through more informal social interaction, do not need to sink in. Baldwinisation would bring little if any fitness advantage for skills that are reliably inherited via a non-genetic route [17], and specialised central mechanisms may be more teachable than specialised peripheral mechanisms. Plausibly, it is easier to learn grammatical constructions than vocal control through conversation, and, in the case of imitation, easier to learn sensorimotor mappings than intrinsic motivation through non-vocal social interaction.

These possibilities warrant further investigation, but the main purpose of this article is to draw attention to empirical work and to encourage testing for Baldwin effects in cognitive science (see [Outstanding Questions](#)). Many nonspecialists assume that research on taste aversion, fear learning, language, and imitation has produced solid evidence of genetically specialised learning mechanisms. This view is outdated. Careful empirical work, starting in the 1970s, has shown that efficiency in these domains depends on genetically specialised input and output processes, and that these cognitive equivalents of scanners and printers are likely to be Baldwin effects.

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(‘overimitation’) than to copy object movements?

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