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A minimal model of plasticity across disciplines

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Abstract

Here, we analyse the concept of plasticity and its application in diverse research fields such as physics, neuroscience, and biology. Historically, plasticity broadly refers to a system's capacity to undergo lasting changes in response to external inputs. This concept has been separated from the concept of elasticity, where changes are considered temporary and reversible. Both concepts were originally developed within physics and engineering, where plastic change happens when a material crosses a yield point. We propose a 'minimal model' to unify the concepts of elasticity, resilience, and plasticity across disciplines by mathematically formalising the transition between elastic and plastic changes. The model defines plasticity as the system's ability to reconfigure its internal parameters when it crosses a yield point, changing how it responds to new inputs. The framework we propose provides a common conceptual tool to facilitate communication across disciplines ranging from engineering to history and art. It can be applied to explain crucial differences between generally applied but still vague concepts, such as resilience and adaptation in different disciplines. Therefore, the model provides a basis for interdisciplinary applications and further exploration of plasticity across disciplines.

Keywords

Plasticity, elasticity, physics, neuroscience, biology, modelling, dynamical systems

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Introduction

Plasticity, as a term, is widely used across several scientific and artistic disciplines. In everyday language, plasticity is often understood as a system's ability to adapt to new demands, or to be reshaped by

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experience, and it is frequently used to signify flexibility, adaptability and the capacity to change. The term is loosely used to describe how individuals may learn new skills, how personalities change in time, how habits form, or how communities adjust to new challenges. This folk notion blends similar concepts such as flexibility, resilience, and growth, treating plasticity as an inherently positive capacity for change. However, it also carries ambivalence, as we speak of someone being 'too plastic' when they seem easily influenced, or of materials losing their shape after being stretched too far. These intuitive uses highlight both the appeal and the conceptual imprecision of the term. They capture a general sense that systems retain a memory of what has happened to them and can shift their structure accordingly, but they often fail to distinguish between reversible adjustments, irreversible transformations, and outright damage.

More specifically, in several scientific disciplines, including physics, biology, psychology, and the social sciences, plasticity has even become a central conceptual framework, promoting a broad understanding of multiple phenomena (Citri et al. 2008; Forsman 2015). In most scientific disciplines, the term plasticity is used to signify transformation, and plasticity refers to the capacity to change in response to external inputs. However, not all changes can be considered plasticity changes.

Although the term 'plasticity' can be intuitively grasped, a formal, broadly applicable, and cross-disciplinary definition has been difficult to propose. This fact presents a challenge to interdisciplinarity. How can we know if, when a scholar from one discipline uses the term, she means the same thing as scholars from other disciplines? If we want to engage in interdisciplinary work, we must at least have a minimal common ground understanding of the term plasticity. There are several ways to overcome this problem. For example, one can suggest a universal conceptual definition for plasticity. However, it is questionable whether this is feasible, as despite its long history, its meaning is far from uniform within some disciplines, and even less so across disciplines. Therefore, rather than seeking uniformity, what may be needed is an interdisciplinary model of plasticity that provides a framework for translation, comparison, and dialogue. Such a model would establish minimal common ground while still allowing for contextual specificity, thus enabling more coherent and productive interdisciplinary engagement.

Here, we aim to advance into such a model by using mathematics to provide an operational definition of plasticity. Developing a mathematical model that grasps the essence of plasticity might pave the way for a minimal integration of the term without the need for a full conceptual integration. Why is a mathematical model superior to a formal definition? In physics, it is common that the same equation can represent phenomena from very different domains. The formal mathematical solution for one phenomenon can be immediately applied to analyse and understand another phenomenon. In analogy, if a minimal model can be applied to understanding the nature of the plasticity process across a broad range of disciplines, the minimal meaning of the process will be carried from one case to the other, and a common interdisciplinary ground will be achieved without the need for a one-size-fits-all definition.

In this paper, we develop a 'minimal model' of plasticity that includes common features of the concept in different fields. We demonstrate the utility of this proposed model by applying it to a variety of domains: plasticity in materials, phenotypes, brains, social structures, and visual art concepts. We use these materials science, biology and neuroscience as examples because they reflect our respective disciplines, but we also discuss our minimal model in the context of other disciplines (social change and conceptualisation of reality representation in the visual arts) to show its general utility. We envision that the model will also be applicable across an even broader range of disciplines and will have the capacity to foster transdisciplinary synergies. These synergies could be conceptual, enabling translation between disciplines that use different but related definitions; methodological, enabling the borrowing and transfer of models and analytical tools; and practical, supporting applied research in areas where disciplines interact. In particular, transdisciplinary synergies could be useful in medical and health sciences, environmental and climate studies, technology and design, and arts and humanities, in which cases they could allow

critical dialogues. In these respects, several audiences such as scholars and researchers, policy makers and educators, professional practitioners, and the general public could benefit from transdisciplinary synergies in the use of plasticity, for whom the term already broadly resonates as an intuitive metaphor for adaptability and transformation. Yet, our main aim in introducing this model is not necessarily to provide a deeper understanding of the plasticity process in each separate discipline. Detailed mechanistic models already exist in most disciplines, so our minimal model is intended at a more abstract level. Hence, our aim is to create a minimal common ground that (1) captures the shared structure of plasticity across disciplines, and (2) formalises how plastic changes generally differ from elastic changes.

A first approach to defining plasticity across scientific disciplines

In this section, we briefly introduce the various meanings 'plasticity' takes on in physics, neuroscience, and biology. We will discuss these disciplines in this order because physics introduces a formal definition of plasticity, neuroscience has given a central role to 'plasticity' in various phenomena, and biology offers a broader application of 'plasticity' across diverse living forms. These disciplines provide the basic ingredients needed for developing the minimal model.

In physics, the term plasticity was first coined in the context of material deformation. Physics formalises a demarcation between elastic changes and plastic changes (Tresca 1869). Elastic changes are changes to physical substrates that reverse once the applied force that caused the change is removed. In contrast, plastic changes are permanent. The point at which an elastic change becomes a plastic change is called the yield point, which varies widely between materials (Figure 1). A material may also break because of plastic deformations, and if this happens when little force is applied, this material is considered 'brittle'.

In neurosciences, the notion of plasticity has been most clearly introduced by William James (1890): 'Plasticity, in the wide sense of the word, means the possession of a structure weak enough to yield to an influence, but strong enough not to yield all at once'. This neuroscientific definition already contains a notion of the 'yield point' as the ability to undergo change and 'brittleness' as the ability not to yield all

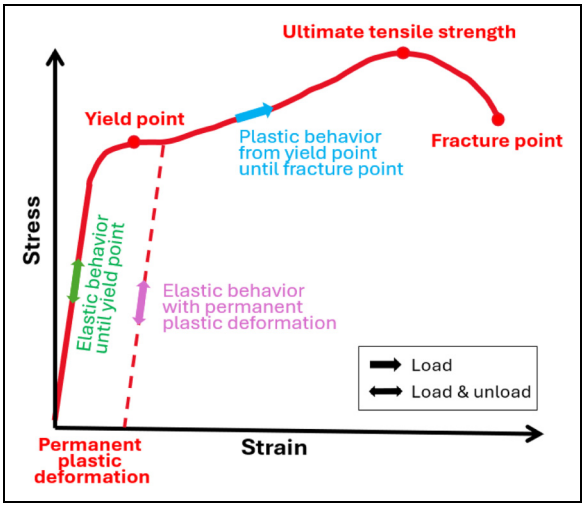

Figure 1. A hypothetical strain–stress curve of a material, indicating its elastic, plastic, and fracture behaviour.

at once (i.e., break as opposed to the elastic behaviour displayed by ductile materials). Notably, however, neuroscience adds a novel component to the definition of plasticity, namely that plastic changes should be a functional adaptation to an external influence, frequently interpreted as a change in the environment.

Biology has primarily developed the notion of phenotypic plasticity along similar lines: plastic changes to the characteristics of an organism that can be beneficial and increase fitness (compared to a non-plastic phenotype) in response to a change in the environment (Schneider 2022). In biology, however, the distinction between 'plasticity' and 'elasticity' is less clear.

In the following sections, we provide a more in-depth analysis of the notion of plasticity across these three disciplines. These sections are relatively technical, and readers mainly interested in our minimal model should feel free to skip to 'A minimal model of plasticity'. However, this detailed analysis will help us construct and apply a minimal model of plasticity in the subsequent sections.

Plasticity in physics and engineering

As stated above, in physics, plasticity describes a material's intrinsic ability to undergo permanent deformation under an applied force. Unlike elastic deformation, where the material returns to its original shape upon removal of the force, plastic deformation results in a lasting change in the material's shape or size. This behaviour is often observed in materials such as metals or polymers.

Plasticity as energy dissipation

Understanding plasticity involves consideration of the energy involved in the process (Chakrabarty 2010). When a force acts on a material, some energy is stored within the intermolecular bonds while some is dissipated as heat, for instance. In this context, the state of the system depends on its history. Hysteresis is a phenomenon in which the state of a system depends not only on its current input or conditions but also on its history of past inputs. In other words, the system exhibits a lag or memory effect, where the output does not immediately follow changes in the input (Mayergoyz 2003). Therefore, hysteresis links directly to the concept of stored and dissipated energy, particularly in physical systems like magnetism, mechanics, and thermodynamics. For example, in magnetic materials, hysteresis describes the relationship between an applied magnetic field and the resulting magnetisation, affecting the energy stored in the material. If the magnetic energy is stored and released back after the external field disappears, we observe a reversible elastic behaviour. Plastic behaviour can be observed when the magnetisation of a permanent magnet remains after the external field is removed. Therefore, this effect shares similarities with plasticity in that both involve a lasting change in the material's state due to energy input.

Understanding plasticity requires probing into the molecular interactions responsible for the observed changes. At this level, plastic deformations take place when the force applied to a material is sufficient to break its interatomic bonds, and energy is consumed and expelled from the system. When the material is unloaded, its shape and structure are permanently altered (Mayergoyz 2003). Referring to our previous example, when a magnetic field is applied to a magnetic material, small, locally magnetised regions align, changing the characteristics of so-called 'domain walls' that are kept in place by the structure of the material and lead to a permanent change in the magnetic properties of the material even after the external field is removed. This results in a 'remanent magnetisation', effectively creating a permanent magnet.

Using plasticity in engineering

In civil engineering, plasticity refers to the behaviour of materials that undergo permanent deformation when subjected to applied loads (Tresca 1869). Materials under load exhibit different behaviours as the

applied stress increases, transitioning from elastic to plastic responses (Figure 1). This transition does not occur instantaneously, but instead follows a progressive continuum governed by intrinsic material properties.

In the initial stage of loading, materials deform elastically. This means that the deformation is temporary and fully recoverable upon the removal of the load (depicted by the green arrow in Figure 1). The material returns to its original shape because the atomic bonds are stretched but not permanently displaced. As stress increases to a certain level, the material reaches its yield point, at which the material begins to deform plastically (von Mises 1913) (i.e., permanently; Figure 1, purple arrow). At the microscopic level, this involves the sliding of atomic planes and the movement of dislocations within the material's crystal lattice. As plastic deformation continues, some materials exhibit strain hardening (or work hardening), where the material becomes stronger and more resistant to further deformation. This occurs due to the increased dislocation density, which impedes further movement of dislocations (Dieter 1986). The material continues to deform plastically until it reaches its ultimate tensile strength – the maximum stress it can withstand. After reaching the ultimate tensile strength, there is a rapid degradation of the load-carrying capacity, resulting in the fracture of the material as indicated by the fracture point in Figure 1.

Ductile materials exhibit significant plastic behaviour after a relatively small elastic deformation, allowing them to undergo considerable permanent deformation before failure. In contrast, brittle materials show little to no plastic behaviour, fracturing suddenly at a critical load with minimal energy absorption. Elastic-plastic material behaviour is the basis for structural design. At the structural level, understanding the elastic-plastic behaviour of materials such as concrete, steel, and glass allows the design of structures that can safely withstand loads beyond the elastic limit by redistributing stresses through controlled plastic deformations. This approach ensures that structures have adequate ductility to prevent sudden, catastrophic failures while being cost-effective.

Other examples include geotechnical engineering, which focuses on soil behaviour, where understanding the plasticity of soils is crucial for predicting settlement, stability, and load-bearing capacity. Pavement engineering concentrates on asphalt, a material that displays viscoelastic and plastic characteristics under repeated traffic loads, affecting pavement performance and longevity. Manufacturing and construction processes leverage plastic deformation principles in methods such as rolling, forging, and extrusion to shape materials into desired forms and improve their mechanical properties (Roberts-Austen 1891). In other disciplines, such as earthquake engineering, plasticity is used as a means to absorb and dissipate seismic energy through controlled plastic deformation (He, Cheng, and Chen 2022). This approach enhances a structure's ability to withstand earthquakes by allowing it to undergo significant deformation without sudden failure, thereby improving safety and resilience.

Plasticity in neuroscience

The brain is one of the most complex and adaptable systems in existence, capable of changing and evolving in response to new experiences and challenges (Bassett and Gazzaniga 2011). This capacity for change, known as brain plasticity, is crucial to understanding the biological and psychological foundation of human adaptability, learning, memory, and recovery from injury and disease. However, in some cases, this powerful mechanism may result in maladaptive changes, resulting in pathologies such as phantom limb pain, focal dystonia, tinnitus, and addiction (Nava and Röder 2011; O'Brien 2009).

Understanding brain plasticity

Brain plasticity, also referred to as neuroplasticity, is commonly defined as the brain's remarkable ability to reorganise and adapt in response to experience. However, the way in which the term 'plasticity' has

been used in connection with the nervous system has changed with time (Berlucchi and Buchtel 2009). The first use of the term plasticity is usually assigned to pioneer scientists such as Adolf Meyer (Lamb 2019) and William James, who conjectured that anything plastic is 'weak enough to yield to an influence, but strong enough not to yield all at once'. From there, through the work of many neuroscientists that culminated with Hebb's neuropsychological theory of behaviour organisation (Hebb 1949), the term plasticity became closely attached to structural changes in synapses and synaptic connections (Berlucchi and Buchtel 2009).

Plasticity was first used to refer to the capacity to change (Eggermont 1990). However, later and more nuanced conceptualisations of the term 'neural plasticity' were introduced by Paillard, Will, and others (Will et al. 2008), who have suggested that to avoid vagueness and misuse of the term, not every change in the nervous system should be called plastic. Paillard supports his argument using three main concepts: (1) In agreement with James' first use of the term, plasticity should refer to the change in structure in response to an external stimulus, and this change in structure should be maintained after the removal of the causing stimulus (cf. elasticity discussed above for material systems). Furthermore, the exclusion of intrinsic processes that drive change is crucial in Paillard's argument, as it excludes changes in the nervous system structure associated solely with growth and development, as those processes do not involve, in principle, external stimuli that cause the change and, therefore, are not contained within the concept of neuroplasticity. (2) Plasticity is different from change in the sense that plasticity refers to changes in a defined structure, its basic principles, or its functions. This is important because Paillard argues that assigning the term plasticity to any change without knowing where the alterations have taken place is problematic. (3) The concept of plasticity assumes that change should be stable and not transient. This is problematic because, depending on the level of observation, even stable systems may change. Therefore, Paillard's concept of plasticity is appropriately applied to the nervous system only when the system achieves a novel, stable, functional state by changing the elements of which it is made or transforming their internal connectivity (Berlucchi and Buchtel 2009).

Mechanisms of brain plasticity

In agreement with the concept of neuroplasticity we discussed in the previous section, two interconnected types of neuroplasticity have been defined: structural and functional. Structural plasticity refers to the brain's ability to change its physical structure, such as the growth of new neurons or the formation of new connections between existing neurons, particularly in response to learning and experience. For this type of neuroplasticity, the process of generating new neurons is essential. Although most of the brain's neurons are formed during early development, certain areas, such as the hippocampus – a region critical for memory and learning – continue to produce neurons into adulthood (Lucassen et al. 2020). Functional plasticity, on the other hand, involves the brain's ability to adapt existing structures to assume new functions by acquiring new 'states' such as the long-term strengthening (long-term potentiation – LTP) or weakening (long-term depression – LTD) of existing synapses through well-characterised cellular processes. Both LTP and LTD enable memory representation and other cognitive functions. Interestingly, short-lived reversible phenomena that typically take place in the order of milliseconds to several minutes such as paired-pulse facilitation and depression are usually classified as short-term plasticity (Citri and Malenka 2008), providing a clear example of how the boundaries of the concept of plasticity in neuroscience drifts away from the concept in physics, where elasticity and plasticity are clearly separated by their (ir)reversibility.

Resilience: psychological stability and recovery

The term resilience is a related concept commonly used to refer to the psychological capacity to endure and even grow in the face of adversity, stress, or trauma (Luthar, Cicchetti, and Becker 2000). Many times, plasticity and resilience appear together, and sometimes they are even conflated. It is therefore interesting to distinguish the two terms. The folk notion of resilience refers to the capacity to withstand or to recover quickly and thus intuitively differs from the concept of plasticity as a long-lasting or irreversible change. However, currently, there is no universally agreed-upon definition of resilience, and therefore, it is unclear how resilience works at the brain level and how it may be related to pathology (Perry 2025; De Vries et al. 2024). For some authors, resilience seems to be a dynamic process that can be developed through experience and is thus associated with neuroplasticity (Yao and Hsieh 2019). For others, resilience may be better assimilated to the concept of elasticity and recovery (IJntema, Schaufeli, and Burger 2023) and may then be a property that individuals may or may not have. Indeed, popular conceptualisations of resilience refer to the ability to 'bounce back' from stressors, i.e., the capacity to return to a previous state following exposure to a stressor. Following this conceptualisation, psychological resilience can be mathematically represented based on physics concepts in relation to elasticity (Den Hartigh and Hill 2022), and thus, a change that is not stable or maintained in the long term. Following this conceptualisation of resilience, we can apply the term resilience to situations of no permanent change, where the system recovers to its previous structure, function, or behaviour, and thus distinguish it from plasticity, which applies to situations of permanent (or long-lasting) change where the system is altered rather than restored to its original state. Thus, our model could help to distinguish between resilience understood as elastic return to a preexisting condition, and resilience understood as a plastic reconfiguration that results in a new, healthier or more functional state.

Phenotypic plasticity in biology

Phenotypic plasticity is a core concept in evolutionary biology, and it has gained increasing prominence because of its potential to render populations less vulnerable to climate change (Forsman 2015; Seebacher, White, and Franklin 2015). Plasticity is formally defined as the expression of different phenotypes (the realised characteristics of an organism) by a single genotype (the genetic makeup encoded in DNA) in response to environmental signals (DeWitt and Scheiner 2004). This definition resembles the one used in neuroscience by linking plasticity to environmental signals. In the evolutionary definition, however, plasticity is defined with reference to genotype rather than to the plastic structure (brain) itself. Hence, each genotype can adopt different phenotypic 'states' in response to environmental change, like the plastic deformation of materials discussed above. In animals with a central nervous system, environmental signals are mostly received by sensing mechanisms and processed by the central nervous system, which initiates endocrine and epigenetic responses (Seebacher and Little 2024). The resulting changes in phenotype can be reversible ('elasticity') or fixed ('plasticity') within the lifetime of the organisms or even across generations.

The concept of plasticity diverges from the dogma that the characteristics of organisms are solely determined by the genetic code on DNA (Laland et al. 2014). Rather, epigenetic ('above genetics') processes alter phenotypes by modifying access to genes on DNA but do not change the DNA sequence (Allis and Jenuwein 2016; Radford 2018). Genes are sections of DNA (nucleotide sequences) that contain information that can be used to synthesise a protein product. However, the information must be read (transcribed) and translated into a sequence of amino acids that are the building blocks of proteins. Transcription of nucleotide sequences occurs by so-called 'transcription factors' that produce a

transcriptional copy of the information in the form of an mRNA molecule, which is then translated into a specific protein.

Epigenetic processes add small chemical groups (e.g., methyl groups) to the DNA molecule or associated proteins and thereby prevent access to the information contained in genes (Luo, Hajkova, and Ecker 2018; Stillman 2018). DNA methylation is among the best-known epigenetic mechanisms that can modify gene expression in response to environmental signals (Loughland, Little, and Seebacher 2021). The reality is far more complex, but the important point here is that epigenetic mechanisms underlie phenotypic plasticity and can alter phenotypes to render individuals less vulnerable to potentially negative impacts of environmental change (Seebacher, White, and Franklin 2015).

Similar to neuroscience, the term 'phenotypic plasticity' in biology is often used to encompass permanent change (plasticity) and reversible changes (elasticity) (Dupont et al. 2024). These responses are not mutually exclusive and may be mediated by diverse epigenetic mechanisms briefly alluded to above. Epigenetically mediated changes in phenotypes can be induced in the gametes (egg and sperm) of parents or even in previous generations and thereby cause lasting changes to offspring phenotypes (Perez and Lehner 2019; Shama and Wegner 2014). Such inter- or transgenerational epigenetic effects thereby induce true plasticity, in the sense that phenotypes are changed non-reversibly. Similarly, environmental conditions experienced during early development can alter epigenetic marks and thereby determine the phenotype for the rest of the organism's life (Burggren 2020). In contrast, 'acclimation' is reversible, and phenotypes move between different states depending on recurring environmental fluctuations (Forsman 2015). Acclimation, therefore, represents elasticity that is well known to occur in response to seasonal changes, for example (Wingfield 2008).

Unlike in engineering, however, the boundaries between these different forms of 'plasticity' are not always clearly defined. Sometimes, transgenerational and developmental plasticity are reversible (Wu and Zhang 2017), and it is not clear whether acclimation is truly reversible or whether there remains a 'memory' of previous states. Part of the reason for this blurring of boundaries lies in the network-like regulation of biological processes, where numerous pathways interact to regulate phenotypic outcomes (Sambamoorthy, Sinha, and Raman 2019). Unlike a particular manufactured material whose plasticity is predictable from its specifications, epigenetic processes are regulated by other epigenetic processes, which in turn are regulated by different hormones, which also interact with each other and with additional regulatory proteins (Jeltsch, Broche, and Bashtrykov 2018; Seebacher and Little 2024). The complexity of these evolved regulatory networks may produce unpredictable results. Similar to neural plasticity, the outcomes – the actual plastic responses – are not always beneficial for the organism. There can be a cost of plasticity, which manifests primarily as a mismatch between phenotype and environment, resulting from different rates of phenotypic and environmental change or from unexpected environmental events (Hoffmann and Bridle 2023).

A minimal model of plasticity

The demarcation between elasticity and plasticity is central to the definition of plasticity, not just in physics but, as we have seen, also in biology and neuroscience. To reiterate, not every change is plastic, and therefore plasticity cannot be accurately defined as the capacity to change. Some changes are a simple consequence of a system being in a particular state and spontaneously (but predictably) changing to a different state. For instance, in the brain, individual cells spontaneously generate (near-)circadian rhythms, continually cycling through a limited set of states, even when external inputs like light and temperature are held constant (Welsh, Takahashi, and Kay 2010). Other changes are responses to some external stressor or input, like a photon hitting a photoreceptor and the photoreceptor going through a cascade of chemical reactions and producing an electrical response (Rieke and Baylor 1998). When

input is suspended, the system will eventually settle into a stationary state or cycle through a limited set of states. And, when a new input or stressor is presented, the system will respond in approximately the same way it did before. These changes are elastic.

When does plasticity enter the story? We propose that the demarcation set by the yield point is crucial to the definition of plasticity. As we discussed before, when the input force to a material exceeds some value, the material starts to change plastically. The configuration of the material has changed rather permanently, which, in turn, changes the way the material responds to new inputs. However, to apply this principle to other disciplines, we need to abstract away from the physical details.

We propose a minimal model that captures the demarcation between elasticity and plasticity, regardless of the level of description. Our model extends beyond simple capacity for change and reversibility, as it posits that the primary characteristic of plasticity is related to an interplay between internal dynamics and external ones. In this sense, our model provides an operational definition to distinguish a reversible change from an irreversible one.

In our model, the behaviour of System S in the elastic regime can be described by the following dynamical system state-space model

$$\frac{dx}{dt} = Ax(t) + Bu(t) \tag{1}$$

Here, S is a linear time-invariant (LTI) dynamical system, with internal state variable x (e.g., length and width of a steel bar, the activity of a group of neurons, etc.), and external inputs u (e.g., load on a steel bar, light falling on the retina). The matrices A and B indicate how the previous state (x_{t-1}) determines the next state (x_t) through A and how external inputs (u) determine the next state through B. That is, external inputs are multiplied by B, and the internal state is multiplied by A, jointly determining the next state of the system (equation 1). If the behaviour of the system can be approximated by this state-space model, it operates in the elastic range. As depicted in Figure 2 (bottom panel), the system changes *plastically* if and only if the system's configuration embodied by A and B is adjusted in response to the system crossing some parameter threshold (the yield point). This threshold may depend on a variety of factors, from external (e.g., temperature) to internal (the A and B matrices of the system), and will mainly depend on the system under study. In physical systems, crossing the yield point leads to a novel configuration of the system. Put differently, the yield point lies somewhere in the state-space that the system has not been in before.

We propose that the structure of A and B changes when the system transitions from the elastic to the plastic regime as a result of crossing some threshold. Hence, the ability to demarcate plasticity processes as such by following the system's history is the defining feature of our model. Or put it more bluntly, in our model, plasticity cannot be defined without a reference to the development of the system's structure or the 'historical' internal states the system occupies.

Formally modelling a system necessarily involves abstraction. It is not always useful to model each individual constituent component (e.g., a molecule) when trying to understand how a system (e.g., a neuron) works. Instead, abstracting away these details by aggregating the effects of many components results in a more tractable description of the system under study. This is no different when trying to understand plasticity. A change in a system may be considered plastic at a high level of description but elastic at a lower level of description. For instance, when an animal is learning to respond to a stimulus by pressing a lever, changes in the behaviour of the animal may be considered plastic (i.e., there are changes in A and/or B in the equation that capture the animal's behaviour). However, when modelling the animal at the level of neurons and connections between neurons, it may well be that these changes turn out to be elastic (i.e., there are no changes in connections between neurons, as mentioned when we explained short-lived phenomena such as paired-pulse facilitation and depression, in previous sections).

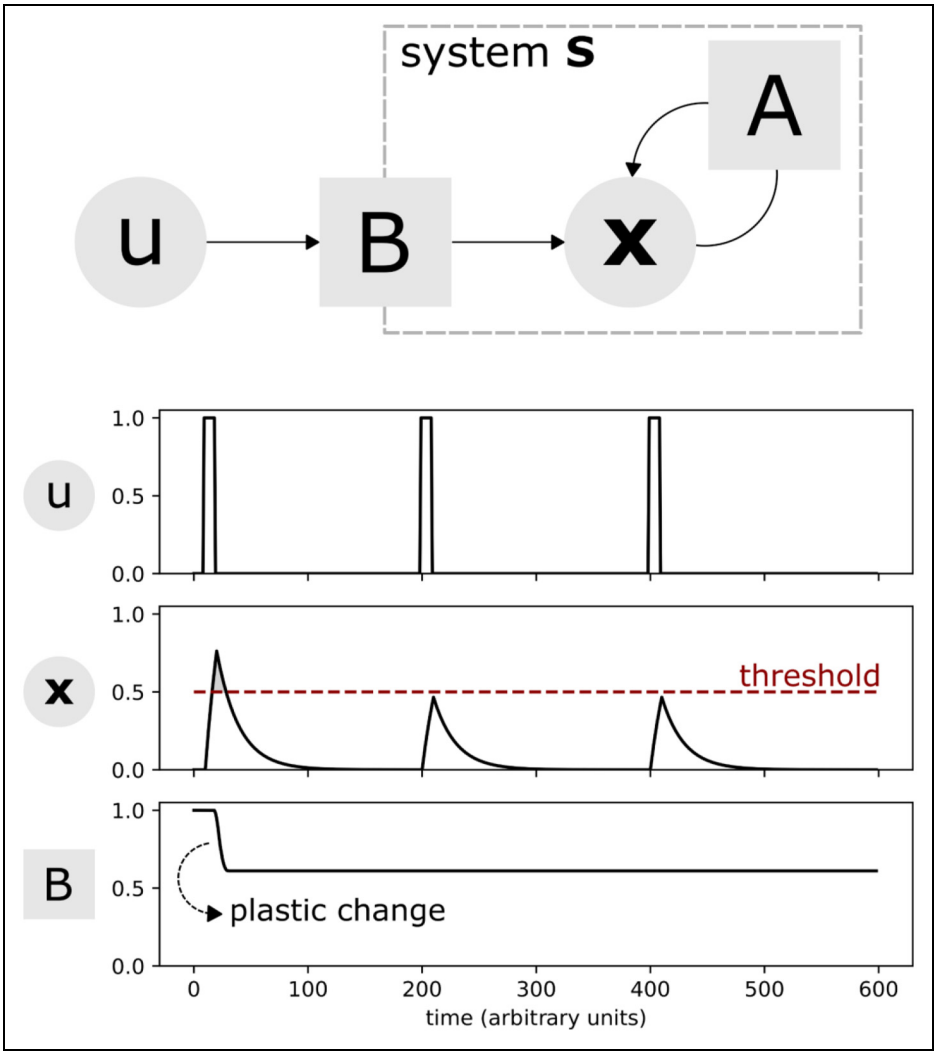

Figure 2. A simple model of plasticity and a toy example. Top: Schematic illustration of a linear time-invariant system. The system S is described by state variables x, and its dependence on external inputs u is governed by B, whereas its dependence on previous states is governed by A. Bottom: Demonstration with a toy example, a leaky integrator ($B = 0.1$, $A = 0.9$). Three identical external input pulses are delivered to the system. The first time, the system's state crosses the 'elastic-to-plastic' threshold, which induces a decrease in B (a plastic change). The response to the next identical pulse, given the same initial conditions, is much reduced. Because the system does not reach the threshold, there is no change in B, and the response to the third pulse is identical. In other words, the second pulse only induces elastic changes.

One possibility is that the animal moves from a portion of the neural state space where it did not respond to the stimulus to a region where it does. In this case, there is only elastic movement in state space but no change in the configuration of the state space. If the level of description is an arbitrary choice, this would seem problematic: the exact same change in a system could be described as plastic or elastic based on nothing more than convenience.

One potential solution to this problem of 'levels of description' is to conclude that plasticity is an emergent property. When a system is described at ever lower levels of description, we are bound to find a level where changes are elastic: these changes are described by some set of *unchanging* laws of nature. This implies that plasticity emerges once we consider the higher-order states a system can occupy and how these states change over time. It is precisely this level of description that most scientists are interested in, often corresponding to a *functional* level of description. There are many different physical configurations a system can be in, but at a higher level of description, they are equivalent. This also points to cases where plastic changes at a lower level may produce elastic changes at a higher level of description. For instance, when a set of neural connections changes plastically, this may well implement a neural circuit that operates the same as before at a functional level. These neural changes may be justifiably viewed as plastic from the perspective of a molecular neuroscientist, while at the same time, a behavioural neuroscientist may justifiably regard them as elastic changes. In sum, the attribution of plasticity to a change in a system is bound by the level of description employed.

In the following sections, we provide examples from within our fields of the model at work, which we will follow with a discussion of its applicability to other disciplines. In each section, we will show how a system can be defined in terms of its internal states, external inputs, and connections between external inputs and internal state (B) and between internal states (A). Through such an identification, we will demonstrate how this minimal model of plasticity can be used to understand plastic materials, plastic phenotypes and plastic brains.

Application of the model to plastic materials

Let us assume a cylinder made of an isotropic material (see Figure 3). Because it is isotropic, it has identical mechanical properties in all directions. The cylinder has cross-sectional area, a, and length, L (these are the 'internal states' x of the system). When an axial force, F (which is an 'external input' u), is applied, the cylinder elongates, and its cross-section becomes narrower. The increment in length and cross-sectional area is denoted by ΔL and Δa, respectively.

Define the axial stress (σ) as the ratio of the force to the cross-sectional area (i.e., $\sigma = \frac{F}{a}$). Assuming the axial stress caused by the force is smaller than the yield stress, the material will respond in an elastic manner. Following Hooke's law for elastic behaviour, the following relation can be established:

$$\sigma = \frac{F}{a} = \frac{E\Delta L}{L}, \tag{2}$$

where E is the Young's modulus characteristic of the material. Therefore, the change in the state variable L in a discrete time step can be expressed as a function of the external input F applied during that time step as follows:

$$\Delta L = \frac{FL}{Ea} \tag{3}$$

Accordingly, the state variable L at time step $t + 1$ is expressed as

$$L_{(t+1)} = L_{(t)} + \frac{\Delta F_{(t+1)} L_{(t)}}{E a_{(t)}} \tag{4}$$

which is a function of the incremental force added in the time step, $\Delta F_{(t+1)} = F_{(t+1)} - F_{(t)}$. Additionally, Poisson's law states that transversal and axial strains are proportional, that is, $\varepsilon_{transversal} = -\upsilon \varepsilon_{axial}$. The coefficient υ is Poisson's ratio, which is characteristic of the material. The

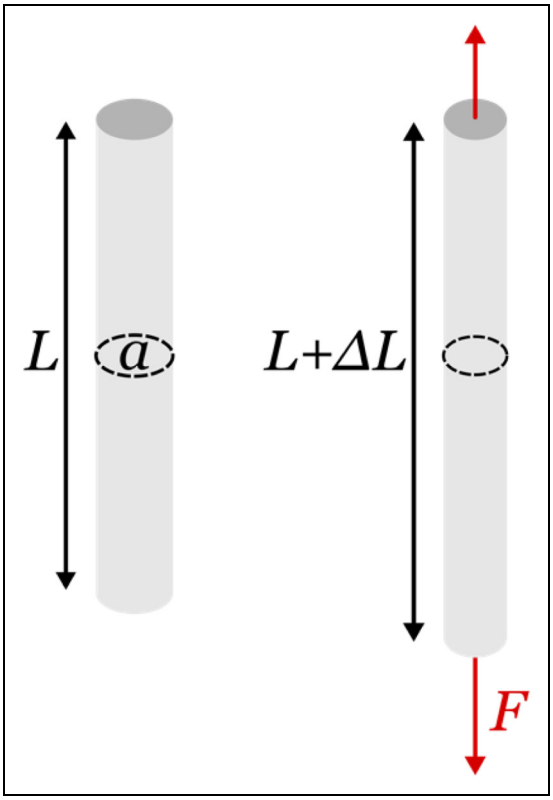

Figure 3. An illustration of an axial deformation of a rod under force. L is the rod length, F is the force applied, and a is the cylinder cross-sectional area. ΔL is the addition to the rod length under force.

negative sign indicates that an increase in one dimension results in a decrease in the other. Accordingly,

$$\frac{\Delta d}{d} = -\upsilon \frac{\Delta L}{L} \tag{5}$$

where d refers to the cylinder's diameter. Assuming that $(\Delta d)^2 = 0$, then $\frac{\Delta d}{d} \frac{\Delta a}{2a}$. Combining equations (3) and (5), the variation in the state variable a in a discrete time step can be expressed as a function of the external input F applied during that time step as follows:

$$\Delta a = \frac{-\upsilon F}{2E} \tag{6}$$

where the negative sign indicates that the area becomes smaller as the axial force increases. Accordingly, the state variable a at time step $t + 1$ is expressed as

$$a_{(t+1)} = a_{(t)} - \frac{\upsilon \Delta F_{(t+1)}}{2E}. \tag{7}$$

Equations (4) and (7) highlight how the state variables at time step $t + 1$, $x_{(t+1)} = \{L_{(t+1)}, a_{(t+1)}\}$ depend on the previous stage $\{L_{(t)}, a_{(t)}\}$ and on the external outputs $u_{(t+1)} = \Delta F_{(t+1)}$.

The presented formulation is only valid in the elastic domain. Once the yield point is reached, the proportional relationship between stress and strain represented in Figure 1 and described by Hooke's law, equation (2), no longer holds. In addition, the assumption of isotropy may also become invalid. Then, more complex models must be used to capture the new behaviour, such as the Ramberg-Osgood equation that describes a nonlinear strain hardening near the yield point as follows:

$$\varepsilon_{axial} = \frac{\sigma}{E} + \alpha \frac{\sigma}{E} \left(\frac{\sigma}{\sigma_y} \right)^{n-1} \tag{8}$$

where α and n are parameters that depend on the material, and σ_y is the yield strength of the material. The Ramberg-Osgood equation adds a second term (plastic part) to the linear behaviour (elastic part) described by Hooke. This equation provides an approximation of complex behaviour characterised by gradual yielding and progressive strain hardening. While defining explicit equations to relate internal states to external outputs beyond the yield point is out of the goal of this work, we note that the state variables at time step $t + 1$can be expressed in terms of the previous state and external outputs. However, this relationship is now governed by updated system matrices A and B, reflecting the plastic response. Thus, as our minimal model suggests, one cannot discuss plasticity without defining a yield point for the system.

Application of the model to plastic phenotypes

In its most basic biological conception, acclimation (e.g., to seasonal temperature changes) exemplifies reversible, elastic changes in biological characteristics (Forsman 2015). On the other hand, phenotypes induced epigenetically by environmental inputs across generations or during early development are more permanent and may represent plastic changes (Burggren 2020). However, the mechanistic basis for this distinction between elasticity and plasticity is not clearly established.

At a physiological level, there is a broad overlap in the mechanistic basis between elastic and plastic responses to the environment. Acclimation is known to be induced by hormone signalling, the autonomic nervous system, histone modifications, and microRNAs (Franke et al. 2024; Little 2021; Sandblom, Zena, and Pichaud 2022; Seebacher and Simmonds 2019). In addition to these mechanisms, plastic changes are also mediated by changes in DNA methylation (Loughland, Little, and Seebacher 2021). However, all these mechanisms are reversible up to a point. For example, hormone signalling is highly context-dependent, acetyl and methyl groups are readily added or removed from histone molecules by dedicated enzymes, and DNA methylation marks can be removed by demethylase enzymes. Most of these mechanisms also interact to achieve phenotypic outcomes (Jeltsch, Broche, and Bashtrykov 2018). In line with the discussion above on different organisational scales, it may be more appropriate to view elasticity and plasticity as emerging properties from network-like interactions that occur in response to specific environmental inputs. Interestingly, there are differences between individuals and between species in their capacity for elasticity and plasticity (Loughland and Seebacher 2020; Schulte and Healy 2022). Some epigenetic processes, such as DNA methylation and histone modifications, are evolutionarily ancient and are present in all organisms (Aliaga et al. 2019; Drazic et al. 2016), while others (e.g., thyroid signalling) are more derived (Zwahlen et al. 2024). These evolutionary differences may explain qualitative differences between distantly related taxa. However, differences between individuals, which all possess the same molecular pathways, must be quantitative. Most parsimoniously, interindividual differences may arise from variance in enzyme activities at multiple points in a biochemical pathway that may elicit major differences in the outcomes of the pathway as a whole. These differences may be viewed as setting the ultimate tensile strength of different taxa, populations, or individuals, beyond which the performance curve (Figure 4A) cannot shift further in response to environmental signals.

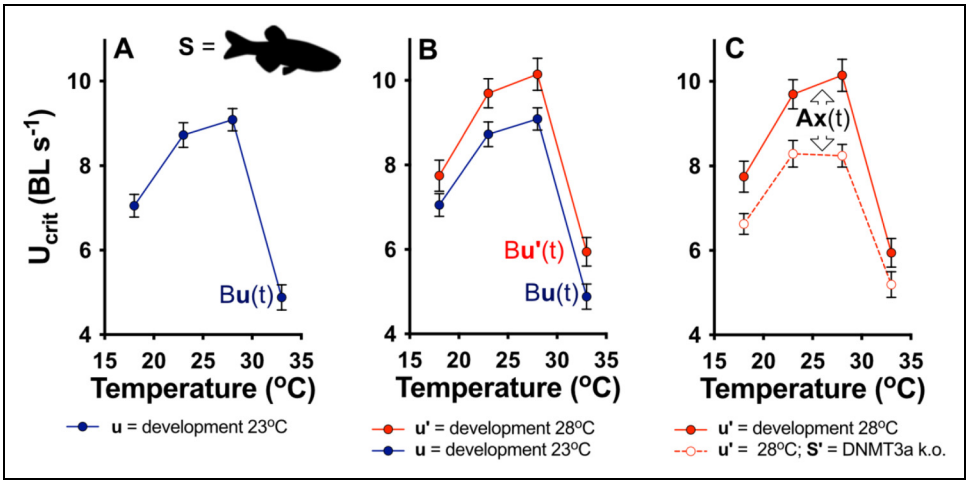

Figure 4. Developmental plasticity of swimming performance in fish. (A) Fish and other ectotherms have a typical performance curve that describes the thermodynamic effects of acute temperature changes on physiological performance. Swimming performance (U_{crit} in body lengths/s) in zebrafish (*S*) shown here [*Bu*(*t*); blue line] depends on the developmental temperature (23°C in this example). (B) When developmental temperature increases to 28°C, the thermal performance curve (*Bu*′(*t*); red line) shifts vertically. (C) A knock-out (k.o.) of DNMT3a (*S*′) – the principal enzyme laying down new DNA methylation code in response to environmental signals – permits an estimate of the 'epigenetic potential' [*Ax*(*t*)] indicated in the decrease of the performance curve of *S*′ (red broken line) compared to *S*. Data redrawn from Loughland, Little, and Seebacher (2021). Image of zebrafish from PhyloPic.

Plasticity or elasticity in biology can be readily interpreted in terms of the model presented above. For example (Figure 4A), the dependence of locomotor capacity (internal state variable, *x*) of an organism (*S*) on temperature (external input, *u*) in its developmental environment are characterised by its thermal performance curve [*Bu*(*t*)]. When exposed to a 'new' environment (*u*'), the 'epigenetic potential', *Ax*(*t*), will determine how the organisms will respond, potentially leading to an altered performance curve [*Bu*′(*t*)] (Figure 4B) that may (elastic acclimation) or may not (e.g., developmental plasticity) return to its original state *Bu*(*t*). *Ax*(*t*) emerges from the network-like interactions of different mechanisms briefly discussed above, and it determines if and by how much the thermal performance curve can change in response to inputs from the environment. All parameters in this model except for *Ax*(*t*) can be readily measured empirically. *Ax*(*t*) can be estimated by manipulating pathways such as thyroid hormone or DNA methylation that are known to be involved in mediating elastic or plastic responses. For example, comparing thermal performance curves between wild-type and a knock-out zebrafish model that limits DNA methylation provides an estimate of the potential of DNA methylation to alter phenotypes in response to developmental temperatures (Figure 4C). As pointed out above, there are responses to the environment that are much closer to elasticity and others that are much closer to plasticity, but sometimes the yield point is not clearly defined. However, responses to developmental environments tend to be more permanent than acclimation, albeit not necessarily fixed.

Application of the model to brain adaptation

The concept of plasticity, as we have defined it earlier, implies the capacity for lasting change. In agreement with this conceptualisation, the term plasticity has been used in neuroscience to describe the capacity of the brain to change and adapt to environmental stimuli (DeFelipe 2002). However, the concept

of plasticity remains ambiguous, especially in how the nature of plastic changes in the brain should be understood (Stahnisch and Nitsch 2002; Dunnett 1999). To progress into this discussion, we need to introduce some additional concepts first. In the neurosciences, stress can be seen as a mismatch between a favourable state, whether physiological, psychological, or social, and the state in which an organism is at any given moment in time (de Kloet and Joëls 2013).

Considering the link between stress and adaptive responses, it is easy to intuitively grasp that stressors may trigger plasticity responses in the brain. However, the nature of these plastic changes remains under intensive investigation. The consensus is that molecular and cellular changes in the brain, including hormones and neurotransmitters, can adapt the function of neuronal networks and thereby impact behaviour (McEwen 2012). Using stressors as an example of environmental factors that disrupt homeostasis, the development of mood disorders has been linked to the disruption of brain plasticity by chronic stress (Pittenger and Duman 2008). This process can be discussed within the framework of the minimal model of plasticity we present here.

Stress, or more accurately, the reaction to a stressor, is classified as acute or chronic, depending on the length of the exposure to the stressor. Acute stress is commonly associated with an adaptive response, while chronic stress is associated with maladaptive responses leading to vulnerable phenotypes (de Kloet, Joëls, and Holsboer 2005). The acute exposure to stressors triggers dynamic, adaptive physiological responses to restore homeostasis. A first arousal phase leads to rapid activation of the sympathetic nervous system and widespread release of (nor)adrenaline. If the arousal triggered by the stressor is appraised as potentially destabilising, a peak in the release of adrenal glucocorticoids follows within minutes, which resolves to pre-stress glucocorticoid levels if the stressor is removed, or the organism's coping response is successful (de Kloet, Joëls, and Holsboer 2005). The reversible glucocorticoid reaction to a stressor is, therefore, a clear physiological example of the concept of elasticity, as defined before. In the absence of stressors, circulating levels of glucocorticoids are determined by well-described circadian oscillatory rhythms (Fitzsimons et al. 2016), which are transiently overwritten by the stressor-induced glucocorticoid peaks in a dynamic equilibration process (Lightman and Conway-Campbell 2010), further emphasising the validity of the application of the concept of elasticity to the dynamic equilibration associated with the adaptive response to acute stressors.

However, one of the most common adaptive responses to stress is glucocorticoid-mediated memory modulation, implying underlying long-term changes in brain function (McReynolds et al. 2014) that may be more easily assimilated to the concept of plasticity, as defined here. Indeed, at the cellular level, brain plasticity is associated with LTP, in which the efficacy of specific synapses is increased following high-frequency stimulation (Shors and Matzel 1997). To induce LTP, a bout of high-frequency stimulation is applied. The existence of specific frequencies and durations necessary to reach LTP intuitively refers to the concept of yield that we discussed in previous sections. As the high-frequency stimulation reaches a certain threshold, the neuron transitions to a different, sometimes permanent, state characterised by a higher firing potential (Abraham et al. 2024).

A straightforward mapping between our model and LTP can be made as follows. When a synapse (S) receives electrical stimulation (u), its internal state variables (x) respond with a certain magnitude (determined by B), and with a certain time course (determined by A). When stimulation is brief, the internal state of the synapse, x, will not have enough time to reach the 'elasticity-to-plasticity' yield point. Any changes in the synapse's internal states will revert to baseline. With prolonged stimulation, however, x may reach the yield point, and the synapse may be potentiated ($B\uparrow$), which can be mechanistically accomplished by adding postsynaptic receptors. Next time this synapse is stimulated by a similar input, it will respond more strongly because of this plastic change in its configuration.

Acute stress and the transient increase in glucocorticoids sometimes result in LTP induction and in some others in inhibition (Bartsch et al. 2021), and factors such as the type of stressor, the phase of the stress response, the type of LTP, and the life history of the organism may determine in which direction LTP is changed (Joëls and Krugers 2007). However, if the stress response is excessive and prolonged (chronic stress), it results in maladaptive changes and may increase disease risk (Kloet, Joëls, and Holsboer 2005). Chronic stress induces a sustained increase in glucocorticoid levels, which in turn suppresses LTP and other forms of brain plasticity, such as the generation of new neurons in the hippocampus, and leads to atrophy and retraction of dendrites (Joëls and Krugers 2007; Gould and Tanapat 1999; Pittenger and Duman 2008). The effects of chronic stress and elevated glucocorticoids on dendritic retraction are reversible to a certain extent but render the brain more vulnerable to harm, suggesting a plastic change towards a different state with a higher susceptibility to damage (Conrad 2008). Indeed, beyond neurobiology, the concept of yield can be applied in psychology to signify a transition to permanent changes induced by stress. Before this point, stress is experienced as elastic strain and is temporary, associated with resilience, while passing the yield point results in plastic strain, a permanent, less reversible impact, such as severe burnout from chronic excessive demands.

Application of the model to psychology, sociology, and cultural evolution

To demonstrate the applicability of our model beyond our home disciplines, we now turn to a process on a higher level, one that is uncovered in the domain of social science research. Showing the relevance of analysing plasticity in the context of our minimal model at this level will hopefully demonstrate the utility of our approach.

In the social sciences, plasticity may be used to understand the capacity of social entities, behaviours, and institutions to change over time and in response to experience or context, rather than being rigid or fixed. In this respect, the concept of plasticity has been used to study the unique events that led to the Industrial Revolution in England (Baumard 2019).

Ever since the Industrial Revolution took place in England in the eighteenth century, Western societies have experienced an unprecedented and sustained rate of economic growth. Why did the Industrial Revolution happen then and there? Some explanations focus on unique plastic changes at the societal level that were either external to England at the time (B), such as trade, or internal factors (A), such as institutions. At this level of description, elastic changes in the overall state of England (x) crossed some threshold that induced structural alterations in how external circumstances (B) or previous internal states (A) drive the future state of England's economy and innovation. For instance, with better institutions in place, England may have fostered a climate in which innovations can accrue more stably (see Baumard 2019).

Alternatively, the Industrial Revolution may have been the result of plastic changes at a lower level of description, namely that of psychology. Baumard (2019) argues that a steadily increasing standard of living induces plastic increases an individual's long-term perspective, optimism, intrinsic motivation, and social trust, which Baumard calls an 'affluence mindset'. In terms of our model, being raised in a stable, high standard of living (as opposed to poverty) structurally changes how an individual relates to their surroundings (B) and their propensity to exhibit specific stable or flexible behaviours (A). These plastic changes are relatively persistent across an individual's lifespan and, to the extent that such a collective affluence mindset fosters innovation at a societal level, may explain why the Industrial Revolution happened in England, where the standard of living exceeded all of its contemporaries (Baumard 2019).

It should be noted, however, that societal- and individual-level explanations of sociocultural phenomena do not necessarily exclude each other. As noted earlier, application of any model requires abstraction, and the level of abstraction may influence whether plastic changes are attributed to a system

or not. Moreover, applying our model at several levels at the same time may clarify how they are interrelated. For instance, plastic changes in individual mindset may also induce plastic changes at higher levels of abstraction, such as at a societal level. Although it is beyond the scope of this manuscript, our model paves the way to demarcate whether the industrial revolution can be cashed out in terms of plasticity and plastic change. Hence, future research should use actual data, as presented in Baumard (2019), to check if indeed a yield point is observed in the economic variables of the society in England in the eighteenth century. In other words, future research should identify changes in the structural parameters that govern economic variables during the industrial revolution to determine if it can be modelled as a plasticity process. We hope to have shown here that conceptualising elastic and plastic changes at the level of individuals and societies in terms of our minimal model may bring some clarity as to whether, and at what level, plasticity plays an explanatory role in social and cultural change.

Discussion of the model in the context of the visual arts

We also suggest that our model can be utilised to foster inter- and transdisciplinary synergies. Here, we briefly discuss its potential in the context of dominant artistic movement change (e.g., Impressionism, Expressionism, Surrealism, Dada, and Hyper-Realism).

Although ontologically different, different spheres of natural (human) systems interact, and plasticity is a unifying concept that connects different real-world components. For example, the material environment, characterised by resources and technology act on individual perception to drive social change, as in our example on the history of the Industrial Revolution above. Changes in historical context also provide the external input to artistic expression. By analogy with our more detailed examples from engineering, neurobiology and biology above, plasticity in art, for example, can be driven by historical context – captured by B(u) in our model – which impacts the internal state (x) of the artist or art movement conditioned by the previous state A(x). Examples include the influence of the First World War on Dadaism and the work of painters such as Otto Dix and George Grosz (Richter 1978) or the plasticity expressed by Disrupted Realism in its reshaping of Realism to introduce another dimension to challenge the viewer (Zmijarević 2025). Art is intrinsically linked to neuroscience via perception, emotion, and cognitive functions. As such, the interaction between neuroscience and art is very much driven by plasticity in either field. Hence, there is a direct link between the disciplines we focussed on here – plasticity in the material world, neuroscience, and biology – and the broader range of culture and history (Malafouris 2013). Our model permits dialogue between these fields, and the likely impact of the First World War on art, for example, likely occurred via neuroplasticity and epigenetics in the first place. A more detailed analysis of these and similar connections requires a common 'language' for comparisons and synthesis, which our model provides.

Discussion

Despite the central role of the concept of plasticity in sciences and arts, currently, its meaning varies substantially across disciplines. In this paper, we have developed a minimal model of plasticity that can be applied to systems under study in various disciplines and at different levels of description. As we have seen, the model emulates the formal demarcation between elasticity and plasticity in the physical sciences. At the same time, the model can make sense of the difference between elasticity and plasticity in more complex systems, such as synapses, brains, and phenotypes, as the model is agnostic as to what constitutes a system. Hence, if the phenomena of interest can be modelled as a dynamical system, as we have done in our application of the model in physics, biology, and neuroscience, our model may

apply to many other disciplines, such as the social sciences. We have also discussed our model in the context of social sciences and art, in which we show how our model can be used to understand persistent transitions in their study systems. Indeed, in other disciplines such as psychology, dynamical systems approaches have a long history (Chemero 2009), and as such, our approach may be fruitfully applied to existing dynamical systems models to explore plastic changes in their respective domains.

When developing this model, we noticed that conceptions of plasticity are often specific to one's discipline. Principles or mechanisms that are central to one discipline may seem obscure or hard to understand to another. Although the term plasticity carries different technical meanings across disciplines, the terminologies surrounding it partially overlap in ways that both enable and complicate interdisciplinary dialogue. In everyday and scholarly contexts alike, plasticity tends to evoke images of flexibility, malleability, and the capacity for transformation. However, the overlap is only partial, and the degree and scope implied by plasticity vary greatly across fields. In such situations, a common ground is needed. In this paper, we developed such a common ground in the form of mathematical equations that can be used to distinguish elastic from plastic changes. During the application of this minimal model, it became much clearer to us what functional role a certain mechanism (such as deformation of molecular bonds, LTP, and DNA methylation) plays in explaining plasticity.

The model developed in this paper is a first attempt at a formalisation of plasticity across disciplines. As such, we have stripped away several complexities. For instance, many real-world systems exhibit nonlinear dynamics. Although these systems should be modelled by non-linear dynamical systems, our reasoning still applies. If the system has changed some of its structural parameters that govern its nonlinear dynamics, this should be considered as a plastic change. Given the complexity of nonlinear dynamics, however, it may be more difficult to tell apart elastic from plastic changes. For instance, elastic changes (i.e., changes in the state of the system, defined within its phase space) may appear to be plastic because the system enters a portion of the state-space that has not been observed before. The new behaviour of the system may naively be ascribed to a change in A and/or B, when there was none.

Our model has a significant added value to the plasticity literature. Its primary focus is on formalising the difference between plastic and elastic changes in general. Thus, we do not necessarily claim that our minimal model can shed light on the details of each plasticity process when viewed from within its discipline. The proposal is that the epistemological strength of our model is achieved by distancing plasticity from simple folk ideas of capacity for change or from simplistic reversible vs. non-reversible criteria (recall that a fracture is also an irreversible change). Its strength lies in its concrete entanglement with plasticity in the domain of time and the interplay between the internal and external through the language of time-dependent differential equations.

The interdisciplinary nature of this work

The collaboration we forged between neuroscience, biology, and physics in this work created exciting opportunities to deepen our understanding of plasticity, but it also presented significant challenges. Each field contributed unique strengths: physics provided a well-characterised and defined framework, based on materials science, on which we could base our conceptualisation, while neuroscience and biology provided application to more complex systems where the variables involved in change are not always explicit or well-characterised. However, merging these disciplines was not always straightforward. Each field has its own language and priorities, which can lead to communication barriers and differing research approaches. A deep and open conversation about these intrinsic limitations helped us to resolve potential contradictions. Bridging our discipline-based perspectives required patience, collaboration, and a willingness to step outside traditional academic comfort zones. Some technical challenges also arose, for example, when discussing and integrating different levels of description, as we discussed

in previous sections. Despite these obstacles, multidisciplinary approaches helped us to advance the general conceptualisation and application of the term plasticity, and therefore we hope that scientists of other disciplines will find our work valuable and applicable to their own discipline-based interpretation of plasticity, but also their interdisciplinary attempts to understand plasticity across fields.

Conclusions

What makes a change plastic? The answer to this question varies widely, depending on whether you ask a physicist, neuroscientist, or biologist (or social scientist, or artist). In this paper, we have developed a 'minimal model' of plasticity that attempts to unify the concept of plasticity through the common ground of mathematics and dynamical systems. Our model can be used to clarify how a system under study changes in the elastic regime, and, after it passes a yield point, experiences a plastic change that modifies the behaviour of the system. We hope that our model will prove useful in an interdisciplinary discussion about a concept that takes such a central role in the sciences and arts.

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