knew what it was like to feel unfairly attacked by the establishment: a leading scientific authority, Peter Medawar, had written a brutal review of Koestler's previous book *The Act of Creation*.

So who was the fraud, Kammerer or Mendel? After Mendel published his paper on peas, he was heavily criticised by botanist Carl Nägeli, who encouraged him to repeat his crossing experiments with hawkweeds. Mendel bred hawkweeds for five years but failed to replicate the pea results and became disheartened. We now know that hawkweeds are exceptional in reproducing by apomixis. By contrast, Kammerer reported in his book *The Inheritance of Acquired Characteristics* that he found his ideas confirmed wherever he looked. Disbelieving critics were blinkered, even though others failed to replicate his findings.

A hallmark of a good scientist is how they respond to criticism — are they prepared to question their own ideas and findings, or do they become defensive and attack their critics? Given Mendel's response to Nägeli's criticism, I doubt that he consciously manipulated his results. He may have been selective about which results he presented, just as scientists commonly publish their best image or exclude data that they consider unreliable, but that is very far from Koestler's accusation of fakery. Kammerer's attitude, by contrast, betrays one of a fraudulent or self-deluded scientist. Koestler's defence may have more to do with his own anger at the scientific establishment for its criticism of him than his having a real case to argue. There are many ingredients that go into making a good scientist, but being self-critical and taking the criticism of others on board are surely important ones.

What do you think are the problems science as a whole is facing today?

On the morning of 9th November 2016 I was due to give a talk at University College London. I'd just heard that Donald Trump had been elected US President. How could so many people have voted for a blatant liar? I'm usually nervous about giving talks, but in this case giving a presentation to a scientific audience was the best therapy that I could have had. I was surrounded by people who evaluated ideas based on evidence and logical consistency, not rants and hearsay. I felt incredibly privileged to belong to a community insulated from the post-truth era.

Science, particularly the peer-review system, is often criticised for its lack of transparency. Surely there is a better way, modelled on social media, with everyone having access to who says what. That assumes, however, that scientists don't take criticism personally. Early on in my career I was discussing a scientist's work with him at a conference. He began to suspect that I had reviewed his paper, which had been recently rejected from *EMBO Journal*. When he asked me outright whether I was a reviewer, I saw no harm in admitting that I was and explaining the reasons for my decision. He was still on my case three hours later.

We invest so much time, effort and emotion into producing a paper that it is understandable that we take it personally if it is rejected or criticised. I get a Pavlovian wrench in my stomach when I see an e-mail from a journal in my inbox, informing me of its decision. A day or so later, I am better placed to understand the reasoning behind the comments, whether it is a lack of clarity on my part or a flaw in the work. The paper invariably ends up improved.

The impact of rejection causes some scientists to blame the system: there are vindictive ignoramuses out there who are hiding behind the blanket of anonymity. Take away the blanket and all will be solved. I doubt it. Worse, it may engender dishonesty for fear of offending influential peers or provide an incentive to flatter them. The more funding that depends on peer review, the more acute the problem is likely to be. I am not saying that the current system is perfect or cannot be improved. Nor am I saying that all reviewers are completely fair. I'm saying that, for all its imperfections, science is a jewel that we should treasure, and we need to tread very carefully when trying to introduce improvements, lest we inadvertently cause untold damage.

DECLARATION OF INTERESTS

The author declares no competing interests.

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Active vision gates ocular dominance plasticity in human adults

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Primary visual cortex (V1) retains a form of plasticity in adult humans: a brief period of monocular deprivation induces an enhanced response to the deprived eye, which can stabilize into a consolidated plastic change[1,2] despite unaltered thalamic input[3]. This form of homeostatic plasticity in adults is thought to act through neuronal competition between the representations of the two eyes, which are still separate in primary visual cortex[4,5]. During monocular occlusion, neurons of the deprived eye are thought to increase response gain given the absence of visual input, leading to the post-deprivation enhancement. If the decrease of reliability of the monocular response is crucial to establish homeostatic plasticity, this could be induced in several different ways. There is increasing evidence that V1 processing is affected by voluntary action, allowing it to take into account the visual effects of self-motion[6], important for efficient active vision[7]. Here we asked whether ocular dominance homeostatic plasticity could be elicited without degrading the quality of monocular visual images but simply by altering their role in visuomotor control by introducing a visual delay in one eye while participants actively performed a visuomotor task; this causes a discrepancy between what the subject sees and what he/she expects to see. Our results show that homeostatic plasticity is gated by the consistency between the monocular visual inputs and a person's actions, suggesting that action not only shapes visual processing but may also be essential for plasticity in adults.

We used a purpose-built altered-reality system that projected the outside world onto two independent monocular screens, with the image for

the dominant eye delayed by 330 ms (Figure 1A). Moving objects elicited diplopic vision, of which participants were readily aware (reporting a smeared tail for moving objects), given that 330 ms delay is much longer than the typical visual integration time. Static portions of a scene (>80%) produced no discrepancy between the two monocular images, hence drove normal binocular fusion. Note that eye movements, which change the point of gaze, do not cause delay-dependent spatial smearing: this occurs only when there is motion or change of the external world. Participants performed a visuomotor coordination task (tower-building with wooden blocks) while wearing the goggles. The delayed-eye image was useless and misleading for this task, as it placed all moving objects in the scene at a systematically erroneous (delayed) position.

After one hour exposure to monocular delay, we found a shift of ocular dominance, measured by binocular rivalry as a boost of the delayed eye (Figure 1B). This effect is similar to that produced by standard two hour monocular deprivation[1], in which the dominant eye is visually deprived with a translucent patch (Figure 1D). The boost following exposure to monocular delay is not the consequence of an automatic suppression of the delayed eye, given that sensitivity in the two eyes remained constant (see control experiment and Figure S1C,E in the Supplemental information), in line with the participants' subjective reports (see Supplemental information). Nor was the boost due to voluntary occlusion of the delayed eye, given that participants reported seeing a smeared tail trailing behind moving objects during the task. Despite the lack of monocular suppression, merely delaying vision in one eye elicited a similar homeostatic plastic response as visual deprivation, with a strong enhancement of dominance of the manipulated eye.

Is it the lack of consistency between participants' actions and the delayed eye that elicits plasticity, or is monocular delay by itself sufficient for the effect? We tested again the same participants in the same altered reality set-up with monocular delay, but without active visuomotor coordination. They passively observed an experimenter performing the tower-building task for one hour.

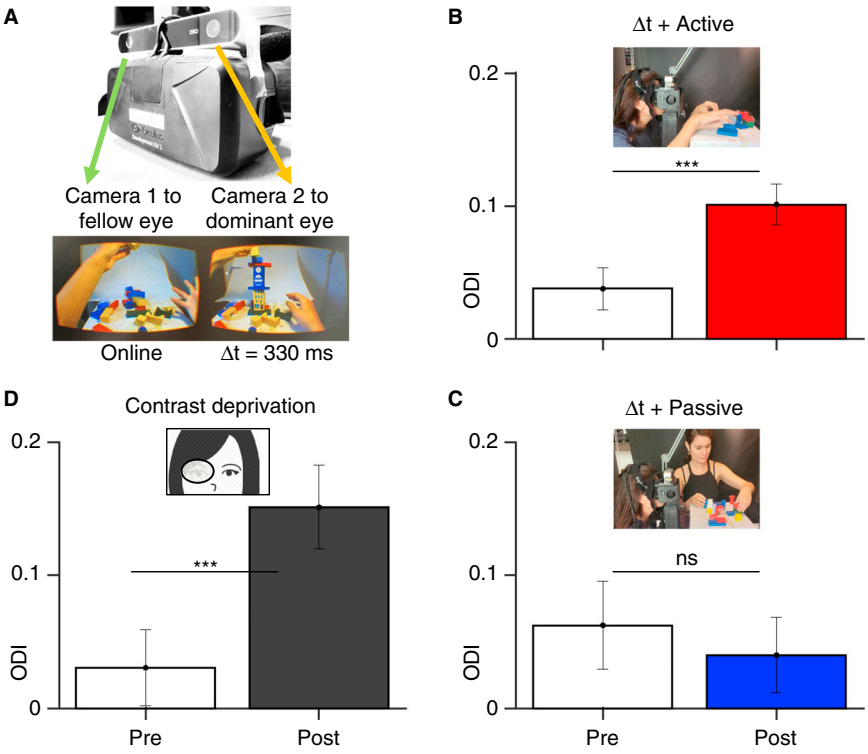

Figure 1. Set-up to deliver delayed vision in one eye and ocular dominance changes after exposure to delayed vision.

(A) An Oculus system equipped with a Zed stereo camera captured two independent monocular views of the participants' visual field. The image delivered to the dominant eye was delayed by 330 ms, producing diplopia for any visual transient or moving object, while ensuring binocular fusion for all static features that covered >80% of the visual image. (B) Participants (N = 27) actively performed a tower-building task with wooden bricks for 1 h. We measured ocular dominance with binocular rivalry and found enhanced dominance of the delayed eye after the exposure to the monocular delay (paired-sample t-test: $t(27) = 4.93$, $p < 0.001$, base-10 logarithm of the Bayes factor or logBF = 2.8). (C) The same 1 h exposure to monocular delay failed to shift ocular dominance in participants (N = 12) who passively viewed an experimenter performing the same tower-building task ($t(11) = -1.36$, $p = 0.202$, logBF = –0.2). (D) The effect of monocular delay in panel B closely resembles the effect of monocular visual deprivation, obtained by patching the dominant eye for 2 h (N = 13, replicating previous studies, $t(11) = 6.77$, $p < 0.001$, logBF = 3.2). Across panels, bars show mean and SEM (see Figure S2 for individual participants' data) and symbols mark the significance of paired t-test comparing ocular dominance indices pre- *versus* post-exposure: ns (non significant), *** ($p < 0.001$).

Despite the monocular delay and similar perceptual reports of the participants, we found no systematic shift of ocular dominance (Figure 1C). The falling wooden blocks produced sounds on both the active and passive conditions; nevertheless, only the active condition elicited an ocular dominance shift, suggesting that audio-visual integration is not sufficient to boost the signal in the delayed eye.

Our findings indicate that the signal gating visual plasticity has a motor or somatosensory origin, related to the voluntary execution of actions. This is likely to be generated outside primary visual cortex, but for this signal to affect ocular dominance it must operate in primary visual cortex, as this is the only cortical area where the two eyes are represented separately[4,5]. In principle, this gating signal does not need to be monocular or to act independently on the neuronal ensemble of the two eyes. It could be agnostic of the neuronal eye preference and could operate indiscriminately on all neurons by regulating their gain. However, only visual neurons that are active, for example responding to visual motion, at the time of arrival of this non-visual signal[8] will be affected (see Supplemental information). Monocular visual responses that are out-of-sync

with the action could be gain-regulated during the dichoptic stimulation, and consequently subject to homeostatic enhancement after normal binocular synchronous vision is re-established. This hypothesis is consistent with previous evidence that suppression from consciousness of monocular signals, without visual deprivation, can also promote a homeostatic shift of ocular dominance[9,10]. More generally, the idea of a top-down signal gating homeostatic plasticity provides a novel framework for understanding the effects of short-term deprivation. We suggest that homeostatic plasticity could be driven by mismatch between contingent sensory signals and sensory signals anticipated by voluntary action.

Clearly, the de-synchronization of contingent and expected visual information produces large errors during voluntary actions, but also monocular contrast deprivation is associated with an increase of visuo-motor error, as the patched eye fails to transmit the visual changes produced by the participant's own movements. Besides reinterpreting the impact of physical exercise and environmental enrichment in modulating primary visual cortical plasticity[1], this model also highlights the importance of predictive and/or multisensory error signals in shaping visual processing based on one's own actions.

In conclusion, our results show that ocular dominance plasticity is not only related to the balance of monocular V1 representations but also gated by internal motor and/or cognitive information, suggesting that homeostatic plasticity is at the service of sensory-motor coordination.

SUPPLEMENTAL INFORMATION

Supplemental information includes two figures and experimental procedures, and can be found with this article online at https://doi.org/10.1016/j.cub.2023.08.062.

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AUTHOR CONTRIBUTIONS

G.S.: Methodology and Resources; C.S.: Investigation; P.B.: Formal Analysis and Resources; P.B., M.C.M.: Writing – first draft; P.B., C.S., M.C.M.: Conceptualization; All authors: Writing – Review & Editing.

DECLARATION OF INTERESTS

The authors declare no competing interests.

INCLUSION AND DIVERSITY

We support inclusive, diverse and equitable conduct of research.

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Genomic insights into the mystery of mouse mummies on the summits of Atacama volcanoes

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Our understanding of the limits of animal life is continually revised by scientific exploration of extreme environments. Here we report the discovery of mummified cadavers of leaf-eared mice, *Phyllotis vaccarum*, from the summits of three different Andean volcanoes at elevations 6,029–6,233 m above sea level in the Puna de Atacama in Chile and Argentina. Such extreme elevations were previously assumed to be completely uninhabitable by mammals. In combination with a live-captured specimen of the same species from the nearby summit of Volcán Llullaillaco (6,739 m)[1], the summit mummies represent the highest altitude physical records of mammals in the world. We also report a chromosome-level genome assembly for *P. vaccarum* that, in combination with a whole-genome re-sequencing analysis and radiocarbon dating analysis, provides insights into the provenance and antiquity of the summit mice. Radiocarbon data indicate that the most ancient of the mummies are, at most, a few centuries old. Genomic polymorphism data revealed a high degree of continuity between the summit mice and conspecifics from lower elevations in the surrounding Altiplano. Genomic data also revealed equal numbers of males and females among the summit mice and evidence of close kinship between some individuals from the same summits. These findings bolster evidence for resident populations of *Phyllotis* at elevations >6,000 m and challenge assumptions about the environmental limits of vertebrate life and the physiological tolerances of small mammals.