rapidly upregulated are almost all those required for synthesis of the translational apparatus. Perhaps most interesting was the identification of approximately 80 transcripts encoding proteins with unknown functions. Thirteen of these genes are defined as encoding cyanobacterial signature proteins [6], and another encodes a putative homolog of a DNA-surveillance protein identified originally in sporulating *Bacillus subtilis* [7].

Over 100 non-coding, small regulatory RNAs are either upregulated or downregulated during resuscitation [3]. Some of these small RNAs might encode peptides but many have predicted antisense functions, consistent with the proposed post-transcriptional control of the dormant state. Recent data from studies on seed germination [8] and control of the hibernation process in higher animals [9] also indicate a key role for small regulatory RNAs in controlling these processes. Other parallels between these processes and chlorosis are striking: the requirement for energy-rich storage compounds; the reprogramming to a hypometabolic state; a drastic reduction in ATP synthesis and expenditure; and the development of stress-tolerant phenotypes.

Understanding dormancy from a mechanistic viewpoint is still in its infancy. The current study [3], however, identifies a clear genetically controlled program of events that governs awakening from the dormant state. The authors have defined a wonderful model system to dissect not only how an organism recovers from the dormant state but also how dormancy is maintained. Future studies on *Synechocystis* will expand our understanding of the bioenergetic processes controlling chlorosis, possibly even revealing new concepts. This field will eventually provide answers to how certain microbial cells (including spores) can remain alive (maintain a membrane potential) for thousands of years or even longer [10,11]. In a broader biological context, this work has obvious relevance to our understanding of the evolution of life, bioenergetics, epigenetics, infection processes (persistence in pathogens and antibiotic resistance) [12], possibly also the regulation of cancer-cell dormancy [13], and even the aging process and longevity [9]. Figuring out how microbial cells enter into, and awaken from, dormancy will shed light on the mechanisms of similar processes in higher organisms.

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Homeostasis: How Neurons Achieve Temperature Invariance

Jan-Hendrik Schleimer[1,2] and Susanne Schreiber[1,2,*]
[1]Institute for Theoretical Biology, Department of Biology, Humboldt-Universität zu Berlin, 10115 Berlin, Germany
[2]Bernstein Center for Computational Neuroscience Berlin, 10115 Berlin, Germany
*Correspondence: s.schreiber@hu-berlin.de
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Temperature influences physiological processes and can corrupt nervous system function. A modelling study shows how regulation of ion channel expression can establish an acute temperature invariance of neuronal responses despite temperature-dependent and variable ionic conductances.

'Heat what you need' seems to be the motto of fast-swimming fish such as opah, tuna and billfish which, although 'cold blooded', actively invest in elevating their muscle temperature [1,2]. Such heating does not always help these fish escape the plates of seafood restaurants across the world, but the resulting increase in swimming speed surely provides a decisive advantage for their predatory life in the open oceans. Temperature is an important parameter that influences not only muscle activity but also the activity of nervous systems.

For the brain, it is not only a question of speeding up performance but rather of maintaining functionality in the first place. In this issue of *Current Biology*, O'Leary and Marder [3] propose a homeostatic mechanism that permits to stabilize the output pattern of a bursting neuron across a wide range of temperatures.

Temperature can impact nervous system function in a variety of ways. Overall, temperature modifies the speed of physiological processes, slowing them down or speeding them up. In the brain, such processes include synaptic transmission or the generation of action potentials, predominantly via ion and receptor channel kinetics and the diffusion properties of ions across the membrane [4]. On short time scales, two effects are most relevant for electrical activity: first, two- to four-fold increases in the speed of ion channel opening and closing on a temperature rise of 10°C; and second, somewhat more moderate increases in total current passing through an open channel [5]. As a consequence, temperature changes can result in acute imbalances of activity levels and timing, with the risk of compromising neural computations. The picture is yet more complex, as many different ion channels contribute to electrical membrane activity, each with their own temperature sensitivity.

Some animals, including mammals and birds, have resorted to an expensive solution to avoid these problems by investing energy to keep their body temperature constant — hence their description as homeotherms. Many species, however, do not possess an elaborate intrinsic heat regulation — these are the so-called poikilotherms. These latter animals are in need of strategies that ensure robust electrical activity in the face of temperature changes, usually across a dozen °C or more. In their new paper, O'Leary and Marder [3] suggest a cellular regulatory mechanism that makes the firing patterns of bursting neurons, such as those in the stomatogastric ganglion of the crab, robust to acute changes in temperature. While the frequency of burst events in these neurons is elevated when temperature rises, the relative fraction of time a neuron actually fires action potentials — the 'duty cycle' — remains constant. This work relies on the authors' [6,7] earlier model for a self-regulatory mechanism of ion channel expression that allows neurons to keep a fixed 'activity set point' even when perturbed. In their new work [3], the authors show how such a regulation can maneuver a cell towards ion channel expression levels where relevant properties of neuronal output are stable if temperature varies.

The work of O'Leary and Marder [3] is an instructive example of how computational studies can deepen our understanding where experimental work would not be feasible (yet). The authors' approach is based on a mathematical model of action-potential generation. Conductances and their dynamics are described by differential equations, just as in the classical Hodgkin–Huxley model [8]. In a first step, the authors numerically explore the space of possible conductance parameters — ion channel peak conductances, opening and closing speeds, and their respective temperature dependencies — to identify those combinations that allow for temperature-invariant duty cycles. Next, they show how model cells can start out from random initial distributions of channel densities and converge towards a temperature-robust solution when primed with parameters derived from one of the robust conductance sets identified in the first step. The regulation mechanism assumes fixed correlations between the expression rates of individual conductances, inspired by the experimental observation that the ratios between conductance expression levels persist over time within a cell, while their absolute values can vary substantially [9]. In this way, once the gene regulating mechanism has reached its steady state, the temperature-robust models have identical ratios of conductance densities.

At the heart of the computationally identified temperature-stable models lies the principle of balancing opposing forces [4,10]. It implies that two or more processes with antagonistic temperature dependence cancel out. In general, this fundamental principle can be exploited at many different organizational levels, of which the single cell seems to suggest itself. As in the work of O'Leary and Marder [3], a few previous studies have identified compensatory mechanisms acting on the cellular level, including robust firing rates [11] and circadian rhythms [10]. For a robustness of firing rates, as observed in locust auditory receptor neurons, the opposing forces were similar to those in the models of O'Leary and Marder [3]: peak conductances and ion channel kinetics [11]. If selectively balancing those parameters with an opposite effect on rate, the net impact of temperature on firing can be alleviated. For a robustness of the circadian rhythm, stabilizing and destabilizing chemical reactions were combined to limit the net effect on oscillation period [10].

These cellular approaches seem adequate when the relevant functional readout is strongly influenced by a single neuron's output, as for example in the case of the pyloric rhythm [12]. At more macroscopic scales or even the behavioural level, it may seem worthwhile to invest in alternative compensatory mechanisms that do not necessarily require absolute temperature stability of cells. What these may be and how single-cell and network compensatory mechanisms interact or even interfere with each other remain to be explored.

As O'Leary and Marder [3] argue, the solution space — the number of possible temperature compensated models — decreases if more constraints need to be fulfilled. For example, only very few models can simultaneously keep both the duty cycle and the period of the burst rhythm constant. In the new study, a neuron is regulated towards a set of ion channel expression levels that, once reached, allow for acute temperature compensation of duty cycle (even if the expression levels do not change any more). It is tempting to speculate that additional homeostatic plasticity mechanisms may enlarge the space of possible solutions and thus increase the flexibility to incorporate additional constraints. Such plasticity mechanisms could continuously adapt gene expression levels for different temperatures on longer timescales (minutes to hours [13]), and — compared to the model suggested by O'Leary and Marder [3] — would require changes in the ratios of channel expression. Whether such ratios of expression levels actually vary across temperatures remains to be seen in experiments.

Returning to mammals and birds, our remarks above may have led the reader to

conclude that temperature compensation is not a problem in these homeothermic organisms. After all, can one not safely assume temperature to be among the least relevant parameters affecting neuronal function in these animals because of their formidable abilities to keep body temperature constant? Far from it! As the work of Moser and colleagues [14] more than twenty years ago has shown, heat from physiological muscle activity is sufficient to warm up a rodent's hippocampus by 2°C and, surprisingly, these temperature changes are sufficient to substantially modulate electrical brain activity, such as synaptic field potentials. While temperature variability in mammals and birds is generally smaller than in poikilothermic species, it can still result in strong repercussions, in particular, if brain dynamics are close to nonlinear transition points. For example, modest temperature increases are known to contribute to the induction of epileptic states, like febrile seizures [15]. In these cases, compensatory mechanisms may fail and further work is required to shed light on the underlying dynamics and to identify additional parameters that steer a system towards or away from those critical points. Altogether, temperature regulation is crucial not just for insects, lizards, or the like, but also affects homeothermic species, including us humans. Quite some work lies ahead to uncover the principles that ensure temperature robustness and homeostasis on functionally relevant levels, ranging from firing rates, timing of spikes [16], noise, metabolic efficiency [17] and network computations [18] to behaviour.

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Plant Biology: Flower Orientation, Temperature Regulation and Pollinator Attraction

Casper J. van der Kooi
University of Lausanne, Department of Ecology and Evolution, CH 1015 Lausanne, Switzerland
Correspondence: Casper.vanderKooi@unil.ch
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The reproductive performance of plants depends on the temperature of the flower. A recent study reports the mechanistic basis of flower head orientation in sunflowers and provides intriguing hints as to its functional significance.

Obtaining the right flower temperature is crucial for plants to reproduce. Increases in plant temperature can occur through absorption of direct solar radiation by plant structures and through thermal radiation of structures surrounding the plant. An effective way to increase absorption of solar irradiance is by tracking the sun, a phenomenon known as heliotropism or solar tracking. Leaf heliotropism is found in many species and regulates the amount of sunlight absorbed for photosynthesis [1]. Some plant species exhibit flower heliotropism, which aids in flower temperature regulation. One of the best-known cases