about the diversity and functions of torpor has been gained from work on free-ranging animals in the wild or using a comparative approach on many species from diverse habitats. Less progress has been made on understanding the mechanisms of the enormous functional differences between homeothermic and heterothermic species. For example, the reasons for the much higher thermal and ischemic tolerance of tissues of hibernators than those of homeotherms are still not understood, although these may have spin-offs for organ storage and other aspects of human medicine. Some potential applications include cardiac surgery or other organ transplants that have to be conducted at relatively high temperatures to avoid tissue damage, but could be better performed at low temperatures. Further, an understanding of the reasons behind the low muscle disuse atrophy in hibernators, despite extremely long inactive phases, has obvious implications for long-term hospital care.

Further reading

Barnes, B.M., and Carey, H.V. eds. (2004). Life in the Cold: Evolution, Mechanisms, Adaptation, and Application. 12th International Hibernation Symposium. Biological Papers of the University of Alaska #27. (Fairbanks: University of Alaska).

Geiser, F. (2004). Metabolic rate and body temperature reduction during hibernation and daily torpor. Annu. Rev. Physiol. *66*, 239–274.

Geiser. F., and Brigham, R.M. (2012). The other functions of torpor. In Living in a Seasonal World. T. Ruf, C. Bieber, W. Arnold, E. Millesi, eds. (Heidelberg: Springer Verlag), pp. 109–121.

Lane, J.E., Kruuk, L.E.B., Charmantier, A., Murie J.O., and Dobson F.S. (2012). Delayed phenology and reduced fitness associated with climate change in a wild hibernator. Nature *489*, 554–557.

Lovegrove, B.G., and McKechnie, A.E. eds. (2008). Hypometabolism in animals: torpor, hibernation and cryobiology. 13th International Hibernation Symposium. (Pietermaritzburg: University of KwaZulu-Natal).

Lyman, C.P., Willis, J.S., Malan, A., and Wang, L.C.H. (1982). Hibernation and Torpor in Mammals and Birds (New York: Academic Press).

Rojas, A.D., Körtner, G., and Geiser, F. (2012). Cool running: locomotor performance at low body temperature in mammals. Biol. Lett. *8*, 868–870.

Ruf, T., Bieber, C. Arnold, W., and Millesi, E. eds. (2012). Living in a Seasonal World. (Heidelberg: Springer Verlag).

Tattersall, G.J., Sinclair, B.J., Withers, P.C., Field, P.A., Seebacher, F., Cooper, C.E., and Maloney, S.K. (2012). Coping with thermal challenges: physiological adaptations to environmental temperature. Compr. Physiol. *2*, 2151–2202.

Centre for Behavioural and Physiological Ecology, Zoology, University of New England, Armidale NSW 2351, Australia.
E-mail: fgeiser@une.edu.au

Correspondence

Spatiotopic neural representations develop slowly across saccades

Eckart Zimmermann[1,*], Maria Concetta Morrone[2,3], Gereon R. Fink[1,4], and David Burr[5,6]

One of the long-standing unsolved mysteries of visual neuroscience is how the world remains apparently stable in the face of continuous movements of eyes, head and body. Many factors seem to contribute to this stability, including rapid updating mechanisms that temporarily remap the visual input to compensate for the impending saccade [1]. However, there is also a growing body of evidence pointing to more long-lasting spatiotopic neural representations, which remain solid in external rather than retinal coordinates [2–6]. In this study, we show that these spatiotopic representations take hundreds of milliseconds to build up robustly.

Aftereffects have proven to be an effective tool to study spatiotopy, as an eye movement between adaptation and test dissociates retinal from external space. We use the tilt aftereffect, previously shown to have a spatiotopic component [2] (but not without controversy [7]). Twelve subjects (11 naïve to the goals of the study) adapted to a tilted (±15°) grating patch, then saccaded on cue to a target 20° to the right. A similar-sized test grating of variable tilt then appeared at either the same screen or retinal position as the adapter, or an unmatched position (Figure 1A). Subjects reported whether it appeared to be tilted clockwise or counter-clockwise, and the proportion of clockwise responses fit with a Gaussian error function to calculate the tilt necessary to annul the illusion (see Figure S1 in the Supplemental Information). The magnitude of the aftereffect was defined as half the difference of tilt to annul the effects of adapting to clockwise compared with counter-clockwise gratings. Data were also collected without eye movements in a matched spatial location ('full adaptation') to give a baseline measure of the effect.

The key manipulation of this study was to display the saccadic target for a variable duration before cueing subjects to saccade (by the extinction of the fixation point; Figure 1A). Figure 1B shows the average strength of the tilt aftereffect, as a function of preview duration. Previewing the target before initiating the saccade reduced slightly the strength of both full adaptation and retinotopic adaptation, presumably because the aftereffect decreased with duration after adaptation (bootstrap sign test between 0 and 1000 ms: $p = 0.03$). However, target preview had the opposite effect in the spatiotopic condition, where adaptation increased with preview duration to reach the strength of retinotopic adaptation at 1000 ms. (bootstrap sign test between 0 and 1000 ms: $p < 0.001$; see Figure S1B in the Supplemental Information for individual results). Figure 1C illustrates this more clearly, plotting retinotopic and spatiotopic adaptation as a proportion of full-adaption strength. Whereas retinotopic adaptation remains quite constant, spatiotopic adaptation builds up over time, from 16% for the reactive saccades (similar to the control condition), to 67% of the full adaptation effect at 1000 ms. Bootstrap sign tests show that the spatial aftereffect is significantly different from the aspecific effect at 500 ms ($p < 0.001$) and 1000 ms ($p < 0.001$), but not at 0 ms ($p = 0.12$).

To be certain that subjects complied with instructions, eye movements were monitored on every trial, and trials discarded if the eyes did not arrive within 4° of target, or were not stationary on probe onset. This ensured that there was no spillage of retinotopic adaptation to the spatiotopic condition, and that in all cases the test overlapped with at least 50% of the adaptor grating. Supplemental Figure S2 plots the scatter of eye positions at the time of probe presentation for the various conditions, showing that there were no systematic biases that could explain the results. There was also no systematic variation in probe presentation time relative to saccade offset, or other aspects of the saccade dynamics were observed that could account for the differences (see Table in Supplemental Figure S2).

The results provide clear evidence for the existence of a spatiotopic representation of orientation, one that can be distorted by adaptation,

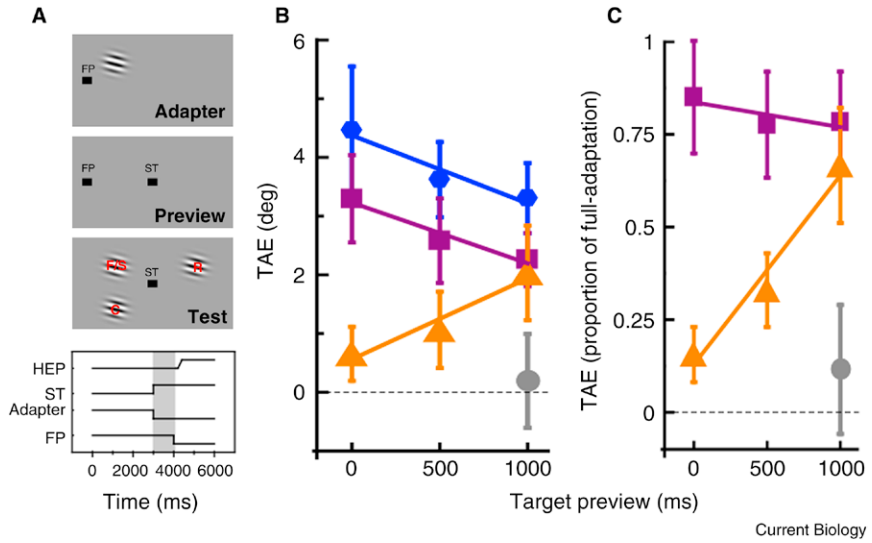

Figure 1. Dependence of tilt aftereffects on target preview.
(A) Timecourse of events in a trial. Each trial started with an adaptation period of 3000 ms in which subjects loosely fixated the fixation point FP, observing an adapter grating patch (0.8 c/deg, vignetted within Gaussian window of $\sigma = 3.5°$) tilted at +15° or –15°. The saccade target (ST) was then presented, to which subjects saccaded on extinction of the fixation point: either at onset of the saccade target, or 500 ms or 1000 ms later. The test target came on 300 ms after extinction of the fixation point, always at least 30 ms after the eyes had landed. The lower panel shows the timecourse of a typical horizontal eye movement (H EP), together with presentation times of stimuli. 300 ms after extinction of fixation point the test patch was presented for 51 ms in the spatiotopic (S), the retinotopic (R), or the control (C) position, and subjects indicated the direction of tilt of the test patch. (B) Tilt-aftereffect for the full-adaptation (blue), retinotopic (purple), spatiotopic (orange) and control (grey) conditions, as a function of preview duration of the saccade target, averaged over all subjects. Error bars represent ±1 SEM. (C) Normalized tilt-aftereffect results for the three eye-movement conditions (colour-coding as for B). Aftereffect magnitude was divided by each subject's full-adaptation magnitude, then averaged over subjects. Error bars represent ±1 SEM.

confirming many previous reports ([2–6]; albeit not Knapen *et al*. [7]). It is not clear why different results have been reported, but one possibility is that the experimental conditions of Knapen *et al*. [7] did not allow sufficient time for the spatiotopy to build.

Our data show that a spatiotopic representation is not available instantaneously, but becomes evident only after the saccadic target has been displayed for at least 500 ms before gaze-change. This suggests that the system needs time to compute the representation in the new reference frame, and a visual reference is necessary for this process: even though all trials in a given session were similar, with the saccadic target always displayed in the same position, spatiotopy did not build up unless the target had been displayed for 500–1000 ms. This is consistent with human electrophysiological recordings in the parahippocampal gyrus showing that activity related to allocentric spatial encoding is evident only 400–600 ms after stimulus onset [8].

Retinotopic adaptation was still very strong after 1000 ms, as strong as the spatiotopic adaptation and not diminished as a proportion of full adaptation, consistent with the fact that adaptation effects in early, retinotopic visual cortex (including V1) are known to persist for some time. That both forms of adaptation occur together points to a dual representation of space, in both retinotopic and spatiotopic coordinates, where the spatiotopic representation builds up slowly. This is consistent with studies showing a clear dissociation of retinotopic and spatiotopic effects for motion-induced adaption of duration [3] and position [6].

A second is a long time for vision, during which we will typically have made three saccades. This means that the time to build a robust spatiotopic representation exceeds that of a typical single fixation, continuing through several fixations, taking the retinal displacement caused by each new saccade into account. A map with such a sluggish timecourse would be of little value for maintaining online visual stability, pointing to the existence of other mechanisms for this purpose. Likely candidates are the proposals for 'predictive remapping' or 'transient spatiotopy' [9,10], based on the transient shifts of the receptive fields of visual neurons in many visual areas [1]. However, it is also clear that a stable spatiotopic representation of the world is constructed over longer periods of time. The precise function of this long-term spatiotopic map and how it contributes to our sense of a stable visual world is yet to be determined.

Supplemental Information

Supplemental information includes details of experimental procedures and two figures, and can be found with this article online at http://dx.doi.org/10.1016/j.cub.2013.01.065.

Acknowledgments

Supported by ERC grant STANIB, and Italian Ministry of Universities and Research.

References

1. Wurtz, R.H. (2008). Neuronal mechanisms of visual stability. Vis. Res. *48*, 2070–2089.
2. Melcher, D. (2005). Spatiotopic transfer of visual-form adaptation across saccadic eye movements. Curr. Biol. *15*, 1745–1748.
3. Burr, D.C., Tozzi, A., and Morrone, M.C. (2007). Neural mechanisms for timing visual events are spatially selective in real-world coordinates. Nat. Neurosci. *10*, 423–425.
4. Zimmermann, E. and Lappe, M. (2011). Eye Position Effects in Oculomotor Plasticity and Visual Localization. J. Neurosci. *31*, 7341–7348.
5. Zimmermann, E., Burr, D., and Morrone, M.C. (2011). Spatiotopic visual maps revealed by saccadic adaptation in humans. Curr. Biol. *21*, 1380–1384.
6. Turi, M., and Burr, D. (2012). Spatiotopic perceptual maps in humans: evidence from motion adaptation. Proc. Biol. Sci. *279*, 3091–3097.
7. Knapen, T., Rolfs, M., Wexler, M., and Cavanagh, P. (2010). The reference frame of the tilt aftereffect. J. Vis. 10(8), 1–13.
8. Bastin, J., Committeri, G., Kahane, P., Galati, G., Minotti, L., Lachaux, J.P., and Berthoz, A. (2012). Timing of posterior parahippocampal gyrus activity reveals multiple scene processing stages. Hum Brain Mapp. Jan 30. http://dx.doi.org/10.1002/hbm.21515. [Epub ahead of print].
9. Melcher, D., and Colby, C.L. (2008). Trans-saccadic perception. Trends Cogn. Sci. *12*, 466–473.
10. Cicchini, G.M., Binda, P., Burr, D.C., and Morrone, M.C. (2012). Transient spatiotopic integration across saccadic eye-movements mediates visual stability. J. Neurophysiol., [Epub ahead of print].

[1]Institute of Neuroscience and Medicine, Jülich, Germany. [2]Department of Translational Research on New Technologies in Medicine and Surgery, University of Pisa, Italy. [3]IRCCS Stella Maris, Calambrone, Italy. [4]Department of Neurology, University Hospital Cologne, Germany. [5]Department of Neuroscience, University of Florence, Italy. [6]Neuroscience Institute, National Research Council, Pisa, Italy.
*E-mail: ec.zimmermann@fz-juelich.de