cannot be directly compared with those seen in the Raman Fe–$N^{\varepsilon 2}$ stretch mode on photolysis[5], as several structural parameters determine its frequency and intensity[12]. However, our results can provide a basis for structural interpretation of the complex changes seen in this important spectroscopic marker.

The structure of the photolysed state provides new insight into the dynamics of the protein. Transient spectroscopic studies have shown that Mb*CO relaxes towards the unligated structure on the subnanosecond timescale at physiological temperature. Associated with this relaxation is a dramatic increase in the enthalpic part of the barrier to ligand rebinding from the pocket[3,4,25]. Comparison of the Mb*CO and unligated conformations allows us to identify the relaxation as an increase in the iron out-of-plane distance, a decrease in the Fe–proximal histidine bond length, a tilting of the proximal histidine and a decompression and motion of the F helix towards its junction with the E helix. These structural changes are functionally important, because an increase in the enthalpic barrier decreases the associated rate coefficient and thus lowers the ligand-binding affinity. The relaxation of the proximal haem environment from Mb*CO to unligated myoglobin is conceptually very similar to the allosteric R→T transition of haemoglobin.

We have used X-ray crystallography at liquid-helium temperatures to obtain snapshots of myoglobin before, during and after its reaction with carbon monoxide. Our results have been compared with information from spectroscopy and computation, and can help to decide the most fruitful approaches to questions about dynamics. Our detailed structural information about the unstable Mb*CO complex may be combined with the results of other approaches to make quantitative structural–kinetic models of the ligand binding process and to build a comprehensive picture of how a simple protein such as myoglobin works. □

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ERRATA

Multiple processing streams in occipitotemporal visual cortex

Edgar A. DeYoe, Daniel J. Felleman, David C. Van Essen & Evelyn McClendon

Nature **371,** 151–154 (1994)

EDITING of the opening sentence of the first paragraph of this Letter introduced a misleading error: the internal subdivisions given in parentheses should correctly read: "('blobs', 'interblobs' and layer 4B in V1; 'thin', 'thick' and 'interstripes' in V2)". □

The ancient regulatory-protein family of WD-repeat proteins

Eva J. Neer, Carl J. Schmidt, Raman Nambudripad & Temple F. Smith

Nature **371,** 297–300 (1994)

IN this Progress article the WD-repeating units for the TUP1 protein shown in Table 1 should have been coloured as shown here, and not all in black as published. Blocks are coloured according to their fit to the regular expression in Fig. 1 (black, no misses; blue, one miss). □

CORRECTION

Somatostatin-induced inhibition of neuronal Ca^{2+} current modulated by cGMP-dependent protein kinase

Stephen D. Meriney, D. Bruce Gray & Guillermo R. Pilar

Nature **369,** 336–339 (1994)

IN this Letter, our citation of the unpublished data of E. Butt and U. Walter was incorrect. We stated that Rp-8-pCPT-cGMPS and Sp-8-Br-cGMPS had been shown to be specific inhibitors and activators of cGMP-dependent protein kinase (cGMP-PK). In fact, Rp-8-pCPT-cGMPS is considered a selective inhibitor of cGMP-PK over cAMP-dependent protein kinase (cAMP-PK). (E. Butt and U. Walter, personal communication). Sp-8-Br-cGMPS is not thought to be selective for cGMP-PK over cAMP-PK as similar analogues do not show significant selectivity in cell-free systems (E.B. and U.W., personal communication). The pharmacological potencies of these compounds have not been determined in intact cells, therefore caution should be used to ensure their appropriate use. □