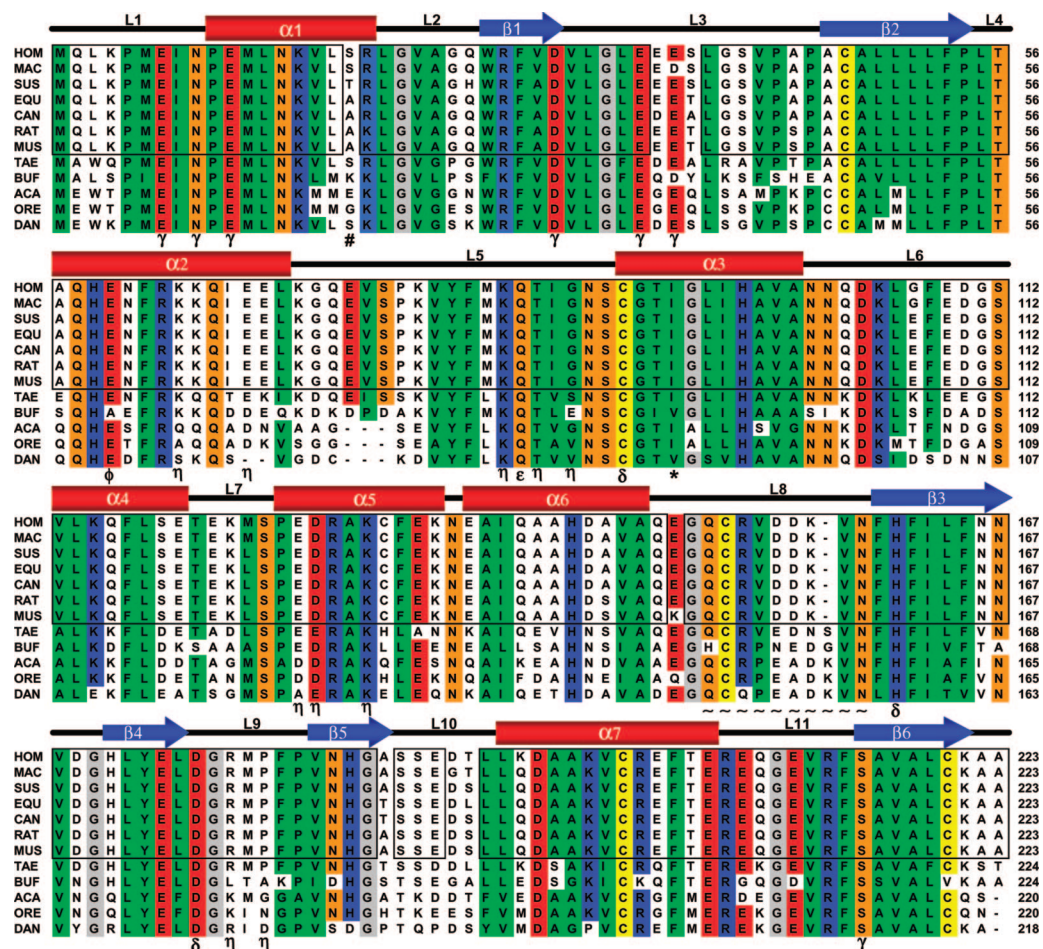




NEUROSCIENCE. For the article “Structural basis for conformational plasticity of the Parkinson’s disease-associated ubiquitin hydrolase UCH-L1,” by Chittaranjan Das, Quyen Q. Hoang, Cheryl A. Kreinbring, Sarah J. Luchansky, Robin K. Meray, Soumya S. Ray, Peter T. Lansbury, Dagmar Ringe, and Gregory

A. Petsko, which appeared in issue 12, March 21, 2006, of *Proc. Natl. Acad. Sci. USA* (103, 4675–4680; first published March 13, 2006; 10.1073/pnas.0510403103), the authors note that Fig. 1 appeared incorrectly. The corrected figure and its legend appear below. This error does not affect the conclusions of the article.



**Fig. 1.** Sequence alignment of UCH-L1 enzymes. Structure-based sequence alignment of UCH-L1 from different species is shown: HOM, *Homo sapiens*; MAC, *Macaca fascicularis*; SUS, *Sus scrofa*; EQU, *Equus caballus*; CAN, *Canis familiaris*; RAT, *Rattus norvegicus*; MUS, *Mus musculus*; TAE, *Taeniopygia guttata*; BUF, *Bufo gargarizans*; ACA, *Acanthogobius flavimanus*; ORE, *Oreochromis niloticus*; DAN, *Danio rerio*. The secondary structure elements of human UCH-L1 are indicated above the primary sequences, and conserved residues are highlighted (green, red, yellow, orange, and gray indicate conserved, hydrophobic, acidic, cysteine, polar, and glycine residues, respectively). Positions are identified as conserved if >80% of the residues are identical, or similar if hydrophobic in nature. δ, catalytic triad; ε, oxyanion hole; φ, H bonding with catalytic H161; γ, ubiquitin binding surface; η, P' cleft; #, mutation site that reduced susceptibility to PD; \*, mutation site that has been reported to cause familial PD; ~, the loop spanning the active site. Mammalian sequences are boxed.

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