

- 67 Maruyama, W. *et al.* (2002) The anti-parkinson drug, rasagiline, prevents apoptotic DNA damage induced by peroxynitrite in human dopaminergic neuroblastoma SH-SY5Y cells. *J. Neural Transm.* 109, 467–481
- 68 Du, Y. *et al.* (2001) Minocycline prevents nigrostriatal dopaminergic neurodegeneration in the MPTP model of Parkinson's disease. *Proc. Natl. Acad. Sci. U. S. A.* 98, 14669–14674
- 69 Le, W. *et al.* (2001) Microglial activation and dopaminergic cell injury: an *in vitro* model relevant to Parkinson's disease. *J. Neurosci.* 21, 8447–8455
- 70 Youdim, M.B. *et al.* (2001) Drugs to prevent cell death in Parkinson's disease. Neuroprotection against oxidative stress and inflammatory gene expression. *Adv. Neurol.* 86, 115–124
- 71 Mouradian, M.M. (2002) Recent advances in the genetics and pathogenesis of Parkinson's disease. *Neurology* 58, 179–185
- 72 Junn, E. and Mouradian, M.M. (2002) Human α -synuclein over-expression increases intracellular reactive oxygen species levels and susceptibility to dopamine. *Neurosci. Lett.* 320, 146–150
- 73 Kruger, R. *et al.* (2002) Parkinson's disease: one biochemical pathway to fit all genes? *Trends Mol. Med.* 8, 236–240
- 74 Chung, K.K. *et al.* (2001) The role of the ubiquitin-proteasomal pathway in Parkinson's disease and other neurodegenerative disorders. *Trends Neurosci.* 24 (Suppl.), S7–S14
- 75 Grunblatt, E. *et al.* (2001) Gene expression analysis in N-methyl-4-phenyl-1,2,3,6-tetrahydropyridine mice model of Parkinson's disease using cDNA microarray: effect of R-apomorphine. *J. Neurochem.* 78, 1–12
- 76 Rosen, D.R. *et al.* (1993) Mutations in Cu/Zn superoxide dismutase gene are associated with familial amyotrophic lateral sclerosis. *Nature* 362, 59–62
- 77 Gurney, M.E. *et al.* (1994) Motor neuron degeneration in mice that express a human Cu,Zn superoxide dismutase mutation. *Science* 264, 1772–1775
- 78 Wong, P.C. *et al.* (1995) An adverse property of a familial ALS-linked SOD1 mutation causes motor neuron disease characterized by vacuolar degeneration of mitochondria. *Neuron* 14, 1105–1116
- 79 Cluskey, S. and Ramsden, D.B. (2001) Mechanisms of neurodegeneration in amyotrophic lateral sclerosis. *J. Clin. Pathol.* 54, 386–392
- 80 Shaw, C.E. *et al.* (2001) Progress in the pathogenesis of amyotrophic lateral sclerosis. *Curr. Neurol. Neurosci. Rep.* 1, 69–77
- 81 Rothstein, J.D. *et al.* (1990) Abnormal excitatory amino acid metabolism in amyotrophic lateral sclerosis. *Ann. Neurol.* 28, 18–25
- 82 Bensimon, G. *et al.* (2002) A study of riluzole in the treatment of advanced stage or elderly patients with amyotrophic lateral sclerosis. *J. Neurol.* 249, 609–615
- 83 Jackson, M. *et al.* (1999) Polymorphisms in the glutamate transporter gene EAAT2 in European ALS patients. *J. Neurol.* 246, 1140–1144
- 84 Trotti, D. *et al.* (2001) Amyotrophic lateral sclerosis-linked glutamate transporter mutant has impaired glutamate clearance capacity. *J. Biol. Chem.* 276, 576–582
- 85 Bristol, L.A. and Rothstein, J.D. (1996) Glutamate transporter gene expression in amyotrophic lateral sclerosis motor cortex. *Ann. Neurol.* 39, 676–679
- 86 Shaw, P.J. and Eggett, C.J. (2000) Molecular factors underlying selective vulnerability of motor neurons to neurodegeneration in amyotrophic lateral sclerosis. *J. Neurol.* 247 (Suppl. 1), 17–27
- 87 Comi, G.P. *et al.* (1998) Cytochrome c oxidase subunit I microdeletion in a patient with motor neuron disease. *Ann. Neurol.* 43, 110–116
- 88 Borthwick, G.M. *et al.* (1999) Mitochondrial enzyme activity in amyotrophic lateral sclerosis: implications for the role of mitochondria in neuronal cell death. *Ann. Neurol.* 46, 787–790
- 89 Strong, M.J. (1999) Neurofilament metabolism in sporadic amyotrophic lateral sclerosis. *J. Neurol. Sci.* 169, 170–177
- 90 Beaulieu, J.M. *et al.* (1999) Late onset death of motor neurons in mice overexpressing wild-type peripherin. *J. Cell Biol.* 147, 531–544
- 91 Ikeda, K. *et al.* (1996) Coadministration of interleukin-6 (IL-6) and soluble IL-6 receptor delays progression of wobbler mouse neuron disease. *Brain Res.* 726, 91–97
- 92 Kurek, J.B. *et al.* (1998) LIF (AM424), a promising growth factor for the treatment of ALS. *J. Neurol. Sci.* 160 (Suppl. 1), 6–13
- 93 Askanas, V. (1995) Neurotrophic factors and amyotrophic lateral sclerosis. *Adv. Neurol.* 68, 241–244
- 94 Apfel, S.C. (2001) Neurotrophic factor therapy – prospects and problems. *Clin. Chem. Lab. Med.* 39, 351–355
- 95 Alvarez, A. *et al.* (1999) Inhibition of tau phosphorylating protein kinase cdk5 prevents β -amyloid-induced neuronal death. *FEBS Lett.* 459, 421–426
- 96 Nguyen, M.D. *et al.* (2001) Deregulation of Cdk5 in a mouse model of ALS: toxicity alleviated by perikaryal neurofilament inclusions. *Neuron* 30, 135–147

Erratum

Please note a correction to the article *Structural pharmacogenomics, drug resistance and the design of anti-infective super-drugs* by Edward T. Maggio, Mark Shenderovich, Ron Kagan, Dean Goddette and Kal Ramnarayan in the print version of *Drug Discovery Today*, 15th December 2002, Volume 7, No. 24, 1214–1220.

Figure 7 on p. 1217 should have been as below:

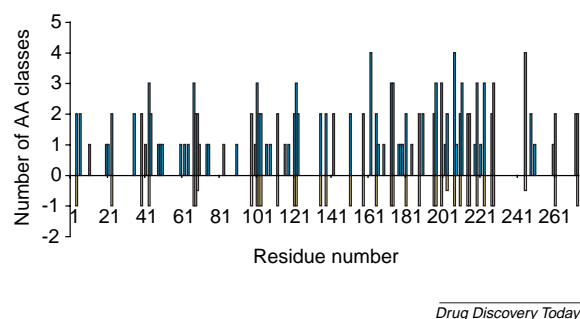


Fig. 7. The number of amino acid classes allowed at each residue position for HIV-1 reverse transcriptase. 38 positions (flagged below the X-axis) frequently exhibit mutation to an amino acid class different from the original class.

We would like to apologize for this inaccuracy and for any confusion that this might have caused.

PII: S1359-6446(02)02577-1