Corrigendum


Frauke Christ*, Zeger Debyser

Laboratory for Molecular Virology and Gene Therapy, Division of Molecular Medicine, KU Leuven, Kapucijnenvoer 33, 3000 Leuven, Belgium

The authors regret that Fig. 1D was unintentionally supplied from a previously published figure in PNAS (102: 17308–17313). The figure with the correct panel appears below.

Fig. 1. Domain organization of LEDGF and HIV-integrase and crystal structure of their interaction. (A) LEDGF/p75 binding to DNA is mediated by the NLS and the nearby located AT-hook DNA binding motives whereas the N-terminal PWWP motive and charged regions (CR1–3) are critical for chromatin recognition (Hendrix et al., 2011; Iiano et al., 2006b; McNeely et al., 2011; Turlure et al., 2006). The C-terminal IBD is essential for integrase binding (Cherepanov et al., 2004; Cherepanov et al., 2005b) and for the interaction with cellular proteins that bind LEDGF/p75 (Bartholomeeusen et al., 2007; Bartholomeeusen et al., 2009; Maertens et al., 2006; Yokoyama and Cleary, 2008). (B) The 325 N-terminal residues of LEDGF/p52 are identical with LEDGF/p75 and therefore both splice variants share their chromatin/DNA binding preferences. The p52 isoform though harbors a unique 8 amino acid sequence at its C-terminus (Singh et al., 2000a). (C) HIV-IN consists of 3 distinct domains: The N-terminal domain (NTD) with the conserved HHC zinc finger motives, the catalytic core domain (CCD) housing the transposase specific catalytic triade (D64, D116, E152) and the C-terminal domain (CTD) involved in multimerization and DNA-binding. (D) The co-crystal structure of LEDGF/p75-IBD (magenta) and the CCD dimer of integrase (green and blue) reveals that the integrase CCD dimer-interface forms a cavity in which the connecting loops of the IBD 5 α-helixes bundle protrudes. (PDB accession code 2B4J; (Cherepanov et al., 2005a)).