Corrigendum

NF- κ B dictates the degradation pathway of I κ B α

Erika Mathes, Ellen L O'Dea, Alexander Hoffmann and Gourisankar Ghosh

The EMBO Journal (2008) 27, 1421. doi:10.1038/emboj.2008.91

Correction to: The EMBO Journal, advance online publication 10 April 2008; doi:10.1038/emboj.2008.73

Since the publication of this paper, the authors have noticed an error in Figure 2B. The X-axis should have been labelled in hours and not minutes.

The correct Figure 2 is shown below.

The authors apologize for any inconvenience caused.

Figure 2 NF- κ B determines the degradation pathway of I κ B α . (A) NF- κ B protects I κ B α from proteasomal degradation *in vitro*. Top: purified 20S proteasome and I κ B α were incubated at 37°C, with or without purified p65. (I) represents the proteasome inhibitor MG132. (B) I κ B α is highly stable *in vivo* in the presence of NF- κ B. Left panel: WB showing WT, IKK phosphorylation and ubiquitination-defective mutants introduced into ikba^{-/-}, where all NF- κ B subunits are present. Cells were treated with cycloheximide (CHX) for different lengths of time (up to 24 h) and the protein levels were visualized by WB. Right panel: this experiment was repeated twice and is represented graphically with error bars signifying $\pm$ s.e.m. (○) Transgenic WT I κ B α , (□) K21, 22R I κ B α , (Δ) KR9 I κ B α and (◇) S32, 36A I κ B α . (C) A model of NF- κ B repression by I κ B α in pre-stimulated cells. There are two processes that control I κ B α degradation. In the resting cell, basal IKK activity phosphorylates bound I κ B α and targets it for ubiquitin-dependent degradation. In addition, free I κ B α is continuously synthesized and degraded in an IKK- and Ub-independent mechanism. This keeps NF- κ B from being activated in the resting cell.