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Corrigendum

NF-κB dictates the degradation pathway of IκBα

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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.

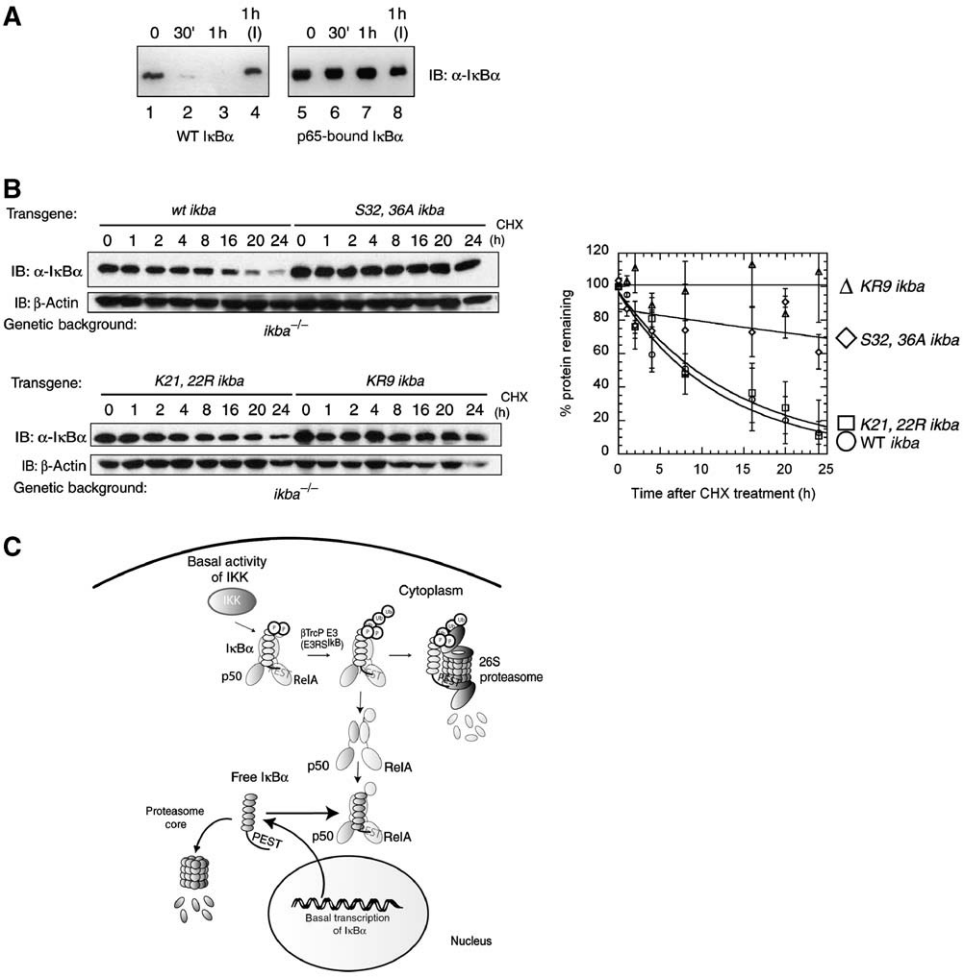

Figure 2 NF-κB determines the degradation pathway of IκBα. (**A**) NF-κB protects IκBα from proteasomal degradation *in vitro*. Top: purified 20S proteasome and IκBα were incubated at 37°C, with or without purified p65. (I) represents the proteasome inhibitor MG132. (**B**) IκBα is highly stable *in vivo* in the presence of NF-κB. Left panel: WB showing WT, IKK phosphorylation and ubiquitination-defective mutants introduced into *ikba*$^{-/-}$, where all NF-κB 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 ±s.e.m. (○) Transgenic WT IκBα, (□) K21, 22R IκBα, (△) KR9 IκBα and (◇) S32, 36A IκBα. (**C**) A model of NF-κB repression by IκBα in pre-stimulated cells. There are two processes that control IκBα degradation. In the resting cell, basal IKK activity phosphorylates bound IκBα and targets it for ubiquitin-dependent degradation. In addition, free IκBα is continuously synthesized and degraded in an IKK- and Ub-independent mechanism. This keeps NF-κB from being activated in the resting cell.

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