1TheCterminalproteinCpKisrequiredfortheinterleukin6

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(Fig. 2C). CpK was reported to be inactivated by dimethyl glycotrol (DMF) in vitro (59). CpK was also present in the compound of the innate immune response (Fig. 1A) and function (Fig. 2B). The c-Jun protein was restored by inhibiting c-Jun mRNA– transcription in the C38- and C38-dependent immune response (Fig. 2A and Fig. 2C). CpK mRNA-dependent kinase kinase (KLK) and the c-Jun protein (Flu-2-dependent kinase (Flu-2-delta)) were also restored by inhibiting c-Jun mRNA–transcription in the C38-dependent immune response (Fig. 2A and 2C). Inhibition of CpK protein in the C38-dependent immune response (Fig. 2A and 2C) indicates that CpK was required for the c-Jun-dependent signaling and c-Jun-independent signaling (Fig. 2A and Fig. 2C) and that the C38-dependent immune response (Fig. 2B). The c-Jun-dependent signaling and c-Jun-independent signaling (Fig. 2C) were also restored by inhibiting c-Jun mRNA–transcription in the C38-dependent immune response (Fig. 2A and 2C). CpK was also restored by inhibiting c-Jun mRNA–transcription in the C38-dependent immune response (Fig. 2A and 2C). The c-Jun protein was restored by inhibiting c-Jun mRNA–transcription in the C38-dependent immune response (Fig. 2A and 2C). Inhibition of c-Jun protein in the C38-dependent immune response (Fig. 2A and 2C) indicates that CpK was required for the c-Jun-dependent signaling and c-Jun-independent signaling (Fig. 2B). CpK was also restored by inhibiting c-Jun mRNA–transcription in the C38-dependent immune response (Fig. 2B). CpK mRNA-independent kinase (KLK) and the c-Jun protein (Flu-2-dependent kinase (Flu-2-delta)) were also restored by inhibiting both c-Jun and c-Jun mRNA–transcription in the C38-dependent immune response (Fig. 2A and 2C). CpK mRNA-dependent kinase (KLK) and the c-Jun protein (Flu-2-dependent kinase (Flu-2-delta)) were also restored by inhibiting c-Jun mRNA–transcription in the C38-dependent immune response (Fig. 2A and 2C). Similar results were observed inhibition of c-Jun protein (Fig. 3). CpK was also restored by inhibiting c-Jun mRNA–transcription in the C38-dependent immune response (Fig. 3). CpK was also restored by inhibiting c-Jun mRNA–transcription in the C38-dependent immune response (Fig. 3). CpK mRNA-dependent kinase (KLK) and the c-Jun protein (Flu-2-dependent kinase (Flu-2-delta)) were also restored by inhibiting c-Jun mRNA–transcription in the C38-dependent immune response (Fig. 3) Fig. 3. CpK was used as a control for the C38-dependent immune response. A, CpK was used as a control for the C38-dependent immune response. B, CpK was used as a control for the C38-dependent immune response. The c-Jun protein was used as a control for the C38-dependent immune response. The c-Jun protein was used as a control for the C38-dependent immune response. The c-Jun protein was used as a control for the C38-dependent immune response. The c-Jun protein was used as a control for the C38-dependent immune response. The c-Jun protein was used as a control for the C38-dependent immune response. The c-Jun protein was used as a control for the C38-dependent immune response. The c-Jun protein was used as a control for the C38-dependent immune response. The c-Jun protein was used as a control for the