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Title: LSC 2013 abstract - NF-κβ acts downstream of EGFR in regulating low dose cadmium induced primary lung cell proliferation

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Body: Studies done so far, regarding cadmium induced cellular proliferation were based on mitogen activated protein kinase and Ca^{2+}/calmodulin dependent protein kinase (MAPK/CaMK) dependent pathways. Cadmium treated mice primary lung epithelial cell proliferation was measured both by cell cycle analysis and Brdu incorporation assay and morphologically by atomic force microscopy. RT-PCR and western blot confirmed the specific signalling pathways. Primary lung epithelial cells were transfected with siEGFR, and molecular interactions of downstream signalling molecules were determined. We have reported RNAi induced silencing of the EGFR, which is over expressed in cadmium induced primary mice lung epithelial cells. Use of siEGFR effectively prevents expression of proinflammatory and cell proliferative markers.We have also transfected the primary lung epithelial cell with siRNA against the regulatory subunit of nuclear factor κβ (NF-κβ) and the data shows that cadmium induced lung cell proliferation is the effect of EGFR mediated NF-κβ activation. Nf-κB regulation of EGFR

Hypothetical Scheme