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EFFECT OF CURCUMIN AND P53 SIGNALING PATHWAY IN RAT THYMOCYTES TOXICITY INDUCED BY MANCOZEB

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Mancozeb, as a dithiocarbamate fungicide, is widely used in agriculture due to its low acute toxicity and short environmental persistence. We examined the protective role of curcumin on Mancozeb-induced toxicity in rat thymocytes and potential mechanisms involved. Rat thymocytes were exposed to Mancozeb (0.01 μg/ml) and/or curcumin (0.3, 1, 3 μM) and levels of cell viability, caspase-3, caspase-9 activity, cytochrome C oxidase, catalase activity, reactive oxygen species (ROS) production and p53 signaling involvement were evaluated after 24 h of incubation. Cells treated with Mancozeb showed increased toxicity, caspase-3, 9 activity and ROS production with decreased cytochrome C oxidase and catalase activity. Inhibition of caspase-3 and 9 activity resulted with reduced rat thymocytes toxicity while inhibition of p53 signaling pathway suppressed caspase-3 activity in cells. Co-treatment with curcumin (1, 3 μM) displayed significantly reduced toxicity, caspase-3, 9 activity and ROS production, together with increased cytochrome C and catalase activity in cells. These findings propose that Mancozeb-induced apoptosis in rat thymocytes is caspase dependent and is partially attributed to p53 signaling pathway. Certain curcumin concentrations may modulate Mancozeb-induced rat thymocytes toxicity, due to its anti-oxidative effect, and may be useful for providing potential thera-peutic strategy in immunomodulation induced by Mancozeb.

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