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Paracetamol treatment increases telomerase activity in rat embryonic liver cells

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Abstract:
Although paracetamol is known to have a damaging effect, this pharmaceutical is widely applied to pregnant and lactating women. Despite substantial progress in our understanding of its hepatotoxicity, some mechanisms, particularly of its embryonal and developmental toxicity, are still unknown. Thus, cell culture assays that investigate its toxicity are of particular interest. We assessed the effects of acute paracetamol treatment on cell viability (LDH assay, MTT assay), glutathione content (GSH assay), metabolic status (albumin and urea assays) and telomerase activity using rat embryonic liver cells (RLC-18 cells).
Incubation with low (6 mmol/l) and high (15 mmol/l) concentrations of toxin for 24 h leads to 20% and 50% cytotoxicity, respectively. Paracetamol exerted its toxicity in a similar pathway (depletion of GSH stores) as in adult liver cells, producing damage at the cellular level. Interestingly, paracetamol treatment significantly enhanced telomerase activity. Mechanisms involved in paracetamol-induced inhibition of cell senescence should be further elucidated. Telomerase activity in RLC-18 cells offers unique opportunities for examining basic biologic mechanisms. Our findings should encourage further studies to investigate a link between telomerase activity and toxicity, implying a role of impaired telomerase activity in human pathology.

Key words:
paracetamol, hepatotoxicity, telomerase activity, liver progenitors