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Genotoxicity of quinocetone in *Salmonella typhimurium* reversed mutation assay

Awais Ihsan
COMSATS Institute of Information Technology, Pakistan

Quinocetone is a novel quinoxaline hetrocyclic-N-dioxide compound which is widely used in China to improve the growth and feed efficiency in food producing animals. It was evaluated for mutagenic potential in six *Salmonella typhimurium* strains (TA97, TA98, TA100, TA102, TA1537 and TA 1538). Five different concentration ranges from 1.0 µg/ plate to 50 µg/ plate were tested in the presence or absence of rat liver homogenate fraction S-9, along with positive and negative controls. Quinocetone was not found mutagenic in *Salmonella typhimurium* TA 102 strain, both in the presence and in the absence of S-9 fraction. QCT produced histadine dependent mutants at 6.9 µg/plate in *Salmonella typhimurium* TA 97 (with or without rat liver S-9 fraction), *Salmonella typhimurium* TA 1537 (with rat liver S-9 fraction). At the dose of 18.2 µg/plate, histadine dependent mutants were produced in *Salmonella typhimurium* TA 100 (with or without rat liver S-9 fraction), in TA 1535 (with rat liver S-9 fraction) and TA 1537 (without rat liver S-9 fraction). At the dose rate of 50µg/plate, histadine dependent mutants were produced only in *Salmonella typhimurium* TA 98 (with or without rat liver S-9 fraction). These results highlighted the genotoxic potential of quinocetone in *Salmonella typhimurium* reverse mutation assay. It also extended knowledge about food safety and security issues of quinocetone.

awais.dr@gmail.com

Microenvironment and autophagy in arsenic lung tumorigenesis

Gang Chen
University of Kentucky, USA

The alterations in lung microenvironment upon chronic arsenic exposure may contribute to arsenic lung tumorigenesis. Immune cells play an important role in mediating the microenvironment in the lungs. Our previous work has shown that long-term arsenic exposure induces transformation of lung epithelial cells in which autophagy functions as a protective mechanism. However, the crosstalk between epithelial cells and immune cells/microenvironment upon arsenic exposure is not clear. Recently using a co-culture system with epithelial cells and immune cells, we determined that long-term arsenic exposure altered the phenotype of certain immune cells. Blocking the alteration decreases arsenic-induced transformation of epithelial cells. In addition, the phenotype changes in immune cells upon arsenic exposure decreases autophagy activity, which may account for increased cell transformation of epithelial cells with co-culture of the immune cells.

gangchen6@uky.edu