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Polycyclic aromatic hydrocarbons could trigger the deregulation of cell- cycle signaling pathways leading to lung cancer

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During the process of ethanol production from sugarcane, a number of carbonaceous compounds in the form of particulate matter along with the polycyclic aromatic hydrocarbons (PAHs) are introduced in the atmosphere. The particulate matter, including PAHs causes DNA damage which involve DNA double- strand breaks (DSBs) as well as single strand breaks (SSBs). DNA damage, either double strand break or single strand break leads to mutation which may be a probable cause for developing lung cancer. The first step in the initiation of activity of DNA damage checkpoints is recognition of DNA damage. Components of checkpoint mechanism include sensors, mediators, transducers and effectors which work cooperatively in different phases of cell cycle. Once DNA damage is sensed, the apical kinases ATM and ATR become activated, which, in turn, phosphorylate several substrates leading the signaling pathway to G1/S and G2/M arrests, respectively. Since, checkpoint kinase 2 and checkpoint kinase 1 play crucial roles in the progression of these signaling pathways leading to G1/S and G2/M arrests. The inhibition of checkpoint kinases 2 and 1 could abrogate the G1/S and G2/M arrests, respectively, which could be proved useful in the therapeutics of anticancer drug development. Thus, the inhibition of checkpoint kinases 2 and 1 could be the ideal candidate targets for anticancer drug development.

Keywords: Polycyclic aromatic hydrocarbons, ATM, ATR, checkpoint kinase.

Biography

I received a Bachelor of Science from Kumaun University, Nainital, India and Master of Science in Biotechnology from Amity University, Noida, India. Currently,I am working as a Project Assistant in Amity Institute for Herbal Research and Studies at Amity University, Noida, India.

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Immunomodulatory activities of chitin microparticles on leishmania major-infected murine macrophages

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Chitin microparticles (CMPs) are found to be potent macrophage stimulators; however, their immunomodulatory effects on the parasite-infected macrophages have not yet been studied. To address this issue, we used a Leishmania major-infected murine macrophage model and characterized the regulatory effects of CMPs on the parasite-infected cells. Mouse peritoneal macrophages were prepared and infected with L. major (MRHO/IR/1975/ER) standard strain. Following cell treatment with CMPs (500 mg/mL) for 48 h, percent of infected macrophages was determined by Giemsa staining and compared with untreated cells. To find the potential mechanisms of the activity of CMPs, TNF-a and accumulated nitrite in the culture supernatants of the treated and untreated cells were also measured by ELISA and colorimetric Griess assays, respectively. According to the obtained results, chitin microparticles reduced the ex vivo parasite infectivity by 12%. However, this inhibitory effect was not directly related to the increased biosynthesis and release of nitric oxide (NO) by macrophages. Instead, we observed a significant increase in the level of TNF-a secretion due to cell treatment with CMPs. Interestingly, this overexpression of TNF-a did not impair cell viability, suggesting the anti-apoptotic effects of the CMPs. These findings show that chitin microparticles have immunomodulatory effects on L. major-infected macrophages and further provide motivations for future studies on the prophylactic use of CMP

Biography

Mostafa Haji Molla Hoseini, has completed his Ph.D at the age of 32 years from Tarbiat ModaressUniversity. He is the assistant professor in Immunology Dept Of Shahid Beheshti University of Medical sciences. He has published 8 papers in reputed journals.

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