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Departmental Seminar

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Seminar Title	: Piperlongumine as a promising phytotherapeutic agent against lung cancer: Targeting ROS, mitochondrial dysfunction, and stemness pathways
Speaker	: Dr Bijesh Kumar Biswal
Supervisor	: Dr Bijesh Kumar Biswal
Venue	: LS Seminar Hall
Date and Time	: 02 May 2025 (16:00 hrs)
Abstract	: Phytochemicals have demonstrated significant potential in cancer therapy. Piperlongumine (PIP), a bioactive alkaloid naturally found in dietary spices, has gained attention for its potent pharmacological properties, particularly its anticancer activity. This study investigated the anti-lung cancer effects of PIP and its underlying mechanisms using in vitro and ex vivo models. The cytotoxic, anti-proliferative, and apoptotic effects of PIP on human lung cancer cells (LCC) were assessed through cell viability, colony formation, cell migration, invasion, comet assay, and various staining techniques. Additionally, a multicellular spheroid assay was used to evaluate its anti-cancer potential ex vivo. Preliminary findings revealed that PIP selectively induced cytotoxic and anti-proliferative effects in LCC by triggering DNA damage and cell cycle arrest. Furthermore, PIP significantly elevated reactive oxygen species (ROS) generation at both cellular and mitochondrial levels, leading to mitochondrial membrane potential (MMP) dissipation and activation of the caspase-dependent apoptotic pathway. Mechanistically, PIP disrupted F-actin organization, inhibiting LCC migration and invasion. This F-actin deformation further influenced epithelial-to-mesenchymal transition (EMT) by modulating marker expression and downregulating stemness-associated proteins, including SOX9, CD-133, and CD-44. Notably, ex vivo studies showed that PIP effectively reduced spheroid size while exhibiting strong apoptotic and cytotoxic effects. Importantly, this study identified SOX9 as a crucial survival factor in LCC, where its inhibition enhanced PIP sensitivity. Overall, this study establishes PIP as a promising therapeutic agent for lung cancer, exerting anticancer effects by increasing ROS levels, disrupting MMP, and inhibiting stemness and EMT pathways.