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Receptor masking bax antagonism and licl supplementation improves homing and engraftment of bone marrow cells

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Effective homing of stem cells is a pre-requisite for successful hematopoietic stem cell transplantation (HCT) when there is a dearth of available hematopoietic stem cells (HSC). However, the transplanted cells face premature differentiation and death while en route to bone marrow. This causes decreased engraftment of transplanted HSC. In order to enhance the homing of stem cells to bone marrow as undifferentiated cells, in this study the mouse bone marrow cells were masked with fibronectin modified alginate (A-F). This biocompatible coat prevented the antibody from binding to its cognate receptor, *i.e*. CD49e, which is present on the cell surface. In addition, coating effectively abolished RBC's clumping by reactive antibodies in haemagglutination assay. Bioluminescence imaging showed significant improvement in the efficiency of coated cells to home in bone marrow. This also provided protection to irradiated BALB/c mice (75% on sublethal irradiation). To further enhance the transplantation efficiency, we designed and evaluated Bax antagonist peptide (BAP-GR23) for decreasing the death of HSC and other tissues, which become morbid on preparatory or undesired radiation exposure. We found that administration of novel BAP and LiCl combination followed by A-F coated bone marrow cells transplantation and ascorbic acid supplementation *Ad libitum* was effective in preventing the imminent death of lethally irradiated BALB/c mice (100% survival). Radiation-induced aplasia was corrected after 10days of treatment. Histologically no toxicity of the treatment was observed. This combination also improved survivability and clonogenicity of cells with a concomitant decrease in DNA degradation in the comet assay. Microarray data analysis showed a specific role of LiCl in upregulation of cell survival genes' expression along with downregulation of bile producing genes (CYP7A1 and FOXA2) having a role in radiation-induced gastrointestinal syndrome. These results suggest the development of a new multi-therapy paradigm to enhance the clinical efficacy of HCT.

Biography

Yogesh Kumar Verma, MNABS, is working as Scientist 'D' in INMAS, DRDO, Delhi, India. He has completed his MSc (Year 2000) and PhD (Year 2008) in Biomedical Science from Delhi University, India. He also has another MSc degree in Bioinformatics from Punjab Technical University, India. In 2008, he received Young Scientist award from DST under SERC scheme for carrying out research work as Principal Investigator. In year 2009, he joined INMAS, DRDO, Delhi, as Scienitst 'C'. He has 23 publications and 04 patents (filed) to his credit. He is presently working in the area of stem cell research, microencapsulation, tissue engineering and omics data mining and analysis for decreasing various types of injuries.

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