

abstract Hematopoietic reconstitution, following bone marrow or stem cell transplantation, requires a microenvironment niche capable of supporting both immature progenitors and stem cells with the capacity to differentiate and expand. Melphalan and VP-16 induced damage of HOB suggests vulnerability of this critical niche to therapeutic agents frequently utilized in pre-transplant regimens and suggests that dose escalated chemotherapy may contribute to post-transplantation hematopoietic deficits by damaging structural components of this supportive niche. This increase was coincident with .reduced HOB capacity to support immature B lineage cell chemotaxis and adherence