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.