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will focus on a new treatment for AML which, unlike allo-HCT, is done as a primary therapy and may overcome some of the significant problems associated with allo-HCT. Our approach to AML is based on several concepts and findings from the literature that suggest that an important early step in the AML disease process is the accumulation of genetic abnormalities in hematopoietic stem cells, that occur prior to the outgrowth of leukemia. These concepts and findings are consistent with the pathogenesis of AML suggested by our observation that normal mice can be

leukemized by transplanting unfractionated bone marrow cells from AML patients. These observations suggest that the majority of leukemic stem cells are present in the bone marrow of patients with AML, and that these cells represent the clonal origin of AML in each patient. We hypothesize that chemotherapy and/or radiotherapy will selectively kill normal hematopoietic stem cells, perhaps even selectively killing AML stem cells, but will not affect leukemic stem cells. Consequently, we propose that the stem cell compartment of AML patients will be largely intact and

unaffected by therapy, allowing patients to be successfully treated with a remission-induction therapy without prior myeloablative therapy. We also hypothesize that the disease-free interval after such therapy will be long because of the essentially non-leukemic nature of the stem cell compartment that has been left intact. To test these hypotheses we have designed a pilot study that will examine the ability of a novel regimen of immunosuppressive therapy with sub-therapeutic doses of the antileukemic agent cytarabine to selectively ablate normal

hematopoietic stem cells. If successful, this study will provide a proof of principle for a phase II trial of this approach as a post-remission therapy f30f4ceada