hematopoetic stem cell transplant (HSCT) outcomes. Poverty is known to be correlated with negative health outcomes in pediatric primary care and subspecialties; it is not known how poverty impacts HSCT outcomes.

Objective: To describe the baseline prevalence of poverty and material hardship in the pediatric HSCT population at a major referral center. To describe transplant-related work disruptions and income losses stratified by federal poverty level (FPL) in this patient population, and explore the relationship between family poverty and clinical outcomes.

Methods: Single institution, cross-sectional survey. Participants included 45 English-speaking, pediatric allogeneic HSCT families whose children had undergone HSCT in the prior twelve months and were alive at time of survey administration. Eighty-seven percent of families approached consented to study participation. This study was approved by the Dana-Farber Cancer Institute Institutional Review Board.

Results: Poverty prior to transplant was prevalent with 18 (40%) families reporting baseline incomes at or below 200% FPL ($44,000 for a family of four). Parental work disruptions due to HSCT were common across all income levels with 39 (87%) families reporting some disruption, and consequent income losses were substantial for all families. Families at or below 200% FPL were disproportionately impacted with 7 (39%) of the poorest families reporting transplant-associated annual income losses of >40% as compared to 2 (18%) of the wealthiest families (p=0.006). Material hardship during the post-transplant period was widespread, with 17 (38%) families reporting either food, housing, or energy insecurity. Baseline family poverty level was not associated with length of transplant admission, unplanned re-admissions, or ICU stay in the 6-month post-transplant period in univariate analysis. Poorer children, however, were more likely to experience Graft Versus Host Disease (GVHD) of any grade in the 6 month post-transplant period with eleven (61%) of those at or below 200% FPL experiencing GVHD as compared with 2 (18%) of the wealthiest (p=0.01).

Conclusion: Baseline poverty is widely prevalent in the pediatric HSCT population, and poverty may be associated with the development of GVHD. Material hardship during the post-transplant period—including food, housing, or energy insecurity—is widespread. Further studies aimed at understanding how these social determinants of health contribute to HSCT outcomes may provide targetable factors to decrease transplant-associated morbidity and mortality.

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Allogeneic Hematopoietic Stem Cell Transplantation (HSCT) for Non Malignant Hematologic Disorders (NMHD), Using a Chemotherapy-Only Cytoreductive Regimen and T-Cell Depleted Grafts from Alternative Donors

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Non-malignant hematologic disorders of childhood comprise a number of various disorders including acquired severe aplastic anemia (SAA), and inherited marrow failure syndromes. Patients with such diseases who do not have matched related donors fare poorly with allogeneic HSCT from alternative donors and are at high risk for developing chronic GvHD. We report 14 patients aged 0.7-18 years of age (median 5.3 years) who received T-cell depleted HSCT from alternative donors between April 2005 and May 2013. Diseases included acquired SAA including PNH (N=6), severe chronic neutropenia (SCN)(N=2), congenital amegakaryotycic thrombocytopenia (N=2), Shwachman Diamond syndrome (N=1), autoimmune hemolytic anemia (N=1), chronic granulomatous disease (N=1) and hemophagocytic lymphohistiocytosis (N=1). Two patients with SAA had constitutional abnormalities and were therefore thought to have possibly genetic diseases despite negative testing for defined disorders. Ten patients had long standing symptomatic disease for > 1 year prior to transplant. Patients with Fanconi anemia and dyskeratosis congenita were treated with reduced intensity regimens and were not included. Patients received one of 3 myeloablative regimens: Busulfan Melphalan and Fludarabine (N=2), Cyclophosphamide Thio Flu (N=3) or Mel, Thio Flu (N=9). Patients received rabbit ATG pre-transplant and filgrastim post transplant to promote engraftment. Donors were unrelated HLA-matched donors (N=5), unrelated HLA-mismatched donors (N=6) or related HLA-mismatched donors (N=2), and related phenotypic matched donor (N=1). Grafts included soybean agglutinin negative, E-rosette negative bone marrow grafts (N=5), Isolex CD34+ E-rosette- peripheral blood stem cell grafts (N = 4), or CliniMACS CD34+ PBSC grafts (N=5).

All 13 evaluable patients engrafted. Neutrophil engraftment occurred on day +12 and platelet engraftment to 20,000 on day +20 post transplant. Four patients died from multi-organ failure and/or infection (three of whom had a duration of their disease of 2-7 years). One patient with SAA fully engrafted with donor cells developed a pancytopenia post transplant with secondary MDS in donor cells; this patient is alive. Excluding one patient who is too early post transplant and with a median follow-up of 2 years, 8 of 13 patients are alive (OS 61%). None of these patients has evidence of GvHD. These results should encourage proceeding to transplant with chemotherapy-only cytoreductive regimens and T-cell depleted stem cell transplants from alternative donors for patients with non malignant hematologic disorders earlier in the course of their disease.

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Lymphopenia in Patients with Hemophagocytic Lymphohistiocytosis: Are B Cells Suppressed in These Patients?

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Background: Hemophagocytic Lymphohistiocytosis (HLH) is an immune regulatory disorder requiring allogeneic hematopoietic stem cell transfusion (HSCT) for long-term survival. More importantly, it is imperative to initiate treatment early as delays in treatment can be associated with significant mortality. Unfortunately, there can be significant overlap of the HLH diagnostic criteria with other non-HLH conditions, thereby considerably delaying diagnosis and therapy for HLH. Hence, surrogate markers are needed to aid in the diagnosis and prevent delays in initiating therapy in this life threatening disorder. Hypogammaglobulinemia has been a less commonly reported feature of HLH. It has been reported in SAP deficient patients +/- HLH. More recently, it has been reported in a few patients with XIAP deficiency and STXBP2 mutations in association with colitis. We have observed B-cell lymphopenia as well as hypogammaglobulinemia in patients with HLH at our institution. However, the