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[ADVANCED](#)[TOP](#) > [Available Issues](#) > [Table of Contents](#) > [Abstract](#)

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[\[PDF \(320K\)\]](#) [\[References\]](#)**Expansion of NK1.1⁻ intermediate TCR cells and granulocytes in mice transplanted with TAP-1-deficient cells**Nakako IZUMI¹⁾, Chikako MIYAJI¹⁾, Hiroki KAWAMURA¹⁾, Toshihiko KAWAMURA¹⁾ and Toru ABO¹⁾

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ABSTRACT

Missing self which lacked the expression of MHC class I antigens was prepared in irradiated B6.Ly5.1 mice (H-2^b) which had undergone bone marrow transplantation (BMT) (depleted of T cells) of TAP-1 (-/-) (Ly5.2, H-2^b) mice. Donor cells (Ly5.2⁺) and recipient cells (Ly5.1⁺) were identified by Ly5 markers. For purposes of comparison, syngeneic (B6.Ly5.2 mice) BMT and allogeneic (BALB/c mice, H-2^d) BMT were also conducted. In the case of missing self cells, the ratio of expanding donor cells increased in the liver, spleen and bone marrow on days 14 and 21 after BMT. Such donor cells were mainly NK cells and NKT cells, especially in the liver. The interacting recipient lymphocytes were NKT cells at the early stage (day 7). However, the major lymphocytes became IL-2R β ⁺CD3^{int} cells which lacking NK1.1 at the fulminant stage (days 14 and 21). At this time, granulocytes expanded prominently. Since IL-2R β ⁺CD3^{int} cells (NK1.1⁻) lacked cytotoxicity, the suppression of expanding donor cells might be mediated by granulocytes. Granulocytes were activated by inflammatory cytokines. These results suggest that in addition to NK1.1-expressing cells (e.g., NK and NKT cells), IL-2R β ⁺CD3^{int} cells lacking NK1.1 may be also the lymphocyte subset which recognizes MHC class I-deficient self.

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