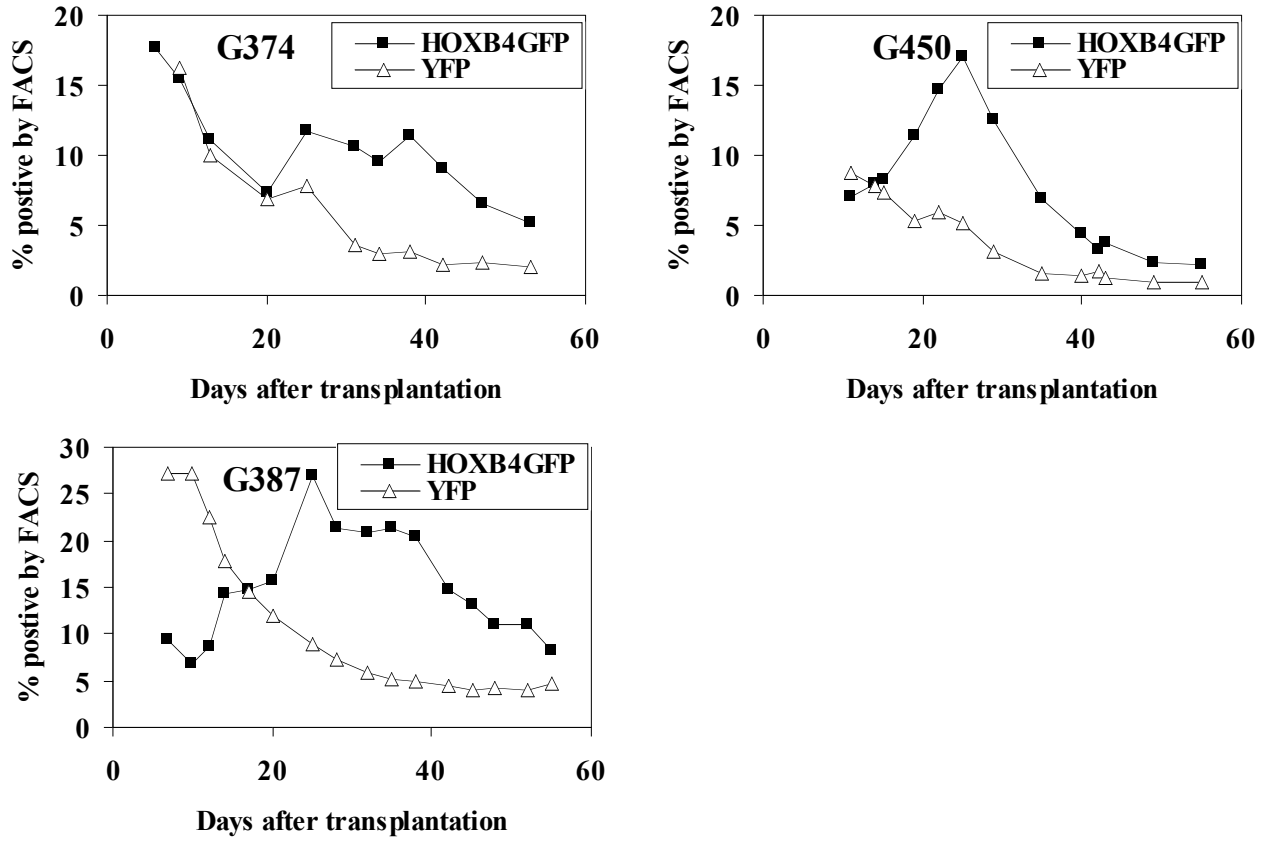
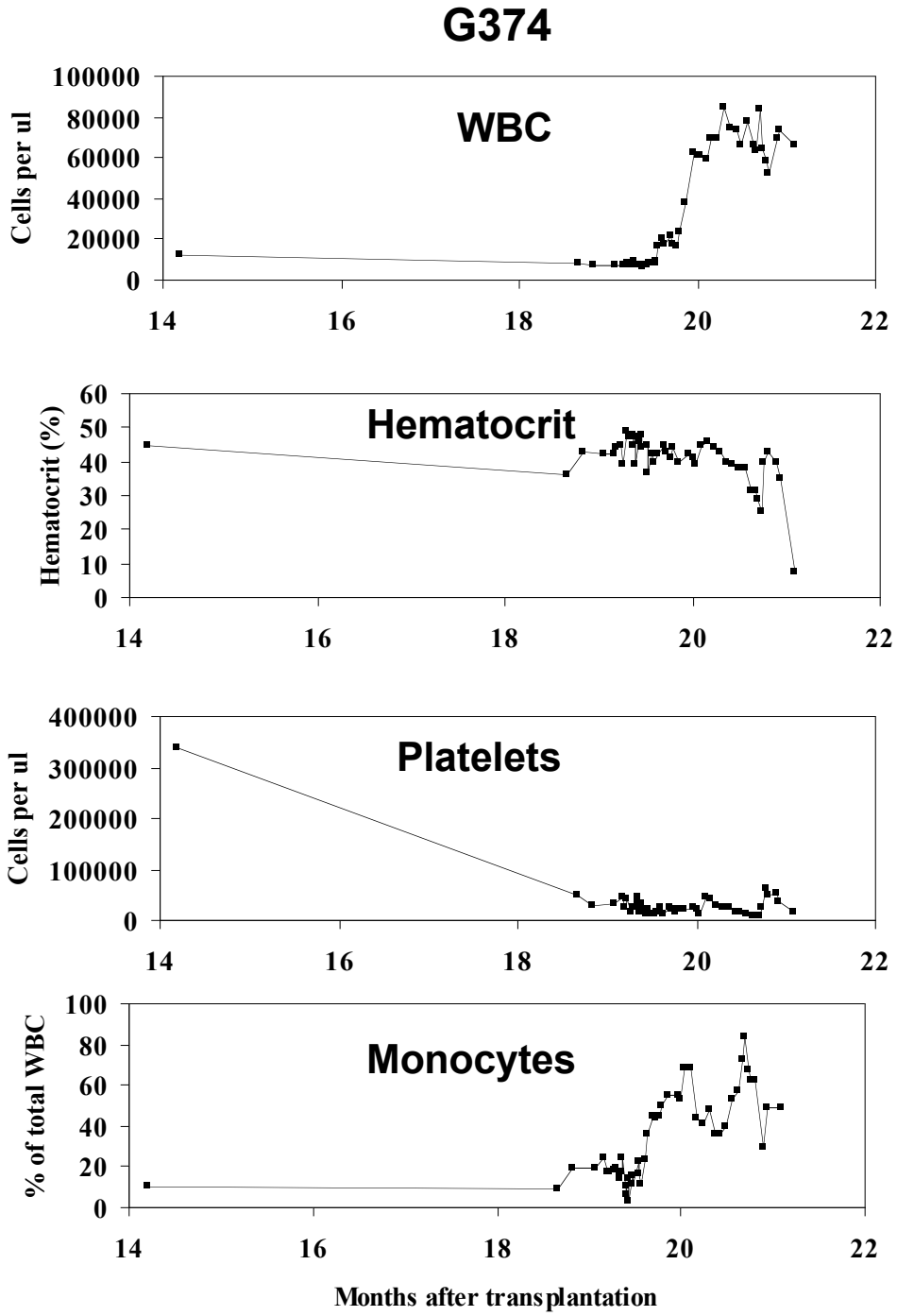
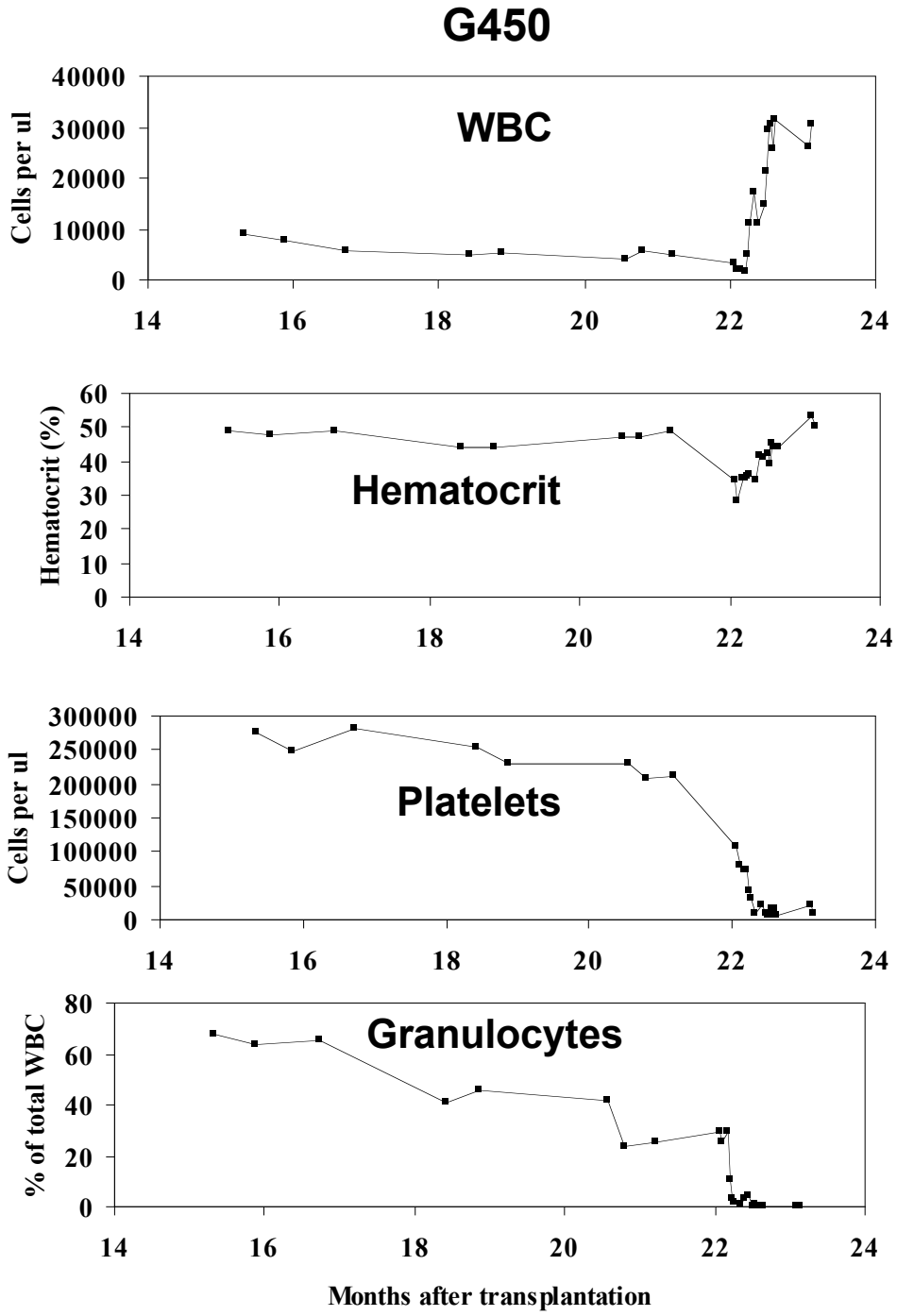


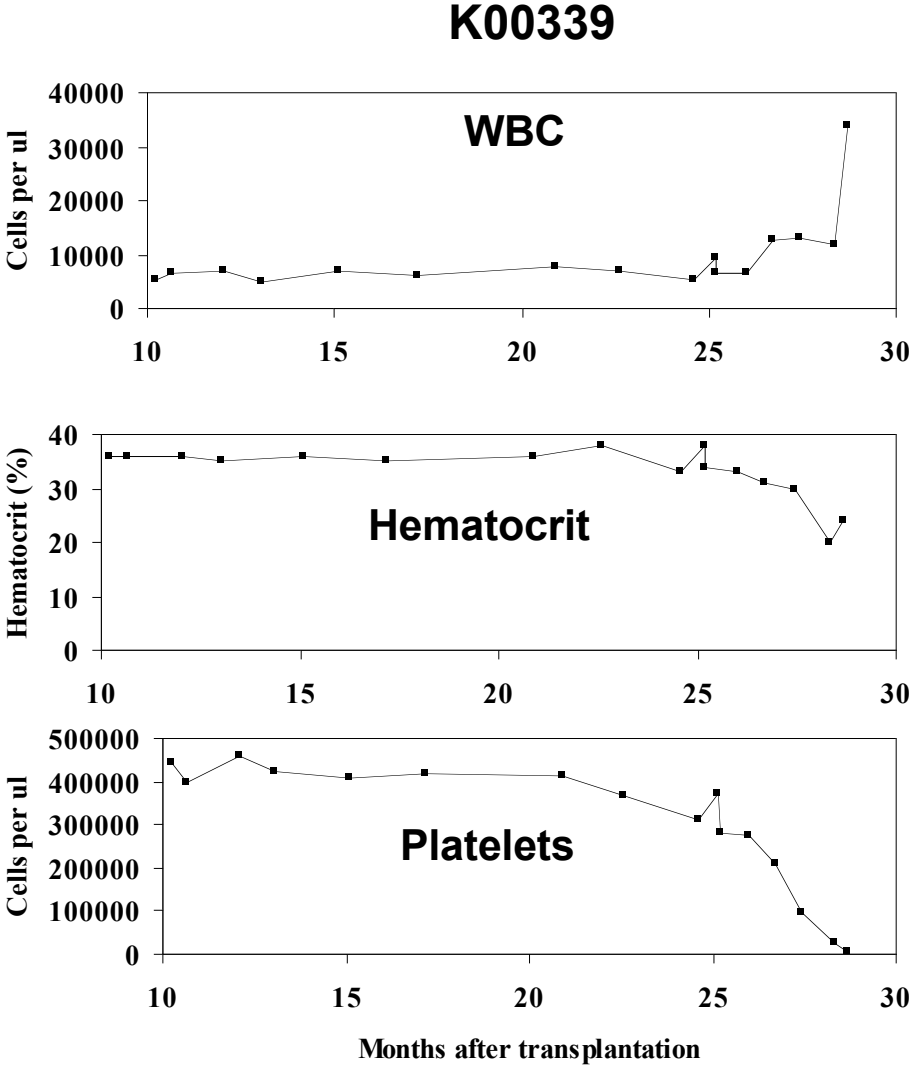
Supplementary Figure 1. Increased contribution of HOXB4-transduced cells to granulocyte engraftment after transplantation in 3 dogs. Equal numbers of $CD34^+$ cells were transduced with HOXB4-expressing or control vectors. HOXB4 marking levels initially increased and by 1 month after transplantation, marking levels for HOXB4 were significantly higher than for the control (14.7% vs. 4.2%, $P = 0.04$), suggesting that HOXB4 promotes the expansion of canine short-term repopulating cells.



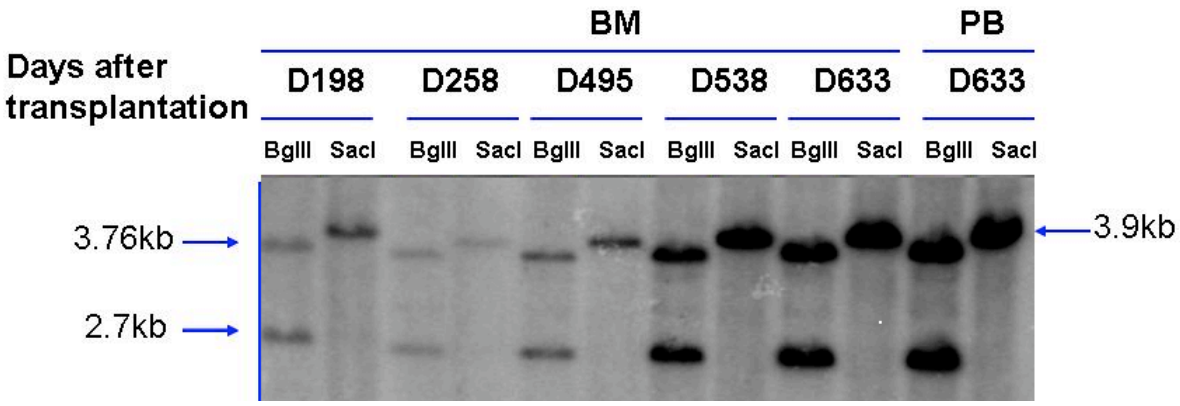
Supplementary Figure 2. Hematology data suggest the development of leukemia in dog G374. At 19 months after transplantation, white blood cell count increased significantly, in particular monocytes, suggesting the development of monocytic leukemia. At 18 months after transplantation, platelet counts had decreased 10-fold relative to normal values. After 20 months, red blood cell counts and hematocrit decreased and the animal became dependent on blood transfusion.



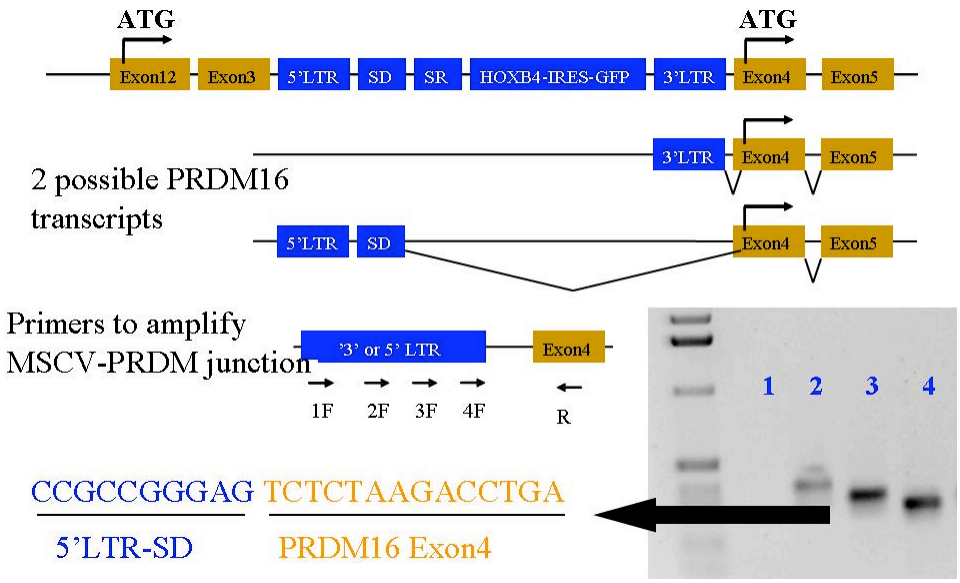
Supplementary Figure 3. Hematology data suggest the development of leukemia in dog G450. Twenty-two months after transplantation, whole white blood counts increased and hematocrit decreased. Platelet and granulocyte counts gradually decreased after 17 months.



Supplementary Figure 4. Hematology data suggest the development of leukemia in macaque K00339. Twenty-five months after transplantation, white blood cell counts increased and hematocrit and platelet counts decreased.



Supplementary Figure 5. Southern blot analysis demonstrating monoclonality in dog G374. Genomic DNA from bone marrow (BM) or peripheral blood (PB) were digested with BglII, which cuts the transgene once, or with SacI, which cuts the transgene twice, releasing a 3.9 kb internal band. The same intensity of the 2 bands over time confirms that this is 1 clone with 2 integration sites.



Supplementary Figure 6. 5'LTR splicing into exon 4 activates the short isoform PRDM16 in dog G374. Virus integration at intron 3 activates transcription of exons downstream of the virus integration site, which encodes a short isoform of PRDM16.