

Data supplements:

Figure S1. AML1-ETO mutant proteins localize in nuclei of circulating hemocytes.

UAS constructs of mutant forms of AML1-ETO (indicated on top of the panels) were over-expressed in hematopoietic cells of *Drosophila* with use of *hml^A-Gal4*. Immunostaining of circulated hemocytes with anti-AML1 antibody has revealed nuclear localization of the AML1-ETO mutant proteins.

Figure S2. Expression of excess Runx partner proteins Brother (Bro) or Big Brother (Bgb) suppresses AML1-ETO-induced hemocyte proliferation.

(A) *UAS-Bro* or *-Bgb* (on X axis) were co-expressed with AML1-ETO in hemocytes (*hml^A-Gal4, UAS-GFP; UAS-AML1-ETO: hml>AML1-ETO*). Number of hemocytes in AML1-ETO background was significantly reduced by loss of both *Bro* and *Bgb* (n=10, P<0.001), while their ectopic expression in wild type hemocytes (*hml*, B) did not significantly affect hemocyte proliferation.

Figure S3. *EcR-B1* is required for AML1-ETO-induced hemocyte proliferation.

Upper panels show hemocyte concentrations (GFP⁺ cells) in larvae of *hml^A-Gal4, UAS-GFP; UAS-AML1-ETO (hml>AML1-ETO)* backgrounds. Lower panels show hemocyte density in larvae of wild type *hml^A-Gal4, UAS-GFP (hml)* backgrounds. Single copy of *EcR* alleles indicated on the top of each panel. Alleles of *EcR-B1* (*Q50st, KG04522, dsRNA*) and dsRNA and dominant negative (DN) alleles of *EcR-A* significantly suppress and enhance AML1-ETO-mediated proliferation of hemocytes, correspondingly, if compared with *w¹¹¹⁸* control background. Single copy loss of both isoforms (*M554fs*, loss of ligand binding) enhances AML1-ETO-mediated proliferation of hemocytes. Alleles of *EcR-B1* and *EcR-A* do not significantly affect hemocyte number (GFP⁺ cells) in wild type backgrounds.

Figure S4. *EcR-B1* function is not required for *hop^{Tum-I}*-induced blood phenotype.

hop^{Tum-I} mutation causes robust enhancement of lamellocyte differentiation and increased number of hemocytes. Single copy loss of the *EcRB1* did not affect the aberrant differentiation of lamellocytes (A), and the proliferation of hemocytes (B) in *hop^{Tum-I}* mutant.

Lamellocytes in the blood samples were revealed after immunostaining with L1 antibody (red). Genotypes are indicated on the top of the corresponding panels.

Figure S5. Functions of several nuclear hormone receptors are required for AML1-ETO-induced hemocyte proliferation.

Each nuclear receptor (on X axis) was inactivated with corresponding *UAS-RNAi* in hemocytes expressing AML1-ETO (*hml^A-Gal4, UAS-GFP; UAS-AML1-ETO: hml>AML1-ETO*). Number of hemocytes in AML1-ETO background was significantly reduced by dsRNA alleles of *E75B, E78C, err, Hr96 and Hnf4* (n=10, * P<0.001). Number of hemocytes in AML1-ETO background was not significantly affected by inactivation of *DHR3, DHR38* but was increased by dsRNA alleles of *DHR39* and *DHR4*.

Figure S6. Increased ROS level caused proliferation of circulating hemocytes.

High ROS level generated by the disruption of complex I protein (ND75) of the mitochondrial electron transport chain in circulating hemocytes (*hml^A-Gal4, UAS-GFP; UAS-dsND75: hml/dsND75*) causes significant increase in number these cells.

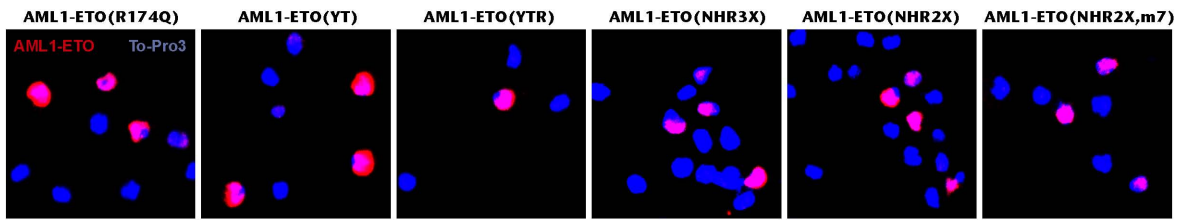


Figure S1

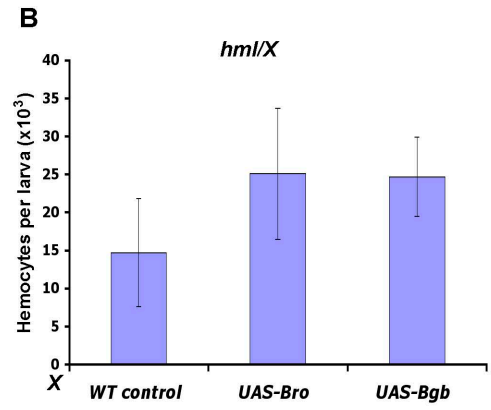
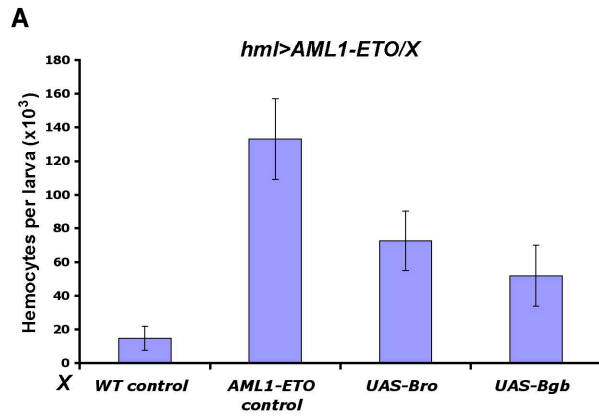


Figure S2

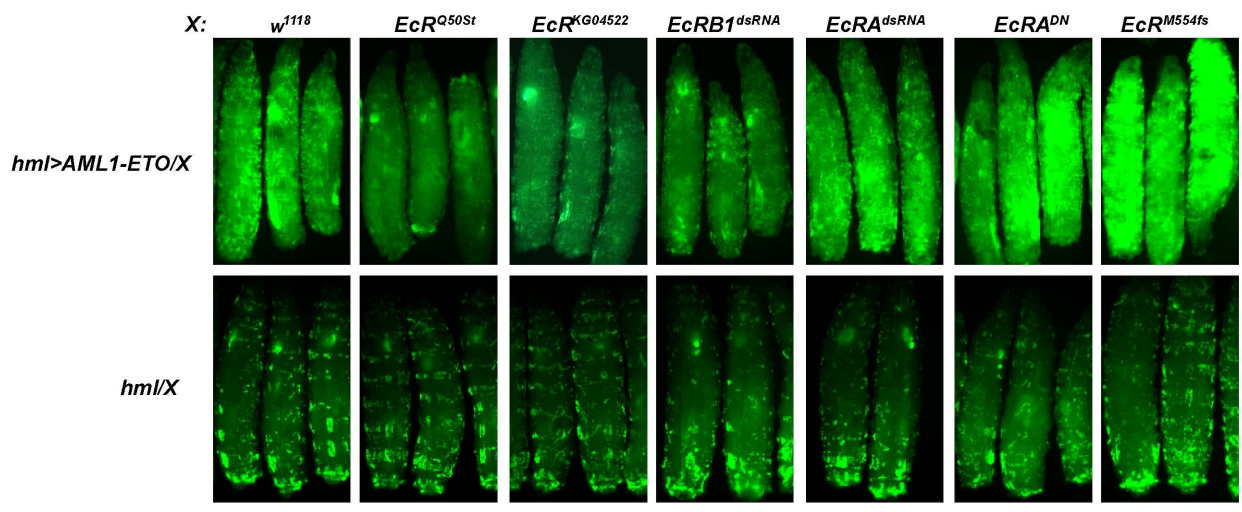
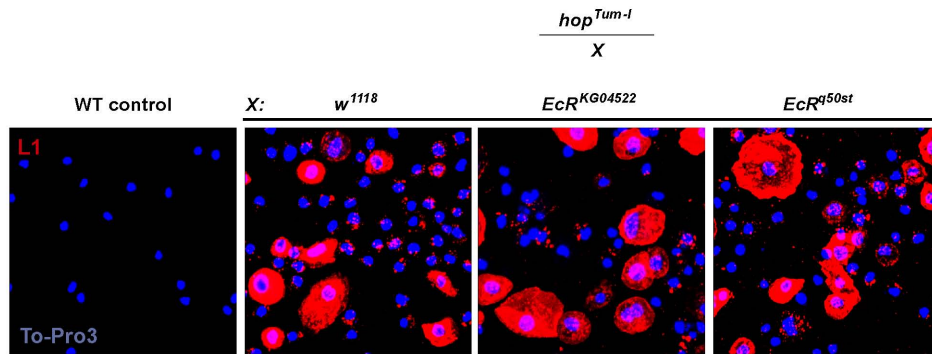


Figure S3

A



B

