

Sustained MEK inhibition abrogates myeloproliferative disease in *Nf1* mutant mice

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Corrigendum

Original citation: *J Clin Invest.* 2013;123(1):335–339. doi:10.1172/JCI63193. Citation for this corrigendum: *J Clin Invest.* 2016;126(1):404. doi:10.1172/JCI85325. The genotype of the *Nf1* mutant mice was incorrectly described. The correct text for Methods appears below. Mice and treatment procedures. Mx1-Cre;Nf1^{tm1Par/tm1Tyj} (referred to as Mx1-Cre;Nf1^{flox/-}) and control mice (Nf1^{flox/+}) were generated and treated with plpC (Sigma-Aldrich) at 3–5 days, as described previously (16). In addition, corrected sentences describing the *Nf1* mutant mice in the Introduction and Results and Discussion appear below. To address this question, we administered 901 to Mx1-Cre;Nf1^{flox/-} mice with MPN. We first assessed the pharmacodynamic properties of 901 in WT and Mx1-Cre;Nf1^{flox/-} (*Nf1* mutant) mice that received an oral gavage dose of 5 mg/kg/d for 5 days. We randomly assigned Mx1-Cre;Nf1^{flox/-} mice (n = 35) and their WT littermates (n = 38) to treatment with 901 (at a daily dose of 5 mg/kg) or control vehicle for 10 weeks or until the mice became moribund. Progressive anemia with elevated reticulocyte counts and massive splenomegaly suggested that Mx1-Cre;Nf1^{flox/-} mice with MPN have ineffective erythropoiesis. In striking contrast, profiling revealed a largely inverted ratio of early-to-late erythroblasts in Mx1-Cre;Nf1^{flox/-} mice, with 10-fold expansion in the percentage of cells in region II, and a reciprocal decline in the number of erythroblasts progressing to region IV (Figure 2C). To further characterize the hematopoietic compartment [...]

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Mice and treatment procedures. *Mx1-Cre;Nf1^{tm1Par/tm1Tyj}* (referred to as *Mx1-Cre;Nf1^{lox/-}*) and control mice (*Nf1^{lox/+}*) were generated and treated with pIpC (Sigma-Aldrich) at 3–5 days, as described previously (16).

In addition, corrected sentences describing the *Nf1* mutant mice in the Introduction and Results and Discussion appear below.

To address this question, we administered 901 to *Mx1-Cre;Nf1^{lox/-}* mice with MPN.

We first assessed the pharmacodynamic properties of 901 in WT and *Mx1-Cre;Nf1^{lox/-}* (*Nf1* mutant) mice that received an oral gavage dose of 5 mg/kg/d for 5 days.

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Progressive anemia with elevated reticulocyte counts and massive splenomegaly suggested that *Mx1-Cre;Nf1^{lox/-}* mice with MPN have ineffective erythropoiesis.

In striking contrast, profiling revealed a largely inverted ratio of early-to-late erythroblasts in *Mx1-Cre;Nf1^{lox/-}* mice, with 10-fold expansion in the percentage of cells in region II, and a reciprocal decline in the number of erythroblasts progressing to region IV (Figure 2C).

To further characterize the hematopoietic compartment in *Mx1-Cre;Nf1^{lox/-}* with MPN, we enumerated KLS (c-Kit⁺lin⁻Sca-1⁺) cells and myelo-erythroid progenitor populations by flow cytometry (11, 12).

In addition, corrected sentences describing the *Nf1* mutant mice in the figure legends appear below.

[Figure 1] 901 reduces myeloproliferation and enhances erythropoiesis in *Mx1-Cre;Nf1^{lox/-}* (*Nf1*) mice.

[Figure 2] Hematopoietic tissues from 6-month-old *Mx1-Cre;Nf1^{lox/-}* (*Nf1*) and WT mice treated with 901 or vehicle were analyzed at the end of the trial.

[Figure 3] 901 normalizes early myelo-erythroid populations in *Mx1-Cre;Nf1^{lox/-}* (*Nf1*) mice.

Finally, the online version of the supplemental data has been updated to indicate the correct genotype of *Nf1* mutant mice in Supplemental Figures 2–5.

The authors regret the errors.