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

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**Corrigendum**


Mice and treatment procedures. *Mx1-Cre;Nf1tm1Par/tm1Tyj* (referred to as *Mx1-Cre;Nf1flox/–*) and control mice (*Nf1flox/+*) 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;Nf1flox/–* mice with MPN. We first assessed the pharmacodynamic properties of 901 in WT and *Mx1-Cre;Nf1flox/–* (*Nf1 mutant*) mice that received an oral gavage dose of 5 mg/kg/d for 5 days. We randomly assigned *Mx1-Cre;Nf1flox/–* 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;Nf1flox/–* mice with MPN have ineffective erythropoiesis. In striking contrast, profiling revealed a largely inverted ratio of early-to-late erythroblasts in *Mx1-Cre;Nf1flox/–* 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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Corrigendum

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The genotype of the Nf1 mutant mice was incorrectly described. The correct text for Methods appears below.

Mice and treatment procedures. Mx1-Cre;Nf1tm1Par/tm1Tyj (referred to as Mx1-Cre;Nf1flox/–) and control mice (Nf1flox/+) 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;Nf1flox/– mice with MPN.

We first assessed the pharmacodynamic properties of 901 in WT and Mx1-Cre;Nf1flox/– (Nf1 mutant) mice that received an oral gavage dose of 5 mg/kg/d for 5 days.

We randomly assigned Mx1-Cre;Nf1flox/– 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;Nf1flox/– mice with MPN have ineffective erythropoiesis.

In striking contrast, profiling revealed a largely inverted ratio of early-to-late erythroblasts in Mx1-Cre;Nf1flox/– 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;Nf1flox/– 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;Nf1flox/– (Nf1) mice.

[Figure 2] Hematopoietic tissues from 6-month-old Mx1-Cre;Nf1flox/– (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;Nf1flox/– (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.