Author Correction: Tumor immunoevasion by the conversion of effector NK cells into type 1 innate lymphoid cell

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The Chief Editor is correcting this article at the request of the authors. An investigation by QIMR Berghofer Medical Research Institute found that Figs. 5e and 6d,e and Supplementary Figs. 5c,d and 8c were based on experiments for which no evidence of their conduct or primary data could be confirmed. As such, the data from the underlying experiments are believed to have been fabricated or are unreliable, respectively. The panels in question have been removed from the figures (see below). The major finding of the paper that TGF-β-driven conversion of NK cells into ILC1s is a novel mechanism by which tumors escape surveillance by the innate immune system remains unaffected. No concerns have been raised regarding other data in the paper.

Due to the age of the paper, the original article can no longer be edited in situ. The following replacement figures, legends and text edits represent the revised version of the article. Replacement Figs. 5 and 6 and Supplementary Figs. 5 and 8 are available in the Supplementary Information accompanying this amendment. The main text edits follow.

Results, "TGF- β limits innate cancer immunosurveillance" subsection:

End of first paragraph, remove "By using a higher dose of MCA (300 μg), we confirmed the greater resistance of *RII*^{FL} mice than that of *Ncr1*$^{cre/wt}$ or *RII*^{WT} mice (Fig. 5e)." Second paragraph, two instances of "(Fig. 5f,g)" to read "(Fig. 5e,f)." Fourth sentence to be replaced by "The subcutaneously transplanted SM1WT1 tumors in *RI*$^{CA-FL}$ mice were characterized with more tumor intILC1s and ILC1s and fewer NK cells ($P < 0.01$; Supplementary Fig. 5b)." Fifth sentence ("SM1WT1 melanomas … (Supplementary Fig. 5d)") to be deleted. End of sixth sentence, remove "and melanoma growth."

Results, "NK cells control experimental metastasis" subsection, middle of first paragraph, remove "Notably, we confirmed, in the RM-1 model, our results showing that treatment with anti-asGM1 reduced the number of lung metastasis in *RI*$^{CA-FL}$ mice (Fig. 6d). Of note, the administration of anti-asGM1 did not affect the number of lung metastasis in *Mcl1*^{FL} mice (Fig. 6e)."

Results, "TNF promotes resistance to innate immunosurveillance" subsection:

Fourth sentence, remove "and Supplementary Fig. 8b." Fifth sentence to be replaced by "Using MCA1956 tumor model, we demonstrated that IFN-γ was important in limiting tumor growth, whereas TNF probably fueled tumor growth, in *RII*^{WT} mice (Fig. 8d)." End of sixth sentence, remove "and Supplementary Fig. 8c."

Results, "Human tumor ILC1-like cells" subsection, first paragraph, third and fourth sentences, replace "(Supplementary Fig. 8d)" and "(Supplementary Fig. 8e)" with "(Supplementary Fig. 8b)" and "(Supplementary Fig. 8c)", respectively.

Methods, "*In vivo* tumor models" subsection, second sentence "…wild-type control mice were given subcutaneous injection of 25 μg or 300 μg MCA," remove "or 300 μg."

Replace Figs. 5,6, Supplementary Figs. 5, 8 legends as follows:

Figure 5. TGF-β signaling limits tumor immune surveillance by converting tumor NK cells into intILC1s and ILC1s. (**a**–**d**) Tumor incidence of indicated transgenic or C57BL/6 mouse strains after s.c. injection of 25 μg of the carcinogen methylcholanthrene (MCA). Some mice were treated with anti-asGM1 or control antibody (ctrl IgG). Sample size (*n*) for each group is depicted in each legend (log-rank test; $^{*}P < 0.05$, $^{**}P < 0.01$). (**a**–**c**) were the same experiment, while (**d**) was an independent experiment. (**e**) Representative flow cytometric plots showing tumor group 1 ILC composition in MCA-induced fibrosarcomas from indicated transgenic mice. (**f**) Corresponding quantification of indicated tumor group 1 ILC subsets ($n = 6$ for *RII*^{WT} and

RII^{FL} mice; $n = 5$ for *RI*$^{CA\text{-}WT}$ and $n = 8$ for *RI*$^{CA\text{-}FL}$ from 7 independent experiments; mean ± s.e.m.; Mann-Whitney *U* test; $^{*}P < 0.05$, $^{**}P < 0.01$).

Figure 6. TGF-β signaling abrogates innate immune control of metastasis.The number of experimental lung metastases in WT or transgenic mice 14 days after i.v. injection of 10^5 (**a**), 10^4 (**b**) and 5×10^3 (**c**) B16F10 melanoma cells. The following number of mice were used for each group: (**a**) WT and *Ncr1*$^{cre/wt}$ $n = 10$, *RII*^{WT} $n = 3$, *RII*^{FL} $n = 6$, *RI*$^{CA\text{-}WT}$ $n = 5$, *RI*$^{CA\text{-}FL}$ $n = 6$; (**b**) *RI*$^{CA\text{-}WT}$ and *RI*$^{CA\text{-}FL}$ $n = 10$, *Mcl1*^{WT}, *Mcl1*^{FL}, WT + ctrl IgG and WT + anti-asGM1 $n = 5$; (**c**) *RI*$^{CA\text{-}WT}$ + ctrl IgG $n = 9$, *RI*$^{CA\text{-}WT}$ + anti-asGM1 $n = 7$, *RI*$^{CA\text{-}FL}$ + ctrl IgG and *RI*$^{CA\text{-}FL}$ + anti-asGM1 $n = 4$. Results in (**a**–**c**) were from one experiment each. Data shown as mean ± s.e.m.; one-way ANOVA and Tukey's multiple comparison test; $^{*}P < 0.05$; $^{**}P < 0.01$, $^{***}P < 0.001$, $^{****}P < 0.0001$.

Supplementary Figure 5

TGF-β signaling affects tumor group 1 ILC components in SM1WT1 tumors.

(a) Representative flow cytometric plots showing group 1 ILC composition in SM1WT1 melanomas harvested from indicated transgenic mice at day 24 after tumor injection. ND, not determined. **(b)** Corresponding quantification of tumor group 1 ILC subsets (mean ± s.e.m.; $n = 8$ for *Ncr1*$^{cre/wt}$ mice, $n = 7$ for *RII*^{FL} mice and *RI*$^{CA\text{-}FL}$ mice of two independent experiments; one-way ANOVA and Tukey's multiple comparison test; $^{***}P < 0.001$, $^{****}P < 0.0001$).

Supplementary Figure 8

Phenotypic features of group 1 ILC subsets SM1WT1 melanomas and in GIST patient PBMCs and TILs.

(a) IFN-γ (left) and TNF (middle) production by tumor group 1 ILC subsets from SM1WT1 melanomas after 4 h stimulation by PMA/ionomycin. Percentages of cytokine producing cells were determined by flow cytometry and ratios of IFN-γ/TNF-producing cells (right) were calculated (mean ± s.e.m.; $n = 7$ tumors from one experiment; one-way ANOVA and Tukey's multiple comparison; $^{*}P < 0.05$, $^{**}P < 0.01$). **(b)** Representative flow cytometric plots showing the gating strategy for the analyses of CXCR6 expression on CD3$^-$CD56^{dim} and CD3$^-$CD56^{bright} human NK cells from PBMC of healthy volunteers (HV) and GIST patients as well as tumor infiltrating lymphocytes (TILs) from GIST patients. **(c)** Representative flow cytometric plots showing the gating strategy for the analyses of CXCR6 expression on NK1.1$^+$NKp46$^+$ tumor group 1 ILCs in MCA1956 tumors ($n = 5$ per group of two independent experiments).

Supplementary information is available in the online version of this amendment.

Additional information

Supplementary information The online version contains supplementary material available at https://doi.org/10.1038/s41590-024-01799-9.