

HUMANIZED CD200/CD200R MICE AS A TOOL FOR EVALUATING NOVEL THERAPEUTICS

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Background Tumor-associated myeloid cells (TAMCs) represent a diverse population of immune cells capable of regulating tumor growth and progression via tumor-specific inflammation. Targeting key pathways that drive inflammation in the tumor microenvironment (TME) via TAMCs is of intense therapeutic interest. In particular, the membrane glycoprotein, CD200, has been shown to be expressed in several cancers and relays an immunoregulatory signal via CD200R, which suppresses anti-tumor immune responses. Therefore, the interaction between CD200 and CD200R plays an important role in regulating the TME via TAMCs.

Methods To explore potential therapeutic strategies targeting CD200/CD200R, we generated a human *CD200/CD200R* knockout/knock-in *in situ* (B-hCD200/hCD200R) mouse model to evaluate the efficacy of anti-human CD200 antibodies. We used flow cytometry to assess CD200 and CD200R protein expression and compare immune cell percentages between B-hCD200/hCD200R and wild-type mice.

Results We confirmed human CD200 and CD200R protein expression in B-hCD200/hCD200R mice by flow cytometry. We next observed that the overall development, differentiation, and distribution of splenic and lymph node immune cells were similar between parental wild-type C57BL/6 and B-hCD200/hCD200R mice by flow cytometry.

Conclusions Our data demonstrates B-hCD200/hCD200R mice are a robust preclinical model for *in vivo* efficacy evaluation of therapeutic candidates.

Ethics Approval All animal studies were reviewed and approved by the Institutional Animal Care and Use Committee (IACUC) of Biocytogen Beijing Co., Ltd.

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