TetOp16 Transgenic Mouse Model for Cell Proliferation (Ref. No. 223-GE)

Background

The pharmaceutical industry currently uses cyclin-dependent kinase (CDK) inhibitors to target cell proliferation for cancer chemotherapy. Such CDK inhibitors have some off target effects. The protein $p16^{INK4a}$ is a potent cell cycle inhibitor and is a relatively selective CDK inhibitor. Induction of p16 inhibits cell proliferation and thus can help to block the growth of tumors in a specific manner.

Summary of the Invention

Dr. Greg Enders developed TetOp16 transgenic mice which would allow for cell, tissue or organ-specific expression of $p16^{INK4a}$, thereby allowing a broad control of cell proliferation in mice. TetOp16 transgenic mice allow for the inducible expression of $p16^{INK4a}$. In these transgenic mice, the nucleic acid sequence encoding human $p16^{INK4a}$ has been linked to an inducible promoter or an operon capable of activation by the reverse tetracycline-controlled transactivator (rtTA), mediating p16 transcription. TetOp16 mice can be mated to mice carrying rtTA transgenes in the tissue of interest. p16 expression can be induced in a regulated fashion in the doubly transgenic mice by administering antibiotic doxycycline or tetracycline.

Applications of the Invention

Studies on cell proliferation are usually associated with deleterious side effects such as DNA damage and inflammation. TetOp16 transgenic mice offer a way to selectively manipulate cell proliferation by allowing for temporal and spatial regulation of $p16^{INK4a}$ expression. Thus, this mouse model is a very useful tool in research studies of cancer, development, tissue renewal, aging, and a variety of chronic diseases that involve cell proliferation.

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