# Reducing the side effects of a multiple sclerosis drug

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The drug FTY720 is approved for the treatment of multiple sclerosis. Although highly effective it can have serious side effects, including reduced lung function and fluid accumulation in the eye.

Understanding the multiple molecular mechanisms by which the drug affects its target (the S1P receptor) could lead to the development of a drug with the same therapeutic efficacy but reduced side effects. In this context, a team of researchers, led by Timothy Hla, at Weill Cornell Medical College, New York, has now detailed the molecular mechanism by which FTY270 causes adverse effects in the lungs of mice.

Specifically, Hla and colleagues outlined a mechanism by which FTY270 causes S1P receptor degradation on the cells lining the blood vessels of the lungs and found that this reduction in S1P receptor levels causes leakage of the blood vessel contents into the lungs, impairing lung function. In contrast, S1P receptor degradation appears not to be required for the effects of FTY720 on immune cells, which are the effects that mediate its therapeutic efficacy. Hla and colleagues therefore suggest that developing a drug that does not act on S1P receptor on the cells lining the blood vessels of the lungs but does target S1P receptor on immune cells may provide a therapeutic with decreased side effects.

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