A Load of Fit

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The conventional wisdom is that drug-induced viral resistance is always evil—the leading cause of treatment failure. But a new Harvard University study offers tentative backing for the hope that treatment smartie Mike Barr first floated in these pages three years ago: that certain mutations may result in a *weaker* virus with a much-reduced ability to replicate. The researchers reported that the D30N mutation of the virus, which causes resistance to nelfinavir (Viracept), substantially reduced its replicative capability. The L90M mutation that confers resistance to saquinavir (Fortovase) resulted in a moderate decrease in viral fitness, while the mutations creating indinavir (Crixivan) resistance left the virus as fit as ever. Although cross-resistance among the meds has long been thought to make the second-choice drugs less effective in those previously PI-treated, changes in viral fitness may turn out to be a major factor. If these test-tube observations can be confirmed with viruses taken from PWAs whose drugs have failed, expect new and improved strategies about which drugs to take, and when.

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