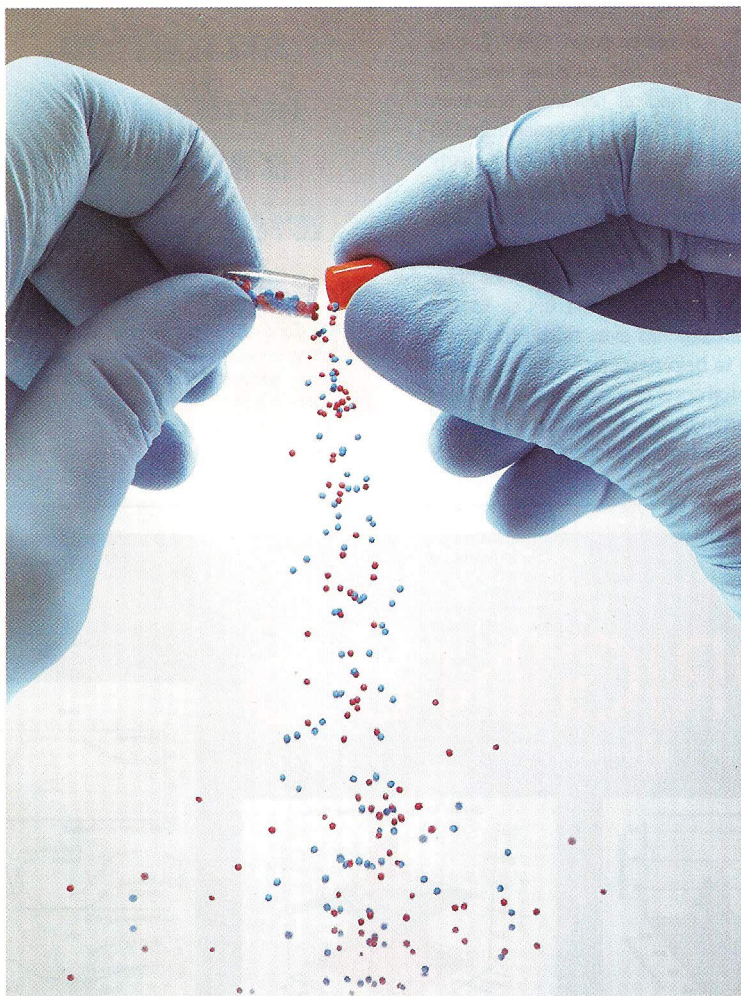


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Curing the Common Cold

Be careful what you wish for. The remedies may be far worse than the illness



Who has not dreamed of a cure for the common cold? It might be a pill that banishes the sniffles, to be taken as soon as you notice the symptoms. Or better yet, a vaccine administered before kindergarten, along with those for measles and mumps. Imagine a world without colds—without weeks of wet Kleenex and phlegmy avalanches in your sinuses. It sounds pretty perfect.

Scientists, in fact, are working toward a vaccine against rhinoviruses—the group that causes 30 to 50 percent of colds. But, ironically, even if it succeeds or if drugs that stop colds in their tracks are found, we may well decide that most of us are better off without these wonder drugs, after all.

Goodness knows our dreams of becoming sniffles-free have

been dashed before. Take the case of the anticold drug pleconaril, which received sensational media coverage in 2002 while it was still in clinical trials. Heralded as “the miracle drug,” “the magic bullet” and “the Holy Grail,” it performed well in cell culture, but its effects in human subjects were not very impressive—it shortened colds by one day. What prompted the U.S. Food and Drug Administration to reject pleconaril, however, were the side effects. It caused some women to bleed between menstrual periods and interfered with hormonal birth control. Indeed, two women in a trial became pregnant while taking it. Numerous other candidates have been abandoned owing to their adverse effects, including nasal inflammation worse than the infection. The common cold, it turns out, is not that bad, compared with some treatments.

Much effort has also gone into vaccines, particularly against rhinoviruses. (Colds are also caused by adenoviruses, coronaviruses and other virus families.) Like HIV, a rhinovirus consists of an RNA genome cocooned in a shell of proteins called a capsid. The virus attaches to the membrane of a host cell, injects its genetic material and then hijacks the host’s machinery to make more of itself. It is the body’s own inflammatory immune response, not viral replication itself, that causes symptoms.

In searching for vaccine candidates against rhinovirus, researchers have concentrated on looking for some piece of the capsid that is the same across all types. A vaccine containing that piece, when given to a healthy person, should, in theory, cause the immune system to produce antibodies against it so that he or she is primed to fend off a later infection by all viral strains bearing that piece. The goal is to pick a shared fragment that does not change much over time; the best vaccines and drugs can be rendered useless by a significant alteration in a target.

For years researchers met disappointment after disappointment in their efforts to find a conserved element in rhinoviruses. Examination of more than 100 variants of the virus had turned up no commonality, says Thomas J. Smith, who studies the structure of the virus at the Donald Danforth Plant Science Center in St. Louis. This variability occurs because, as an RNA virus, a rhinovirus is prone to mutations. The enzymes that replicate RNA do not have any of the proofreading mechanisms possessed by the enzymes that replicate DNA, so each new virus may be peppered with changes in its code, and each type

