Thus, Starko’s intriguing hypothesis fails the test of dose-response. That is to say, in countries such as the United States, where salicylates were more available, mortality was much lower compared with regions where salicylates were less readily available. These observations are at the ecological level, and such comparisons are notoriously susceptible to confounding. However, if the salicylate hypothesis applies universally, then the ecological confounding would have to operate such that the salicylate-influenza connection is stronger in countries with less access to aspirin, which seems a priori unlikely. Indeed, the overwhelming majority of the millions of Indian peasants who were killed by the flu certainly had no access to salicylates whatsoever. If the salicylate hypothesis only works in the United States and in similar settings, then we question its validity given the worldwide scope of severe mortality in 1918–1919.

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population, the number of deaths attributable to aspirin could be substantial. One step in proving or disproving the hypothesis is comparison of outcomes for those treated and those not treated with aspirin in 1918. Although I am unaware of any study of influenza with 1918 aspirin doses, a 1983 study of 47 college students with influenza A/Brazil/78 H1N1 comparing daily doses of 3.25 g aspirin to 100- and 200-mg doses of amantadine found worse symptom scores at 48 and 72 h in the aspirin and the 200 mg amantadine groups as well as a 35% discontinuation rate for bothersome symptoms in the aspirin group [6]. Meticulous records, such as those kept by the military in 1918, may be an excellent source of additional information.

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