writing was originally done with the left hand. The alternative explanation, that naturally left-handed persons invented phonetic scripts, is even more far-fetched. One possible avenue of exploration could be that phonetic scripts were conceived in the hemisphere (known to be associated with musical ability) opposite to Broca's area, finding their outlet via the left hand. The transition from left-handed to right-handed writing occurred gradually in ancient Greece, exemplified by several texts (such as on Apollo's temple at Delphi) written in zig-zag form with normal and mirror script alternating. As many Greek (and Roman) capital letters are bilaterally symmetrical, it is not difficult to read mirror script. By contrast, establishing mirror versions of asymmetrical scripts such as Semitic would have been tantamount to creating a new script, which is perhaps why the original version was retained after the more dexterous hand took over writing. Indeed, the practice of suppressing left-handed writing might well have had its origins in an effort to establish a single writing style rather than in a mystical aversion to the left hand, as is widely believed.

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Virus release evaluation

SIR — There have been several recombinant approaches to increase the speed by which insect-specific viruses kill pest species; recent data indicate that insect viruses can be engineered to increase dramatically the speed of kill[1–4]. This advance makes these viruses far more attractive for use in agriculture. Williamson's Scientific Correspondence[5] addressing the News and Views article by Hochberg and Waage[6] is valuable in focusing attention on the safety of these genetically engineered organisms. Although these prototype viruses offer numerous potential benefits[6], possible risks need to be addressed, certainly by discussions between scientists and the public, and by design of rigorous experiments to evaluate safety.

These viruses are being designed as insecticides, not as biological control agents that will become permanently established. As augmentative biological control relies increasingly on inundative release of commercially available agents, the strategies being developed tend to be like those for insecticides. In such releases it is important to adopt a more holistic approach cognizant of environmental consequences, not only for natural and engineered pathogens, but also for predaceous and parasitic anthropods[7,8]. By referring both to the wild-type and engineered viruses as viral insecticides, we remind ourselves not to repeat the mistakes that were made with synthetic chemical insecticides.

This terminology also has a positive connotation regarding Williamson's legitimate worry that nontarget lepidopterous larvae could be permanently endangered by these engineered viruses. Because these materials are being designed for an insecticide-type application and not as 'biological control' agents that will replicate in the environment, this concern is in part overcome. The fact that these viruses kill insects much more quickly than wild-type viruses leads to far fewer infective virus particles being produced, because the larvae are killed when they are very small and at an early stage in viral replication. Thus, by their mechanism of these actions, recombinant viruses will be less competitive than wild-type organisms. The risk to nontarget species seems more analogous to classical synthetic insecticides in that it arises primarily from the initial application. Recombinant viruses appear to vanish rapidly from the ecosystem, and the risk to nontarget species following direct application of large doses is being systematically addressed by the Oxford NERC group referred to by Williamson. Data so far indicate an intrinsically greater specificity of the recombinant viruses for target insects than either synthetic chemical insecticides or *Bacillus thuringiensis*. It is critical that such studies on stability and specificity of the viruses continue.

Certainly, it is important to disable the viruses. But the approach of using polyhedron-negative constructs as suggested by Williamson results in viruses so severely disabled that they are of little value in agriculture. Fortunately, there are other means to limit replication of engineered viruses in the field. Further research into the molecular mechanism of host specificity will enhance our ability to target more pest species, with greater safety to nontarget species.

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Mongols and soap

SIR — Cowart[1] has recently explained how to test the clinical disorders of smell. The earliest record of a disturbing soap-eating experience appears in the Thirteenth century, when the Mongol envoys came to the court of French king Philip *Le Bel* with the goal of discussing the joint Crusade in the Middle East[2]. Upon arrival, the Mongol ambassadors were offered a bar of soap and a towel to wash off the dust from their long journey. The Mongols, assuming that the presentation of a piece of dried cheese was also a customary homage in the land of the Franks, had a good bite of it. It is unlikely that the Mongols were anosmic. It is also questionable that Philip IV was a notorious olfactory psychophysicist and was conducting chemosensory measurement experiments.

We do not know of the fate of the washroom attendant at the hands of sixteen deceived Barbarians whose tribe had just conquered half of the world. There is little doubt that the Mongols preferred to conclude the incident by demonstrating their skill in martial arts rather than in diplomacy. The influence of this episode on the gloomy destiny of the Crusades is not yet measured, but since then *savon* became the only known French word adopted by the Mongol vocabulary.

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Diabetes and heat shock protein

SIR — Cornall *et al.*[1] identify a new susceptibility gene, IDD5, for insulin-dependent diabetes in the non-obese mouse. The identity of the gene product in this region, however, remains unknown, although linkage has been identified to the interleukin-1 receptor and to the *Lsh/Ity/Bcg* gene. This gene is involved in the immune response to several organisms such as mycobacteria in which the major antigen involved is heat-shock protein 65. The human equivalent of IDD5 seems to be located within the 2Q32 region[1].

We have previously identified human heat-shock protein 65 as being a β-cell antigen in insulin-dependent diabetes in man and have identified the presence of binding antibodies to this antigen in the serum of a large proportion of newly diagnosed insulin dependent diabetic patients[2]. We have used a human heat-shock protein 65 gene probe λC5 to map the location of this gene by fluorescence *in situ* hybridization with a biotin-