followed by similar observations in the HL-A system. It is very likely that antisera against public antigens of the HL-A system—if such antigens in this system exist—are discarded as too complex and therefore unsuitable for serological analysis.

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Does Virus Infection have Evolutionary Significance?

ANDERSON's theory[1] that "evolution depends largely" on the transfer of genetic material by viral transduction may be plausible for microbes, but in cellular organisms there are some serious objections.

First, the argument that susceptibility to viral infection must have a selective advantage because organisms do not have the complete resistance which they could evolve is equally valid for susceptibility to bacterial infections, parasites and even predators. That an organism has not reached an imagined "perfection" is insufficient reason to impute a function to the existing "imperfection".

Second, it is improbable that the "interchange of genes 'on approval'" would promote parallel evolution in cellular organisms to any significant degree, because of the integration of genotypes (coadaptation)[2]. It is unlikely that an alien gene or block of genes will perform well in a genetic environment with which it has never had any previous interaction.

The author's parliamentary analogy falls on the same point. Populations harbour vast amounts of genetic variation[3-5]. Thus to be closer to reality, the analogy should be to a parliament with a vast library of variant versions of laws, nearly all of which are being tried out constantly, in a country where many statutes interlock. An entirely new statute would have little chance of being compatible with the pre-existing complex system and would almost certainly cause chaos if it were introduced even in a somewhat altered form. The question is whether there is any "need" for entirely new statutes while the vast library of variants exists.

In this connexion, Anderson supposes that "plants and animals which are free from virus infection would evolve very slowly if at all". I suggest that the important kind of variation for evolutionary change is that exemplified by isozymes[3,4], as well as by concealed recessives detected in *Drosophila*[5], and that this sort of variation is abundant in nature. The very recently published evidence of a similarly large variation in *Limulus*[6], an animal long thought to have had an extraordinarily stable evolutionary history, further argues against any simple relationship between the amount of genetic variation and the speed of evolution.

My last and most serious objection concerns the mechanism by which cellular organisms would evolve susceptibility to virus infection. Anderson believes that the selective advantage of infectability is in allowing novel genes to be taken in and tried out. A novel gene would have to increase the reproductive success of the host and also have to be incorporated into the host's germ cell line if genes for susceptibility are to increase in frequency in the host population. If the expression of the novel gene did not increase the fitness of the individual into which it was transferred but only that of some descendant, then there would be no selection at the level of the individual and no increase in genes for susceptibility. The only mechanism left would be group selection, a tenuous hypothesis of doubtful validity[7].

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Hangover Effect of Hypnotics in Man

DRUGS used to induce sleep—the hypnotics—are among the most widely used of all medicaments. It has been estimated that "At a very rough reckoning about one night's sleep in every ten in Britain is hypnotic-induced"[1]. This assertion stems from the number of prescriptions each year for barbiturate (20 million) and non-barbiturate hypnotics (5 million)[1,2] and emphasizes the importance of studying the detailed clinical pharmacology of such drugs. But so far there has been little attention to the residual or hangover effect detectable the next morning. Significant impairment of performance on a battery of psychological tests was found up to 15 h after a hypnotic dose (200 mg) of chlorpromazine or quinalbarbitone given at night[3]. Similarly, behavioural impairment and electroencephalographic changes have been reported 12 h or more after nitrazepam or amylobarbitone sodium[4]. In these studies, however, subjects were forbidden caffeine-containing drinks for the period of the study and were thus undergoing some degree of caffeine withdrawal.

We have investigated the hangover effects of two hypnotics each given in two doses compared with a placebo: butobarbitone sodium (100 and 200 mg) and nitrazepam (5 and 10 mg), a new non-barbiturate hypnotic, which is widely used and safe in overdoses[5]. Ten normal subjects each received all five treatments (placebo, and two drugs in two doses) at weekly intervals as part of a fully balanced design, using double blind procedures. They were told not to drink alcohol on the evenings when they took the sleeping tablets but were allowed their normal intake of caffeine-containing beverages both night and morning. The drug was taken at 23.00 h and the physiological and psychological tests included the electroencephalogram both at rest and during an auditory reaction time task, and palmar sweat gland activity. Psychological tests included key-tapping rate (a measure of simple motor speed), the digit symbol substitution test (a measure of coding and associative skills) and linear scales (100 mm) on which the subjects rated themselves for quality