point of discord: whereas the chicken genes for TAP, the supplier of class I-binding peptides, are in the class I region, the human ones lie in the class II region[2,4].

The vast sprawl of the human MHC means meiotic recombination between polymorphic genes occurs at rates of up to 2%. This mechanism generates new HLA haplotypes and is the source of the enormous variety of HLA phenotypes in the human population. By contrast the compact size of the chicken MHC essentially eliminates meiotic recombination as a force for generating MHC diversity, explaining why previous pedigree analysis had failed to find a single recombinant MHC haplotype[6]. As emphasized by Kaufman *et al.*[4], absence of recombination raises the probability that alleles for different genes within the same haplotype have co-evolved to cooperate in function. One way this could be manifest is for TAP allotypes to specialize in the delivery of peptides that bind well to the linked class I allotypes. On the downside, low-frequency recombination and small numbers of class I and II genes are properties that make the chicken MHC less plastic and adaptable to environmental change than its human counterpart. These features could therefore explain why associations of MHC haplotype with resistance and susceptibility to infectious disease are much stronger in inbred chickens than other laboratory animals or humans[7].

Some 40% of the expressed genes in the human MHC work for the immune system[2]. Although this level of commitment is unlikely to be a uniform feature of the human genome, other parts of it are clearly conscripted to defence. Well established at centre stage, along with the MHC, are the large arrays of rearranging gene segments that determine B-cell immunoglobulins and T-cell receptors. Now emerging from the wings are two families of conventional, non-rearranging genes that provide receptors for natural killer (NK) cells. These large lymphocytes provide defence from the start of a pathogen's attack, a response distinguishing them from B and T cells which only become useful after days of infection.

One form of NK-cell receptor resembles a C-type lectin and is specified by gene families in the natural killer complex (NKC) on human chromosome 12 (ref. 8). A second form of NK-cell receptor is constructed from immunoglobulin-like domains and is specified by gene families in the leukocyte receptor complex (LRC) on human chromosome 19 (ref. 9). Whereas some gene families in the LRC exhibit haplotypic and allelic polymorphism[10] those of the NKC are relatively conserved. An NK cell expresses several receptors drawn from these families and diversity within a person's NK-cell population arises from the expression of different receptor combinations. Some NK-cell receptors bind polymorphic determinants of HLA class I molecules and appear sensitive to the effects that infections have upon them. Because the MHC, NKC and LRC are on different chromosomes their polymorphisms segregate independently in the human population. Consequently some people express NK-cell receptors for which they have no MHC class I ligand, while others have the ligand but not the receptor. The drawback to this arrangement is allayed by having NK cells express several different receptors, so at least one of them binds a person's MHC class I.

Although human genes for NK-cell receptors and MHC class I ligands are strictly partitioned between different chromosomes, that is not so in the chicken. Kaufman *et al.*[4] describe two genes in the chicken MHC which specify proteins resembling the lectin-like receptors of mammalian NK cells. Such juxtaposition could also have fostered co-evolution producing NK-cell receptors that preferentially interact with class I allotypes encoded by the same MHC haplotype. Kaufman *et al.* take this idea one step further in their speculation that polymorphisms affecting NK-cell function are at the heart of the chicken's MHC-determined susceptibility to infection by the herpes virus that causes Marek's disease[7]. The idea is not so wild because an NK-cell-deficient patient presented with life-threatening herpes virus infections[11], and susceptibility to mouse cytomegalovirus, another type of herpes virus, maps to the NKC[8].

The two papers in this issue[2,4] vividly illustrate that working MHCs come in all shapes and sizes. The HLA complex is the sort of muddled mix of chaotic junk and organized function that one has come to expect from diverse and changeable selection by pathogens. By contrast, the simple elegance of the chicken MHC appears more the work of a single selective pressure which may or may not have anything to do with defence against pathogens[5]. Nevertheless, having crossed that particular road the chicken is seen to have put a lot of immunological eggs in a rather small basket. ■

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Daedalus

Nuclear autumn

Global warming is generally blamed on the emission of greenhouse gases by human activities, chiefly the burning of hydrocarbons. But one theory blames the declining intensity of cosmic rays. As they traverse the atmosphere, they nucleate water vapour to cloud droplets, just as in a particle-detecting cloud chamber. Clouds reflect sunlight; so the lower the cosmic ray flux, the fewer clouds, the more sunlight hits the Earth, and the higher its temperature. So to counter global warming, we need more atmospheric radiation.

The nuclear industry will welcome this argument. Nuclear power not only reduces the need to burn hydrocarbons; it generates radioactive waste as well. If instead of storing the waste in careful seclusion, we released it as fine dust into the atmosphere, it would soon be wafted up to cloud altitudes. It would nucleate new clouds with splendid efficiency.

This simple proposal would arouse almost the ultimate in environmental outrage. But Daedalus reckons that conventional fuel-burning can do the job instead. It too releases particles into the atmosphere. The finest and most efficient nuclei are probably those released in annoying clouds by diesel engines, a growing segment of the automotive market. Many engineers are trying to modify the diesel engine to reduce its particulate emissions; but DREADCO engineers are modifying the fuel so as to optimize those emissions. Their aim is to reduce the size of the particles to much less than a wavelength of light. They will then be invisible, and will no longer annoy the public. They will also be ideal condensation nuclei.

The project has many hopeful clues to follow. Carbon black is made by burning fuel oil. For the finest soot particles, special alkali metal compounds are added to the oil. Fullerene chemists have their own tricks for burning fuels to very small carbon particles. So with good fortune DREADCO's clean-exhaust, high-nucleation, save-the-planet diesel fuel will soon hit the market. Ecologically aware consumers will rush to buy it. Its invisible exhaust particles, wafted aloft by atmospheric turbulence, will encourage global cloud cover and counter global warming. Sadly for cold, humid Britain, their nucleation properties will be even more effective at ground level. In dense winter traffic, the relentless nucleation of innumerable exhausts will create appalling fog on the motorways. **David Jones**