2. **M.R.Smallman-Raynor and A.D.Cliff, Acquired Immune Deficiency Syndrome (AIDS) : literature, geographical origins and global patterns, Progress in Human Geography, Vol. 14, 1990.**

This paper is designed to introduce geographers to a field which is naturally dominated by medical scientists. We attempt to identify those areas in which geographers can best employ their particular skills; in so doing, we identify research questions which may permit geographers to make an effective contribution to knowledge about the disease.

The origins of HIV/AIDS

Given the apparent recent emergency of HIV infection as a public health problem, there has been intense interest in the geographical Origins of the disease Researchers have sought to establish how far it is a 'new' disease, its spatial hearth(s), and the corridors of spread from any heartland areas identified by its present global distribution. This debate in the medical literature is readily recognized within geography as a classic issue in spatial diffusion and, as noted earlier, it is beset with what Renee Sabatier of the Panos Institute, London, calls the 'blaming others syndrome'. Despite the fact that nearly 60% of the world's AIDS cases reported to WHO by March 31, 1989 originated from the USA, most origin hypotheses have focused outside the developed west.

AIDS in the 'pre-AIDS' era

Prior to the isolation of HIV in 1983-84, speculation about a 1970s geographical source for a viral agent for AIDS focused upon Haiti, with the suggestion that the virus had mutated from the causative agent of African swine fever.

The limited scientific evidence to support the hypothesis that HIV had been imported from Haiti into the USA led some workers to propose a reversal of the supposed direction of spread, namely that Haitians were infected by vacationing American homosexuals. However, as attention has turned from one origin hypothesis to another, the geography of AIDS-related stigmatization and the concomitant deleterious implications for both peoples and researach has merely changed focus from one area to another, currently Africa.

The geographical spread of HIV

When knowledge of the structure of HIV appeared, similarities between HIV and certain virus found in African green monkeys were soon recognized. As a consequence, Africa emerged (replacing Haiti) as the main candidate for the hearth of the disease.

The African origin hypothesis

If retrospective serological analysis points to an African hearth (and this is by no means certain given the problematical interpretation of results and the limited number of tests conducted prior to the mid-1970s on western sera), the question then arises of how HIV appeared. Two main strains of the human immunodeficiency virus have been identified so far - HIV-1 and HIV-2. The arguments about the origins of the virus are complex and

full of contention. One possible model, outlined by Essex and Kanki (1988), is that the virus jumped the species barrier to HIV-2 from a related simian immunodeficiency virus, coded SIVagm, found in green monkeys. The virus then mutated to the highly pathogenic strain, HIV-1, most commonly found in man. If HIV-2 is, as Essex and Kanki propose, an intermediate to HIV-1, then west Africa may be the place of origin of HIV. In fact, Clavel (1987) suggests that Guinea-Bissau may be the geographical epicentre of HIV-2.

An alternative hypothesis sees HIV-2 developing in central Africa (Gallo, 1987), as well as in West Africa, with termination in HIV-1 reached earlier in central than west Africa. The possibility of independent development of HIV in two areas of the continent has not been addressed. One problem confounding both a spread hypothesis from west Africa to central Africa, or an independent evolution hypothesis, is the lack of evidence of HIV- 2 infection in central Africa (for example, Kanki et al., 1987); some suggestion of HIV-2 infection in that region would be expected under both hypotheses. The isolation of a possible HIV- 3 from Gabon (Marx, 1988) may be a key link in the story. With regard to spread, Saimot et al. (1987) see the historical links of Portuguese with both west and central Africa as in need of assessment.

The Global spread of HIV-1: hypothesis and evidence

The only general model for the global spread of HIV-1, is that proposed by Robert C.Gallow, codiscoverer of HIV-1. Following the early work of Kanki and collabortors regarding the simian origin hypothesis, Gallo (1987) suggested that SIVagm entered the human race in the vicinity of Lake Victoria, that a 'series of mutations' yielded an 'intermediate' virus (HIV-2) before 'terminating in the fierce pathology' of HIV-1. The lack of concordance of regions of endemic HIV-1 and HIV-2 infection is a major geographical conundrum in this model.

Given the uncertainties surrounding possible AIDS cases before 1980 and the controversy spawned by the African origin hypothesis for HIV, it is impossible to be sure of the transmission corridors followed in the spread of HIV-1 between Africa, the USA and western Europe under the Gallow model. However, a conventional spatial diffusion model seems a much more likely candidate to account for the present global distribution of the disease than a model which postulates independent origins. While independent evolutions of HIV may theoretically have occurred on different continents, this is inherently unlikely given the complex structure of the virus.

The global spread of HIV-2

HIV-2, first isolated in France in 1986 from individuals native to the Cape Verde islands and Guinea-Bissau (Clavel et al., 1986), is now known to be endemic in many West African states, most notably Senegal, Cape Verde, Guinea-Bissau, Guinea, Ivory Coast, Ghana and Burkina Faso (Fleming, 1988). Despite sporadic reports of HIV-2 infection in other countries of Africa, west Africa is the only known area of HIV-2 endemicity. Because Guinea-Bissau is the country of most extensive infection, Clavel (1987) has suggested that HIV-2 may have evolved there.

Many practical problems surround the detection of HIV-2 infection. Tests have also been hindered by cross-reactivity between antibodies to HIV-1 and HIV-2. The problem is particularly acute when attempts are made to determine dual HIV- 1/2 infections. In essence, tests for HIV-2 may give positive results due to the presence of antibodies to HIV-1.

The global distribution of HIV-2 is likely to be far wider than is currently known. Yet, because of the apparently restricted geographical extent of HIV-2, observation of future spread may offer a better testbed for those modelling spread than its sister virus, HIV-1.

Conclusion : further research

This paper has attempted to introduce geographers to the vast and rapidly growing medical literature on the AIDS pendemic.

The more general issue has been raised of which research questions can be most helpfully pursued by geographers, working in a field properly dominated by medical scientists. There is no doubt that the medical literature on AIDS/HIV is weak on all aspects of dynamic and cross-sectional mapping. Individual papers are also frequently narrowly conceived. There is, therefore, enormous scope for the traditional geographical skills of careful mapping and interpretation of maps paying particular attention to any casual inferences which may be made. Linking studies are also immensely valuable. The material presented on the geographic origins of the two main strains of the virus, HIV-1 and HIV-2, along with model of spread from its suspected African hearth to the present global distribution is illustrative of the work envisaged.

Such maping leads naturally to other questions. How certain are we of the African origin hypothesis? Can we develop time-space forecasts at different geographical scales of the arrival, speed of growth and ultimate size of the epidemic? What are the implications of the extraordinarily long incubation period of the disease for conventional spatial diffusion/epidemic models? Can we eventually generate forecasts with sufficient accuracy to guide policy decisions on investment in appropriate health care, targeting educational programmes of particular risk groups or, hopefully at last, patterns of vaccination?