the cold and others at 37° C, present in patients with SLE, rheumatoid arthritis (RA) and infectious mononucleosis. Messner (Albuquerque) reported the presence of cytotoxic antibodies against lymphocytes in the majority of family members of patients with SLE, irrespective of consanguinity. A virus or other transmissible agent may be involved.

F. Dixon (Scripps Clinic, La Jolla) emphasised the analogy between the role of endogenous virus antigens in mice and the appearance of SLE in humans. In all mouse strains virus-specific antigens are found, and in some strains complexes of these antigens and antibodies, as well as nucleic acids and antibodies, accumulate in the renal glomeruli.

R. Lerner (Scripps Clinic, La Jolla) presented evidence obtained in his laboratory and that of E. Boyse in New York that a mouse thymocyte differentiation marker, G_{IX}, is the same as a glycoprotein (gp70) in the virions of murine oncornaviruses. The oncornavirus genomes are integrated in the host genomes, and the expression of gp70 is under control of factors both genetic (manifestation on the membrane of thymocytes in G_{IX}^+ strains only) and environmental (manifestation only under the influence of the thymus). In G_{IX}^- strains the antigen is present only in leukaemic cells. Thus the distinction between virus and host gene products becomes tenuous, and whether immune responses to them are regarded as autoimmune is a question of semantics. As in mice and chickens, all human cells seem to have in their genomes lengths of DNA hybridising with oncornavirus nucleic acid, and the number of copies of oncornavirus DNA is increased in SLE and leukaemia.

B. Pernis (Basel Institute for Immunology) pointed out that most human B lymphocytes have surface IgM as well as IgD, the proportion varying from cell to cell. These turn over at different rates: when they are lost after capping or protease treatment IgM is resynthesised much more rapidly than IgD. W. Hijmans (Institute for Experimental Gerontology, Rijswijk) reported that the only surface immunoglobulin of lymphocytes in young human foetuses is IgM, which invalidates the view that IgD is the first to appear.

H. Muller-Eberhard (Scripps Clinic, La Jolla) described the amino-acid sequence of the complement component C3. The cleavage product C3a requires a high α-helical structure for activity. When C-terminal arginine is removed from C3a, the product binds to mast cell receptors and norepinephrine receptors of blood vessels and blocks their activation. K. Austen (Harvard) emphasised the symmetry of the steps involved in activation of complement by the classical and alternate pathways. Both pathways are activated in the joint fluids of rheumatoid arthritic patients. Austen concludes that the alternative pathway is activated *ab initio* but another explanation, that there is feedback activation from the classical pathway, is difficult to exclude.

J. Natvig (Institute of Immunology and Rheumatology, Oslo) reviewed recent observations on the heterogeneity of amyloid. A non-immunoglobulin AA protein from tissues of different patients shows identity over most of the 76 residues but some variation in the C-terminal region. Levels of the related serum component (SAA) are increased in sera of patients with rheumatoid arthritis and in old people. An immunoglobulin isolated from amyloid shows homology with λ-chains over the first 45 residues and seems to represent a new λ-variable subgroup.

L. Glynn (Kennedy Institute, London) discussed his model of persistent monoarticular arthritis following intra-articular injection of antigen into previously immunised animals. Complexes of antigen and antibody are found in menisci, but it is not clear whether these have any pathogenetic role. Release of hydrolytic enzymes from mononuclear phagocytes exposed to immune complexes or products of activated lymphocytes, discussed by A. Allison (Clinical Research Centre, Harrow), may be involved in joint damage in rheumatoid arthritis. There is still no convincing evidence of any causal role of an infectious agent in rheumatoid arthritis.

Immunopathology of parasitic infection

F.E.G.C. REPLIES to the responses in Matters Arising last week (**252,** 509; 1974) to his News and Views article (**246,** 187; 1973).

My statement that "most parasites are capable of evoking immune responses in their hosts but these are seldom effective in eliminating the infection" implies that some parasites do mount effective immune responses. Professor Urquhart and Dr Miller both refer to lungworm disease of cattle thus stressing the comparative rarity of the latter situation. It is true that there are well defined immune responses that eventually result in some degree of protective immunity against parasitic infections. But it is equally true that the actual elimination of the parasites as a result of an immunological response is not commonplace. The major parasitic diseases of man (malaria, sleeping sickness, Chagas' disease, American leishmaniasis, schistosomiasis and filariasis) are characterised by infections that last months or years. To most people this indicates a failure of the immune response to eliminate the infection. The immune response may ameliorate the disease symptoms but transmission of the parasite still occurs. Domesticated animals are more fortunate than man in being able to mount effective immune responses but in piroplasmosis and fascioliasis these responses are not very effective. It must also be remembered that helminth infections may diminish as a result of the natural death of the worms and not an immune response. This aspect of recovery from infection has been little studied.

My statement that "the possibilities of developing useful methods of immunisation [against parasitic diseases] fade further and further into the distance" does not imply that immunisation is always going to be impossible. Vaccination against cattle lungworm, canine hookworm and certain kinds of piroplasmosis has been achieved but the vaccines used, irradiated larvae or infected blood, are unacceptable in the field of human medicine. With the development of a vaccine against cattle lungworm nearly twenty years ago, it was felt by many workers that the solution to the problem of vaccination against parasitic infections was just round the corner. This early optimism has not been justified. As more and more is discovered about immunity to parasitic infections further complications are uncovered. The development of vaccines against many parasitic diseases may not be possible before the diseases are eliminated by drugs or public health measures. Trichinellosis is a case in point. There have been encouraging reports of possible immunisation procedures against *Trichinella* but its decline in the United States has been brought about by legislation designed to prevent swine fever. Studies on the immunological responses to parasites have been amply rewarded by discoveries in pure immunology, the development of immunodiagnostic techniques and an understanding of many immunopathological conditions. Vaccines will also be developed as a result of these studies—but not for many years to come.