

SECTION B - ABSTRACTS

1. GENERAL (INCLUDING LAND USE)

5925 Antwerp Trypanosomiasis Causal Modelling Group, 1989.

Constructing a causal model of African human trypanosomiasis. *Annales de la Société belge de Médecine tropicale*, **69** (Suppl. 1): 49-72. (See also **12**: no. 5938.)

Prince Leopold Institute of Tropical Medicine, Nationalestraat 155, B-2000 Antwerp, Belgium.

Following the workshop at Antwerp in January 1988, a group of scientists at the Prince Leopold Institute of Tropical Medicine, Antwerp, started to develop a causal model of human African trypanosomiasis. The group hypothesised a series of relations between determinants and associated factors of the prevalence of sleeping sickness. These relations were pictured in a logically structured hierarchical representation of the causal web of sleeping sickness.

Authors' abstract

5926 **Baker, R.D., 1989.** Calculating the basic reproductive rate R_0 when there are 2 or more pathogens. *Annales de la Société belge de Médecine tropicale*, **69** (Suppl. 1): 99-107. (See also **12**: no. 5938.)

Academic Information Services (Computing), University of Salford, Salford, M5 4WT, UK.

Some reasons why observed prevalences of the three major trypanosomes in cattle and livestock animals might not be independent are discussed. Apart from genuine interactions between the infections, averaging over heterogeneous populations produces an apparent correlation between pathogens. More subtly, chemotherapy and mortality both induce a positive correlation between pathogens. The calculation of the basic reproductive rate R_0 for one pathogen in the endemic presence of another is explained, and illustrated by a simple calculation. Although multi-pathogen calculations are difficult, the calculation of R_0 is both useful and feasible.

Author's abstract

5927 **Barrett, J.C., 1989.** *Tsetse control, land use and livestock in the development of the Zambezi Valley, Zimbabwe: some policy considerations.*

Addis Ababa; ILCA, African Livestock Policy Analysis Network (ALPAN Network Paper no. 19).

Tsetse Control Branch, Department of Veterinary Services, P.O. Box 8283, Causeway, Harare, Zimbabwe.

Major rural development projects are being planned and implemented in the tsetse-infested Zambezi Valley in Zimbabwe. This semi-arid region is considered a fragile eco-system with generally limited agricultural potential, where the sustainability of traditional agro-pastoral farming systems may be questioned. This paper examines issues relating to the introduction of livestock into the Zambezi Valley and their implications for tsetse control policy. Emphasis is placed on the need for economic and land use planning inputs to tsetse control programmes.

J. C. B.

5928 **Beghin, I., Muynck, A. de, Vanderstuyft, P. and Mentens, H., 1989.**

Can the causal model approach contribute to the study of the epidemiology and the control of sleeping sickness? *Annales de la Société belge de Médecine tropicale*, **69** (Suppl. 1): 31-47. (See also **12**: no. 5938.)

Prince Leopold Institute of Tropical Medicine, Nationalestraat 155, B-2000 Antwerp, Belgium.

A causal model links together, in an hierarchical manner, a set of hypotheses about the causes of, and mechanisms leading to, a phenomenon under study. Initially used in nutrition studies for identifying the variables to be observed, such models have proven useful in the choice and evaluation of interventions, as well as in the selection of relevant special studies to be carried out inside a broader research programme. The authors describe a technique of model building used by nutritionists, and argue that this approach could be of benefit in the study and control of African trypanosomiasis. Their hypothesis is that although causal models do not substitute for mathematical models, the latter ones, and epidemiological models in general, would be (1) more correct logically; (2) fitting reality more closely; and (3) more useful for the analyst, the decision maker and the field worker, if they were built from a preceding causal model.

Authors' abstract

5929 **Brouwere, V. de and Pangu, K.A., 1989.** Réflexions sur la flexibilité

d'un service de santé intégré dans la lutte contre la trypanosomiase à *Trypanosoma brucei gambiense*. [Reflections on the flexibility of an integrated health care service in the fight against trypanosomiasis due to *T. b. gambiense*.] *Annales de la Société belge de Médecine tropicale*, **69** (Suppl. 1): 221-229. (See also **12**: no. 5938.)

Institut de Médecine Tropicale Prince Léopold, Nationalestraat 155, B-2000 Antwerp, Belgium.

The difficulties encountered by the vertical control programme for *gambiense* human African trypanosomiasis in various countries, and the achievements of primary health care policies on the other hand, justify the consideration of an alternative operational management model for this disease. The alternative model presented here is the integrated model. The authors define the concept of integration, discuss its justifications and describe the operational model.

Integration is not a goal in itself and could be completed by a vertical approach to the problem according to the local situation. Integration appears both as a rational response from the health care service to the needs of the population and as a research instrument aiming at answering more adequately the sleeping sickness problem.

Authors' abstract

5930 **Dietz, K., 1989.** Density-dependence in parasite transmission dynamics. (Summary.) *Annales de la Société belge de Médecine tropicale*, **69** (Suppl. 1): 17-19. (See also **12**: no. 5938.)

Universität Tübingen, Institut für Medizinische Biometrie, Westbahnhofstrasse 55, D-7400 Tübingen 1, Federal Republic of Germany.

The transmission of vector-borne parasites is complex, yet to a large extent this complexity can be unravelled through the insights gained from simple mathematical models of the transmission system. However, the importance of

some degree of density-dependence in regulating the increase or decrease in parasite numbers tends to be neglected. Density-dependence may operate on the parasite population within any of its intermediate or definitive host or vector species. Five simple models are presented which combine different levels of density-dependence in the human and the vector host. Such quantitative investigations should assist in the rational planning of parasitic disease control programmes.

From author's abstract

5931 **Gettinby, G., 1989.** Understanding infectious diseases: modelling approaches for the trypanosomiasis. *Annales de la Société belge de Médecine tropicale*, **69** (Suppl. 1): 21-30. (See also **12**: no. 5938.)

ILRAD, P.O. Box 30709, Nairobi, Kenya.

In recent years, modellers have been enchanted by the prospect of using mathematical equations to describe the transmission of trypanosomiasis and other infectious diseases. Models have been proposed but their value to controlling disease has not been properly understood. Many of these models have been constrained by the limitations of classical modelling techniques. Advances in computer hardware and software offer the prospect of describing disease systems using computer simulations, databases and knowledgebases. These developments point towards better models for the understanding and prediction of African trypanosomiasis.

Author's abstract

5932 **Ghogomu, A.N., 1989.** Trypanosomiasis: a public health priority in Cameroon. *Annales de la Société belge de Médecine tropicale*, **69** (Suppl. 1): 231-236. (See also **12**: no. 5938.)

Department of Preventive Medicine, Ministry of Public Health, P.O. Box 2041, Messa, Yaoundé, Cameroon.

From a historical standpoint and the present distribution of the disease, human trypanosomiasis, in spite of its comparatively low incidence, still remains a public health priority in Cameroon, requiring both national and international support for control measures. The geographical position of the country within the *Glossina* belt, certain cultural practices, agro-industrial activities, and an improving transport system are current factors favouring the spread of the disease. Similarly the changing pattern of the disease, ignorance and the lack of valid diagnostic and safe chemotherapeutic tools are some obstacles to current control measures.

Author's abstract

5933 **Habtemariam, T., 1989.** Utility of epidemiologic simulation models in the planning of trypanosomiasis control programs. *Annales de la Société belge de Médecine tropicale*, **69** (Suppl. 1): 109-124. (See also **12**: no. 5938.)

International Center for Tropical Animal Health, School of Veterinary Medicine, Tuskegee University, Tuskegee, AL 36088, USA.

To understand the behaviour of complex epidemiologic systems, it is useful to devise computer-based simulation models by approximating the interactions via biomathematical expressions. Without doubt, these models could be over-simplifications of complex interactions but they could be useful in comparison to classical laboratory and/or field experimental approaches which may not be practical or feasible. In the past, obtaining solutions to biomathematical equations with any degree of complexity has been impossible. However, the

availability of powerful computers has now made the quantitative analysis of such systems feasible and indeed practical. With this in mind, research to develop computer simulation models involving *Trypanosoma* has been under way at Tuskegee University. Based on static and dynamic computer models, several alternatives for the control of trypanosomiasis have been examined. The integration of multivariate systems analysis and optimisation models in epidemiologic problem solving and decision making pertaining to the trypanosome-tsetse complex has been valuable. Its potential application to other epidemiologic problems is an asset that should be exploited in future analytical studies.

Author's abstract

5934 **Jenni, L., 1989.** Brief thoughts on dynamics of host-parasite relationships. (Summary.) *Annales de la Société belge de Médecine tropicale*, **69** (Suppl. 1): 205-206. (See also **12**: no. 5938.)

Swiss Tropical Institute, Socinstrasse 57, CH-4002 Basel, Switzerland.

Whenever we consider a host-parasite relationship, we are dealing with a complex and dynamic system in a changing environment. Instead of the normally applied analytical approach, what is needed is a systemic approach which concentrates on interactions of elements and results of interactions, modifies several variables at the same time, and includes space and time. In this approach, the evaluation of facts is based on the comparison of the function of a model with reality. It is useful for non-linear and strong interactions, and leads to inter- and multidisciplinary studies and objective-orientated actions. It reaches good knowledge about objectives and goals but not about details. The systemic approach could lead to new mathematical models which may be sensitive enough to develop a better understanding of host-parasite relationships in open systems and strategies for control.

From author's abstract

5935 **Jordan, A.M., 1989.** Importance of land use on the incidence of *Trypanosoma brucei gambiense* sleeping sickness. (Summary.) *Annales de la Société belge de Médecine tropicale*, **69** (Suppl. 1): 254. (See also **12**: no. 5938.)

TRL, University of Bristol, School of Veterinary Science, Langford House, Langford, Bristol BS18 7DU, UK.

Much destruction of flora and fauna in Africa has occurred in recent years as a result of the rapidly increasing human population. These changes are affecting the vectors of *T. b. gambiense* in different ways. Towards their drier limits, shifting cultivation is tending to become more permanent and the riparian vegetation is becoming more degraded and less suitable as a habitat for species of the *Glossina palpalis* group. At their wetter limits, these species often adapt to man-made habitats, such as cocoa and coffee plantations, as the natural vegetation is removed. Under such circumstances the flies change from feeding mainly on wild mammals and reptiles to feeding mainly on man and his domestic animals. Although the increasing numbers of man are having less profound effects on the vectors of *T. b. gambiense* than on the *morsitans* and *fusca* groups of *Glossina*, nevertheless, changes in land use are affecting the incidence of the disease. As numbers of humans continue to increase, these effects are likely to become more pronounced.

Author's abstract

5936 **Kuzoe, F.A.S., 1989.** Current knowledge on epidemiology and control of sleeping sickness. *Annales de la Société belge de Médecine tropicale*, **69** (Suppl. 1): 217-220. (See also **12**: no. 5938.)

Parasitic Diseases Programme, WHO, 20 avenue Appia, 1211 Geneva 27, Switzerland.

African trypanosomiasis (sleeping sickness) occurs in some 200 known foci within tsetse-inhabited areas in sub-saharan Africa, where an estimated 50 million people are at risk of acquiring infection. Only 25,000 new patients are reported each year but this is an underestimate. Where medical surveillance was inadequate or lacking, serious epidemics could occur and this potential risk makes the disease one of the serious health problems in sub-saharan Africa. The chronic *gambiense* form of sleeping sickness occurs in West and Central Africa, while the acute *rhodesiense* form occurs in East and Southern Africa. Recent advances in science have improved knowledge on the epidemiology of the disease and provided tools for improved diagnosis, treatment and vector control. The current strategy for control of the disease is based upon continuous suppression through diagnosis and treatment and limited vector control with community participation. Nevertheless, a long-term solution to the problem of African trypanosomiasis lies in effective land-use management and rural development.

Author's abstract

5937 **Milligan, P.J.M., 1989.** Epizootiology of trypanosomiasis: some aspects of mathematical modelling. *Annales de la Société belge de Médecine tropicale*, **69** (Suppl. 1): 89-98. (See also **12**: no. 5938.)

Department of Biological Sciences, University of Salford, Salford, M5 4WT, UK. The formulation of trypanosomiasis transmission models is complicated by heterogeneity, due to (a) different feeding preferences of vectors for a range of host species, (b) the presence of different genetic lines in the parasite population, and (c) variations in susceptibility and infectivity among hosts and vectors.

Simple models are used to explore qualitative effects of changes in host range of vectors on the basic reproductive rate (section 1), of migration of vectors on disease persistence (section 2), and of gradual acquisition of immunity to different trypanosome serodemes (section 3). Many implicit and explicit assumptions need to be validated by fitting models to field data as part of multidisciplinary projects.

Author's abstract

5938 **Muynck, A. de and Rogers, D.J. (eds), 1989.** *Workshop on modelling sleeping sickness epidemiology and control*, Antwerp, 25-29 January 1988. Organised by the UNDP/World Bank/WHO Special Programme for Research and Training in Tropical Diseases (TDR) in collaboration with the Prince Leopold Institute of Tropical Medicine, Antwerp, Belgium. *Annales de la Société belge de Médecine tropicale*, **69** (Suppl. 1): 256 pp.

Department of Epidemiology, Prince Leopold Institute of Tropical Medicine, Nationalestraat 155, B-2000 Antwerp, Belgium; Department of Zoology, Oxford University, South Parks Road, Oxford OX1 3PS, UK.

The organisers of this workshop sought to bring together field workers and modellers from five African countries, Europe and America with experiences ranging from the organisation of sleeping sickness control services in affected countries, through the various disciplines of parasitology, immunology, entomology and veterinary studies to epidemiology. The workshop was held in

the knowledge that so far models have made little impact on control programmes in the field. Control workers were invited to give their experiences, voice their needs and see what modelling is, and how models can inspire rational control strategies that could direct their future activities. The modellers in turn could appreciate the difficulties of the field situation, the better able to tailor their models to relevant problems. The workshop was arranged in plenary sessions, attended by all, and parallel working groups on modelling, vectors, the human host, parasites and the animal reservoir. Many of the discussions of the various working groups were directed towards control of the trypanosomiasis. The organisers purposefully avoided having a separate working group on this topic, preferring that the control experiences of the various participants should be shared equally among the other groups, in order that their discussions should be fully informed of the many practical difficulties that arise during field research and control projects. The papers presented at the workshop are grouped in three sections: Modelling (see nos. 5925, 5926, 5928, 5930, 5931, 5933, 5937, 5940, 5943, 5970), Epidemiology: parasites, hosts and vectors (see nos. 5934, 5953, 5960, 5964, 5966, 5967, 5968, 5971, 5972, 5978, 5979, 5991) and Control (see nos. 5929, 5932, 5935, 5936, 5944), with an introductory presentation (see no. 5942). Brief reports of the discussion groups are also included, with recommendations for future research.

5939 **Overseas Development Administration and University of Bristol, 1989.** *Tsetse Research Laboratory Annual Report 1988*. Bristol; ODA and University of Bristol. 53 pp. TRL, Langford House, Langford, Bristol BS18 7DU, UK. In 1988 aspects of the nutrition and reproduction of tsetse continued to be studied, particularly in relation to trap-orientated behaviour of tsetse in the field and the evaluation of biases inherent in trapping techniques. Studies on tsetse behaviour sought to improve the efficiency of traps and increase the frequency and/or duration of contact of tsetse with insecticide-impregnated targets using host odour attractants. A major re-analysis of published data sought to clarify age-dependent sampling bias. Recent research on the vectorial capacity of tsetse has been concerned with determining at which stage in the trypanosome life cycle within the fly the signal for maturation operates. Research on trypanosomes included isoenzyme characterisation of *Trypanozoon* isolates, production of monoclonal antibodies, field diagnostic studies, and the discovery of a probable new *Nannomonas* species in The Gambia.

5940 **Paling, R.W., 1989.** The network approach: a means for the collection of integrated data following standardized protocols. *Annales de la Société belge de Médecine tropicale*, **69** (Suppl. 1): 137-143. (See also **12**: no. 5938.) Faculty of Veterinary Medicine, University of Utrecht, P.O. Box 80.163, 3508 TD Utrecht, Netherlands.

The understanding of the epidemiology of a vector-borne disease, involving various vector and host species in a defined area, requires a multidisciplinary approach. It is essential that specialists obtain data relevant to common objectives. In the case of African trypanosomiasis this means that the observations being made on the health status of the human and domestic and wild animal populations are made over the same period of time as those on the tsetse population to which these hosts are exposed. Standardisation of methodology is a pre-condition for reliable comparison of observations. The creation of a network of research situations is one possibility for the fulfilment of this pre-condition; while at the same time it is a suitable means for the collection of integrated data. The African Trypanotolerant Livestock Network, created in 1982, is one example of such a network. Examples of conclusions which could be drawn after analysis of data collected over a two-year period through this Network are presented.

Author's abstract

5941 **Putt, S.N.H., Jordan, A.M., Koeman, J.H., Mulder, P. and Shaw, A.P.M., 1989.** *Evaluation of the EEC-funded Tsetse and Trypanosomiasis Control Projects in Malawi, Mozambique, Zambia and Zimbabwe.* Pan Livestock Services Limited and the Centre for Development and Cooperation Services of the Free University of Amsterdam. 313 pp.

This report is concerned with the findings of the first of two evaluation missions for the EEC-funded Regional Tsetse and Trypanosomiasis Control Programme. Besides evaluating the RTTCP, the report aims to clarify and resolve some of the complex issues which have aroused so much controversy. The report recommends that the Preparatory Phase of the RTTCP should be extended by three years with a review after the second year to decide on whether the operational phase of the programme should be implemented. The prime objective during this time should be the development of a comprehensive strategic plan to consider not only the technical issues involved in tsetse control but also the land use and economic implications, and the budgetary, staffing and training requirements. A Regional Planning Unit should be established within the Office of the Regional Co-Ordinator to achieve this objective, and administration should be streamlined. Research on target and aerial spraying technology, development of simpler protocols for the evaluation of candidate insecticides and of ELISA techniques for use in epidemiological studies should be continued. Since aerial spraying is likely to receive less emphasis in future, the environmental monitoring input should be reduced. Increased emphasis should be placed on the training of tsetse and trypanosomiasis control staff in the countries concerned. Separate recommendations for the four national programmes are given.

5942 **Raadt, P. de, 1989.** Epidemiological models for African trypanosomiasis: a waste of time? *Annales de la Société belge de Médecine tropicale*, **69** (Suppl. 1): 7-10. (See also **12**: no. 5938.)

Trypanosomiasis and Leishmaniasis Unit, Parasitic Diseases Programme, WHO, 20 avenue Appia, CH-1211 Geneva 27, Switzerland.

Since trypanosomiasis risk is as yet impossible to quantify by practical epidemiological parameters, control programmes are generally founded on *ad hoc* decisions taken on the basis of probably not very accurate sleeping sickness prevalence rates. An epidemiological model would probably serve to identify the correct parameters for answering some of the questions pertinent to control, but the feasibility of developing a model which can be used in practice depends partly on whether our present state of knowledge would be adequate to feed such a model. Many epidemiologists, mathematicians and biologists are naturally intrigued by the intellectual challenge to bring some logical order into the multitude of variables with their complicated interrelationships which are typical for the transmission cycles of parasitic diseases and difficult to place in an overall perspective. However, there may well be too many imponderables in trypanosomiasis epidemiology to expect results directly applicable to control in the near future.

5943 **Rogers, D.J., 1989.** The development of analytical models for human trypanosomiasis. *Annales de la Société belge de Médecine tropicale*, **69** (Suppl. 1): 73-88. (See also **12**: no. 5938.)

Department of Zoology, Oxford University, South Parks Road, Oxford OX1 3PS, UK.

The idea of biological equilibria is presented and applied first to the case of single species populations. Difference and differential descriptions of population changes over time are compared and contrasted. The complications that arise when describing interactions between species are then illustrated for the case of vector-borne diseases, such as malaria. A simple model for malaria is explored and later extended to the case of the African trypanosomiasis where more than one vertebrate host species is involved. Appropriate parameter values obtained from the literature are listed, and equilibrium predictions given. These predictions broadly coincide with field experience. A brief description of the analysis of age-prevalence data is given and applied to several sets of data from sites in West, Central and East Africa. In each case the force of infection is extremely low, indicating that a non-human reservoir must be involved in the maintenance of the disease throughout Africa. A final plea is made to integrate the modelling exercise into on-going programmes for disease control or eradication.

Author's abstract

5944 **Shaw, A.P.M., 1989.** Comparative analysis of the costs and benefits of alternative disease control strategies: vector control versus human case finding and treatment. *Annales de la Société belge de Médecine tropicale*, **69** (Suppl. 1): 237-253. (See also **12**: no. 5938.)

Veterinary Epidemiology and Economics Research Unit, University of Reading, Department of Agriculture, P.O. Box 236, Early Gate, Reading RG6 2AT, UK. Recent estimates of the costs of detection and treatment and vector control for human trypanosomiasis are summarised and the main factors influencing their levels discussed. In order to proceed to an economic analysis a benefit unit is defined as equivalent to one year's infection avoided due to the control strategy for one person. Different monetary values could be assigned to this unit and it

has the advantage of being easily integrated into epidemiological models, being represented by the difference in prevalence with and without control. A simple spreadsheet-based economic model was developed to analyse the relative economic performance of the two control strategies in terms of the cost per benefit unit obtained. The results demonstrate the sensitivity of the economic performance of each strategy to the epidemiological assumptions made, especially with respect to the existence of an animal reservoir and the evolution of the disease situation in the absence of control. They point to the need to integrate economic and epidemiological models in order to evaluate control options.

Author's abstract

5945 **Stevenson, S.R., 1988.** *Land use implications of the EEC-funded Regional Tsetse and Trypanosomiasis Control Programme of Malawi, Mozambique, Zambia and Zimbabwe.* Zimbabwe; International Union for Conservation of Nature and Natural Resources. 163 pp.

IUCN, Regional Office for Southern Africa, P.O. Box 745, Harare, Zimbabwe.

This study was commissioned by the RTTCP and presents advice to the Governments of Malawi, Mozambique, Zambia and Zimbabwe on means to ensure that the RTTCP leads to sustainable development in the region. Chapters 1 to 9 are mainly descriptive, covering the natural characteristics of the common fly-belt, the effect of man's various activities, policy, planning and institutions, and general attitudes to land use and tsetse control. The situation in the four countries is described in detail. The main analysis, conclusions and recommendations of the study are presented in Chapters 10 and 11. Technological capability to eradicate tsetse has surpassed the land use planning (LUP) capability and present resources for agricultural extension in Zambia, Mozambique and Malawi, and the recent rate of control has severely strained LUP and extension resources in Zimbabwe. It is recommended that large-scale, rapid and irreversible changes to the environment should not be induced prior to intensive scrutiny and enlightened decision-making by a broad spectrum of government agencies, assisted by other bodies such as conservation committees, university departments and international agencies with expertise in environmental science. LUP should be undertaken in advance of tsetse control and should incorporate meaningful involvement at local level, as is practised in Zimbabwe. LUP capability urgently needs to be developed in Mozambique and strengthened in Malawi and Zambia. Conservation education should be improved at local level. Wherever possible, tsetse control, and indeed LUP, should be an element of integrated rural

development rather than an isolated effort, as at present. Immediate establishment and funding of a small institution, parallel to the RTTCP but independent of it, staffed by environmental experts, and primarily concerned with minimising environmental damage and promoting sustainable development in the programme area, is recommended.

5946 **Wellde, B.T., 1989.** *Trypanosomiasis in the Lambwe Valley, Kenya.* *Annals of Tropical Medicine and Parasitology*, **83** (Suppl. 1): 220 pp. Walter Reed Project, Veterinary Research Laboratory, Ministry of Agriculture and Livestock Development, Kabete, Kenya. Present address: Department of Immunology, Walter Reed Army Institute of Research, Washington, DC 20307, USA.

Dr B.T. Wellde and his co-workers of the Walter Reed Project have carried out a comprehensive in-depth study of trypanosomiasis in the Lambwe Valley, the results of which are presented in this special issue of the *Annals of Tropical Medicine and Parasitology*. Their studies cover the history of sleeping sickness in the area, demographic characteristics of the human population, epidemiology, diagnosis, clinical symptoms and treatment of the disease (see nos. 5947-5949, 5969, 5974-5977, 5981, 5982, 5984). Aspects of animal trypanosomiasis include a parasite survey of wild animals, and studies on *Trypanosoma vivax* and *T. congolense* in cattle (see nos. 5987, 5988, 5992-5994, 5998). Other papers cover experimental infection and treatment (see nos. 5995, 5999, 6011) and a review of tsetse control measures (see no. 5959).

5947 **Wellde, B.T., Chumo, D.A., Waema, D., Reardon, M.J. and Smith, D.H., 1989.** A history of sleeping sickness in Kenya. *Annals of Tropical Medicine and Parasitology*, **83** (Suppl. 1): 1-11. (See also **12**: no. 5946.) Walter Reed Project, Veterinary Research Laboratory, Ministry of Agriculture and Livestock Development, Kabete, Kenya; *ibid.*; *ibid.*; *ibid.*; Department of Tropical Medicine and Infectious Diseases, Liverpool School of Tropical Medicine, Pembroke Place, Liverpool L3 5QA, UK.

Gambian sleeping sickness entered what is now Kenya from Uganda in about 1901 and quickly spread along the Kenyan shores and islands of Lake Victoria, reaching Tanzania in 1902. By 1910 the disease had spread 25 miles inland along the Kuja and Migori rivers and their tributaries. Sleeping sickness waxed and waned in these areas despite attempts to control tsetse fly populations by various methods. It was not until 1950, when the use of insecticides (DDT) applied by backpack sprayer proved successful against *Glossina fuscipes* at Kibigori, that eradication of *G. fuscipes* and Gambian sleeping sickness seemed possible. Subsequently the Kuja-Migori endemic area was cleared of flies and disease, as well as the South and Central Nyanza lake shores and islands. By 1965 Gambian sleeping sickness had virtually disappeared from Kenya. A more virulent form of

the disease, Rhodesian sleeping sickness, may have also spread to Kenya from Uganda, although its appearance in diverse areas of the Gambian disease suggest that local ecological factors may have played a role in enhanced virulence of trypanosome stocks. The Rhodesian form of sleeping sickness appeared in the Lambwe Valley, South Nyanza, in about 1959, and despite attempts to eradicate this disease it still persists as the only remaining endemic area in Kenya at this time. The usual transmission of Rhodesian sleeping sickness by *G. pallidipes* in Kenya was altered when an outbreak occurred at Alego, in Central Nyanza, in 1964. It was discovered that *G. fuscipes* was the vector and that domestic cattle were an important reservoir of infection. *G. fuscipes* was also the vector of Rhodesian sleeping sickness in an outbreak in Samia in 1976 and another along the lakeshore in South Nyanza in 1981. Sleeping sickness has been restricted primarily to the Western and Nyanza Provinces of Kenya.

Authors' abstract

5948 **Wellde, B.T., Reardon, M.J., Chumo, D.A., Waema, D., Smith, D.H., Koech, D., Siongok, T.A. and Wanyama, L., 1989.** Demographic characteristics of the Lambwe Valley population. *Annals of Tropical Medicine and Parasitology*, **83** (Suppl. 1): 29-42. (See also **12**: no. 5946.)

Walter Reed Project, Veterinary Research Laboratory, Ministry of Agriculture and Livestock Development, Kabete, Kenya; *ibid.*; *ibid.*; *ibid.*; Department of Tropical Medicine and Infectious Disease, Liverpool School of Tropical Medicine, Pembroke Place, Liverpool L3 5QA, UK; Division of Disease Control, Ministry of Health, Nairobi, Kenya; *ibid.*; *ibid.*

Thirty-four per cent of the population (11,079) in the Lambwe Valley study site was under the age of 10, and 59% were under the age of 20. The population was equally divided among males and females (M/F 0.99). The crude birth rate averaged 45‰; the death rate was 8‰ and the natural increase averaged 37‰. Infant and child mortality was 66‰ and 108‰ respectively. The average household size was 8.4 individuals. The population migrated to the Lambwe Valley primarily from other areas in Kenya, although 13% were from Tanzania. The predominant tribal group was Luo (59%) followed by the Abasuba (38%). A few Luyha and Kisii were also encountered. Population increased by 3.5-fold in the 1960s, followed by a 2.5-fold increase in the 1970s. Due to an aerial spray tsetse control programme, sleeping sickness had a low prevalence in October 1981 (one of 5749). Of 339 individuals confirmed as sleeping sickness patients by hospital records from within the study site 320 (94.3%) were traced to their homes. Mortality in domestic cattle (40%), goats (47%), sheep (55%) and dogs (50%) had occurred over the previous two years during an outbreak of trypanosomiasis.

Authors' abstract

5949 **Wellde, B.T., Waema, D., Chumo, D.A., Reardon, M.J., Adhiambo, A., Olando, J. and Mabus, D., 1989.** The Lambwe Valley and its people. *Annals of Tropical Medicine and Parasitology*, **83** (Suppl. 1): 13-20. (See also **12**: no. 5946.)

Walter Reed Project, Veterinary Research Laboratory, Ministry of Agriculture and Livestock Development, Kabete, Kenya; *ibid.*; *ibid.*; *ibid.*; *ibid.*; Division of Disease Control, Ministry of Health, Homa Bay, Kenya; Division of Community Development, South Nyanza District, Homa Bay, Kenya.

By 1936 the Lambwe Valley, which had been heavily populated in the early years of this century, was nearly devoid of people. Population since that time has increased markedly as a result of a settlement scheme and efforts made to control and eradicate *Glossina pallidipes* and trypanosomiasis. The formation of a game reserve (now a National Park) prevented the completion of a tsetse eradication programme and has provided an unmolested habitat for both *G. pallidipes* and large numbers of game animals which act as a reservoir for trypanosomiasis. Rhodesian sleeping sickness, as well as animal trypanosomiasis, have been severe problems for the local farmers who live around the boundaries of the National Park.

Authors' abstract

5950 **World Health Organization, 1988.** Urban vector and pest control. (Eleventh report of the WHO Expert Committee on Vector Biology and Control, Geneva, 15-21 September 1987.) *WHO Technical Report Series*, no. 767: 77 pp. (ISBN 92 4 120767 1. Price Sw. fr. 9.)

WHO, 1211 Geneva 27, Switzerland.

The ecological requirements of tsetse flies are such that they are hardly suited to an urban environment, but some cities may contain small wooded areas (parks) and these may harbour quite considerable tsetse populations. This is the case with *Glossina palpalis gambiensis* in Ouagadougou, Burkina Faso, and *G. fuscipes* in Brazzaville, Congo. As cities are extended, the outlying districts may impinge on tsetse breeding sites. This accounts for the cases of sleeping sickness transmitted by *G. palpalis* that have occurred in the cities of Douala (Cameroon), Bamako (Mali) and Matadi (Zaire).

2. TSETSE BIOLOGY

(a) REARING OF TSETSE FLIES

5951 **Langley, P.A., 1989.** Laboratory colonization of the tsetse fly *Glossina pallidipes* Austen (Diptera: Glossinidae) using an *in vitro* feeding method. *Bulletin of Entomological Research*, **79** (3): 429-435.

TRL, Langford, Bristol BS18 7DU, UK.

Attempts to establish a laboratory colony of *G. pallidipes* from adults and puparia of Uganda origin (Lugala) sent from a colony previously established in Amsterdam have been highly successful. Flies are maintained at 25 ± 0.5°C, L:D 12:12 and 85% r.h. They are fed 6 days per week on whole defibrinated pig

blood collected aseptically and irradiated at 1.5 kGy before storage at 4°C. The blood is presented through silicon rubber membranes at 37°C. Virgin female flies are mated when 7-9 days old with equal numbers of 9-12 day old males in a 30 × 30 × 10 cm cage covered with white Terylene netting and illuminated with a fluorescent light from above, providing 300-500 lux on the cage bottom. The sexes are left together for 40-48 h, during which time they are offered food twice. After separation of the sexes, 30 females are housed in a cage 15 cm in diameter and two males are added. Flies are kept for 90 days before discarding. The establishment of this colony is discussed in terms of the mating behaviour of the species, highlighting differences in the laboratory between *G. pallidipes* from Uganda and Zimbabwe and between *G. pallidipes* and *G. morsitans morsitans*.
Author's abstract

(b) TAXONOMY, ANATOMY, PHYSIOLOGY, BIOCHEMISTRY

(c) DISTRIBUTION, ECOLOGY, BEHAVIOUR, POPULATION STUDIES

5952 **Doku, C. and Brady, J., 1989.** Landing site preferences of *Glossina morsitans morsitans* Westwood (Diptera: Glossinidae) in the laboratory: avoidance of horizontal features? *Bulletin of Entomological Research*, **79** (3): 521-528.

Imperial College, Silwood Park, Ascot, Berks, SL5 7PY, UK. (Correspondence to Brady.)

Landing site preferences of fed males of *G. morsitans* were measured in c. 50 cm wide arenas in the laboratory. Flies were presented with simple black shapes of 225 cm² against a white background. Preference ran in the order: circle < square < triangle (whether the targets were black shapes on white or white on black). However, the triangle on its side (i.e. rotated through 90°) or the square presented as a diamond (i.e. rotated 45°) were both greatly preferred over their original orientations, whereas inverting the triangle (by rotating it 180°) greatly decreased its attractiveness. These preferences were retained whether the targets were compared paired or singly. The most obvious correlation was between preference and the relative lack of horizontal features in the target. When the diamond (the most attractive target) was split horizontally by a narrow white band, its attractiveness was reduced by c. 60%, whereas when split by a vertical band the attractiveness was reduced by only c. 25%. Similarly, when an additional narrow black bar was placed horizontally beneath the diamond its attractiveness was cut by 47%, whereas when the bar was placed vertically beside it the attractiveness was cut by only 28%. These reductions occurred even though the targets should have been more attractive because of their greater edge length and area. When the effect was tested with a series of rectangles, the greater the ratio of the vertical:horizontal edge length, the greater the attractiveness. The relative inhibition of landing by the presence of horizontal features in a laboratory-tested landing site thus seems clear, notwithstanding that horizontal resting sites in nature are the most preferred.

Authors' abstract

5953 **Dransfield, R.D. and Brightwell, R., 1989.** Problems of field testing theoretical models: a case study. *Annales de la Société belge de Médecine tropicale*, **69** (Suppl. 1): 147-154. (See also **12**: no. 5938.)

ICIPE, P.O. Box 30772, Nairobi, Kenya.

The problems of field-testing models are discussed with reference to a population simulation model for the tsetse fly *Glossina pallidipes* developed by us at Nguruman in south-western Kenya. Model predictions showed a reasonable fit to the changes in the biconical trap index of population size, but tended to overestimate some changes and underestimate others. A more rigorous test for the model is to manipulate one of the population parameters and compare model predictions with the observed population trends. This revealed that some changes in population size resulted more from fly movement than from changes in mortality rates. Fly movement must therefore be incorporated in the model for it to be a useful tool in the development of appropriate control strategies.

Authors' abstract

5954 **Madubunyi, L.C., 1989.** Estimation of the interval between feeding and capture in peridomestic *Glossina tachinoides*. *Medical and Veterinary Entomology*, **3** (4): 327-332.

Department of Veterinary Parasitology and Entomology, Faculty of Veterinary Medicine, University of Nigeria, Nsukka, Nigeria.

From successive 24-hourly dissections of non-teneral field-caught *G. tachinoides* which had fed on a guinea-pig in the laboratory, the progressive movement of a bloodmeal through the midgut was monitored and five stages (categories) in the trophic cycle were identified. The frequency distribution in these trophic categories of *G. tachinoides* subsequently caught in the peridomestic agro-ecosystem at Orië-Orba near Nsukka by means of the biconical trap revealed that males and females had fed 2.8 ± 0.4 and 2.4 ± 0.4 days respectively preceding their capture.

Author's abstract

5955 **Warnes, M.L., 1989.** Response of tsetse flies (*Glossina* spp.) to compounds on the skin surface of an ox: a laboratory study. *Medical and Veterinary Entomology*, **3** (4): 399-406.

TRL, ODA/University of Bristol, Langford House, Langford, Bristol BS18 7DU, UK.

The behaviour of male *G. morsitans morsitans* and *G. pallidipes* alighting on targets with or without ox sebum was compared. The presence of ox sebum did not increase significantly the number of flies alighting on the target in either species. However, after contact with the sebum-coated target, both species showed an increase in flight activity, and *G. m. morsitans* showed a greater tendency to return to the target. This behaviour resulted in a number of short flights which may reflect the search for a feeding site on a host. The duration of each visit to the target was significantly reduced when sebum was present for *G. m. morsitans* but not for *G. pallidipes*. This is explained by documented differences in the resting behaviour of the two species which shows that *G. m. morsitans* normally rests for longer periods on the surface of an untreated black target than does *G. pallidipes*. Other experiments showed that the presence of sebum elicited a probing response in *G. m. morsitans* and *G. pallidipes*. The results are discussed with reference to the possible use of host sebum to improve trap catches in the field.

Author's abstract

3. TSETSE CONTROL (INCLUDING ENVIRONMENTAL SIDE EFFECTS)

[See also 12: nos. 5938, 5945, 5965.]

5956 **Carpels, G.I. and Greathead, D.J., 1989.** A record of *Exhyalanthrax abruptus* (Loew) (Diptera, Bombyliidae), a tsetse parasitoid from the Luangwa Valley, Eastern Province, Zambia. *Annales de la Société belge de Médecine tropicale*, **69** (2): 157-159.

Prince Leopold Institute of Tropical Medicine, Nationalestraat 155, B-2000 Antwerp, Belgium; CAB International Institute of Biological Control, Imperial College, Silwood Park, Ascot, Berks, SL5 7PY, UK.

In September and October 1986, a total of 176 *Glossina morsitans morsitans* pupae were collected from holes at the bases of trees near Kakumbi Tsetse Research Station, situated just outside the South Luangwa National Park, Eastern Province, Zambia. After incubation, 16% of pupae were found to be parasitised with *E. abruptus* (24% if dead pupae excluded). *E. abruptus* emerged in two distinct groups, spaced by a dormancy of 91 days.

5957 **Ethiopia Ministry of Agriculture, National Tsetse and Trypanosomiasis Investigation Centre, 1988.** *Annual Report 1987-1988.*

38 pp.

National Tsetse and Trypanosomiasis Investigation Centre, P.O. Box 15, Illubabor-Bedelle, Ethiopia. In recent years tsetse (*Glossina morsitans submorsitans*) have advanced up the Didessa Valley and are spreading to higher altitudes, resulting in an ever-increasing trypanosomiasis problem in the area at a time when population growth is causing an increasing demand for new land. A pilot tsetse control trial was carried out in ten villages in the Upper Didessa Valley in an area where most of the livestock population had died following recent tsetse infestation. Targets and monoconical traps baited with octenol, acetone and cattle urine and sprayed with deltamethrin were positioned along drainage line vegetation at a density of 3-10 targets/km² from the end of November to January (dry season) and were looked after by the farming communities. Within 4 months, the tsetse population had declined to undetectable levels throughout most of the area. Targets are being maintained in remaining pockets of infestation and as a barrier to reinvasion. Research on odour baits and visually attractive devices indicated that a blue/black/blue target design baited with cattle urine, octenol and acetone is the most effective bait system for the control of savanna *G. m.*

submorsitans whilst the cattle urine/octenol combination can be applied for the control of riverine *G. tachinoides*. 5958 **Kaaya, G.P. and Darji, N., 1989.** Mortality of adult tsetse, *Glossina morsitans morsitans*, caused by entomopathogenic bacteria. *Journal of Invertebrate Pathology*, **54** (1): 32-38.

ICIPE, P.O. Box 30772, Nairobi, Kenya.

Mortality in adult tsetse, *G. m. morsitans*, caused by *Pseudomonas aeruginosa*, *Serratia marcescens*, *Bacillus sphaericus*, *Bacillus cereus*, *Bacillus thuringiensis* H-14, *B. thuringiensis* 1, *B. thuringiensis* 5, *B. thuringiensis* var. *israelensis* and *Providentia rettgeri*, was determined. When bacteria were smeared on rabbit skin and tsetse allowed to feed only once on the contaminated area, mortality 8 days post-ingestion was significantly higher ($P < 0.01$) in tsetse fed on *P. aeruginosa*, *S. marcescens*, *B. thuringiensis* 1 and *P. rettgeri* and increased when tsetse were allowed to feed for the second time on the contaminated skin. With this smear technique, however, mortalities were generally not remarkable. In artificial membrane feeding experiments using low concentrations of bacteria (10^6 /ml of blood), the *B. thuringiensis* strains caused low mortality, except *B. thuringiensis* H-14, which caused 59% mortality. However, at this concentration, *P. aeruginosa*, *S. marcescens*, *B. cereus* and *P. rettgeri* caused highly significant ($P < 0.01$) mortality (64-96%). When higher concentrations of bacteria (10^7 /ml) were used, all the bacteria tested, except *B. sphaericus*, caused high mortality ranging from 70 to 98%. Thus, mortality depended on the species of bacteria, the dose ingested, and time post-ingestion.

Authors' abstract

5959 **Wellde, B.T., Waema, D., Chumo, D.A., Reardon, M.J., Oloo, F., Njogu, A.R., Opiyo, E.A. and Mugutu, S., 1989.** Review of tsetse control measures taken in the Lambwe Valley in 1980-1984. *Annals of Tropical Medicine and Parasitology*, **83** (Suppl. 1): 119-125. (See also **12**: no. 5946.) Walter Reed Project, Veterinary Research Laboratory, Ministry of Agriculture and Livestock Development, Kabete, Kenya; *ibid.*; *ibid.*; *ibid.*; Tsetse Control Section, Veterinary Research Laboratory, Ministry of Agriculture and Livestock Development, Kabete, Kenya; KETRI, Ministry of Health, Muguga, Kenya; *ibid.*; *ibid.*

During an outbreak of Rhodesian sleeping sickness in the Lambwe Valley in 1980 initial tsetse control measures consisted of applications of dieldrin to the periphery of the Ruma National Park. This activity had a marked effect on the prevalence of sleeping sickness. Concern about the use of dieldrin caused the cessation of this programme and justified an aerial spray programme using endosulfan. Although the Lambwe Valley did not appear to be a good candidate for aerial spray, the endosulfan had a marked effect on tsetse fly levels and on the prevalence of sleeping sickness. Sleeping sickness cases were detected in decreasing numbers for eight months following the endosulfan programme, but the subsequent five months yielded no cases of sleeping sickness in the area. Some flies persisted, however, and they had regained high levels in about a year. As the prevalence of sleeping sickness increased another aerial spray programme was initiated in 1983, using pyrethrum as insecticide. The pyrethrum aerial spray programme did not make significant reductions in the *Glossina pallidipes* population or in the prevalence of sleeping sickness. A subsequent ground

control programme using insecticides (dieldrin and cypermethrin) and bush clearing, conducted primarily within the National Park, has subsequently limited the prevalence of sleeping sickness to low levels.
Authors' abstract

4. EPIDEMIOLOGY: VECTOR-HOST AND VECTOR-PARASITE INTERACTIONS

[See also **12**: nos. 5925, 5926, 5928, 5930, 5931, 5933, 5934, 5937, 5938, 5940, 5941, 5942, 5943, 5947.]
5960 **Akulep, C., 1989.** Factors enhancing man-fly contact in African human trypanosomiasis. (Summary.) *Annales de la Société belge de Médecine tropicale*, **69** (Suppl. 1): 209. (See also **12** no. 5938.)

National Sleeping Sickness Control Programme, P.O. Box 1241, Jinja, Uganda. Sleeping sickness endemic and epidemic states in Africa have persisted due to continuing contact of man with tsetse flies infected by trypanosomes, either *Trypanosoma brucei gambiense* in West Africa or *T. b. rhodesiense* in East Africa. The abundance of the necessary macro- and micro-environments for the breeding and spread of the tsetse fly, its longevity, and its adaptability to ecological changes both biotic and abiotic, ensures the persistence of the fly. In many cases, man's natural activities and innovations bring him in close contact with the fly, especially where control measures are inadequate, inappropriate or lacking.

Author's abstract

5961 **Dukes, P., Rickman, L.R. and Maudlin, I., 1988.** The acquisition of human serum resistance during cyclical passage of a *Trypanosoma brucei brucei* clone through tsetse maintained on human serum: a retraction. (Letter.) *Transactions of the Royal Society of Tropical Medicine and Hygiene*, **82** (6): 944. TRL, Department of Veterinary Medicine, University of Bristol, Langford House, Langford, Bristol BS18 7DU, UK.

The authors reported the acquisition of resistance to human serum by a clone of *T. b. brucei*, apparently as a result of maintaining infected *Glossina morsitans morsitans* on human serum (see *TTIQ*, **7** (4): no. 3575). This suggestion is now withdrawn. Further investigation has shown that a low level of contamination with *T. b. rhodesiense* (< 0.001%) was the cause of this unexpected result.

5962 **Food and Agriculture Organization of the United Nations, 1986.**

Training manual for tsetse control personnel. (Edited by J.N. Pollock.) Volume III: Control methods and side effects. Revised edition. Rome; FAO. 139 pp. FAO, Via delle Terme di Caracalla, 00100-Rome, Italy. This updated version describes the use of targets for tsetse control, and gives details of survey methods. (See *TTIQ*, **5** (4): no. 2522 for details of first edition.)

5963 **Joshua, R.A., 1989.** Occurrence of human serum-resistant *Trypanosoma congolense* in goats and sheep in Nigeria. *Veterinary Parasitology*, **31** (2): 107-113.

Department of Veterinary Medicine, University of Ibadan, Ibadan, Nigeria.
 An assessment of the role of dogs, goats and sheep as reservoir hosts of African trypanosomes infective for humans (sleeping sickness) was carried out in Nigeria during a 2-year study period. Twelve stocks of *T. (Trypanozoon) brucei*, 10 stocks of *T. congolense* and 11 stocks of *T. vivax* were isolated from a total of 699 animals, comprising 286 sheep, 221 goats and 192 dogs. The potential infectivity of the isolates for man was tested *in vitro* using the blood incubation infectivity test. None of the *T. brucei* group was resistant to the trypanocidal action of human serum; three of the *T. congolense* group were resistant to human serum. A parallel study of the trypanocidal action of test serum on authenticated *T. b. brucei* and *T. b. gambiense* showed that the human serum behaved as expected. The possibility is discussed that *T. congolense* might produce infections in man and should, therefore, be handled carefully both in the laboratory and by veterinarians in the field.

Author's abstract

5964 **Kageruka, P., 1989.** Réservoir animal de *Trypanosoma (Trypanozoon) brucei gambiense* en Afrique Centrale. [Animal reservoir of *T. b. gambiense* in Central Africa.] *Annales de la Société belge de Médecine tropicale*, **69** (Suppl. 1): 155-163. (See also **12**: no. 5938.)

Département Vétérinaire, Service de Santé Animale, Institut de Médecine Tropicale Prince Léopold, Nationalestraat 155, B-2000 Antwerp, Belgium.

The data currently available, from Central as well as from West Africa, allow us to assert that animals are naturally carriers of *T. b. gambiense*. Strains have been isolated from pigs, sheep and goats. However, the importance of animals in the epidemiology of Central-West African sleeping sickness is still to be clarified.

Author's abstract

5965 **Laveissière, C. and Hervouet, J.-P., 1988.** *Epidémiologie et contrôle de la trypanosomiase humaine en Afrique de l'Ouest.* [Epidemiology and control of human trypanosomiasis in West Africa.] Paris; ORSTOM (TDM no. 42). 156 pp. (This work represents a synthesis of 41 published papers and was the subject of the first author's 1986 thesis; see *TTIQ*, **10** (4): no. 5033.)

IPR/OCCGE, B.P. 1500, Bouaké, Côte d'Ivoire.

In West Africa, human trypanosomiasis is a disease connected with the presence of water, which favours vector species of *Glossina*. A close ecological relationship between such species and man leads to the dissemination of the parasite among human populations close to fly habitats. The relatively simple epidemiological situation which results in savanna is, however, complicated in the forest zone where tsetse habitats are not geographically limited, and where all biotopes represent, at different levels, zones of risk for transmission. Man is the main agent responsible for the creation of vector habitats, but his degree of contact with tsetse populations depends greatly on his social and agricultural activities. On the one hand,

agriculture of a collective type, involving frequent population movements, co-operative work and often life in the midst of plantations, leads to the transmission of the parasite among the entire ethnic group concerned; it may also permit the spread of the disease to other areas which are geographically dissimilar. In contrast, a individualistic, familial pattern of agriculture limits man-fly contact and confines the spread of the trypanosome, where this occurs, to the familial group. Control of endemic sleeping sickness requires medical surveillance and vector control. Experience shows that vector control works best when the rural communities themselves are mobilised, and involved in the application of the control technique. At the moment trapping is the only method suitable for this approach in forest areas, because its simplicity and speed of implementation allow its distribution by the national health services and its use by peasant farmers. In fact, its efficiency, moderate cost and harmlessness to the environment are qualities which should make trapping a generally useful tool for the control of *Glossina* species of medical and veterinary importance. For the suppression of sleeping sickness, simple and cost-effective methods are needed but it would be preferable to take adequate measures to prevent or limit the spread of the disease. Agricultural planning and health education allow man-fly contact to be reduced considerably.

Authors' abstract

5966 **Le Ray, D., 1989.** Vector susceptibility to African trypanosomes. *Annales de la Société belge de Médecine tropicale*, **69** (Suppl. 1): 165-171. (See also **12**: no. 5938.)

Department of Protozoology, Institute of Tropical Medicine, Nationalestraat 155, B-2000 Antwerp, Belgium.

Susceptibility of tsetse flies to trypanosomes depends on two distinct barriers controlling respectively colonisation of the midgut and migration to the salivary glands. These barriers are modulated by barely known factors pertaining to the physiological status of the fly as well as to cytoplasmic and nuclear inheritance. Quantification of colonisation (p) and migration (m) rates provides a way to calculate intrinsic vectorial capacity (IVC) as a product $IVC = p \times m$, and to undertake comparative analysis of the underlying factors.

Author's abstract

5967 **Mbulamberi, D.B., 1989.** Possible causes leading to an epidemic outbreak of sleeping sickness: facts and hypotheses. *Annales de la Société belge de Médecine tropicale*, **69** (Suppl. 1): 173-179. (See also **12**: no 5938.)

National Sleeping Sickness Control Programme, P.O. Box 1241, Jinja, Uganda.

Sleeping sickness epidemics have been noted to occur with some degree of periodicity and the question as to why this is so has been asked for quite a long

time. These epidemics have been partially controlled in the past using the conventional methods of bush clearing, mass diagnostic surveys and treatment. Political, social and economic upheavals have been found to be very important factors in the recurrence of these epidemics. In addition, a number of facts and hypotheses have been advanced as possible causes of epidemic outbreaks of sleeping sickness. This paper presents a brief account of factual epidemic outbreaks of sleeping sickness in south-eastern Uganda (Busoga) and then proceeds to discuss, in general terms, a number of hypotheses that have been put forward to date as possible causes that might lead to an epidemic outbreak of the disease.

Author's abstract

5968 **Mehlitz, D., 1989.** Assessing the importance of the animal reservoir and its possible relevance to the medium of human trypanosomiasis. (Summary.) *Annales de la Société belge de Médecine tropicale*, **69** (Suppl. 1): 210. (See also **12**: no. 5938.)

Department of Veterinary Medicine, Bernhard-Nocht-Institut, Bernhard-Nocht-Strasse 74, D-2 Hamburg 4, Federal Republic of Germany.

Recent studies have re-emphasised the zoonotic character of sleeping sickness due to *Trypanosoma brucei rhodesiense*. Evidence for the existence of animal reservoir hosts for *T. b. gambiense* is given by the following observations: 1. Domestic and wild animal carriers with human infective trypanosomes have been identified in West and Central Africa. 2. These trypanosomes can occur as mixed infections together with *T. b. brucei* and other non-human infective *Trypanosoma* spp. in naturally exposed animals. 3. *T. b. gambiense* multiplies, persists and retains its infectivity for the vector in non-human hosts over long periods. To clarify further the epidemiological significance of animal reservoir hosts and the transmission dynamics of the parasites, especially in the *T. b. gambiense* complex, the improvement of sampling and isolation techniques for parasites from human and non-human hosts has been stressed in terms of sensitivity and in providing representative samples of the parasite populations for characterisation.

Author's abstract

5969 **Roberts, L.W., Welde, B.T., Reardon, M.J. and Onyango, F.K., 1989.** Mechanical transmission of *Trypanosoma brucei rhodesiense* by *Glossina morsitans morsitans* (Diptera: Glossinidae). *Annals of Tropical Medicine and Parasitology*, **83** (Suppl. 1): 127-131. (See also **12**: no. 5946.)

Walter Reed Project, Veterinary Research Laboratory, Ministry of Agriculture and Livestock Development, Kabete, Kenya.

Interrupted feedings of teneral, laboratory-reared *Glossina morsitans morsitans* were used to study mechanical transmission of *Trypanosoma brucei rhodesiense*. Intervals between exposure of individual flies on parasitaemic rats and re-feeding on clean rats were varied from 5 min to 24 h. Direct transmissions were demonstrated at each interval up to 160 min after exposure. Proboscis dissections showed that active trypanosomes were present up to 320 min after exposure. No mechanical transmissions from bovine to bovine occurred in 39 attempts, when groups of 20-120 flies exposed on parasitaemic bovines were transferred immediately to uninfected cattle, but two of 40 individual flies exposed on parasitaemic bovines mechanically transmitted trypanosomes to clean rats. Proboscis dissections made immediately after flies were exposed to a bovine with a parasitaemia of 4.8×10^4 trypanosomes/ μ l of blood showed that 11 of 20 (55%) had active trypanosomes in the food canal. The mean number of trypanosomes per proboscis was 29.4 (\pm 20.5). Of 20 flies exposed on a bovine with a low parasitaemia, however, only one trypanosome was seen in proboscis dissections. The parasitaemia of the infected donor was an important factor in mechanical transmission. The mechanical transmission of trypanosomes from one host to another may largely depend on the infectivity threshold of the recipient host, and individual mechanically-infected tsetse flies may not transmit an infective dose.

Authors' abstract

5970 **Sékétéli, A., 1989.** Estimation de l'incidence de la trypanosomiase humaine à *Trypanosoma brucei gambiense* à partir de paramètres épidémiologiques essentiellement liés au vecteur: cas d'un foyer de Côte d'Ivoire. [Estimation of *T. b. gambiense* human trypanosomiasis incidence from epidemiological parameters principally linked to the vector: case study from a focus in Côte d'Ivoire.] *Annales de la Société belge de Médecine tropicale*, **69** (Suppl. 1): 125-135. (See also **12**: no. 5938.)

OMS-Programme de Lutte contre l'Onchocercose en Afrique de l'Ouest, B.P. 2279, Bamako, Mali.

The entomo-epidemiological studies carried out from 1980 to 1985 in the human trypanosomiasis focus at Bouaflé (Côte d'Ivoire) were designed to estimate the main epidemiological parameters related to *Glossina palpalis palpalis*, the sleeping sickness vector in the zone considered. This paper defines the mathematical relationships which can exist between these parameters, leading to the production of a simple model enabling estimates to be made of the incidence of human trypanosomiasis caused by *T. b. gambiense* in this focus. The model predicted a mean annual incidence of 1.50% in the Bouaflé focus. In the zones with high pig densities which were characterised by high densities of tsetse flies with weak anthropophily, the model predicted an incidence of 1.22%. However, a disease incidence of 1.76% was estimated for zones where domestic pigs were absent or at low density and characterised by low densities of tsetse flies with strong anthropophily. The author also emphasises the relationships between

mathematical models and realities in the field and the usefulness of these models to the field worker in planning control campaigns.

Author's abstract

5971 **Tait, A., 1989.** The epidemiological relevance of trypanosome strain variation. *Annales de la Société belge de Médecine tropicale*, **69** (Suppl. 1): 197-203. (See also **12**: no. 5938.)

Wellcome Unit of Molecular Parasitology, Department of Veterinary Parasitology, Faculty of Veterinary Medicine, Beardsen Road, Glasgow G61 1QH, UK.

The study of strain variation in *Trypanosoma brucei rhodesiense* has yielded a lot of information which suggests that these trypanosomes are a sub-set of the stocks circulating in tsetse and non-human hosts and that each focus constitutes a separate set of human infective stocks. These conclusions remain to be fully established by extensive studies in specific foci. Whether the maintenance of these foci or the expansion of them into epidemics is dependent on variation in the trypanosome stocks generated by mutation or genetic exchange is unanswered. In order to examine such questions, detailed long-term studies using *large* numbers of isolates are needed. The analysis of strain variation using existing methods requires considerable manpower and equipment and suffers from the need for growth of trypanosomes in laboratory animals with resultant selection of the stocks under study. The question of whether strain variation in *T. b. rhodesiense* is epidemiologically relevant remains unanswered; variation certainly occurs but the existing studies are not able to answer this question. It is possible that this question could be answered by detailed studies in two or more foci over a long period using new reagents for the detection of variation (e.g. monoclonal antibodies), large trypanosome stock collections and the measurement of parameters such as virulence, tsetse transmissibility, etc.

Author's abstract

5. HUMAN TRYPANOSOMIASIS

(a) SURVEILLANCE

[See also **12**: nos. 5938, 5947, 5948.]

5972 **Kazyumba, G.L., 1989.** Dépistage de la maladie du sommeil. Expérience du Zaïre. [Detection of sleeping sickness cases. Experience of Zaïre.] (Summary.) *Annales de la Société belge de Médecine tropicale*, **69** (Suppl. 1): 207-208. (See also **12**: no. 5938.)

Bureau Central de la Trypanosomiase, B.P. 41, Kinshasa, Zaïre.

Since the late 1960s, the number of new cases of sleeping sickness reported annually has risen from 3,247 to around 10,000. The main reason for this increase is the recrudescence and extension of foci as a result of the mobility of the population, the distribution of potential vectors, and the lack of adequate surveillance. At independence, 6 million people were regularly under surveillance by 240 mobile teams. Today there are only 25 mobile surveillance teams for a much larger population at risk, of around 10 million people. There is a need to intensify and co-ordinate active and passive surveillance, using

serological field techniques as well as classical methods. At the same time, vector control should be strengthened and maintained.

From author's abstract

5973 **Simarro, P.P., Sima, F.O. and Mia, M., 1989.** African trypanosomiasis and *S. intercalatum* infection in Equatorial Guinea: comparative epidemiology and feasibility of integrated control. *Tropical Medicine and Parasitology*, **40** (2): 159-162.

Centro Control Tripanosomiasis, Cooperacion Technica Espanola, Apartado de Correos 560, Bata, Equatorial Guinea.

A preliminary assessment of trypanosomiasis and schistosomiasis (due to *Schistosoma intercalatum*) in Equatorial Guinea is presented. Between 1968 and 1980 no surveillance for trypanosomiasis was undertaken, which resulted in this disease reappearing as an important health problem. Surveillance has now been re-established in all endemic areas of the country. A survey of schistosomiasis distribution and morbidity has recently been undertaken by the trypanosomiasis staff to consider the feasibility of integrating surveillance and control of the two diseases. At present it appears that integration would not be operationally feasible since the two diseases are quite distinct in geographical and age distribution.

5974 **Smith, D.H., Bailey, J.W. and Welde, B.T., 1989.** Immunodiagnostic tests on cerebrospinal fluid in the diagnosis of meningoencephalitic *Trypanosoma brucei rhodesiense* infection. *Annals of Tropical Medicine and Parasitology*, **83** (Suppl. 1): 91-97. (See also **12**: no. 5946.)

Department of Tropical Medicine, Liverpool School of Tropical Medicine, Pembroke Place, Liverpool L3 5QA, UK; *ibid.*; Walter Reed Project, Veterinary Research Laboratory, Ministry of Agriculture and Livestock Development, Kabete, Kenya.

Fourteen cerebrospinal fluid (CSF) samples obtained from Rhodesian sleeping sickness patients from the Lambwe Valley at relapse were positive for the presence of anti-trypanosomal antibody by both IFAT and ELISA. The mean optical density (o.d.) in the ELISA test was 0.804 ± 0.362 and ranged from 0.258 to 1.363. CSF from five patients from the same area without evidence of meningoencephalitis were all negative by ELISA (mean o.d. 0.023 ± 0.016 , range 0.011-0.051). Control CSF samples from UK patients without Rhodesian sleeping sickness but with elevated levels of CSF total protein were also negative. Antibody detected by ELISA declined after Mel-B treatment of relapse and most samples had returned to negative within two years of treatment. We present evidence that serological evaluation of the CSF by ELISA and/or IFAT can provide supportive evidence of the trypanosomal origin of the infection. This is especially important at the time of relapse, when parasitological diagnosis may be impossible and records of treatment for the primary infection may not be available.

Authors' abstract

5975 **Welde, B.T., Chumo, D.A., Hockmeyer, W.T., Reardon, M.J., Esser, K., Schoenbechler, M.J. and Olando, J., 1989.** Sleeping sickness in the Lambwe Valley in 1978. *Annals of Tropical Medicine and Parasitology*, **83** (Suppl. 1): 21-27. (See also **12**: no. 5946.)

Walter Reed Project, Veterinary Research Laboratory, Ministry of Agriculture and Livestock Development, Kabete, Kenya; *ibid.*; *ibid.*; *ibid.*; Department of Immunology, Walter Reed Army Institute of Research, Washington DC, USA; *ibid.*; Division of Disease Control, Ministry of Health, Homa Bay, Kenya.

Even though tsetse control measures were discontinued in the Lambwe Valley in 1974, the prevalence of Rhodesian sleeping sickness remained at low levels. A survey conducted in 1978 verified a low prevalence of disease (0.1%). Thirty-four per cent of the individuals tested were positive for malaria with the highest prevalence (44%) in children aged 0-9 years. Thirteen of 1340 individuals (0.97%) tested and found negative for sleeping sickness in 1978 developed the disease by 1985. Fourteen individuals with moderate titres (2+) in the IFAT but who showed no evidence of disease were traced and found to be alive and well seven years later. Three of these patients still had positive titres but the others had converted to negative. Sera from four patients infected and treated in 1978 were also positive, but only one of five patients treated in 1977 reacted in the test. The complement fixation test as described did not appear useful as a diagnostic test.

Authors' abstract

5976 **Welde, B.T., Chumo, D.A., Reardon, M.J., Nawiri, J., Olando, J., Wanyama, L., Awala, J., Koech, D., Siongok, T.A. and Sabwa, C., 1989.**

Diagnosis of Rhodesian sleeping sickness in the Lambwe Valley (1980-1984). *Annals of Tropical Medicine and Parasitology*, **83** (Suppl. 1): 63-71. (See also **12**: no. 5946.)

Walter Reed Project, Veterinary Research Laboratory, Ministry of Agriculture and Livestock Development, Kabete, Kenya; *ibid.*; *ibid.*; Division of Disease Control, Ministry of Health, Nairobi, Kenya; *ibid.*; *ibid.*; *ibid.*; *ibid.*; *ibid.*; Chemotherapy of Trypanosomiasis Research Project, Veterinary Research Laboratory, Ministry of Agriculture and Livestock Development, Kabete, Kenya. In primary Rhodesian sleeping sickness patients, parasitological diagnosis was best performed by rodent inoculation of blood (98.5%+) followed by Giemsa-stained thick blood smears (93.3%+). Parasitological diagnosis in relapse patients was sometimes impossible and clinical diagnosis based on CSF examination was necessary. Early during a disease outbreak in 1980, 89% of the infections were detected by mobile field teams, but once established in the endemic area a stationary diagnostic facility detected most of the cases. A total number of 23,751 examinations for Rhodesian sleeping sickness and malaria were made by mobile field teams during 1980-84; 102 primary cases (0.43%) and 25 (0.10%) relapse cases were diagnosed. A total of 9339 individuals (39%) had patent malaria infections. The IFAT was positive in 89% of the primary sleeping sickness patients and 77% of the relapse patients. Seventy-nine per cent of the primary patients were positive in a complement fixation test, and 77% of the relapse patients were considered positive.

Authors' abstract

5977 **Wellde, B.T., Chumo, D.A., Reardon, M.J., Waema, D., Smith, D.H., Gibson, W.C., Wanyama, L. and Siongok, T.A., 1989.** Epidemiology of Rhodesian sleeping sickness in the Lambwe Valley, Kenya. *Annals of Tropical Medicine and Parasitology*, **83** (Suppl. 1): 43-62. (See also **12**: no. 5946.)

Walter Reed Project, Veterinary Research Laboratory, Ministry of Agriculture and Livestock Development, Kabeta, Kenya; *ibid.*; *ibid.*; *ibid.*; Department of Tropical Medicine and Infectious Diseases, Liverpool School of Tropical Medicine, Pembroke Place, Liverpool L3 5QA, UK; Department of Pathology, University of Bristol, Langford, Bristol BS18 7DU, UK; Division of Disease Control, Ministry of Health, Nairobi, Kenya; *ibid.*

A total of 912 cases of sleeping sickness have been recorded from the Lambwe Valley from 1959 to 1984. After a period of decreasing prevalence in the 1970s an outbreak of disease occurred between 1980 and 1984. The incidence of disease for this five-year period was highest in areas adjoining the Ruma National Park, reaching 54 per thousand in Area I. Attack rates were highest in the 50+ age group (125) and children had significantly lower attack rates (8‰) in this area of peridomestic transmission. Sex ratios of patients (M/F) were near 1.0 in areas in closest proximity to the thickets in the National Park, while in distant areas the ratios rose to 6.0. The distribution of the number of patients within different households was studied; fewer households than expected had 0 or one patient, and more than expected had three or more patients. No differences in attack rates were found between Nilotic and Bantu groups. Twelve different zymodemes were found in 136 stocks of *Trypanosoma brucei rhodesiense*. Four new zymodemes appeared in 1980 in the latest outbreak and accounted for 73% of the stocks isolated from man during this outbreak. Neutralisation tests

indicated that each trypanosome zymodeme may also represent a different serodeme.

Authors' abstract

5978 **Wéry, M. and Mulumba, M.P., 1989.** Detection of *Trypanosoma brucei gambiense* in the host and alternative sleeping sickness diagnostic approaches. *Annales de la Société belge de Médecine tropicale*, **69** (Suppl. 1): 181-187. (See also **12**: no. 5938.)

Laboratory of Protozoology, Institute of Tropical Medicine, Nationalestraat 155, B-2000 Antwerp, Belgium; Service de Parasitologie, Faculté de Médecine UNIKIN, B.P. 747, Kinshasa XI, Zaire.

The laboratory diagnostic methods of human African trypanosomiasis are reviewed and described: demonstrations of parasites in the blood, lymph and spinal fluid; specific antibody titrations in the serum and spinal fluid; cytological and chemical parameters of blood, plasma and spinal fluid. Some evaluation studies of these methods are mentioned.

Authors' abstract

(b) PATHOLOGY AND IMMUNOLOGY

5979 **Meirvenne, N. van, 1989.** Acquired protective immunity in African trypanosomiasis. (Summary.) *Annales de la Société belge de Médecine tropicale*, **69** (Suppl. 1): 211. (See also **12**: no. 5938.)

Prince Leopold Institute of Tropical Medicine, Nationalestraat 155, B-2000 Antwerp, Belgium.

African trypanosomes evade the immune response of their mammalian host by producing a virtually unlimited series of variable antigen types (VATs). The successive VATs are eliminated by highly specific antibodies that bind to the surface of the live trypanosomes. The interplay between antigenic variation and tardy immune response explains the relapsing nature of the infection. As the infection progresses the host acquires antibody mediated protective immunity against the whole spectrum of VATs experienced. *Trypanosoma brucei rhodesiense* displays a large diversity of both metacyclic and blood form VAT repertoires. *T. b. gambiense* on the other hand exhibits high conservation in its blood form repertoires but little is known about its metacyclic trypanosomes.

From author's abstract

5980 **Pentreath, V.W., 1989.** Neurobiology of sleeping sickness. *Parasitology Today*, **5** (7): 215-218.

Department of Biological Sciences, University of Salford, Salford M5 4WT, UK.

The advanced stages of sleeping sickness are correlated with a spread of trypanosomes into the CNS, producing a disseminated encephalitis.

Inflammatory reactions extend along the blood vessels causing perivascular cuffing, which consists of infiltrations and proliferations of lymphocytes and also increased numbers of astrocytes and microglia. Progress in our understanding of the functions of astrocytes suggests that they are efficient antigen-presenting cells, initiating and regulating the intracerebral inflammatory response and limiting parasite spread to the perivascular spaces.

Author's abstract

5981 **Wellde, B.T., Chumo, D.A., Reardon, M.J., Mwangi, J., Asenti, A., Mbwabi, D., Abinya, A., Wanyama, L. and Smith, D.H., 1989.** Presenting features of Rhodesian sleeping sickness patients in the Lambwe Valley, Kenya.

Annals of Tropical Medicine and Parasitology, **83** (Suppl. 1): 73-89. (See also **12**: no. 5946.)

Walter Reed Project, Veterinary Research Laboratory, Ministry of Agriculture and Livestock Development, Kabete, Kenya; *ibid.*; *ibid.*; *ibid.*; *ibid.*; KETRI, Ministry of Health, Alupe, Kenya; Division of Disease Control, Ministry of Health, Homa Bay, Kenya; *ibid.*; Department of Tropical Medicine and Infectious Diseases, Liverpool School of Tropical Medicine, Pembroke Place, Liverpool L3 5QA, UK.

During a recent outbreak of Rhodesian sleeping sickness in the Lambwe Valley no asymptomatic Rhodesian sleeping sickness patients were found although 54% of the primary patients had mild symptoms and 9% were stuporous or comatose at presentation. The duration of symptoms was 3 months or less in 90% of the patients. Headache, weakness, joint and back pains and weight loss were claimed by at least 75% of the patients, while 82% of the females reported amenorrhoea and 70% of the males claimed impotency. Physical examination revealed lymphadenopathy in 86% but fever in only 36% of the patients, while chancres were found in only 16%. Patients had significantly lower levels of haemoglobin and thrombocytes than controls and their erythrocyte sedimentation rates were elevated. A comparison of both blood group and haemoglobin type between patients and controls yielded no significant differences. Fifty-seven per cent of the primary patients reporting mild symptoms had abnormal levels of leucocytes in their CSF. All relapse patients had abnormal CSF parameters. Levels of serum urea nitrogen were significantly elevated in patients, but serum glutamic oxaloacetic transaminase, serum glutamic pyruvic transaminase and total bilirubin were not. Levels of albumin and β -globulin in patients were significantly lower than controls while γ -globulin was elevated. Mean serum IgM levels in patients were elevated to nearly three-fold those of controls, but 35% of the individual patient values fell within the 95% range of control values. Some patients had extended prothrombin and thrombin times while fibrinogen levels were significantly elevated. No patients reported haemorrhage, and none was seen.

Authors' abstract

(c) TREATMENT

5982 **Bales, J.D. jr, Harrison, S.M., Mbwabi, D.L. and Schecter, J.P., 1989.** Treatment of arsenical refractory Rhodesian sleeping sickness in Kenya. *Annals of Tropical Medicine and Parasitology*, **83** (Suppl. 1): 111-114. (See also **12**: no. 5946.)

Walter Reed Project, Kenya Medical Research Institute, Nairobi, Kenya; *ibid.*; Human Treatment Facility, KETRI, Alupe, Kenya; Merrill Dow Research Institute, Strasbourg Cédex, France.

Case histories of three Rhodesian sleeping sickness patients who relapsed after Mel-B therapy are presented. Repeated Mel-B therapy was clinically effective but not curative, and all three patients subsequently relapsed again and required further treatment.

Authors' abstract

5983 **Pepin, J., Guern, C., Ethier, L., Milord, F., Mpia, B. and Mansinsa, D., 1989.** Trial of prednisolone for prevention of melarsoprol-induced encephalopathy in *gambiense* sleeping sickness. *Lancet*, no. 8649: 1246-1250.

Pepin: MRC Laboratories, P.O. Box 273, Banjul, Gambia; other authors: Zone de Santé Rurale, Nioki, Zaire; Guern, Ethier, Milord also: University of Sherbrooke, Sherbrooke, Canada.

In a prospective randomised trial, 620 patients who had *Trypanosoma brucei gambiense* trypanosomiasis with CNS involvement were treated either with prednisolone plus melarsoprol or with melarsoprol only. 598 patients were evaluable: morbidity and death associated with melarsoprol-induced encephalopathy was reduced in patients who were given prednisolone. The two groups did not differ either in the incidence of other complications of melarsoprol therapy or in relapse rate after melarsoprol therapy. The cost of prednisolone would be outweighed by savings on the treatment of encephalopathies in such patients.

Authors' abstract

5984 **Wellde, B.T., Chumo, D.A., Reardon, M.J., Abinya, A., Wanyama, L., Dola, S., Mbwabi, D., Smith, D.H. and Siongok, T.A., 1989.** Treatment of Rhodesian sleeping sickness in Kenya. *Annals of Tropical Medicine and Parasitology*, **83** (Suppl. 1): 99-109. (See also **12**: no. 5946.)

Walter Reed Project, Veterinary Research Laboratory, Ministry of Agriculture and Livestock Development, Kabete, Kenya; *ibid.*; *ibid.*; Homa Bay District Hospital, Ministry of Health, Homa Bay, Kenya; *ibid.*; *ibid.*; KETRI, Ministry of Health, Alupe, Kenya; Department of Tropical Medicine and Infectious Diseases, Liverpool School of Tropical Medicine, Pembroke Place, Liverpool L3 5QA, UK; Division of Disease Control, Ministry of Health, Nairobi, Kenya.

In a study of 269 sleeping sickness patients treated with Mel-B, 14 (5.2%) died during treatment. With total dosages of at least 30 ml (1.08 g), 1.4% relapsed and another 6.4% died, mostly of unknown causes, within three years of treatment, giving a success rate of 92.1% over the three years. Mel-B was used to treat 55 relapses after suramin therapy with 1.8% deaths during treatment, 3.6% relapses and 92.7% success over at least three years. Apparent drug resistance to Mel-B was found in three patients who continued to relapse after repeated treatments. During 1980, 51 patients were treated with suramin on the basis of clinical condition without benefit of CSF analysis. Subsequently 49% of these patients relapsed within three years of treatment. When 29 patients were treated on the basis of CSF evaluation only two (7%) relapsed.

Authors' abstract

6. ANIMAL TRYPANOSOMIASIS

(a) SURVEY AND DISTRIBUTION

5985 **Diall, O., Coumare, A., Diarra, B., Sanogo, Y. and Coulibaly, Z., 1987.** Note sur la trypanosomose *T. evansi* dans le secteur de Nara (Mali) [et] dans le Sud Mauritanien. [Note on *Trypanosoma evansi* infection in the Nara district (Mali) and in southern Mauritania.] *Camel Forum, Working Paper*, no. 18: 97-99.

Diall: Institut National de la Recherche Zootechnique Forestière et Hydrobiologique, Bamako, Mali.

A survey was undertaken to discover the incidence of *T. evansi* infection in 195 camels in the Nara district of

Mali and at Adel-Bagrou and Néma in Mauritania. Infection rates varied according to season: 3% in the dry season and 20% in the rainy season (90% of the latter were animals aged 0-5 years). Infected animals had significantly lower PCV levels. The high infection rate found in the rainy season was undoubtedly due to an abundance of tabanids at this season.

5986 **Guleed, H.A. and Bornstein, S., 1987.** Pilot study of the health of Somali camel herds. *Camel Forum, Working Paper*, no. 23: 10 pp.

A preliminary report is given of a small pilot study of nine camel herds (775 heads) in southern Somalia (Lower Shabelle region, Wanlewyen district). Both acute and chronic trypanosomiasis were common, along with many other diseases. All herds showed low fertility, frequent abortion and rather high mortality rate, particularly in calves and young stock.

Based on authors' abstract

5987 **Kariuki, D.P., Injairo, R., Boyce, W.L., Welde, B.T. and Ngethe, S., 1989.** Parasite survey of eight wild animals in the Ruma National Park. *Annals of Tropical Medicine and Parasitology*, **83** (Suppl. 1): 115-118. (See also **12**: no. 5946.)

Veterinary Research Department, Kenya Agriculture Research Institute, P.O. Box 32, Kikuyu, Kenya; Pathology Section, Veterinary Research Laboratory, Ministry of Agriculture and Livestock Development, Kabete, Kenya; Walter Reed Project, Veterinary Research Laboratory, Ministry of Agriculture and Livestock Development, Kabete, Kenya; *ibid.*; Wildlife Conservation and Management, Ministry of Tourism, Nairobi, Kenya.

Eight game animals representing seven species in the Ruma National Park in South Nyanza, Kenya, were examined for the presence of blood protozoa, ectoparasites, and helminthic and coccidian endoparasites using standard parasite-identification methods. Haematological parameters were also determined. The oribi was positive for *Trypanosoma brucei* ssp. and the reedbuck for *T. congolense*. No other blood protozoans were found. Strongyle eggs were found in the faeces of all species except the waterbuck. Five of eight animals harboured liver flukes and five were parasitised by ticks of the genus *Amblyomma*. One roan antelope was anaemic, but the other animals had haemoglobin levels within the normal range and appeared to be in a good state of health.

Authors' abstract

(b) PATHOLOGY AND IMMUNOLOGY

5988 **Boyce, W.L., Welde, B.T., Reardon, M.J., Bhogal, M.S. and Chumo, D.A., 1989.** Effects of splenectomy on *Trypanosoma congolense* infection in cattle. *Annals of Tropical Medicine and Parasitology*, **83** (Suppl. 1): 195-200. (See also **12**: no. 5946.)

Walter Reed Project, Veterinary Research Laboratory, Ministry of Agriculture and Livestock Development, Kabete, Kenya.

The role of the spleen in cattle infected with *T. congolense* was studied by comparing levels of parasitaemia, blood cell values, and body weights of intact and splenectomised cattle. A total of 28 Zebu \times Hereford steers were used in two separate experiments. Seven animals were splenectomised at least 4 weeks prior to infection and two others were splenectomised 128 days after infection. Splenectomised animals were compared to 12 intact infected animals and seven intact uninfected controls. The splenectomised animals suffered no less severe anaemia and no higher parasitaemia than the intact, infected cattle. Splenectomy in two animals during infection had no effect of PCV, parasitaemia or survival. Splenectomised cattle have lower levels of circulating lymphocytes following treatment than intact animals; also the splenectomised steers lost more weight during the active infection.

Authors' abstract

5989 **Igbokwe, I.O. and Anosa, V.O., 1989.** Leucopenia in *Trypanosoma vivax* infection of sheep. *Revue d'Elevage et de Médecine vétérinaire des Pays tropicaux*, **42** (2): 219-221.

Department of Veterinary Pathology, University of Maiduguri, Nigeria;
Department of Veterinary Pathology, University of Ibadan, Ibadan, Nigeria.
Experimental *T. vivax* infection of sheep produced a moderate leucopenia associated with a lymphopenia and eosinopenia. The total white blood cell counts of adult mice were not significantly depressed when inoculated with plasma from *T. vivax*-infected sheep. These observations suggested that the plasma of the infected sheep did not have a factor which could depress leucopoiesis *in vivo*.

Authors' abstract

5990 **Kyewalabye Kaggwa, E. and Lawal, I.A., 1989.** *Babesia equi* and *Trypanosoma vivax* infections in donkeys. *Revue d'Elevage et de Médecine vétérinaire des Pays tropicaux*, **42** (2): 205-210.

TDR, P.O. Box 71769, Ndola, Zambia; Department of Parasitology and Entomology, Faculty of Veterinary Medicine, Ahmadu Bello University, Zaria, Nigeria.

Six donkeys (*Equus asinus*) were purchased locally. To screen them before and during *T. vivax* infection, thin and thick blood smears, temperature, HCT, PCV, white blood cell counts and IFAT were done for *B. equi*. For the IFAT, an anti-horse conjugate was used. In spite of patent *B. equi* or *T. vivax* parasitaemia, the donkeys' temperatures remained below 38.5°C; PCV was depressed more in *B. equi* infection than in *T. vivax* infection. Four out of the 6 donkeys had *B. equi* antibodies while 2 of them had detectable parasitaemia. Treatment with either Berenil or Imizol cleared the detectable *B. equi* parasitaemia, and IFAT was negative at 35-45 days post-treatment. However, relapses occurred within 60-70 days after the treatment. In two circumstances serological titres were below 1:40 (negative) while there was detectable parasitaemia.

Authors' abstract

5991 **Murray, M., 1989.** Factors affecting duration and intensity of trypanosome infection of domestic animals. *Annales de la Société belge de Médecine tropicale*, **69** (Suppl. 1): 189-196. (See also **12**: no. 5938.)

Department of Veterinary Medicine, University of Glasgow Veterinary School, Bearsden Road, Glasgow G61 1QH, UK.

Domestic animals can become infected with several species of trypanosomes that are transmitted by tsetse, including *Trypanosoma congolense*, *T. vivax*, *T. brucei*, *T. simiae*, *T. rhodesiense* and *T. gambiense*. The intensity and duration of the resulting parasitaemia are affected by the parasite, the host and detection methods. Techniques currently used to detect parasites range in sensitivity from one to 10^5 organisms per ml. At the same time, parasite kinetics vary greatly both within and between species of trypanosomes, although as a rule, at least for cattle, it is usual for *T. vivax* to give rise to higher parasitaemias ($> 10^6$ per ml) than *T. congolense* ($< 10^6$ per ml) which is generally more persistent, while *T. brucei* results in only scanty transient parasitaemia ($< 10^5$ per ml). It should also be appreciated that both *T. vivax* and *T. brucei*, unlike *T. congolense*, invade tissues. Species of the host infected has a significant effect on parasitaemia patterns, e.g., in contrast to cattle, dogs develop high parasitaemias ($> 10^7$ per ml) when infected with *T. brucei*. Certain animals possess a remarkable inherent capacity to control, reduce and even self-cure infections, irrespective of the species of infecting trypanosome. This is best demonstrated by trypanotolerant breeds of cattle such as the N'Dama and West African Shorthorn, as well as by most species of wild Bovidae and Suidae. Physiological and environmental factors can probably influence the kinetics of infection but these are poorly defined and await quantification. Factors which may be involved include nutritional status, age, sex, previous exposure to infection and stress in relation to reproduction, overwork and intercurrent infection. In conclusion, detection and persistence of trypanosomes in domestic animals are affected by many variables, with the result that failure to detect parasites on any one occasion cannot be taken as an indication of absence of infection.

Author's abstract

5992 Wellde, B.T., Chumo, D.A., Onyango, F.K., Reardon, M.J., Roberts, L.M., Njogu, A.R. and Opiyo, E.A., 1989. *Trypanosoma vivax*: disseminated intravascular coagulation in cattle. *Annals of Tropical Medicine and Parasitology*, **83** (Suppl. 1): 177-183. (See also **12**: no. 5946.)

Walter Reed Project, Veterinary Research Laboratory, Ministry of Agriculture and Livestock Development, Kabete, Kenya; *ibid.*; *ibid.*; *ibid.*; *ibid.*; KETRI, Muguga, Kenya; *ibid.*

Five crossbred cattle infected with *Trypanosoma vivax* (Likoni) by *Glossina morsitans* developed capillary haemorrhages at the onset of parasitaemia, followed by the presence of occult blood in faecal samples and eventually melena. Two animals required treatment to survive, on days 13 and 38 respectively. The other three animals cleared their parasitaemias without treatment. PCV levels decreased in all animals to levels ranging from 7.5 to 17%. Relapse in a treated animal initiated marked haemorrhage and a loss of 14 PCV units during a six-day period. Thrombocytopenia was common to all animals, and thrombocytes decreased to levels of 4000/ μ l of blood. All animals developed increased levels of fibrinogen and fibrin monomer. Prolonged prothrombin times were found in all animals, and activated partial thromboplastin times were also extended in the two animals with high parasitaemias.

Authors' abstract

5993 Wellde, B.T., Preston, J.M., Kovatch, R.M., Higgs, J. and Chumo, D.A., 1989. *Trypanosoma congolense*: erythrocyte indices, plasma iron turnover

and effects of treatment in infected cattle. *Annals of Tropical Medicine and Parasitology*, **83** (Suppl. 1): 201-206. (See also **12**: no. 5946.)

Walter Reed Project, Veterinary Research Laboratory, Ministry of Agriculture and Livestock Development, Kabete, Kenya; Preston: Merck Sharp & Dohme Research Laboratories, Hoddesdon, UK.

Early during the course of *T. congolense* infection in cattle, decreases in PCV occurred and coincided with increases in both mean corpuscular volume (MCV) and mean corpuscular haemoglobin (MCH). The indices reached highest levels between 8 and 12 weeks post-infection. By week 20 of infection MCV and MCH had decreased to pre-infection levels even though a substantial anaemia persisted. Serum iron levels were elevated at 8 weeks post-infection (infected 271 mg dl⁻¹ v. control 140 mg dl⁻¹) but decreased to low levels in infected animals by Week 28 (63 mg dl⁻¹). At 8 weeks post-infection plasma iron turnover rate (PITR) was elevated in infected animals (infected 2.6 mg dl⁻¹ day⁻¹ v. control 0.82 mg dl⁻¹ day⁻¹). By Week 28, however, PITR had fallen in infected animals to 0.62 mg dl⁻¹ day⁻¹ indicating a severe dyshaemopoiesis since PCV levels averaged only 22%. Cattle which were treated with Berenil or that self-cured the infection had normal iron parameters when tested 61 weeks post-infection. Cattle treated early

during the course of infection showed a significantly greater PCV response than those treated later during infection.

Authors' abstract

5994 **Wellde, B.T., Reardon, M.J., Chumo, D.A., Kovatch, R.M., Waema, D., Wykoff, D.E., Mwangi, J., Boyce, W.L. and Williams, J.S., 1989.** Cerebral trypanosomiasis in naturally-infected cattle in the Lambwe Valley, South Nyanza, Kenya. *Annals of Tropical Medicine and Parasitology*, **83** (Suppl. 1): 151-160. (See also **12**: no. 5946.)

Walter Reed Project, Veterinary Research Laboratory, Ministry of Agriculture and Livestock Development, Kabete, Kenya.

Surveys in Zebu cattle in the Lambwe Valley in 1980 indicated that many (up to 70%) were infected with trypanosomes. The predominant parasite was *Trypanosoma brucei* ssp. followed by *T. congolense*. CSF analysis showed a high proportion of animals with pleocytosis and elevated total CSF protein.

Trypanosomes were detected in CSF and signs of a CNS disease were observed.

Histopathological lesions in the CNS were identical to those found in experimentally-infected cattle and consisted of perivascular infiltrations, swollen endothelium of vessels, infiltration of the vascular wall, and perivascular oedema. The severest cases showed rarefaction, astrocytosis and areas of necrosis.

Infected cattle transported to the Veterinary Research Laboratory were studied for up to four years. Absence of trypanosomes from the peripheral blood was common, and even subinoculation of lymph node aspirates and CSF were usually negative. Death was preceded by a period of weight loss and the development of severe CNS signs. An attempt to cure animals with Mel-B treatment failed.

Serum from naturally-infected cattle neutralised *T. b. rhodesiense* stocks collected in the same area.

Authors' abstract

5995 **Wellde, B.T., Reardon, M.J., Kovatch, R.M., Chumo, D.A., Williams, J.S., Boyce, W.L., Hockmeyer, W.T. and Wykoff, D.E., 1989.** Experimental infection of cattle with *Trypanosoma brucei rhodesiense*. *Annals of Tropical Medicine and Parasitology*, **83** (Suppl. 1): 133-150. (See also **12**: no. 5946.)

Walter Reed Project, Veterinary Research Laboratory, Ministry of Agriculture and Livestock Development, Kabete, Kenya.

Infection of cattle with various stocks of *T. b. rhodesiense* indicated that 49% developed a fatal CNS disease comparable to that found in man. Duration of disease ranged from 85 to 1613 days post infection. All eight stocks of *T. b. rhodesiense* tested, including those from Ethiopia and Tanzania, induced CNS disease. Blood became positive 3-5 days after inoculation, and after an initial peak of parasitaemia remained positive for 3-5 months. Subinoculation of blood into rodents subsequently became negative, although trypanosomes persisted in

the lymph nodes for at least 56 to 1613 days. Only animals with CNS disease had detectable parasites in the CSF, usually after the animals had undergone severe deterioration. At post mortem examination trypanosomes could usually be found in the lymph nodes and CSF, and occasionally in the blood. Clinical signs included fever, hyperkinesia, weight loss, cerebellar ataxia, tremor, salivation and hyperaesthesia. A mild to moderate anaemia accompanied a transient thrombocytopenia and leucopenia. Animals subsequently developed leucocytosis. A pleocytosis and elevated total protein in the CSF was found, which persisted in some animals for long periods. Histopathological examination of the brain showed prominent generalised perivascular infiltrates consisting mainly of lymphocytes and plasma cells. Mott's cells were regularly observed. Vascular changes were characterised by swollen endothelium, infiltration of the vascular wall by inflammatory cells, and in some instances perivascular oedema. In the most severe cases evidence of ischaemia consisted of large numbers of astrocytes, rarefaction of the parenchyma, and areas of necrosis with loss of normal architecture. Demyelination was limited to perivascular areas. Occasionally a moderate to severe pancarditis was found.

Authors' abstract

(c) TRYPANOTOLERANCE

5996 **Maillard, J.C., Kemp, S.J., Leveziel, H., Teale, A.J. and Quéval, R., 1989.** Le complexe majeur d'histocompatibilité de bovins ouest africains.

Typage d'antigènes lymphocytaires (BoLA) de taurins Baoulé (*Bos taurus*) et de zébus soudaniens (*Bos indicus*) du Burkina Faso (Afrique Occidentale). [The major histocompatibility complex of West African cattle. Lymphocyte antigen (BoLA) typing on Baoulé taurine cattle (*Bos taurus*) and Sudanian zebu (*B. indicus*) in Burkina Faso (West Africa).] *Revue d'Elevage et de Médecine vétérinaire des Pays tropicaux*, **42** (2): 275-281.

Maillard, Quéval: Service Immunogénétique du CRTA, 01 B.P. 454, Bobo-Dioulasso 01, Burkina Faso; Kemp, Teale: ILRAD, P.O. Box 30709, Nairobi, Kenya; Leveziel: Laboratoire de Génétique Biochimique, INRA-CRZ, 78350 Jouy-en-Josas, France.

Lymphocyte antigen (BoLA) typing on 247 Baoulés and 106 Sudanian zebu allowed the determination of gene frequencies of 43 class 1 specificities, international 'W' and European 'EU' as well as local African 'KN' from Kenya and 'BF' from Burkina Faso. Comparison of these frequencies showed that some specificities could be considered as significant breed markers for Baoulé taurines and zebu.

Authors' abstract

5997 **Mutayoba, B.M., Gombe, S., Waindi, E.N. and Kaaya, G.P., 1989.**

Comparative trypanotolerance of the Small East African breed of goats from different localities to *Trypanosoma congolense* infection. *Veterinary Parasitology*, **31** (2): 95-105.

Sokoine University of Agriculture, P.O. Box 3017, Morogoro, Tanzania; Reproductive Biology Unit, University of Nairobi, P.O. Box 30197, Nairobi, Kenya; *ibid.*; ICIPE, P.O. Box 30772, Nairobi, Kenya.

Differences in susceptibility of the Small East African breed of goats to an experimental *T. congolense* infection were investigated. The goats were obtained from different areas of East Africa, Morogoro and Arusha (Tanzania), Imbo and Lambwe Valley (Kenya). Morogoro goats were found to be more tolerant, followed by Arusha, Lambwe Valley and Imbo goats, in that order. The Imbo goats had highest parasitaemia, more severe anaemia, marked weight losses and highest mortality rate. The Morogoro goats showed a milder infection with lower parasitaemia, less severe anaemia, smaller weight losses and lowest mortality rate. The Lambwe Valley and Arusha goats were intermediate to the Morogoro (resistant) and Imbo (susceptible) goats. The significance of these findings in relation to existence of heterogeneity in the Small East African goats is discussed. Authors' abstract

5998 **Wellde, B.T., Reardon, M.J., Onyango, F., Chumo, D.A., Muriithi, R.M., Roberts, L.M., Njogu, A.R. and Kamar, K.K., 1989.** Natural and acquired resistance to *Trypanosoma vivax* in cattle. *Annals of Tropical Medicine and Parasitology*, **83** (Suppl. 1): 185-194. (See also **12**: no. 5946.)

Walter Reed Project, Veterinary Research Laboratory, Ministry of Agriculture and Livestock Development, Kabete, Kenya; *ibid.*; *ibid.*; *ibid.*; *ibid.*; KETRI, Ministry of Health, Muguga, Kenya; *ibid.*

Zebu \exists European (Z \exists E) crossbred cattle suffered a more severe course of disease than Boran cattle when infected with *Trypanosoma vivax* (Likoni) by *Glossina morsitans*. All Z \exists E animals in this study required Berenil treatment while all Borans self-cured the infection without treatment. The more severe disease in Z \exists E animals was characterised by longer periods of patent infection and fever, more severe anaemia and greater likelihood of haemorrhage. Cattle previously infected and cured with Berenil showed resistance and self-cured challenge infections. After self-cure cattle remained immune to tsetse fly challenge with the homologous trypanosome stock for long periods. Immunity induced by infection and drug or self-cure appeared to be specific for the homologous stock, since cattle immune to *T. vivax* (Likoni) showed no resistance when challenged with stocks of *T. vivax* isolated in Lugala, Uganda or Galana, Kenya. Severe haemorrhages, most prominent in the digestive tract, were seen in some infected cattle before treatment.

Authors' abstract

(d) TREATMENT

[See also **12**: no. 6011.]

5999 **Wellde, B.T., Reardon, M.J., Chumo, D.A., Muriithi, R.M., Towett, S. and Mwangi, J., 1989.** Experimental infection of goats with *Trypanosoma*

brucei ssp. and effects of treatment with suramin and Mel-B. *Annals of Tropical Medicine and Parasitology*, **83** (Suppl. 1): 161-169. (See also **12**: no. 5946.)

Walter Reed Project, Veterinary Research Laboratory, Ministry of Agriculture and Livestock Development, Kabete, Kenya.

A stock of *Trypanosoma brucei* ssp. isolated from a naturally infected goat in the Lambwe Valley, Kenya, induced cerebral trypanosomiasis in experimentally infected goats. Six of nine goats with cerebral trypanosomiasis induced by this stock were cured by a single high dose of suramin (50 mg kg⁻¹). Two other goats appeared to be cured with this dosage of suramin but later developed abnormal CNS signs and parasitaemia. Parasites first appeared in the CSF and then in the blood and lymph nodes. Mel-B was also effective against primary and relapse cerebral trypanosomiasis in goats.

Authors' abstract

7. EXPERIMENTAL TRYPANOSOMIASIS

(a) DIAGNOSTICS

6000 Moser, D.R., Cook, G.A., Ochs, D.E., Bailey, C.P., McKane, M.R. and Donelson, J.E., 1989. Detection of *Trypanosoma congolense* and *Trypanosoma brucei* subspecies by DNA amplification using the polymerase chain reaction. *Parasitology*, **99** (1): 57-66.

Department of Internal Medicine (Cook), Department of Biochemistry, and Diabetes and Endocrinology Research Center (others), University of Iowa, Iowa City, IA 52242, USA.

(b) PATHOLOGY AND IMMUNOLOGY

6001 Black, S.J., Murray, M., Shapiro, S.Z., Kaminsky, R., Borowy, N.K., Musanga, R. and Otieno-Omondi, F., 1989. Analysis of *Propionibacterium acnes*-induced non-specific immunity to *Trypanosoma brucei* in mice. *Parasite Immunology*, **11** (4): 371-383.

Black, Kaminsky, Musanga, Otieno-Omondi: ILRAD, P.O. Box 30709, Nairobi, Kenya; Murray: University of Glasgow Veterinary School, Glasgow, G61 1QH, UK; Shapiro: University of Illinois at Urbana-Champaign, Urbana, IL 61801, USA; Borowy: Max-Planck-Institut für Biologie, D-7400 Tübingen 1, Federal Republic of Germany.

6002 Emeribe, A.O. and Anosa, V.O., 1988. Haematology of experimental *gambiense* trypanosomiasis. I. Platelet and other haemostatic changes. [Rabbits.] *Central African Journal of Medicine*, **34** (12): 286-290.

Department of Haematology, College of Medical Sciences, University of Calabar, Calabar, Nigeria; Department of

Veterinary Pathology, University of Ibadan, Ibadan, Nigeria.

6003 **Mahan, S.M. and Black, S.J., 1989.** Differentiation, multiplication and control of bloodstream form *Trypanosoma (Duttonella) vivax* in mice. *Journal of Protozoology*, **36** (4): 424-428.

ILRAD, P.O. Box 30709, Nairobi, Kenya.

6004 **Seed, J.R. and Sechelski, J.B., 1989.** African trypanosomes: inheritance of factors involved in resistance. *Experimental Parasitology*, **69** (1): 1-8.

Department of Parasitology and Laboratory Practice, School of Public Health, University of North Carolina, Chapel Hill, NC 27599-7400, USA.

Work on mice demonstrated that survival time is inherited as a recessive trait, whereas the ability to produce antibody to the first variant antigen population is inherited as a dominant trait. Survival time could therefore not be correlated with ability to produce antibody. Neither was it possible to link the animal's ability to control its early parasitaemia, or change in haematocrit, with either antibody production or survival time.

From authors' abstract

6005 **Sileghem, M., Darji, A., Hamers, R. and Baetselier, P. de, 1989.** Modulation of IL-1 production and IL-1 release during experimental trypanosome infections. [*T. brucei*.] *Immunology*, **68** (1): 137-139.

ILRAD, P.O. Box 30709, Nairobi, Kenya; Instituut voor Moleculaire Biologie, Vrije Universiteit Brussel, B-1640 St-Genesius-Rode, Belgium; *ibid.*; *ibid.*

6006 **Sileghem, M., Darji, A., Hamers, R., Winkel, M. van de and Baetselier, P. de, 1989.** Dual role of macrophages in the suppression of interleukin 2 production and interleukin 2 receptor expression in trypanosome-infected mice. [*T. brucei*.] *European Journal of Immunology*, **19** (5): 829-835.

Sileghem: ILRAD, P.O. Box 30709, Nairobi, Kenya; Darji, Hamers, Baetselier: Instituut voor Moleculaire Biologie, Vrije Universiteit Brussel, B-1640 St-Genesius-Rode, Belgium; Winkel: Faculteit Geneeskunde, Vrije Universiteit Brussel, Jette, Belgium.

6007 **Verducci, G., Perito, S., Rossi, R., Mannarino, E., Bistoni, F. and Marconi, P., 1989.** Identification of a trypanocidal

factor against *Trypanosoma equiperdum* in normal human serum. *Parasitology*, **98** (3): 401-407.

Department of Experimental Medicine and Biochemical Sciences, Microbiology Section (Verducci, Perito, Bistoni, Marconi), Institute of General Pathology (Rossi) and Division of Internal Medicine, Policlinico, Montelucente (Mannarino), University of Perugia, Via del Giochetto, 06100 Perugia, Italy.

(c) CHEMOTHERAPEUTICS

6008 **DeLoach, J.R. and Wagner, G.G., 1988.** Some effects of the trypanocidal drug isometamidium on encapsulation in bovine carrier erythrocytes. *Biotechnology and Applied Biochemistry*, **10** (5): 447-453.

Veterinary Toxicology and Entomology Research Laboratory, ARS, USDA, P.O. Drawer GE, College Station, TX 77841, USA; Department of Microbiology and Parasitology, College of Veterinary Medicine, Texas A & M University, College Station, TX 77843, USA.

6009 **Igweh, A.C. and Onabanjo, A.O., 1989.** Chemotherapeutic effects of *Annona senegalensis* in *Trypanosoma brucei brucei*. *Annals of Tropical Medicine and Parasitology*, **83** (5): 527-534.

Department of Pharmacology, Chemotherapy Unit, College of Medicine, University of Lagos, Lagos, Nigeria.

Aqueous root extracts of *A. senegalensis* were shown to be therapeutically effective against *T. b. brucei* infection in mice, substantiating the claims of Nigerian practitioners of traditional medicine that it is effective against trypanosomiasis in man.

6010 **Kaminsky, R., Chuma, F. and Zweygarth, E., 1989.** *Trypanosoma brucei brucei*: expression of drug resistance *in vitro*.

[Diminazene aceturate, isometamidium chloride.]

Experimental Parasitology, **69** (3): 281-289.

ILRAD, P.O. Box 30709, Nairobi, Kenya; KETRI, P.O. Box 362, Kikuyu, Kenya.

6011 **Reardon, M.J., Welde, B.T., Muriithi, R.M., Chumo, D.A., Towett, S. and Mwangi, J., 1989.** Effectiveness of WR163577 against animal trypanosomes in goats and mice. *Annals of Tropical Medicine and Parasitology*, **83** (Suppl. 1): 171-175. (See also **12**: no. 5946.)

Walter Reed Project, Veterinary Research Laboratory, Ministry of Agriculture and Livestock Development, Kabete, Kenya.

A bisquinaldine, 1,6-bis-(6-amino-2-methyl-4-quinolyamino) hexane, was tested against *Trypanosoma brucei* ssp. in goats and against *T. brucei*, *T. congolense* and *T. vivax* in mice. At doses of 25 and 100 mg kg⁻¹, the drug protected goats for a least 90 days against blood challenge with *T. brucei* ssp. Fifty to sixty per cent of

goats challenged 180 days after treatment were protected, but all goats challenged 270 days after treatment became infected. In mice, bisquinaldine also had a marked effect on *T. brucei*, but only a minimal effect on *T. vivax* and no apparent effect on *T. congolense*. No drug toxicity was noted in mice even at doses of 2000 mg kg⁻¹. Both short-term (25 and 100 mg kg⁻¹) and long term (100 mg kg⁻¹) toxicity was apparent in goats treated with bisquinaldine.

Authors' abstract

6012 **Wosu, L.O. and Ibe, C.C., 1989.** Use of extracts of *Picralima nitida* bark in the treatment of experimental trypanosomiasis: a preliminary study. *Journal of Ethnopharmacology*, **25** (3): 263-268.

Department of Veterinary Medicine, University of Nigeria, Nsukka, Nigeria.

The effect of a boiling water extract of *P. nitida* bark against trypanosomes (*Trypanosoma brucei*) in rats was compared with that of diminazene aceturate at 8 mg/kg i.p. Results showed that the extract had a definite trypanocidal effect which was statistically comparable to that of diminazene aceturate. Further work is needed to identify the active pharmacological agent and to standardise the usage of the agent in practice.

Authors' abstract

8. TRYPANOSOME RESEARCH

(a) CULTIVATION OF TRYPANOSOMES

6013 **Schwarz, R.T., Mayor, S., Menon, A.K. and Cross, G.A.M., 1989.**

Biosynthesis of the glycolipid membrane anchor of *Trypanosoma brucei* variant surface glycoproteins: involvement of Dol-*P*-Man. *Biochemical Society Transactions*, **17** (4): 746-748.

Medizinisches Zentrum für Hygiene, Philipps-Universität Marburg, Robert-Koch-Strasse 17, 3550 Marburg, Federal Republic of Germany; Rockefeller University, 1230 York Avenue, New York, NY 10021, USA; *ibid.*; *ibid.*

(b) TAXONOMY, CHARACTERISATION OF ISOLATES

[See also **12**: no. 5977.]

6014 **Richner, D., Schweizer, J., Betschart, B. and Jenni, L., 1989.**

Characterization of West African *Trypanosoma* (*Trypanozoon*) *brucei* isolates from man and animals using isoenzyme analysis and DNA hybridization. *Parasitology Research*, **76** (1): 80-85.

Swiss Tropical Institute, Socinstrasse 57, CH-4051 Basel, Switzerland. (Reprint requests to Jenni.)

The 18 stocks investigated could be separated into two distinct groups according to their isoenzyme and DNA patterns: a homogeneous group of *T. b. gambiense* and a very variable non-*gambiense* group consisting of *T. b. brucei* and human pathogenic *T. b. rhodesiense*.

(c) LIFE CYCLE, MORPHOLOGY, BIOCHEMICAL AND MOLECULAR STUDIES

6015 **Affolter, M., Rindisbacher, L. and Braun, R., 1989.** The tubulin gene cluster of *Trypanosoma brucei* starts with an intact β -gene and ends with a truncated β -gene interrupted by a retrotransposon-like sequence. *Gene*, **80** (1): 177-183.

Institut für Allgemeine Mikrobiologie, Universität Bern, Baltzer-Strasse 4, 3012 Bern, Switzerland.

(Correspondence to Braun.)

6016 **Bacchi, C.J., Garofalo, J., Santana, A., Hannan, J.C., Bitonti, A.J. and McCann, P.P., 1989.** *Trypanosoma brucei brucei*: regulation of ornithine decarboxylase in procyclic forms and trypomastigotes. *Experimental Parasitology*, **68** (4): 392-402.

Haskins Laboratories and Department of Biology, Pace University, 41 Park Row, New York, NY 10038, USA; *ibid.*; *ibid.*; *ibid.*; Merrell Dow Research Institute, 2110 Galbraith Road, Cincinnati, OH 45215, USA; *ibid.*

6017 **Bülow, R., Griffiths, G., Webster, P., Stierhof, Y.-D., Opperdoes, F.R. and Overath, P., 1989.** Intracellular localization of the glycosyl-phosphatidylinositol-specific phospholipase C of *Trypanosoma brucei*. *Journal of Cell Science*, **93** (2): 233-240.

Department of Medical Microbiology, Sherman Fairchild Building, Stanford, CA, USA; European Molecular Biology Laboratory, Postfach 10.2209, D-6900 Heidelberg, Federal Republic of Germany; ILRAD, P.O. Box 30709, Nairobi, Kenya; Max-Planck-Institut für Entwicklungsbiologie, Spemannstrasse 35, D-7400 Tübingen, Federal Republic of Germany; International Institute of Cellular and Molecular Pathology, Université Catholique de Louvain, B-1200 Brussels, Belgium; Max-Planck-Institut für Biologie, Corrensstrasse 38, D-7400 Tübingen, Federal Republic of Germany. (Correspondence to Overath.)

6018 **Coquelet, H., Tebabi, P., Pays, A., Steinert, M. and Pays, E., 1989.** *Trypanosoma brucei*: enrichment by UV of intergenic transcripts from the variable surface glycoprotein gene expression site. *Molecular and Cellular Biology*, **9** (9): 4022-4025.

Department of Molecular Biology, University of Brussels, 67 rue des Chevaux, B-1640 Rhode-Saint-Genèse, Belgium. (Correspondence to E. Pays.)

6019 **Davis, C.E., Colmerauer, M.E.M., Kim, C.-H., Matthews, B. and Guiney, D.G., 1989.** *myc*-related proteins and DNA

sequences in *Trypanosoma brucei*. *Microbial Pathogenesis*, **7**
(1): 45-53.

Departments of Pathology (Davis, Colmerauer), Medicine (Kim, Matthews, Guiney) and Center for Molecular Genetics (Davis, Guiney), University of California, San Diego, CA 92103, USA. (Correspondence to Davis at UCSD Medical Center H811F, 225 Dickinson Street, San Diego, CA 92103, USA.)

- 6020 **Doering, T.L., Masterson, W.J., Englund, P.T. and Hart, G.W., 1989.** Biosynthesis of the glycosyl phosphatidylinositol membrane anchor of the trypanosome variant surface glycoprotein: origin of the non-acetylated glucosamine. [*T. brucei*.] *Journal of Biological Chemistry*, **264** (19): 11168-11173.

Doering, Englund, Hart: Department of Biological Chemistry, Johns Hopkins University School of Medicine, Baltimore, MD 21205, USA; Masterson: Biochemistry Department, Medical Sciences Institute, University of Dundee, Dundee DD1 4HN, UK.

- 6021 **Gottlieb, M., 1989.** The surface membrane 3'-nucleotidase/nuclease of trypanosomatid protozoa. [Incl. *T. b. rhodesiense*.] *Parasitology Today*, **5** (8): 257-260.

Department of Immunology and Infectious Diseases, Johns Hopkins University School of Hygiene and Public Health, 615 N. Wolfe Street, Baltimore, MD 21205, USA.

- 6022 **Greef, C. de, Imberechts, H., Matthyssens, G., Meirvenne, N. van and Hamers, R., 1989.** A gene expressed only in serum-resistant variants of *Trypanosoma brucei rhodesiense*. *Molecular and Biochemical Parasitology*, **36** (2): 169-176.

Greef, Imberechts, Hamers: Instituut voor Moleculaire Biologie, Vrije Universiteit Brussel, Paardenstraat 65, B-1640 St Genesius Rode, Belgium; Matthyssens: Plant Genetic Systems, Ghent, Belgium; Meirvenne: Instituut voor Tropische Geneeskunde Prins Leopold, Laboratorium Serologie, Antwerp, Belgium.

- 6023 **Grondal, E.J.M., Evers, R., Kosubek, K. and Cornelissen, A.W.C.A., 1989.** Characterization of the RNA polymerases of *Trypanosoma brucei*: trypanosomal mRNAs are composed of transcripts derived from both RNA polymerase II and III. *EMBO Journal*, **8** (11): 3383-3389.

Max-Planck-Institut für Biologie, Molecular Parasitology Unit, Spemannstrasse 34, 7400 Tübingen, Federal Republic of Germany.

- 6024 **Hide, G., Gray, A., Harrison, C.M. and Tait, A., 1989.** Identification of an epidermal growth factor receptor homologue in trypanosomes. [*T. brucei*.] *Molecular and Biochemical Parasitology*, **36** (1): 51-59.

Wellcome Unit of Molecular Parasitology, Department of Veterinary Parasitology, University of Glasgow, Bearsden Road, Glasgow G61 1QH, UK.

- 6025 **Hsu, M.P., Muhich, M.L. and Boothroyd, J.C., 1989.** A developmentally regulated gene of trypanosomes encodes a homologue of rat protein-disulfide isomerase and phosphoinositol-phospholipase C. [*T. brucei*.] *Biochemistry*, **28** (15): 6440-6446.

Department of Microbiology, University of Iowa, Iowa City, IA 52242, USA; Division of Chemistry 147-75, California Institute of Technology, Pasadena, CA 91125, USA; Department of Microbiology and Immunology, Stanford University School of Medicine, Stanford, CA 94305-5402, USA. (Correspondence to Boothroyd.)

- 6026 **Jess, W., Hammer, A. and Cornelissen, A.W.C.A., 1989.** Complete sequence of the gene encoding the largest subunit of RNA polymerase I of *Trypanosoma brucei*. *FEBS Letters*, **249** (1): 123-128.

Max-Planck-Institut für Biologie, Molecular Parasitology Unit, Spemannstrasse 34, 7400 Tübingen, Federal Republic of Germany. (Correspondence to Cornelissen.)

- 6027 **Kiira, J.K. and Njogu, R.M., 1989.** Evidence for glycerol 3-phosphate:glucose transphosphorylase activity in bloodstream *Trypanosoma brucei brucei*. *International Journal of Biochemistry*, **21** (8): 839-845.

Department of Biochemistry, University of Nairobi, P.O. Box 30197, Nairobi, Kenya.

- 6028 **Kooy, R.F., Hirumi, H., Moloo, S.K., Nantulya, V.M., Dukes, P., Linden P.M. van der, Duijndam, W.A.L., Janse, C.J. and Overdulve, J.P., 1989.** Evidence for diploidy in metacyclic forms of African trypanosomes. [*T. b. brucei*, *T. simiae*, *T. congolense*, *T. vivax*.] *Proceedings of the National Academy of Sciences of the United States of America*, **86** (14): 5469-5472.

Kooy, Overdulve: Institute of Infectious Diseases and Immunology, Department of Tropical Veterinary Medicine and Protozoology, University of Utrecht, P.O. Box 80.165, 3508 TD Utrecht, Netherlands; Hirumi, Moloo, Nantulya: ILRAD, P.O. Box 30709, Nairobi, Kenya; Dukes: TRL, Department of Veterinary Medicine, Langford House, Langford, Bristol BS18 7DU, UK; Linden: Department of Radiotherapy, University Hospital Utrecht, Heidelberglaan 100, 3584 CX Utrecht, Netherlands; Duijndam: Department of Cytochemistry and Cytometry, University of Leiden, Wassenaarseweg 72, 2333 AL Leiden, Netherlands; Janse: Department of Parasitology, University of Leiden, P.O. Box 9605, 2300 RC Leiden, Netherlands.

- 6029 **Krakow, J.L., Doering, T.L., Masterson, W.J., Hart, G.W. and Englund, P.T., 1989.** A glycolipid from *Trypanosoma brucei* related to the variant surface glycoprotein membrane anchor. *Molecular and Biochemical Parasitology*, **36** (3): 263-270.

Krakow: Department of Pharmaceutical Chemistry, School of Pharmacy, University of California, San Francisco, CA 94143, USA; Doering, Hart, Englund: Department of Biological Chemistry, Johns Hopkins University School of Medicine, Baltimore, MD 21205, USA; Masterson: Biochemistry Department, Medical Sciences Institute, University of Dundee, Dundee DD1 4HN, UK.

- 6030 **Laird, P.W., 1989.** *Trans* splicing in trypanosomes - archaism or adaptation? [*T. brucei.*] *Trends in Genetics*, **5** (7): 204-208.

Division of Molecular Genetics H-4, Netherlands Cancer Institute, Plesmanlaan 121, 1066 CX Amsterdam, Netherlands.

- 6031 **Markos, A., Blahusková, A., Kalous, M., Bysková, E., Byska, K. and Nohynková, E., 1989.** Metabolic differentiation of bloodstream forms of *Trypanosoma brucei brucei* into procyclic forms. Effect of hydroxyurea, arabinosyl adenine, and serum omission. *Folia Parasitologica*, **36** (3): 225-238.

Department of Physiology and Developmental Biology, Faculty of Sciences, Charles University, Prague, Czechoslovakia; *ibid.*; *ibid.*; *ibid.*; *ibid.*; Postgraduate Medical Institute, Prague, Czechoslovakia.

- 6032 **Mellors, A. and Samad, A., 1989.** The acquisition of lipids by African trypanosomes. (Review.) *Parasitology Today*, **5** (8): 239-244.

Department of Chemistry and Biochemistry (Mellors) and Department of Biomedical Sciences, Ontario Veterinary College (Samad), University of Guelph, Guelph, Ontario N1G 2W1, Canada.

- 6033 **Michels, P.A.M., 1989.** The glycosome of trypanosomes: properties and biogenesis of a microbody. (Review.) *Experimental Parasitology*, **69** (3): 310-315.

Research Unit for Tropical Diseases, International Institute of Cellular and Molecular Pathology, Avenue Hippocrate 74, B-1200 Brussels, Belgium.

- 6034 **Parsons, M. and Smith, J.M., 1989.** Trypanosome glycosomal protein P60 is homologous to phosphoenolpyruvate

carboxykinase (ATP). *Nucleic Acids Research*, **17** (15):
6411.

Seattle Biomedical Research Institute, 4 Nickerson Street, Seattle, WA 98109, USA; Department of Pathobiology SC38, University of Washington, Seattle, WA 98195, USA.

- 6035 **Patzelt, E., Perry K.L. and Agabian, N., 1989.** Mapping of branch sites in *trans*-spliced pre-mRNAs of *Trypanosoma brucei*. *Molecular and Cellular Biology*, **9** (10): 4291-4297.

Bender+CO, Ges mbH, Vienna, Austria; Intercampus Program, Molecular Parasitology, University of California, San Francisco and Berkeley, Laurel Heights Campus, Suite 150, Box 1204, San Francisco, CA 94143-1204, USA; *ibid.* (Correspondence to Agabian.)

- 6036 **Pays, E., Coquelet, H., Pays, A., Tebabi, P. and Steinert, M., 1989.** *Trypanosoma brucei*: posttranscriptional control of the variable surface glycoprotein gene expression site. *Molecular and Cellular Biology*, **9** (9): 4018-4021.

Department of Molecular Biology, University of Brussels, 67 rue des Chevaux, B-1640 Rhode-Saint-Genèse, Belgium.

- 6037 **Pearson, T.W. and Jenni, L., 1989.** Detection of hybrid phenotypes in African trypanosomes by high resolution two-dimensional gel electrophoresis. [*T. b. brucei*, *T. b. gambiense*.] *Parasitology Research*, **76** (1): 63-67.

Department of Biochemistry and Microbiology, University of Victoria, Victoria, BC, Canada V8W 2Y2; Swiss Tropical Institute, Socinstrasse 57, CH-4051 Basel, Switzerland.

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Department of Biological Chemistry and Molecular Pharmacology, Harvard Medical School, Boston, MA 02115, USA; Johns Hopkins School of Medicine, Baltimore, MD 21205, USA. (Correspondence to Englund.)

- 6039 **Schlaepfli, K., Deflorin, J. and Seebeck, T., 1989.** The major component of the paraflagellar rod of *Trypanosoma brucei* is a helical protein that is encoded by two identical, tandemly linked genes. *Journal of Cell Biology*, **109** (4): 1695-1709.

Institut für Allgemeine Mikrobiologie, CH-3012 Bern,
Switzerland.

- 6040 **Schlaeppli, K. and Seebeck, T., 1989.** A silent open reading frame of *Trypanosoma brucei* coding for a protein which shares epitopes with the major structural protein of the paraflagellar rod. *Nucleic Acids Research*, **17** (13): 5399.

Institut für Allgemeine Mikrobiologie, Universität Bern, Baltzerstrasse 4, CH-3012 Bern, Switzerland.
(Correspondence to Seebeck.)

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Schneider: Biozentrum, Klingelbergstrasse 70, CH-4056 Bern, Switzerland; Institut für Allgemeine Mikrobiologie (Hemphill, Seebeck) and Zoologisches Institut (Wyler), Universität Bern, CH-3012 Bern, Switzerland. (Correspondence to Seebeck.)

- 6042 **Shapiro, S.Z. and Webster, P., 1989.** Coated vesicles from the protozoan parasite *Trypanosoma brucei*: purification and characterization. *Journal of Protozoology*, **36** (4): 344-349.

Department of Veterinary Pathobiology, University of Illinois, 2001 South Lincoln Avenue, Urbana, IL 61801, USA; ILRAD, P.O. Box 30709, Nairobi, Kenya.

- 6043 **Stuart, K., 1989.** Trypanosomatids: mitochondrial RNA editing. (Review.) [Incl. *T. brucei*.] *Experimental Parasitology*, **68** (4): 486-490.

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