



Mantophasmatodea Now in South Africa

THE DRAMATIC DISCOVERY OF EXTANT representatives of the new insect order Mantophasmatodea in Namibia and Tanzania ("Mantophasmatodea: a new insect order with extant members in the Afrotropics," Reports, K.-D. Klass *et al.*, 24 May, p. 1456; "Mantophasmatodea: a new insect order?," E. Tilgner, Technical Comments, 2 Aug., and "Response," K.-D. Klass, www.sciencemag.org/cgi/content/full/297/5582/731a) galvanized entomologists into searching for this group in other parts of the world.

Two of us (M.D.P. and J.F.C.) had previously noted unusual orthopteroid insects in the semiarid Succulent Karoo (part of the Karoo-Namib region) of South Africa. Their identity became apparent after the description of Mantophasmatodea was published by Klass *et al.* A search for additional material in South African Museums procured 29 pinned specimens, collected in South Africa (1890 to 1994) from 13 localities (one pair has been preserved in copula).

Males were on average smaller (body length 12.8 ± 2.22 mm) and more slender than females (body length 16.9 ± 1.79 mm). Interestingly, a specimen from a series of five individuals collected in 1890 bears the label "*Ograbiesia ferox* n.g. & sp. P." in Louis Albert Péringuey's (director of the South African Museum, 1906–24) handwriting. This manuscript name was, however, never published. Field collecting has shown that Mantophasmatodea are abundant in the Western Cape Province of South Africa, where they hatch and develop during the wet winter months, reaching maturity in spring. Laboratory colonies are providing details of the life history, mating (see figure), and egg-laying behavior. Specimens from museum and field collections are currently known from 25 localities (see figure), some of which are 600 km apart; they appear to represent at least three species.

Our records show that Mantophasmatodea are widespread within the Cape Faunal Zone (1) [the phytogeographical equivalent being the combined Karoo-Namib region and Cape Floral Kingdom (2)]. The exceptional species richness and endemism of many plant (2) and animal groups (3) of this globally im-

portant biodiversity hotspot (4) are attributed to the unique combination of ecological, geological, and climatic variables of the area. The current distribution of the Mantophasmatodea is centered within the Cape Faunal Zone, with the single preserved Tanzanian specimen representing a geographical outlier.

Both putative sister taxa of the Mantophasmatodea (Grylloblattodea and Phasmatodea) are small orders. Moreover, the Mantophasmatodea had, until recently, escaped detection. We therefore suggest that the extant Mantophasmatodea are likely to be a small, relictual order, with a restricted global distribution. Further examination of the unusual, gracile Western Cape species will probably reveal deeper taxonomic diversification within the order [only a single family, Mantophasmatidae, is currently recognized (see Klass *et al.*)]. A survey of the systematics, distribution, ecology, and conservation of South African species is now under way.

MIKE D. PICKER,^{1*} JONATHAN F. COLVILLE,¹

SIMON VAN NOORT²

¹Zoology Department, University of Cape Town, Rondebosch 7700, South Africa. ²Natural History Division, South African Museum (Iziko Museums of Cape Town), Cape Town 8000, South Africa.

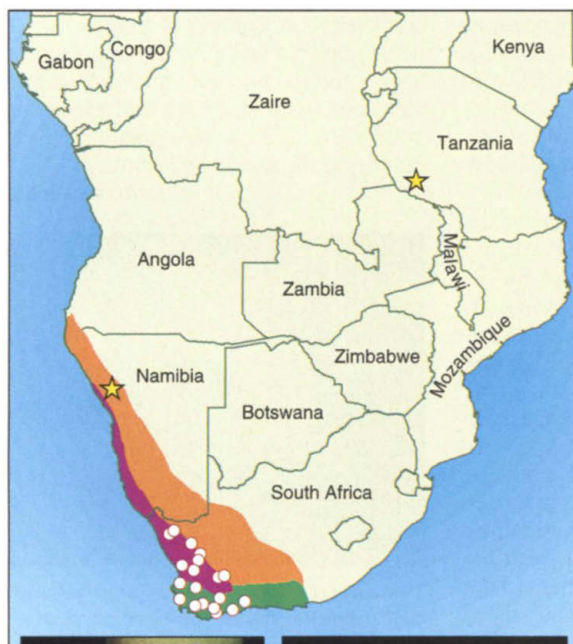
*To whom correspondence should be addressed. E-mail: mpicker@botzoo.uct.ac.za

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Mycobacterium leprae and Demyelination

RAMBUKKANA ET AL., USING AN IN VITRO NERVE tissue culture model, report that *Mycobacterium leprae* attachment to myelinated Schwann cells induces rapid demyelination ("Contact-dependent demyelination by *Mycobacterium leprae* in the absence of immune cells," Reports, 3 May, p. 927). This process of nerve damage was mediated, at least in part, by the *M. leprae*-specific cell wall constituent phenolic glycolipid-1. Second, in vivo intraneural injection of *M. leprae*



Current global distribution of Mantophasmatodea. New records from South Africa (○) are located in the Cape Floral Kingdom (green) and Succulent Karoo (pink). Previous records (☆) are restricted to Namibia (Karoo-Namib zone, orange) and Tanzania. (Inset) Two of the new species of Mantophasmatodea from South Africa (nymph of gracile species at left, mating pair of Karoo species at right).

organisms or cell walls in *Rag-1* knockout mice, which lack mature T and B cells, led to significant myelin breakdown early during infection, comparable to the level observed in wild-type mice. From this, they conclude that *M. leprae*-induced demyelination can occur in the absence of immune cells.

These findings, however, do not explain two major clinical observations in leprosy. First, it is well known that nerve damage in leprosy occurs particularly during (upgrading) type-1 reactions. Type-1 leprosy reactions are idiopathic episodes of strongly increased inflammation and cell-mediated immune reactivity and are often accompanied by acute inflammation of peripheral nerves. The latter complication frequently leads to extensive and irreversible nerve damage. The treatment of choice is rapid administration of immunosuppressive drugs to prevent further nerve damage (1).

Second, although patients with lepromatous leprosy display demyelination and nerve damage, these processes are chronic and relatively slow, despite the presence of high bacillary indices. Thus, in the absence of an adequate cellular immune response, symptomatic neurodegeneration progresses relatively slowly, despite the abundant presence of *M. leprae* organisms.

These combined clinical and epidemiological data suggest that inflammatory immune reactions play an important role in leprosy nerve damage and that the mere presence of *M. leprae* itself does not explain the full phenotype of leprosy nerve damage. Indeed, inflammatory responses seem to be needed for the complete manifestation of demyelination and associated neurological symptoms in other neurodegenerative diseases, as Rambukkana *et al.* point out. In my view, this likely applies also to leprosy: During a first and early phase of *M. leprae*-specific targeting of peripheral nerves, contact-dependent myelin breakdown takes place, and this may continue to progress further. During episodes of enhanced immunity and inflammation, acute and extensive additional nerve damage can take place, to which cytokines and immune effector cells contribute (2, 3), although the precise mech-

anisms involved remain to be elucidated.

Thus, in my view, the human immune response plays a significant role in the full manifestation of leprosy neuritis and nerve damage. It is important to emphasize this point, given its implications for the effective treatment and management of nerve damage in leprosy control.

TOM H. M. OTTENHOFF

Department of Immunohematology and Blood Transfusion, Building 1, L3-33, Leiden University Medical Center, Albinusdreef 2, 2333 ZA Leiden, Netherlands. E-mail: t.h.m.ottenhoff@lumc.nl

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Response

IT IS KNOWN THAT ALMOST ALL CLINICAL features of leprosy are associated with immune responses (1), but we know nothing about the early events, preclinical stage, and associated pathology in nerve injury. It is impossible to explain all features of a complex spectral disease like leprosy in one paper. Our report explains one aspect of this complex pathology, the early consequence of *M. leprae* interaction with myelinating Schwann cells.

The key message of our report is that demyelination is an early step in nerve injury after the attachment of *M. leprae* and its components to myelinated Schwann cells (2). It certainly does not explain the immune-mediated clinical features of leprosy; rather, it is a model system to study the molecular basis for early nerve damage. In the absence of immune cells, *M. leprae* attachment to Schwann cell-axon units, the earliest interaction of the invading pathogen (3), results in significant demyelination. Such myelin damage could set forth the foundation to recruit immune cells in later infection. We neither concluded nor stated our finding as the major mechanism of nerve damage in leprosy. In fact, we stated that an immune response is needed for the complete manifestation of nerve damage in leprosy and other neurodegenerative diseases. Because the clinical manifestation of nerve injury, mainly due to inflammatory responses, occurs years after a slow infectious process, it is obvious that other events are occurring before the immune system comes into play. In fact, we know nothing about the early or preclinical stage of leprosy (certainly not after 72 hours postexposure, as in our study).

Thus, in early *M. leprae* infection, mechanism(s) other than immune responses are certainly involved in nerve injury. Because the Schwann cell is a privileged site for *M. leprae* that eludes surveillance by immune cells (3), we concluded that such non-im-

mune-mediated mechanisms may play critical roles in nerve damage in the very early stage of the infectious process, although it may not be able to manifest clinically.

ANURA RAMBUKKANA,^{1*} GEORGE ZANUZZI,²

NIKOS TAPINOS,¹ JAMES L. SALZER²

¹Laboratory of Bacterial Pathogenesis and Immunology, The Rockefeller University, New York, NY 10021, USA. ²Departments of Cell Biology and Neurology, New York University Medical Center, New York, NY 10016, USA.

*To whom correspondence should be addressed. E-mail: rambuka@mail.rockefeller.edu

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Mistaken Identity

MARTIN DELANEY'S REVIEW OF JOHN Crewdson's book *Science Fictions* ("Double jeopardy for Gallo," *Books et al.*, 31 May, p. 1615) is excellent and to the point. However, I am somewhat confused by the photograph picturing Robert Gallo and Luc Montagnier. The accompanying caption identifies Gallo at the left and Montagnier at the right. They are now good friends, but did they really exchange identities?

EDWARD DE MAEYER



The correct caption: Montagnier (left) and Gallo (right) embrace after being honored at a 2000 ceremony in Spain for identifying HIV.

The Ups and Downs of Global Research Centers

HERE, WE PRESENT AN ANALYSIS OF THE changing performance of the largest "research centers" of the world. Of the total 2,790,179 papers listed by the Science Citation Index (SCI) for the period 1996–98, 38.3% have at least one author from the top 40 research centers. This formidable concentration pattern of research output is reinforced in 1999–2001, with 39.6% of the 2,929,977 papers associated with the world's 40 largest research centers.

Here, we define a center as a "greater

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