Blood disease was first reported about 80 years ago from southern Sulawesi (formerly Celebes), Indonesia, where it caused the abandonment of dessert banana plantations being developed on the adjacent Salayar islands. The disease was the subject of extensive investigations by Ernst Gäumann in the early 1920s, who showed that it was caused by a Gram negative bacterium which he named *Pseudomonas celebensis*. Gäumann found the disease widely distributed throughout southern Sulawesi, and observed symptoms in wild *Heliconia* and *Musa* spp. in which it was apparently endemic. It was not found in Java or other islands. A quarantine order restricting movement of banana fruit and vegetation from Sulawesi was probably instrumental in limiting further spread of the disease until 1987, when an outbreak was confirmed in West Java. There are recent, unconfirmed reports from Kalimantan and the northern Maluku islands.

Blood disease is commonly seen in the Pisang Kepok cultivar (ABB/BBB. 'Saba') but other groups are also affected. Symptoms are similar to those of Moko disease in Latin America and vary according to the growth stage of the plant and the route of infection. Fully expanded leaves of plants of all ages show a conspicuous transient yellowing, followed by loss of turgor, desiccation and necrosis. In mature plants, the base of the petiole collapses causing wilted leaves to hang down around the pseudostem. The youngest leaves cease emerging and develop whitish and later necrotic panels in the lamina. Daughter suckers may show general wilting, but infection is not always systemic and healthy suckers are sometimes produced. Internally, vascular bundles exhibit a reddish-brown discoloration which, depending on the mode of infection, may extend throughout the plant or may be confined to the central fruit stem. If kept moist, cut vascular tissues exude droplets of bacterial ooze which can vary in color from white to reddish-brown or black.

There is convincing evidence that infection occurs via inflorescences, and that blood disease is transmitted by insects in a similar way to Moko disease in Latin America. Blackening and shrivelling of male flowers is frequently found in mature plants, and vascular discoloration can be traced into the peduncle and down the fruit stem. Blackening sometimes extends into the lower fruit bunches, but these often appear outwardly healthy. Internally, fruits in all bunches are usually uniformly discolored reddish-brown and rotten. Insect transmission would account for the rapid spread of the disease (over 25km per annum in some areas) since it was first noted in Java. Spread can also occur in infected planting material and the pathogen can probably persist...
in soil or plant debris. Fruits from infected plants may also be a source of infection. Because affected bunches can appear normal, these may be marketed and subsequently discarded by the consumer near backgarden banana plants.

Although there are many similarities in the symptoms and epidemiology of blood and Moko diseases, the causal bacteria show distinctive phenotypic and genetic differences. On first isolation on commonly used culture media, distinctive features of the blood disease bacterium are: production of non-fluidal colonies, which are smaller and develop more slowly than those of *P. solanacearum*; utilization of, and production of acid from, galactose and glycerol, but not glucose and other carbohydrates used to differentiate strains of *P. solanacearum*; and failure to reduce nitrate. Motility has not been observed in any of the isolates examined so far, and attempts to demonstrate intracellular accumulation of poly β-hydroxybutyric acid (a key diagnostic feature of *P. solanacearum*) have given equivocal results. Unlike Moko strains, blood disease isolates are not pathogenic to solanaceous hosts, including tomato, tobacco and *Capsicum*, but symptoms are readily reproduced by mechanical inoculation to the corm or pseudostem of banana plants of all ages. Genetic analyses, by whole genome RFLP groupings, comparison of partial 16s ribosomal DNA sequences and analysis of tRNA consensus primer amplification products, indicate that the blood disease bacterium is closely related to, but distinct from, strains of *P. solanacearum*. The precise taxonomic status of the pathogen requires further study but, as the specific epithet *P. celebensis* is no longer valid in the international bacteriological nomenclature, it is recommended that the trivial name “blood disease bacterium” be used at present to distinguish the pathogen from Moko strains (*P. solanacearum* race 2) which also occur in south east Asia.

Until recently, the distribution of blood disease was very limited, but it is now spreading rapidly in Java and poses a serious threat to neighbouring islands. Plant quarantine regulations, including controls on the movement of fruits, need to be strictly enforced to limit further spread. Within affected areas, sanitation measures developed for Moko are likely to be effective against blood disease, particularly disinfection of cutting tools, field sanitation and selection of disease-free planting materials (including, where possible, avoidance of cultivars with dehiscent male flower bracts, which are considered particularly vulnerable to infection via newly-exposed bract scars). Removal of male flower buds (denavelling) may be effective, but it should be noted that Gäumann reported evidence for infection via both male and female flowers.

Further research is particularly needed on aspects of the ecology and epidemiology of the disease: there is little information on survival or persistence of the pathogen in soil, and nothing is known about the role of insects in transmission of the disease in south east Asia. In his early studies, Gäumann found no resistance to blood disease in over 100 banana varieties, but preliminary experiments by the author suggest that differences in response can be demonstrated following mechanical inoculation.

The Natural Resources Institute is one of INIBAP’s key partners and is carrying out further comparative studies in the UK on the characteristics and mechanisms of pathogenicity of bacteria causing blood, Moko and bugtok diseases and welcomes collaboration with groups working on the epidemiology and control of these diseases. Contact Simon Eden-Green (NRI, Chatham, Kent ME4 4TB, UK; fax 44 1634 880066/77).