Levels of Ag8 in cultural media varied considerably depending on periods of cultivation of bacteria. Additionally, the level of Ag8 varied among strains of B. pseudomallei and B. mallei. Among 61 strains of B. pseudomallei from the museum collection (most of which were isolated in Southeast Asia and northern Australia), three had increased ability to produce Ag8. These strains had been isolated from clinical specimens (blood, abscesses of hospitalized melioidosis patients) in Vietnam. The strains gave results typical of B. pseudomallei species in all routine serologic tests (agglutination test, immunofluorescence assay, immunodiffusion test). In contrast, the B. pseudomallei glanders agent (16 strains from the museum collection) had reduced ability for Ag8 production; ELISA titers of Ag8 were a thousandfold less in culture fluids in these strains.

The ELISA technique not only facilitates diagnosis of disease but also provides a rational basis for selecting strains for vaccine production. It also has considerable utility for studying the pathogenicity of B. pseudomallei.

Natalja P. Khrapova, Nikolay G. Tikhonov, Yelena V. Prokhvatilova
Volgograd Antiplague Research Institute, Volgograd, Russia

References
1. Dodin A. La mélioidose: un problème mondial. Recherche 1976;7(68):574-7.
2. Galimand M, Dodin A. Le point sur la mélioidose dans le monde. Bull Soc Pathol Exot 1982;75,4:375-83.
3. Larionov GM, Pogasiy NI. A methodology for determination of reservoirs of the causative agents of sapronoses. Med Parazitol (Mosk) 1995;4:45-9.
4. Piven NN, Ilyukhin VI. Antigenic structure of Pseudomonas pseudomallei. Zh Microbiol Epidemiol Immunobiol 1981;4:78-82.
5. Kapliev VI, Piven NN, Khrapova NP. Determination of antigens on melioidosis agents with the use of monodonal antibodies at the ultrastructural level. Mikrobiol Z 1992;54,1:85-9.
6. Voller A, Bidwell DE, Bartlett A. Enzymeimmunoassays in diagnostic medicine. Bull World Health Organ 1976;53,1:55-65.

Forging New Perspectives on Disease Surveillance

To the Editor: Recognizing disease emergence as a paradigm uniquely influenced by human activity demands a reevaluation of traditional disease surveillance systems. Part of a surveillance program should be focused on the areas of human activity where disease emergence is most likely to occur. A system that monitors areas known to be involved in disease emergence, such as development projects, agriculture, climate, and refugee movements, may greatly increase our ability to detect and prevent outbreaks.

Large development projects entail ecologic upheavals that can facilitate disease emergence. Construction of a dam in 1987 in Mauritania resulted in increased mosquito breeding sites and in an explosion in the mosquito population; epidemics of Rift Valley fever quickly followed (1). The Southeastern Anatolia Irrigation Project on the Euphrates and Tigris Rivers in Turkey, which will provide irrigation for 1.7 million hectares, has already increased malaria and leishmaniasis cases in the local population (2). The massive Three Gorges Dam Project on the Yangtze River in China, which will create a reservoir 760 km long, must be evaluated for its impact on local disease. With knowledge of endemic diseases and their reservoirs and vectors in these areas of ecologic change, public health workers can anticipate disease epidemics and implement prevention measures.

Incorporating climate predictions into a disease surveillance system would supplement resources in an area known to affect disease emergence. The U.S. Agency for International Development's (USAID) Famine Early Warning System monitors the African continent for two major factors implicated in emergence: temperature and precipitation. Focused on countries at high risk for food shortages and famine, the early warning system is an example of a predictive and preventative surveillance system. Precipitation, temperature, and plant health data from satellites are evaluated as indicators of crop failure. These data are supplemented by information from field representatives who directly observe agricultural production. USAID's system and other global monitoring systems can provide a base level of surveillance that can add to our knowledge of climatologic influence on disease emergence.

The beginning of the Zairian refugee crisis in 1994 illustrates the need for surveillance among refugee populations. In July 1994, 500,000 to 800,000 Rwandan Hutus fled into the North Kivu region of Zaire. In the month between July 14 and August 14, 48,347 of these refugees died,
predominantly of infectious diseases (3). Similar infectious disease epidemics occurred among Kurdish refugees in 1991 (4), Somali refugees in 1992 (5), and Burundian refugees in Rwanda in 1993 (6). In the wake of intense political, social, or physical disruption, the movement of large numbers of people creates ideal conditions for disease outbreaks. When moving into new areas, refugees may not be equipped immunologically against endemic diseases. Most refugee camps are overcrowded, with inadequate sanitation or medical care (7). Refugees often have severe shock or stress, which in combination with poor nutrition, weakens immune defenses. Therefore, refugee populations are extremely vulnerable and should be closely monitored for infectious disease outbreaks. The United Nations High Commissioner for Refugees, the International Rescue Committee, and the Human Rights Watch can provide rapid notification about disease emergence in refugee populations.

Recognizing human involvement as a common critical factor in emergence creates the possibility of refining international disease surveillance. The Centers for Disease Control and Prevention, the World Health Organization, and national governments should foster relationships with organizations already placed to provide disease emergence information in populations and locations implicated in disease emergence. These relationships will increase the scope and efficiency of our efforts to prevent human disease.

Harley Feldbaum
Wesleyan University, Middletown, Connecticut, USA

References
1. Monath T. Arthropod-borne viruses. In: Morse S, editor. Emerging Viruses. New York: Oxford University Press; 1993. p. 144.
2. Aksoy S, Ariturk S, Armstrong MYK, Chang KP, Dortbudak Z, Gottlieb M, et al. The GAP Project in Southeastern Turkey: the potential for emergence of diseases. Emerg Infect Dis 1995;1:62-3.
3. Goma Epidemiology Group. Public health impact of Rwandan refugee crisis: what happened in Goma, Zaire, in July, 1994? Lancet 1995;345:339-44.
4. Yip R, Sharp TW. Acute malnutrition and high childhood mortality related to diarrhea. JAMA 1993;270:587-90.
5. Moore PS, Martin AA, Quenemoen LE, Gessner BD, Ayub YS, Miller DS, et al. Mortality rates in displaced and resident populations of central Somalia during 1992 famine disaster. Lancet 1993;42:913-7.
6. Centers for Disease Control and Prevention. Health status of displaced persons following civil war—Burundi, December 1993–January 1994. MMWR Morb Mortal Wkly Rep 1994;43:701-3.
7. Wilson ME. Travel and the emergence of infectious diseases. Emerg Infect Dis 1995;1:39-46.

Provide a Context for Disease Emergence

To the Editor: When a disease emerges, the trend is to assume that another important and spreading infection is about to devastate humans or animals. Some qualification of the term “emergence” is needed to put emerging diseases into a context for each target species. There may be a cause for alarm and further action or, alternatively, no real change except in knowledge. In Australia, for example, an old disease “emerged” in a new area, while in another, a disease new to the continent emerged. The two disease agents were Ross River virus (which causes fever and polyarthritis in humans) and bluetongue virus (which often causes fatal disease in sheep). Both causative viruses are insect-borne.

Ross River virus is probably of very ancient lineage as an infection transmitted between marsupials and indigenous mosquitoes and was on the Australian continent long before humans first entered (some tens of thousands of years ago). In 1975, the infection was not known to occur in Tasmania, the state separated from the Australian mainland by a wide stretch of sea. In that year, my group detected a clear-cut seroconversion to Ross River virus in sentinel cows in northern Tasmania (1). Cooperative investigations found antibody-positive sera first in marsupials and then in persons who had never left Tasmania. The existing clinical condition of polyarthritis was linked to Ross River virus only after the causative virus was recognized indirectly (2). The marsupial populations of mainland Australia and Tasmania were continuous until the seas rose at the end of the last ice age. Ross River disease had emerged in Tasmania, but only from obscurity.

Bluetongue viruses occur widely in southern and eastern Asia (3). This general picture has been established only since the discovery of bluetongue virus in Australia. Overt disease occurs in sheep on the fringes of the endemic-disease region and in susceptible sheep imported into various countries within the region (3). In