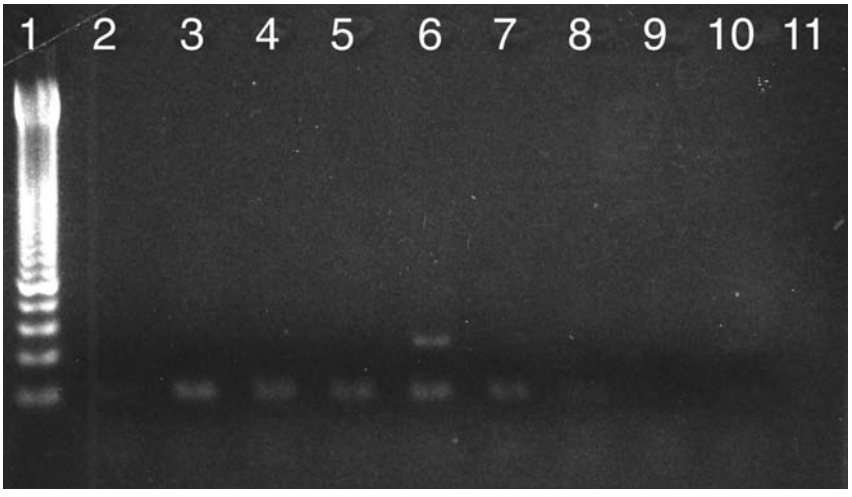


Figure. PCR amplification of a 120-bp fragment of kinetoplastid mitochondrial DNA of *Leishmania* spp. in Egyptian and Nubian mummies. Lane 1, 50-bp ladder; lanes 2–8, mummy samples; lanes 9,10, extraction controls; lane 11, PCR controls. Lane 6 provides a positive amplification product of the expected size.

*Leishmania* DNA–positive samples in the Nubian mummies (12.9%) suggests that leishmaniasis was endemic in Nubia during the Early Christian period and, in light of the data on the ancient Egyptian mummies, probably already several thousand years before. Taken together, our results support the theory that Sudan could have been indeed the original focus of visceral leishmaniasis (*4*).

Our study shows a completely new aspect of molecular paleopathology. The detection of ancient pathogen DNA is not only used to identify a certain disease and gain information on its frequency and evolutionary origin but also to trace back cultural contacts and their role in the transmission and spread of infectious diseases.

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# Tickborne Encephalitis Virus, Northeastern Italy

**To the Editor:** Approximately 3,000 cases of tickborne encephalitis virus (TBEV) disease are registered annually in Europe (*1*). In Italy, indigenous TBEV infection cases have been only sporadically recorded from 1975 through 2001; in addition, serologic investigations in populations at risk in northern Italy have shown only a low prevalence of specific antibodies (0.6%–5%) (*2*,*3*). A surveillance system for TBEV infections was started after autochthonous TBEV was recognized in late summer and fall 2003 in Friuli-Venezia Giulia (FVG), a small region of northeastern Italy with nearly 1 million inhabitants (*4*). Surveillance is based on systematic microbiologic screening of all patients referred to the emergency departments of regional hospitals for suspected community-acquired central nervous system infections or fever and headache with a history of tick bite in the past 6 weeks. Screening for TBEV was performed on sera or cerebrospinal fluid (CSF) by enzyme immunoassay (Enzygnost Anti-TBE virus Ig, Dade Behring Marburg GmbH, Marburg, Germany) and repeated on convalescent-phase sera. Demonstration of specific immunoglobulin M (IgM) in serum or CSF in the acute phase or ≥4-fold rise in serum antibody titer in the convalescent phase was interpreted as an indicator of recent TBEV infection. For surveillance purposes, TBEV infection was defined when hemagglutination inhibition antibody test and neutralization assay by a reference laboratory confirmed ELISA results (*5*). Data were collected at a regional reference center, where cases were classified as possible, probable, and confirmed, according to the new TBEV case definition (*6*).

From July 2003 through November 2005, 20 cases of TBEV infection were detected; their demographic, epidemiologic, and clinical characteristics are given in the Table. Cases occurred throughout the year, with a biphasic peak in June and September–November. A biphasic clinical course was reported in 10 patients. The median period between tick bite and date of referral to hospital was 22 days (range 15–46 days). Seventeen cases were classified as confirmed, 2 as probable, and 1 case could not be classified because symptoms started after tick season (December) (*6*). Two patients were coinfected with *Borrelia burgdorferi*.

The most common symptoms were fever, headache, nausea, vomiting, and myalgia; the most common central nervous system signs were stiff neck, irritability, and limb paresis. Five patients only reported headache and fever without neurologic signs. Lumbar puncture, performed in 15 patients, showed mild pleocytosis with neutrophil predominance in 13 patients, elevated protein level in 14 patients, and normal glucose level in all.

The clinical syndrome was classified, in accordance with Kaiser et al., into febrile form (4 cases), aseptic meningitis (3 cases), encephalitis (2 cases), meningoencephalitis (8 cases), and meningoencephalomyelitis (3 cases) (*7*). None of the patients died, but 3 required respiratory support in the intensive care unit. Outcome was favorable for 9 patients; major neurologic sequelae were observed in 6 and minor sequelae in 5.

During the past 20 years, TBEV has reemerged in several European areas that had been disease free (*1*,*8*). In FVG, which borders disease-endemic areas such as Slovenia and Austria, the first cases of TBEV infection were documented recently (*4*). Several explanations, in addition to the well-established role of climate change, can be proposed (*1*). First, in Slovenia, after the end of the Communist regime, recreational activities increased considerably, with the creation of natural parks and hunting grounds, densely populated with deer, chamois, rodents, foxes, and other wild animals that can easily cross national borders (*9*). Second, after the 1976 earthquake that destroyed a large number of mountain villages in FVG, economic activities were progressively concentrated in the plains of the region, which rapidly increased urbanization of the plains towns. As a consequence, the mountains in the northern part of the region were progressively abandoned by humans and returned to wilderness. A final possible explanation is that TBEV cases were undiagnosed because awareness among local physicians was low; however, this variable likely played a minor role, since a recent serologic survey of persons at high risk (forest rangers) yielded a low positivity ratio (*3*). If even workers at risk had a low seroprevalence, TBEV cases were likely uncommon in the region.

The implementation of a regional active surveillance system allows the highest sensitivity in assessing the epidemiologic features of TBEV infections, which are characterized by highly disease-endemic microfoci in areas free of the problem (*10*). Precisely defining areas where risk is particularly will lead to optimal use of

Table. Demographic, epidemiologic, and clinical data for 20 patients with TBEV infection in Friuli-Venezia Giulia*

| Patient | Sex | Age (y) | Tick bite | Hospitalization date (length of hospitalization [d]) | Definitive diagnosis | Sequelae |
|---|---|---|---|---|---|---|
| 1 | F | 36 | Yes | 2003 Jul 28 (31) | MEM | UL paresis |
| 2 | M | 58 | Yes | 2003 Oct 13 (15) | E | Absent |
| 3 | F | 42 | Yes | 2003 Oct 17 (19) | ME | Absent |
| 4 | F | 27 | No | 2003 Dec 30 (25) | ME | UL paresis, paresis of VII cranial nerve |
| 5 | M | 16 | Yes | 2004 Apr 28 (21) | ME | UL tremors |
| 6 | F | 53 | Yes | 2004 Jun 21 (18) | ME | Diplopia |
| 7 | M | 43 | Yes | 2004 Jul 17 (0) | FF | Absent |
| 8 | M | 62 | Yes | 2004 Oct 10 (10) | ME | UL paresthesia |
| 9 | M | 35 | Yes | 2004 Nov 8 (15) | ME | Absent |
| 10 | F | 77 | Yes | 2004 Nov 22 (0) | FF | Absent |
| 11 | F | 36 | Yes | 2005 May 8 (19) | MEM | UL paresis |
| 12 | M | 12 | Yes | 2005 May 13 (27) | ME | Absent |
| 13 | M | 64 | Yes | 2005 Jun 10 (11) | FF | UL paresthesia, hearing impairment |
| 14 | M | 59 | Yes | 2005 Jun 20 (12) | M | Absent |
| 15 | M | 15 | Yes | 2005 Sep 1 (10) | ME | Absent |
| 16 | F | 39 | Yes | 2005 Sep 8 (8) | M | Absent |
| 17 | M | 70 | Yes | 2005 Sep 16 (53) | MEM | UL paresis, RI, VAP |
| 18 | M | 75 | No | 2005 Oct 18 (10) | FF | UL tremors |
| 19 | M | 20 | No | 2005 Nov 2 (7) | M | UL tremors |
| 20 | M | 61 | Yes | 2005 Nov 26 (13) | E | UL tremors, ataxia, opsoclonus |

*TBEV, tickborne encephalitis virus; MEM, meningoencephalomyelitis; UL, upper limbs; E, encephalitis; ME, meningoencephalitis; FF, febrile form; M, meningitis; RI, respiratory insufficiency; VAP, ventilator-associated pneumonia.

prevention programs and design of educational programs for residents, tourists, and healthcare workers.

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## Alex Langmuir and CDC

**To the Editor:** We were surprised and disappointed by the brevity of your article commemorating the 60th anniversary of the establishment of the Communicable Disease Center (CDC) (*1*). We realize that the accomplishments of the center and its derivative agencies are vast and that to give them full recognition would require far more space in Emerging Infectious Diseases than might be feasible. Nevertheless, your article that appropriately identified Joe Mountin as the administrative "father" of the center omitted any mention of Alex Langmuir, arguably the most influential of the infectious disease leaders over the years. Langmuir's creation and direction of the Epidemic Intelligence Service epitomized CDC's role in infectious diseases. His legacy deserves recognition in any chronicle of CDC, no matter how short.

**Warren Winkelstein, Jr,* and Arthur L. Reingold***

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**In response**: We thank Drs. Winkelstein and Reingold for their comment regarding our article on the 60 years of CDC's progress in the area of infectious diseases. We certainly agree that Dr. Alexander Langmuir has made enormous contributions to this area, which we fully respect, We further agree that a more comprehensive approach would have required far more space than allotted for these types of commentaries. Allow us to emphasize that the omission of Dr. Langmuir and many other outstanding colleagues was not an oversight but an effort on our part to abbreviate an exceptionally long list of these deserving persons. We are looking forward to opportunities to provide a more comprehensive overview in which many of them will be appropriately recognized.

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