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研究報告の概要

バングラデシュ中部でニパウイルス感染が再興しており、40名以上の患者が脳炎を伴う重症症状を呈し、14名が死亡した。ニパウイルスもその近縁のヘンドラウイルスもフルーツバットが自然宿主となり、馬が中間宿主となる。ヘンドラウイルスが初めて種の壁を越えてヒトに感染したのは1994年であり、この時の死亡者は2名であった。ニパウイルスがマレーシアで大流行した際は276名の患者中105名が死亡した。バングラデシュでは2001年と2003年に小規模の流行が確認されてきた。これまでCDCの調査ではニパウイルス抗体は検出していたもののウイルスは分離していなかったために「ニパ様」ウイルスと名づけて来たが、今回はウイルスの分離に成功したため、このウイルスがニパウイルスか、あるいは近縁ウイルスかについて結論がでそうである。疫学的には今回の流行とマレーシアの流行は異なるという。マレーシアでは患者のほとんどが養豚業者であったが、バングラデシュではブタは介在しておらず、患者のほとんどは子供である。現時点では中間宿主の特定には至っておらず、患者はフルーツバットの糞等により直接感染した可能性もある。現時点ではニパウイルスの治療法は存在しないが、ワクチンは開発段階にある。

報告企業の意見	今後の対応
バングラデシュ中部でニパウイルス感染が再興しているとの報告である。	これまで、本製剤によるニパウイルス感染の報告はない。本製剤の製造工程には、平成11年8月30日付医薬発第1047号に沿ったウイルス・プロセスバリデーションによって検証された2つ以上の異なるウイルス除去・不活化工程が含まれていることから、本製剤の安全性は確保されており、特別の対応を必要としないが、今後も情報の収集に努める。

使用上の注意記載状況・その他参考事項等

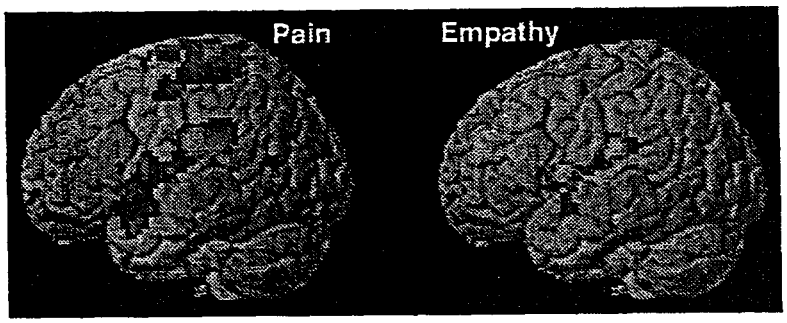
クロスエイトM250
クロスエイトM500
クロスエイトM1000
血液を原料とすることに由来する感染症伝播等
理論的な vCJD 等の伝播のリスク

NEUROSCIENCE

Imaging Studies Show How Brain Thinks About Pain

When you see someone getting hurt, you flinch. And so does your brain. Indeed, when we empathize with another person's pain, we use many of the same brain areas that are activated by our own experience of pain, a new brain-imaging study on page 1157 has shown.

Researcher Tania Singer of the Institute of Neurology at University College London, U.K., and her team set up an experiment using 16 couples who were romantically involved and presumed to be acutely sensitive to each other's pain. Keeping both partners in the same room, they put the female in a magnetic resonance imaging machine and watched her brain while a 1-second electric shock was delivered to the back of either her hand or her partner's. She could not see his face but could see from an indicator which one of them was going to be zapped and whether it would be a weak shock or a sharp, stinging one.



Picture of pain. Empathy for pain mirrors the suffering—but not the physical pain—in the same brain regions.

When the woman received sharp shocks, well-known pain regions in the limbic system were activated, including the anterior cingulate cortex, the insula (which is involved in relaying information from the cortex), the thalamus, and the somatosensory cortices, which relay the physical nature and location of the pain. Many of the same regions were activated in subjects when their partners got the painful shock. But empathy alone failed to activate the somatosensory cortices, for instance. The fact that the same affective brain areas respond to both experienced and imagined pain, claims Singer, is the root of empathy.

Neuropsychiatrist Helen Mayberg of Emory University in Atlanta, Georgia, calls the study "brilliant." Using a "very fundamental system like pain," she says, the researchers have captured both sensory and emotional aspects of the experience and provided new insights on how they interact.

Singer's study is part of a growing body of research exploring mind states—including empathy, imitation, and "theory of mind"—which have in common the creation of an interior representation of what another individual is experiencing. This type of representation has been shown at a cellular level by the discovery, in monkeys, of "mirror neurons": brain cells that are activated both when monkeys observe another individual grasping something or when they are doing the grasping.

Singer says the same sets of neurons that are activated by empathy are also set in motion by the anticipation of pain. That fits with another piece of the pain puzzle, presented by a second imaging study in this issue (see p. 1162), showing that anticipation of pain relief is closely tied to the placebo response.

In this study, headed by Tor Wager of the University of Michigan, Ann Arbor, subjects were given an inert salve that they were told was being tested as an analgesic cream. They were then given a shock or painful heat stimulus on the wrist. Those who showed increased activity in the prefrontal cortex prior to the stimulus also showed the biggest reduction of activity in pain-sensitive brain regions and reported the greatest pain reduction—suggesting that anticipation of pain relief is intimately tied with actual pain reduction. Co-author Richard Davidson of the University of Wisconsin, Madison, says this indicates that cognitive control may be crucial for downregulating pain circuitry. Presumably, he says, more prefrontal activity reflects "the active maintenance of a [mind]set" associated with pain relief.

Mayberg, who has done brain-imaging studies on placebo effects with depressed patients, says the study supports the notion that it may be possible to predict response to medication by looking at the "expectation component" in patients' brain scans.

—CONSTANCE HOLDEN

CREDIT: T. SINGER ET AL.

EMERGING INFECTIOUS DISEASES

Nipah Virus (or a Cousin) Strikes Again

An enigmatic, highly lethal group of viruses has struck again. More than 40 people in central Bangladesh appear to have fallen ill with encephalitis, and 14 have died. Tests point to the Nipah virus, which debuted during a devastating outbreak in Malaysia in 1999. Dozens more cases are under investigation, according to the World Health Organization. The disease has occurred in several clusters, and many of the patients are children, says senior scientist Robert Breiman of the Centre for Health and Population Research in Dhaka.

The Nipah virus and an Australian cousin, Hendra, both naturally infect *Pteropus* fruit bats. Using horses as an intermediate host, Hendra first jumped to humans in 1994, killing two. Nipah made its way to humans in Malaysia after causing a massive outbreak in pigs, killing 105 of its 276 victims (*Science*, 16 April 1999, p. 407). Grouped into a new genus—the *Henipaviruses*—within the Paramyxovirus family, the duo's high mortality and ability to jump species barriers have attracted close attention.

Bangladesh had similar, smaller outbreaks in 2001 and 2003. Because researchers at the U.S. Centers for Disease Control and Prevention (CDC) in Atlanta, Georgia, could detect antibodies against Nipah antigens in patients but could not isolate the virus, they dubbed it "Nipah-like." This time, the virus has been isolated, Breiman says, and CDC studies should soon make clear whether it's Nipah or a close relative.

Epidemiologically, "it's a very different disease than in Malaysia; that's what makes it so fascinating," Breiman says. Most Malaysian victims were pig farmers; in Bangladesh, there has been no pig outbreak, and many of the patients were young boys. Tests in Bangladesh fruit bats have shown that they, too, carry a Nipah-like virus; whether there is an intermediate host is under intense investigation. It's also possible that the victims were exposed directly to infectious bat droppings, Breiman says.

There's no cure for Nipah, but a vaccine is in development. In the January issue of the *Journal of Virology*, French and Malaysian researchers reported that vaccinia viruses, engineered to express either one of two Nipah's surface glycoproteins, protected golden hamsters from a lethal challenge of Nipah. Because antibodies against Nipah and Hendra cross-react, Pasteur Institute senior virologist Vincent Deubel says he's "quite confident" that the vaccine would also protect against the Bangladesh virus.

—MARTIN ENSERINK

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研究報告の概要

捕獲され、販売されている野生のプレーリードッグの間で、*Francisella tularensis* B型による野兎病が大流行した。プレーリードッグと接触のあった1名は*F. tularensis*凝集価が、最近感染したことを示唆している。これらの結果は、プレーリードッグからヒトへ野兎病が感染するという初の科学的根拠を示すもので、エキゾチックペットの取引がヒトの健康に対して潜在的リスクとなる可能性を示した。

報告企業の意見	今後の対応
プレーリードッグからヒトへ野兎病が感染するという初の科学的根拠を示すもので、エキゾチックペットの取引がヒトの健康に対して潜在的リスクとなる可能性を示した報告である。	日本赤十字社は問診時にペット等の動物による刺咬を質問しており、傷が治癒してから3ヶ月間は採血しないとしている。しかし、エキゾチックペット等の飼育の有無については、質問している。エキゾチックペット等からヒトへの感染症について、情報収集に努め、対応を協議する。

使用上の注意記載状況・その他参考事項等

濃厚血小板「日赤」
照射濃厚血小板「日赤」
濃厚血小板 HLA「日赤」
照射濃厚血小板 HLA「日赤」
血液を介するウイルス、細菌、原虫等の感染
vCJD 等の伝播のリスク

First Reported Prairie Dog–to-Human Tularemia Transmission, Texas, 2002

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A tularemia outbreak, caused by *Francisella tularensis* type B, occurred among wild-caught, commercially traded prairie dogs. *F. tularensis* microagglutination titers in one exposed person indicated recent infection. These findings represent the first evidence for prairie-dog-to-human tularemia transmission and demonstrate potential human health risks of the exotic pet trade.

Tularemia is a zoonosis affecting more than 150 wildlife species, including prairie dogs, squirrels, cats, and humans (1–3). Tularemia is caused by the bacterium *Francisella tularensis*, which exists in two main types. Type A is found almost exclusively in North America and is highly virulent in humans. Type B exists throughout North America, Asia, and Europe and is less virulent in humans (4). Tularemia vaccines have been used to protect military and laboratory personnel at high risk for exposure but are not available for the general population (5).

Humans can acquire tularemia through contact with infected animals (2,3,6). Although not previously documented, transmission to humans from prairie dogs is a concern because thousands of wild prairie dogs are captured annually in the United States and sold as exotic pets worldwide (7).

In mid-July 2002, a die-off began among wild-caught, black-tailed prairie dogs (*Cynomys ludovicianus*) (Figure 1) at a commercial exotic pet distributorship in Texas (facility A). On July 29, one of the dead prairie dogs tested positive for *F. tularensis* (8). Hundreds of potentially infected prairie dogs had already been distributed to other states and exported internationally. Epidemiologic and microbiologic investigations were initiated on August 1. We report on the epidemiologic findings; the microbiologic investigation is reported separately (9).

The Study

Animal Investigation

Facility A's purchasing and shipping records were reviewed and the staff interviewed. All involved states and countries were notified of the outbreak, asked to identify the status of prairie dogs from the suspected shipments, and submit tissue samples for testing.

All prairie dogs at facility A, prairie dogs distributed within Texas from facility A since June 2002, and other dead and free-roaming exotic species at facility A were retrieved; live animals were euthanized; and all were tested for *F. tularensis* by direct fluorescence assay (DFA) and culture on cysteine heart agar with 9% chocolatized blood media (9). All recovered isolates were subtyped by using a polymerase chain reaction (PCR) assay (9).

Trappers who supplied prairie dogs to facility A in May and June 2002 were interviewed, and prairie dogs from their respective facilities in Texas and South Dakota were euthanized and tested for tularemia. South Dakota trapping sites suspected to be a potential source of the outbreak were also investigated.

Investigation of facility A on August 2 indicated a variety of exotic species crowded within a 2,500 square foot building. We found 163 remaining prairie dogs in four groups: sick and dying prairie dogs (bin 1), healthy-appearing prairie dogs (bin 2 and cages), prairie dog carcasses (frozen), and escaped prairie dogs roaming free

Figure 1. Black-tailed prairie dogs (*Cynomys ludovicianus*).

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around the facility. The bins were metal, uncovered, 2.5 feet tall and 5 feet in diameter, with 50–100 prairie dogs per bin. In addition, several other exotic animals were found roaming free or dead.

According to shipping records, approximately 3,600 prairie dogs passed through facility A during January through July 2002. In July, an estimated 250 prairie dog deaths occurred compared with approximately 25 deaths over the previous 6 months (Figure 2). On August 1, shipments to and from facility A were halted.

Necropsies on all 163 prairie dogs remaining in facility A indicated clinical signs of oropharyngeal tularemia in all the dead and most of the euthanized sick animals, suggesting transmission through ingestion. Many of the dead animals had been cannibalized. *F. tularensis* was isolated from 61 animals (Table 1). Of these, 60 isolates came from prairie dogs remaining in facility A, including one prairie dog roaming free in the facility, and one isolate came from a privately owned prairie dog purchased from a Texas pet shop supplied by facility A. All of the isolates were identified as type B.

During June through July 2002, more than 1,000 prairie dogs were distributed from facility A to locations in 10 U.S. states and 7 other countries (Table 2). By early August, 100 prairie dogs, those shipped to the Czech Republic, remained unsold: of these, approximately 30 were dead on arrival, 30 were ill, and evidence of cannibalism had been noted within the shipment. All living animals were euthanized.

Of the prairie dogs distributed from facility A to other U.S. states, specimens were received from two prairie dogs sent to Michigan; serum samples from both tested negative for tularemia (Table 1). The Netherlands and Belgium retrieved 4 and 10 prairie dogs, respectively, for serologic testing and culture of tissue samples; all were reported to be negative. The Czech Republic tested six prairie dogs for tularemia: one was positive by isolation of *F. tularensis* in culture, and five were presumptively positive by polymerase chain reaction (PCR). The Czech *F. tularensis* isolate was identified as type B, indistinguishable from the

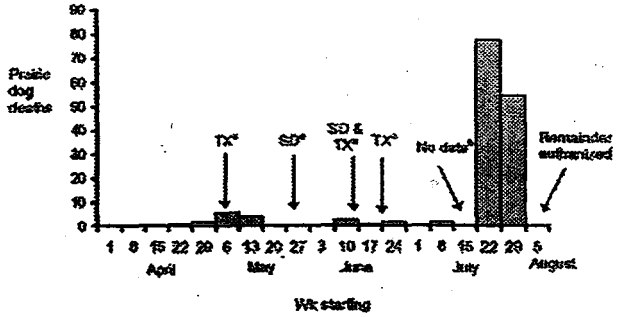

Figure 2. Weekly prairie-dog deaths at facility A, Texas, April–August, 2002. [a]Arrows represent prairie dog shipments arriving at facility A from Texas (TX) and South Dakota (SD). [b]No data are available for the week of July 15, when the outbreak was first noticed by facility A staff.

Texas isolates by restriction fragment length polymorphism analysis (9).

All healthy-appearing prairie dogs in bin 2 and cages, as well as other exotic animals roaming free or found dead in facility A tested negative for tularemia, demonstrating that outbreak propagation required direct contact with infected prairie dogs. Prairie dogs collected from Texas trappers, South Dakota trappers, and trapping sites all tested negative.

Human Investigation

A human case was defined as a fourfold change in serial *F. tularensis* antibody titers from serum samples obtained at least 14 days apart, with at least one titer ≥1:128, in an exposed person. Paired serum samples were tested with an *F. tularensis* microagglutination assay. Anyone who transported, handled, bought, or cleaned the cages of prairie dogs from facility A since June 2002 was considered exposed. Exposed persons in Texas and other U.S. states were given a standardized questionnaire to assess infection risk factors and symptoms during the 2 weeks after their exposure. To enhance case finding, periodic follow-up was maintained with health authorities in involved U.S. states and foreign countries.

Table 1. Diagnostic results for all animals tested in association with tularemia outbreak in prairie dogs, Texas, 2002

Location	Species	No. animals tested	Confirmed positive[a]
Facility A	Prairie dogs	163	61
Retrieved from other Texas facilities	Prairie dogs	7	1
Czech Republic	Prairie dogs	6	1
Trapper facility, TX	Prairie dogs	8	0
Trapper facility, SD	Prairie dogs	2	0
Michigan	Prairie dogs	2	0
Facility A	Chinchilla, sugarglider, hedgehog, red squirrel, eastern chipmunk	16	0
Field investigation, Mellette County, SD	Prairie dogs, deer mice, white-footed mice, grasshopper mice, ground squirrel, jack rabbit, meadow vole	90	0

[a]Prairie dogs were confirmed positive on recovery of an isolate with characteristic growth on cysteine heart agar with 9% chocolatized blood and positive testing of the isolate by direct fluorescent antibody or polymerase chain reaction.

Table 2. Numbers of prairie dogs distributed from facility A to U.S. states and countries in Europe and Asia, June–July, 2002

Locations	No. prairie dogs
United States	
Texas	115
Illinois	26
Ohio	20
Washington	18
Arkansas	12
Nevada	12
West Virginia	12
Michigan	2
Florida	1
Mississippi	1
Europe	
the Netherlands	400
Belgium	250
Czech Republic	100
France	2
Portugal	1
Asia	
Japan	328
Thailand	2

Twenty-two exposed persons were identified in Texas: 5 worked at facility A, 13 worked at other Texas facilities supplied by facility A, 3 worked at a veterinary care center and necropsied a prairie dog originating from facility A, and 1 privately owned an infected prairie dog originating from facility A. In interviews with 20 of 22 exposed persons, 6 (32%) reported recent prairie-dog bites, 7 (37%) ate or drank without handwashing after contact with prairie dogs, and 13 (67%) handled prairie dogs or cleaned cages barehanded. Although gloves and soap were available to employees, none of the involved Texas facilities had formal written policies enforcing proper handwashing, wearing gloves, or prohibiting eating or drinking in animal care areas.

During their exposure interval, 14 of 20 exposed persons interviewed reported having ≥2 nonspecific symptoms that can be consistent with tularemia: headache, sore throat, myalgias, stiff neck, fever, chills, cough, and swollen glands. Initial serologic testing on blood samples obtained 1 week to 2 months after initial exposure from 19 of 22 persons in Texas identified a positive *F. tularensis* titer of 1:128 in a 24-year-old man, who was an animal handler at facility A. All other persons tested negative, and no new positive titers were identified from follow-up samples obtained 1–2 months later from 9 of 19 persons. Except for the animal handler, other symptomatic persons had spontaneous resolution of symptoms or other diagnoses for their symptoms. The animal handler's 1-month follow-up titer persisted at 1:128; however, a fourfold decline in titer, from 1:128 to 1:32, was documented for samples obtained 4 and 6 months after the initial titer, indicating recent exposure to *F. tularensis*. The animal handler had begun working at facility A in June 2002 and had handled dead and dying prairie dogs barehanded. He denied prior potential tularemia exposures, such as hunting, having tick bites, or owning a pet. Additionally, he denied having received a tularemia vaccine, which could have explained the elevated titer. During our investigation, the animal handler reported having an afebrile upper respiratory infection-like illness atypical of tularemia, with sore throat, cough productive of green sputum, and mild chest discomfort but no interruption of work or leisure activities. His symptoms began 12 days after the last prairie dog shipment arrived at facility A and 1 week before the die-off, and they resolved after oral fluoroquinolone therapy.

Health authorities in other states and countries reported no illness in exposed persons. Six months after the outbreak occurred, follow-up calls to health authorities in the involved U.S. states indicated no new human cases. No serologic testing was performed on exposed persons outside of Texas.

Conclusions

Our investigation demonstrated the first evidence that prairie dogs can transmit tularemia to humans. The animal handler's atypical symptoms and unclear route of infection might be because he was exposed to the less virulent subspecies type B. Studies have documented higher rates of *F. tularensis* seropositivity among animal trappers from tularemia-endemic areas, and many of the trappers were asymptomatic (10).

This outbreak highlights health risks to humans who handle wild-caught animals and underscores the speed with which exotic species and virulent pathogens can be transported worldwide (11). A number of public health risks associated with the exotic pet trade were observed at facility A. Prairie dogs were crowded in large bins, allowing unnaturally close contact and propagation of the outbreak through cannibalism. A variety of wild-caught and captive-bred exotic animals were also held in close quarters, providing opportunity for diseases to jump species. This risk for disease transmission between species was heightened because several exotic animals were able to roam free and comingle.

Until recently in the United States, no federal regulations existed to protect humans from the domestic distribution and sale of infected, wild-caught animals; a ban against transport and sale of prairie dogs and certain other rodent species was implemented on June 11, 2003, in response to a monkeypox outbreak in the Midwest (12). Many states forbid capture and sale of native wildlife species, including prairie dogs; however, states that do permit trapping and sale do not have regulations to address the human risk of acquiring zoonoses.

This incident and others, such as transmission to humans of plague from prairie dogs, monkeypox from prairie dogs, and salmonellosis from African pygmy hedgehogs, highlight the importance of developing strategies to reduce human risk from the domestic and international sale of infected, wild-caught animals (13–16). Strategies might include educating the public, standardizing exotic animal husbandry practices, restricting trade to animals bred in captivity, or banning sale of wild-caught animals. As a result of this investigation, Japan banned prairie dog importation as of March 2003. We recommend that the United States and other countries review and strengthen their regulations governing the transport and sale of prairie dogs and other exotic pets.

Acknowledgments

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Dr. Avashia is an internist and pediatrician working as an Epidemic Intelligence Service Officer with the Centers for Disease Control and Prevention assigned to the Texas Department of Health. Her research interests include infectious disease epidemiology.

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