Could Schizophrenia Be a Viral Zoonosis Transmitted From House Cats?

by E. Fuller Torrey and Robert H. Yolken

Abstract

Studies have suggested that some cases of schizophrenia may be caused by viruses. We hypothesize that such cases may be cases of viral zoonosis transmitted primarily from house cats. Epidemiological aspects of schizophrenia and a case-control questionnaire support this hypothesis.


Several lines of research have suggested that some cases of schizophrenia may be caused by viruses. Known cerebral viral infections occasionally present with symptoms of schizophrenia (Torrey 1986), and immunological and virological abnormalities have been reported in some individuals with this disease (Stevens and Hallick 1992; Kirch 1993). There is also evidence that prenatal and perinatal events may affect the later development of schizophrenia (Weinberger 1987; McNeil 1988; Murray et al. 1992), including studies suggesting that prenatal exposure to influenza is a risk factor (Mednick et al. 1988; Barr et al. 1990; O’Callaghan et al. 1991; Sham et al. 1992).

A viral theory may also account for epidemiological aspects of schizophrenia. Past studies using admissions to public psychiatric hospitals—these studies have methodological limitations, but they are the only ones available—suggest that the incidence of admissions began increasing sharply in the 1870s (Stroup and Manderscheid 1988) (figure 1); schizophrenia was the single largest diagnostic group accounting for this increase. The disease occurs more commonly in northern areas of Europe and America and is comparatively rare in most tropical countries (Torrey 1980). A high incidence has been reported, for example, in western Ireland (Torrey et al. 1984), in northern Sweden (Book et al. 1978), among Afro-Caribbeans in England (Harrison et al. 1991), and among the offspring of Dutch women who were pregnant during the 1944-45 famine in Holland when Nazi troops cut off all food supplies (Susser and Lin 1992). Conversely, a very low incidence has been reported among groups such as the Hutterites who live in rural areas of the United States and Canada (Eaton and Weil 1955).

There are also suggestions that urban living and crowding are risk factors (Torrey and Bowler 1990), and an excess winter and spring birth seasonality has been demonstrated in more than 40 studies.
The authors hypothesize that an infectious agent transmitted primarily from house cats could account for these observations of schizophrenia. Transmission of toxoplasmosis from cats to pregnant women has been well documented and can result in the transplacental infection of the fetus; presumably, a virus could be similarly transmitted either during pregnancy or after birth. Exposure to influenza, other concurrent infections, or perinatal trauma resulting in disruption of placental barriers might increase the risk of transmission.

Cats were rarely kept as pets in Europe or America until the 19th century; before then they were generally associated with the devil, often burned on religious holidays, and kept primarily in barns and granaries to control rodents (Mery 1967). During the second half of the 19th century, cats increased rapidly in popularity as pets, the first English cat show took place in London's Crystal Palace in 1871, and the first show in America was in Bangor, Maine, in 1884 (Simpson 1903). The American increase was referred to as a "cat craze" or "cat cult" (Repplier 1892); in advertising it was said that "the 1850s would prove to be the beginning of the boom, for cats would be seen everywhere until the late 1920s" (Lynnelee 1990, p. 25).

House cats, like schizophrenia, are common in northern areas in Europe and America and much less common in tropical countries. Northern European countries are said to be "famous for their love of cats" (Mery 1967, p. 288), and house cats are abundant in western Ireland and northern Sweden. It is possible that exposure to English house cats accounts for the high incidence of schizophrenia among Afro-Caribbean immigrants, especially since the increased incidence is more marked in the second-generation immigrants who are born in England (Harrison et al. 1988; Wessely et al. 1991). Regarding the reported increased incidence of schizophrenia among the offspring of the 1944-45 Dutch famine, it is known that the Dutch people, presumably including pregnant women, ate cats as the famine progressed (Dr. Ezra Susser, personal communication, March 1994).

In the United States, studies since 1880 have consistently shown the New England and Pacific Coast States to have comparatively high prevalences of schizophrenia (Torrey and Bowler 1990). Maine, well known for its coon cats, was a leader in popularizing house cats in America, as were the New England States in general (Winslow 1900, Tibbetts 1903). Large numbers of cats also accompanied miners to the Pacific Coast States during the Gold Rush years (Bretinar 1978). Even today, the New England and Pacific Coast States have a high incidence of cat ownership in comparison with other States (American Veterinary Medical Association 1992). By comparison, the rural Hutterites, among whom the prevalence of schizophrenia is very low (Eaton and Weil 1955), are reported to restrict cats to the barn and almost never keep them as house pets (Dr. John A. Hostetler, personal communication, October 1993).

Urban living might be a risk factor for schizophrenia, because cats are kept as house pets more commonly in cities than in rural areas, where they often remain outdoors or in the barn. Crowding might increase the exposure of pregnant women and young children to house cats by increasing cat-human contacts. The observed excess winter-spring seasonality of schizophrenia births might be due to cats and people spending more time indoors during the cooler and
If schizophrenia is a feline zoonosis, it could be caused by a known feline infectious agent, a yet-to-be-characterized infectious agent, or an environmental factor associated with exposure to cats. Infectious diseases known to be capable of being spread from cats to humans include diseases caused by the following: viruses (rabies, cowpox), bacteria (anthrax, campylobacter infection, cat scratch disease, diphtheria, leptospirosis, listeriosis, Lyme disease, plague, salmonellosis, shigellosis, streptococcal infection, tularemia, and yersinia infection); rickettsiae (Q fever); fungi (blastomycosis, dermatophytoses, sporotrichosis); helminths (cutaneous and visceral larval migrans); cestodes (dipylidiasis, echinococcus); protozoa (amebiasis, cryptosporidiosis, giardiasis, toxoplasmosis, trichomoniasis); and ectoparasites (scabies) (Lappin 1993). Transmission of these agents from cats to humans may occur through direct contact (e.g., a bite in rabies or a scratch in cat scratch disease), exposure to urine or feces (e.g., inhaling sporulated oocysts from a cat litter box in toxoplasmosis), intermediate vectors (e.g., flea bites in plague), or contaminated foods (e.g., milk in Q fever), or through a combination of these contacts. Feline zoonosis usually involves more than one animal reservoir, especially in rural areas where farm animals are common.

In addition to these known causes of feline zoonoses, numerous feline retroviruses have been identified that cause systemic and neurological feline diseases. Human infection with the type C exogenous retrovirus feline leukemia virus has been suspected because of the fact that the virus can infect human cells in vitro, but human transmission has not been proven (Loar 1987). Another feline retrovirus, the feline immunodeficiency virus, has been intensively studied regarding its possible transmission to humans (Sparger 1993). One strain (Hardy Zuckerman-5) of a related feline retrovirus, the feline sarcoma virus, has nucleotide sequence homology to human receptors for the cytokine colony-stimulating factor (CSF-1). This virus contains within its genome coding region a 30-nucleotide CAG triplet repeat similar to the nucleotide triplet repeats associated with Huntington’s disease, fragile X syndrome, myotonic dystrophy, and other human genetic diseases with behavioral manifestations (Ross et al. 1993).

The hypothesis that schizophrenia is a feline viral zoonosis is also compatible with the genetics of schizophrenia, since virtually all viral infections are known to have genetically determined susceptibilities. A possible interaction of viruses and genes might occur if a feline retrovirus became incorporated into the human genome. Alternatively, some familial clustering of schizophrenia that appears to be genetic may in fact be caused by a chronically infected family cat, a type of “typhoid tabby,” as has been documented to account for some clusters of human toxoplasmosis (Teutsch et al. 1979).

To test the hypothesis that schizophrenia is a feline zoonosis, a questionnaire was administered to 165 parents who were members of the National Alliance for the Mentally Ill and whose child had been diagnosed as being seriously mentally ill, mostly with schizophrenia. Under a case-control method the parents were asked to have a friend whose child had not become mentally ill complete an identical questionnaire. A question regarding exposure to cats during pregnancy or childhood (up to the age of 10) was included in a two-page questionnaire covering such subjects as breastfeeding, developmental milestones, coordination, childhood social interaction, family history of serious mental illnesses, rheumatoid arthritis, and multiple sclerosis. The only two questions on which there were differences between the groups were on breastfeeding (mentally ill individuals were more likely to have been breastfed, \( p = 0.002, \chi^2 \) and on exposure to cats. Among the individuals with serious mental illnesses, 84 of the 165 (51%) had had a house cat in childhood versus 65 (38%) of the 165 case controls \( \chi^2 = 0.02, p < 0.01 \).

In summary, it is suggested that house cats may be an important environmental factor in the development of schizophrenia. It should be emphasized that this suggestion is preliminary and that there is no reason at this time for anyone to change behavior toward cats or cat ownership. Future studies should be directed at confirming this epidemiological association. If it is confirmed, additional efforts should be directed at characterizing the agents involved in disease transmission and in developing methods for reducing or eliminating the environmental risk.

References

Veterinary Medical Association, 1992.


An Invitation to Readers

Providing a forum for a lively exchange of ideas ranks high among the Schizophrenia Bulletin's objectives. In the section At Issue, readers are asked to comment on specific controversial subjects that merit wide discussion. But remarks need not be confined to the issues we have identified. At Issue is open to any schizophrenia-related topic that needs airing. It is a place for readers to discuss articles that appear in the Bulletin or elsewhere in the professional literature, to report informally on experiences in the clinic, laboratory, or community, and to share ideas—including those that might seem to be radical notions. We welcome all comments.—The Editors.

Send your remarks to:

At Issue
Research Projects and Publications Branch
National Institute of Mental Health
5600 Fishers Lane, Rm. 18C-06
Rockville, MD 20857

The Authors

E. Fuller Torrey, M.D., is Director, Twin Study Unit, National Institute of Mental Health Neuroscience Center, St. Elizabeths Hospital, Washington, DC. Robert H. Yolken, M.D., is Professor of Pediatrics and Director of the Stanley Neurovirology Laboratory, The Johns Hopkins University School of Medicine, Baltimore, MD.