Evaluation of Torrey and Yolken’s Feline Viral Zoonosis Theory of Schizophrenia

by Royce W. Waltrip II

Abstract

A formal method of evaluation is applied to a theory presented by Drs. Torrey and Yolken, which asserts that cases of viral schizophrenia are due to a zoonosis from house cats. A formal method of theory evaluation is described, and the Torrey and Yolken theory is subjected to analysis by the method. The theory is found to be weak in several areas stemming from inadequate description of both the relevant clinical population and the viral pathogenesis, as well as an incomplete examination of available data bearing on a hypothesized association between schizophrenia and cat ownership. At this point, further work is indicated at the level of theory development before proceeding with research or clinical activity.


Torrey and Yolken (1995, this issue) present a causal theory of schizophrenia. The theory focuses on a subset of individuals who are presumed to have a viral etiology for schizophrenia and states that house cats have played a role in the transmission of the agent that is causing the illness. The task of this article is to examine the strength of Torrey and Yolken’s theory development; a formal method of theory analysis is described first, then the Torrey and Yolken model is evaluated according to the method.

A Method of Theory Analysis

The process of theory development has been formalized by several authors, including the recent exposition of a method of evaluating theory and its relationship to research (Fawcett and Downs 1992). Fawcett and Downs suggest that the propositions or axioms—and the subsequently derived theory and hypotheses and the resulting research—should be specified, hierarchized, and evaluated for the following: (1) significance to the professional discipline and society and to the understanding and precision of predicting a phenomenon; (2) internal consistency, including semantic clarity, consistency, and nonredundancy of concepts and structural consistency of propositions; (3) parsimony—providing a clear precise statement of the theory, as in Occam’s razor, without oversimplification; (4) testability, including empirically observable concepts, measurable propositions, and falsifiable hypotheses; (5) operational adequacy, with a sample that is representative of the population of interest, valid and reliable empirical indicators, and appropriate research procedures and data analysis; (6) empirical adequacy, with congruence between empirical evidence and theoretical claims and also the appropriate consideration of alternative theories; and (7) pragmatic adequacy—evidence of research findings that are related to the problem of interest and the possibility of innovative actions with favorable outcomes resulting from the theory.

Evaluation of the Torrey and Yolken Theory

The hierarchical structure of the Torrey and Yolken theory is based...
on deductive reasoning and is as follows:

Proposition 1: Some cases of schizophrenia may be caused by viruses.

Proposition 2: The rate of schizophrenia is correlated with exposure to house cats.

Proposition 3: The viral cases of schizophrenia are a viral zoonosis from house cats.

Theorem: Some cases of schizophrenia result from exposure to house cats.

Significance. If there were cases of a viral schizophrenia and a common animal such as the house cat were a vector for the causative agent, then this theory would be of considerable importance to both the field and society. The connection between viruses and schizophrenia that is presented in this article draws on concepts that have been summarized in the literature as arguments for the plausibility of a viral causation of schizophrenia—viral infections with schizophrenia-like presentations, immunological and virological abnormalities in schizophrenia patients, increased perinatal events in schizophrenia, geographic variations in the prevalence of schizophrenia, and the season of birth effect (Torrey and Peterson 1976; DeLisi and Crow 1986; Torrey and Kaufmann 1986; DeLisi 1987).

Rather than develop a novel mechanistic argument for a particular schizophrenia subgroup having a specific viral etiological factor, Torrey and Yolken have presented the connection between viruses and schizophrenia at a general level of plausibility. Unfortunately, this presentation compromises the predictability and therefore the significance of the Torrey and Yolken model, because we do not know how to define the subgroup to whom this theory applies. One of the effects is that we cannot estimate the number of cases involved. Also, the lack of definition of these cases compromises another criterion of significance: improving the understanding of the phenomenon of interest. Given that there is an empirical study, the authors have the opportunity to address it in post hoc exploratory analyses of the clinical characteristics of the cat-exposed group. The study does not attempt to clinically characterize cat-exposed cases by post hoc exploratory analysis and actually detracts from the authors' aims by studying a patient cohort that is not well defined diagnostically at the outset.

Internal Consistency. The theory concepts are stated reasonably clearly and consistently throughout the paper. There are no redundant concepts or proposition sets; however, there are problems with internal consistency because of incomplete evaluation of existing data bearing on propositions 2 and 3, thus jeopardizing the deductive chain of reasoning. Proposition 2 involves a coincidence of cat ownership and schizophrenia both geographically and in time. The argument regarding the association of geographic differences in schizophrenia and cat ownership is not compelling because of the lack of good data on the geography of cat ownership outside this country and the lack of large differences in schizophrenia prevalence across cultures in a World Health Organization 10-country study (Jablensky et al. 1992). The authors suggest, because the cat was vilified in Europe until the mid-1800s, that cat ownership was scarce. They also suggest that schizophrenia began to increase at that time. Superstition usually seems to cut both ways, and in addition to fear of cats, there is also considerable evidence of the veneration of cats during the same time period (Dale-Green 1963, pp. 1-67). In the art of the period, cats were also commonly portrayed as companions (Beadle 1977, pp. 75-88; Lynnlee 1990, pp. 39-53), and there are well-known examples of historical figures who were cat owners before the mid-1800s (Beadle 1977, pp. 75-88). There is evidence of cat domestication since 2600 B.C., and tomb paintings since 1600 B.C. depicted cats as pets (Beadle 1977, pp. 61-74). There is paleontological evidence of cats being associated with human settlements as early as 9,000 years ago and of cats being tamed and domesticated in a neolithic settlement in Cyprus by 6000 B.C. (Morris 1987, p. 133).

The authors argue that there was an increase in the incidence of schizophrenia in concert with an increase in cat ownership in this country. To argue the increase in the incidence of schizophrenia, Torrey and Yolken use data on the number of hospitalized cases of mental illness. Figure 1 in Torrey and Yolken, based on data from Stroup and Manderscheid (1988), indicates that there was a great increase in hospitalized cases of mental illness from 1850 to 1950 and implies that the increase was due to the rising popularity of cats in this country. Stroup and Manderscheid (1988) provide data for the number of hospitalized cases to 1980 as well, which we have used to extend the graphical depiction in figure 1 in this article. Referring to figure 1, the number
of hospitalized cases is at a peak in 1950 and falls dramatically thereafter. It seems reasonable to consider that the major force influencing the data in this curve is simply the availability of beds. Hospitals became more plentiful between the 19th century and the 20th (Stroup and Manderscheid 1988); however, bed availability has been dramatically curtailed by deinstitutionalization practices of the past 20 years. Torrey and Yolken would need to provide data supporting the influence of cat ownership as a more powerful force than bed availability on mental hospital bed occupancy in the past 130 years. Torrey and Yolken mention a high rate of schizophrenia in Ireland and note that house cats are “abundant” there. Waddington and Youssef (1994) have recently reported a decreasing rate of schizophrenia in women in rural Ireland, which should be explainable in the Torrey and Yolken model as a change in cat ownership preferences by females in rural Ireland.

A survey of 55,143 respondent households by the Center for Information Management, American Veterinary Medical Association (1992) generated data on the geography of cat ownership, indicating a greater rate of households with cats in New England. Torrey and Yolken point out that fact as coinciding with a higher prevalence of schizophrenia in the same geographic area. The same survey, however, also contains a report of a direct relationship between household income and cat ownership: 24.7 percent of households earning less than $12,500 have cats, while 34 percent of households earning $60,000 or more have cats (American Veterinary Medical Association 1992). According to this information and the Torrey and Yolken theory, schizophrenia should be more prevalent in higher socioeconomic levels, but it has not been proven so (Eaton 1985).

Torrey and Yolken (1995, this issue) also state that “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” (p. 168). This statement raises the question of how this line of reasoning might apply to the suburbs, where house cat ownership is common and families are affluent. The interest in the association between schizophrenia and lower socioeconomic class that began with the work of Faris and Dunham (1939) has led to competing hypotheses of the relative roles of disease processes in schizophrenia versus factors related to the inner city in creating the association. These opposing explanations have been termed the “social drift versus breeder” hypotheses; controversy exists as to the relative importance of each (Silverton and Mednick 1984; Freeman 1994). The interface of a cat vector theory of schizophrenia with the relationship of schizophrenia and social class should be able to either clarify or accommodate controversial epidemiological observations.

Parsimony. The criteria of parsimony is not met due to oversimplification of concepts. As
noted earlier, the patient group cannot be defined in this study, because there is no description of defining features for the hypothesized viral subgroup of patients. Nor is there any mechanistic hypothesis regarding the pathogenesis of the catborne disease.

**Testability.** The testability of the theory is also compromised by the lack of a definition of which cases of schizophrenia are viral and therefore which cases are due to contact with house cats. The authors have included a study that inquired about cat ownership from gestation through age 10 in patients with severe mental illness. Testing the theory outlined in their article would require collecting reliable and valid data on cat ownership from discrete time epochs before the onset of schizophrenia. Presuming there is an effect to be detected, one would get information on the time period of risk of cat exposure. That information would provide a basis for generating a hypothesis regarding the pathogenesis of cat-contact schizophrenia. The authors also have not specified what conditions would constitute falsifiability of the theory. Again, greater elaboration of details of the theory would be helpful in specifying conditions of falsifiability.

**Operational Adequacy.** Torrey and Yolken report a study in their presentation that has several problems in meeting the criteria of operational adequacy. Their sample comprises the parents of patients with severe mental illness who are members of the National Alliance for the Mentally Ill (NAMI). Members of NAMI are a highly informed group regarding etiological theories of schizophrenia; Dr. Torrey has a long and active relationship with NAMI and is a vocal proponent of a viral etiology of schizophrenia. From the presentation, one would have to suppose that the sample in this study was not blind to the study hypotheses and was highly likely to have a response bias in favor of the hypothesis. Also, since the parents selected the controls, any biases that did exist were further compounded. The sampling criteria are also procedurally compromised in that an unspecified (and unascertained) proportion of the study cohort has schizophrenia among a group with severe mental illness. The empirical indicator for this study is a questionnaire that is not described in detail for the reader to ascertain content validity, and there is no description of instrument development, including internal consistency reliability and test-retest reliability. Data analysis is difficult to evaluate, given the sparse description of the instrument used. Bonferroni correction was done for the "number of questions asked" (Torrey and Yolken 1995, this issue, p. 169) requiring a $p < 0.01$, implying a correction for five questions on a two-page questionnaire.

**Empirical Adequacy.** The results reported are consistent with the theoretical claims, implying empirical adequacy. However, the criteria for empirical adequacy are not met, because the authors have not appropriately considered alternative theories. An alternative theory is that cat ownership is related to asociality, which is regarded as a schizophrenia spectrum trait. Therefore, of the two most common pets in our society, people with some degree of asociality might choose the relatively aloof cat over the more interpersonally demanding and affectionate dog. Given the tremendous degree of cat exposure in our society, the authors have not provided appropriate theoretical development for why the rate of schizophrenia is so low. For example, one could propose that there may be a schizophrenogenic agent in something that is used infrequently in cat food.

**Pragmatic Adequacy.** Pragmatic adequacy is not achieved by Torrey and Yolken, because the theory and the associated research do not suggest a clinical course of action or provide a compelling basis for choosing a particular research direction. Weakness in the evidence supporting the theory, and lack of elaboration of the theory are obstacles to the implementation of a plan of action based on Torrey and Yolken’s presentation.

**Summary**

Torrey and Yolken have put forth a theory proposing the house cat as a vector for an unspecified agent that is causing schizophrenia and a study to test the theory. Each of these elements has significant problems in several areas of the relationship of theory to research. The theory has problems in predictability, internal consistency, parsimony, and testability that largely result from (1) lack of sufficient specification of the clinical group and the viral pathogenesis and (2) weak evidence for the association between schizophrenia and cat ownership. The study suffers from several types of sampling bias and empirical connection to the theory itself, due to sampling an unspecified study cohort and using an uncharacterized
instrument. With greater specification of the subgroup of schizophrenia patients who are hypothesized to have a viral etiology, and with specification of aspects of the pathogenesis of the hypothesized virus, a logical way to proceed on the idea of house cats being a vector for an agent causing schizophrenia may become evident.

References


Hare, E. Was insanity on the increase? British Journal of Psychiatry, 142:439-455, 1983.


Waddington, J.L., and Youssef, J.A. Evidence for a gender-specific decline in the rate of schizophrenia in rural Ireland over a 50-year

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Schizophrenia: Questions and Answers

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