

A Critique of the Finnish Adoptive Family Study of Schizophrenia

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This paper evaluates the ongoing Finnish Adoptive Family Study of Schizophrenia. The Tienari, Lahti et al. (1987) study is the most recent attempt to use adoptees as a way of testing the hypothesis that schizophrenia carries a genetic component, and the purpose here is to present what is probably the first in-depth critical analysis of its findings. The published reports of Tienari and associates are the primary focus of analysis, while problems with other schizophrenia adoption studies using similar research designs are also discussed. Because of factors including the selective placement of adoptees, the low variance explained by the two major hypothesized predictor variables for schizophrenia, the invalidity of the schizophrenia spectrum concept, and the failure to find an index schizophrenia rate greater than general population expectations, it is concluded that the Finnish study cannot be regarded as having produced evidence in favor of the genetic theory of schizophrenia.

The genetic theory of schizophrenia has virtually become an accepted fact in psychiatry, and this view has been widely accepted in the related fields as well. This consensus has been established on the basis of results from family, twin, and adoption studies. Here, we will examine the most recent attempt to study adoptees as a way of testing for the operation of genetic factors in schizophrenia. For many, the carefully planned study of Pekka Tienari and associates constitutes the final piece of evidence in favor of the genetic theory, but like all family, twin, and adoption studies, this study contains theoretical and methodological problems deserving serious attention.

A previous paper (Joseph, 1998a) discussed the theoretical fallacy of the use of the so-called "classical twin method" for the study of possible genetic components of schizophrenia and other psychiatric disorders. Dissatisfaction with the twin method by some genetically oriented researchers has led to the

study of adopted individuals, who do not share common genes with the family who raised them, as an ideal way of separating genetic and environmental influences. However, we will see that the theoretical separation of genes and environment is not as clear-cut as it appears at first glance.

Adoption studies are difficult to perform because of the scrupulously guarded records of both adoptees and their biological families. Unusual circumstances, such as the existence of national registers (utilized by the Danish/American schizophrenia adoption researchers) or the intervention of a U.S. senator (who aided the author of the first schizophrenia adoption study; Heston, 1966) are often necessary to enable researchers to have access to needed records.

The Danish/American schizophrenia adoption studies (Kety, Rosenthal, Wender, and Schulsinger, 1968; Kety, Rosenthal, Wender, Schulsinger, and Jacobsen, 1975; Kety et al., 1994; Rosenthal, Wender, Kety, Welner, and Schulsinger, 1971; Wender, Rosenthal, Kety, Schulsinger, and Welner, 1974) have produced the most widely cited evidence in favor of the genetic theory of schizophrenia, and have been the subject of critical analysis by a number of authors (most notably, Boyle, 1990; Cassou, Schiff, and Stewart, 1980; Joseph, in press; Lewontin, Rose, and Kamin, 1984; Lidz, 1976; Lidz and Blatt, 1983; Lidz, Blatt, and Cook, 1981; Pam, 1995). A recent lengthy review of the Danish/American studies (Joseph, 1998b) has demonstrated that the conclusions drawn by their authors are faulty, and that the studies are likely invalidated on the grounds that index adoptees were placed into inferior rearing environments on the basis of a family history of mental illness. In their critical analysis of the schizophrenia adoption studies, the French team of Cassou, Schiff, and Stewart (1980) repeatedly referred to these reports as "studies of abandoned children" ("*Les Études d'Enfants Abandonnés*"). As Pam (1995) has noted, this phrase indicates "the irony of using throwaway kids as proof that schizophrenia is genetically transmitted" (p. 31).

The present discussion centers on the most recent schizophrenia adoption study: the Finnish Adoptive Family Study of Schizophrenia. To the best of my knowledge, there exists no in-depth critical analysis of this study. The design utilized by the lead researcher of this project, Pekka Tienari, is based on the so-called "Adoptees" method, which compares the rate of schizophrenia among the adopted-away children of schizophrenic biological parents to the rate found in a matched control group consisting of the adopted-away children of biological parents with no record of psychiatric hospitalization. The first study of this type was published by Heston (1966), and was followed by the Danish/American reports of Rosenthal and associates (Rosenthal et al., 1968, 1971). Both Heston and Rosenthal claimed to have found a significantly greater concentration of schizophrenia or "schizophrenia-related disorders" among their index adoptees, but there is good reason to doubt these

claims (see Lewontin et al., 1984; Lidz, Blatt, and Cook, 1981; Joseph, in press; Pam, 1995).

Overview of the Finnish Study

Tienari has made the study of schizophrenia his life's work — beginning with his well-known twin study (Tienari, 1963), and culminating in a longitudinal study of adoptees who had been given away by their schizophrenic biological mothers. Tienari is unique among schizophrenia adoption researchers because in addition to genetics, he places significant etiological importance on the psychological environment of the adoptive families whom he has studied. In many ways, the Finnish study is the most comprehensive and best planned of the schizophrenia adoption studies. Therefore, its claim to have shown a genetic component for schizophrenia must be carefully examined.

Tienari has criticized the Danish/American studies as having looked at their participants' family environments "in a very limited manner" (Tienari, Sorri, Lahti, Naarala, Wahlberg, Moring et al., 1985, p. 21), and as having been "misinterpreted as a final proof of genetics, though they only propose suggestions and trends" (Tienari et al., 1994, p. 25). On the other hand, his study is definitely the product of an era in which the question of a genetic component for schizophrenia is considered "closed." Tienari and associates have based the Finnish study on the idea that the genetic component of schizophrenia is real but overemphasized, and that familial environmental factors have been given insufficient attention. "The major goal of the Finnish Adoptive Family Study," wrote Tienari et al., "is to reassess genetic contributions to schizophrenia and to add measures of the adoptive family rearing environment" (Tienari, Sorri et al., 1987, p. 477).

The specific methods of the Finnish study will soon be discussed, but the general approach of every update of the ongoing work has been:

- (1) To present evidence that the adopted-away children of schizophrenic mothers manifest schizophrenia or "psychosis" at a significantly higher rate than their matched controls, thereby establishing the genetic component, then,

- (2) To note that virtually all adoptees diagnosed as schizophrenic have been raised in chaotic or disturbed adoptive families. Thus, "genetically predisposed" children may be more vulnerable when raised in such families, while healthy rearing families may serve to protect them from becoming schizophrenic. Both genetic background and family environment are seen as predictor variables in the etiology of schizophrenia.

The major focus of the present study's analysis of the Finnish adoption study will be to determine whether its findings are consistent with a genetic/familial component to schizophrenia.

Method

The Finnish Adoptive Family Study began obtaining data in the late 1960s. There is no national adoption register in Finland, and the arduous task of collecting the names of hospitalized schizophrenic women netted a total of 9,832 such patients between 1969 and 1972 (Tienari, Sorri et al., 1987). Because this sample was deemed insufficient to provide enough children adopted away at an early age, a second sample was collected, which included the entire psychiatric population of Finland, plus consecutive admissions from 1960–1979. These two samples yielded the names of and relevant information for a total of 19,447 schizophrenic women. Through the use of local population registers, it was determined which of these women had given up a child for adoption. A list of 289 offspring of schizophrenic mothers was obtained in this way. All offspring had been placed in non-relative homes and had gone through formal adoption procedures. From this group, 94 participants were excluded from the study for various reasons, leaving a total of 184 adopted-away offspring of 171 schizophrenic mothers. Some of the children had lived with their biological parents until the age of four years, eleven months.

A matched control group was established on the basis of sex, age of placement, age of adoptive parents (10 year maximum difference), and social status. Adoptees were eligible to be controls if there was no record that their biological mother had been treated for psychosis. About one-half of the biological fathers were identified, and adoptees were excluded if these fathers were found to have been treated for psychosis (Tienari, Sorri et al., 1987). The matching of index and control participants was performed independently by persons not involved in the study.

Joint interviews were performed with the entire family, after which separate interviews were undertaken with the adoptive couple only. All interviews were tape-recorded for the purpose of future blind evaluation. Both parents and adoptees were given a battery of psychological tests, which included the Rorschach and the WAIS. In addition, all index adoptees completed an MMPI. Where possible, the biological parents were also interviewed and tested. The interviewers and testers were blind to the status of the families they worked with.

Tienari and associates outlined five different types of adoptive families, whose status was determined through interviews and testing: (1) "healthy," (2) "mildly disturbed," (3) "neurotic," (4) "rigid, syntonic," and (5) "severely disturbed" (Tienari, Lahti et al., 1987, pp. 40–41). The descriptions of each type of family could be criticized on several grounds (for example, there is no mention of violence, child abuse, alcoholism, etc.), but they are acceptable as a general ranking of family types from healthy to unhealthy. And the cate-

gorizing of different family types is important for the additional reason that, as a longitudinal study, the researchers are able to observe family interaction *before* a child is diagnosed as schizophrenic. This is one of the unique aspects of the study:

The Finnish program will be the first attempt in schizophrenia research to combine the direct adoptive family strategy and the risk research strategy to study adoptive families and adoptees prospectively, beginning prior to the onset of illness in the offspring. (Tienari, 1991, p. 465)

Results and Discussion

At the time of this writing, the Finnish Adoptive Family Study is still a work in progress, and the final results are due to be published in the near future. The most recent update of the study's findings was presented in 1994. Diagnostic results were obtained through blind evaluations and were published in a table (Tienari et al., 1994, p. 21). The results led Tienari et al. to conclude that "there was a clear-cut tendency for the offspring of schizophrenics to have more schizophrenia, functional psychoses in general and serious psychological disturbances . . ." (p. 22).

There are several points worth making about the data provided in the Tienari et al. 1994 paper. First, we see that the researchers have pulled together a "schizophrenia spectrum" in the tradition of the Danish/American team — and unfortunately for many of the same reasons. No justification is provided for why these conditions deserve to be counted with DSM-III-R schizophrenia when comparing index and control groups.¹ In addition to "delusional disorders," this new spectrum includes the schizophreniform diagnosis, which is equivalent to the acute schizophrenia condition determined by the Danish/American team to be unrelated genetically to chronic schizophrenia (Kety, 1985; Kety, Rosenthal, and Wender, 1978). A recent detailed critique of the Danish/American schizophrenia spectrum (Joseph, 1998b) has demonstrated that the spectrum concept is invalid on empirical, statistical, and historical grounds. Therefore, Tienari's spectrum is no more valid than the Danish/American version. In a recent abstract produced by the study, Tienari et al. (1997) reported that the schizophrenia spectrum concept has been enlarged to include "schizo-affective disorder," "psychosis NOS," and the DSM-III-R latent schizophrenia equivalent, "schizotypal personality disorder." Thus, Tienari's concept of "schizophrenia and schizophrenia-related

¹The 1994 report was the first to make diagnoses on the basis of standard "operationalized" criteria (in this case, DSM-III-R).

disorders" has come to resemble its Danish/American antecedent — accompanied by the same erroneous conclusions:

The results reported here for detailed DSM-III-R diagnoses lend support to a hypothesis of genetic vulnerability that is broader than for narrowly defined schizophrenia and includes a broad spectrum of nonschizophrenic psychotic illnesses and schizotypal personality disorder. (Tienari et al., 1997, p. 43)

The publication of a full presentation of the results will be necessary in order to comment further on this new spectrum definition.

The rejection of the Tienari et al. spectrum means that the 19 offspring of biological mothers diagnosed with non-DSM-III-R schizophrenia spectrum disorders (1994, p. 21) cannot be counted when comparing index and control groups. Because the spectrum concept is of dubious validity, the total number of cases of chronic schizophrenia found among the adopted-away children of chronic schizophrenic biological parents is the only legitimate measure of the genetics of schizophrenia. Tienari et al. (1994) have identified six such cases. When compared with controls, the difference is significant (6/136 index vs. 1/186 control: Fisher's Exact Test, one-tailed, $p = .024$). However, we must examine how these six cases compare with the expected population rate before this finding can be generalized to the non-adoptee population. There are six cases of chronic schizophrenia among the 136 adopted-away offspring of schizophrenic biological mothers (4.4%). Based on a population prevalence rate of 0.8–1.0% (Gottesman, 1991; Rosenthal, 1970; Slater and Cowie, 1971), we would expect to find one case of chronic schizophrenia among a random sample of 136 members of the Finnish population. Thus, the comparison between cases of DSM-III-R schizophrenia among adoptees with similarly diagnosed mothers is not statistically significant when compared with the general population expectation (6/136 index vs. 1/136 population: Fisher's Exact Test, one-tailed, $p = .060$, n.s.). One can therefore conclude that, apart from the other problems with this study which shall be discussed shortly, the results pertaining to chronic schizophrenia are not generalizable to the non-adoptee population. In fact, no schizophrenia adoption researcher has ever recognized that index biological relatives must have schizophrenia (or even a "schizophrenia spectrum disorder") in numbers significantly greater than population expectations — yet the necessity of finding a higher rate is essential in order to be able to generalize findings to the non-adoptee population. This was never a problem with the twin studies, where even a 30% MZ concordance rate demonstrated that an identical twin of a schizophrenic is about 30 times more likely to become schizophrenic than a randomly selected member of the population.

Boyle (1990) has argued that if index adoptees are significantly more vulnerable to schizophrenia because of the less "schizophrenogenic" rearing

environments of the more thoroughly screened control adoptive parents, then what may have been found is a significant difference between index and control environments. It cannot be concluded that the genetic theory of schizophrenia has been supported. Boyle noted that "a simple comparison of two groups of biological relatives does not indicate how similar each is to the general population" (p. 144).

The information provided in the 1994 table also demonstrated that there is no significant difference between index and control adoptees receiving no psychiatric diagnosis.² Many genetic theories of schizophrenia emphasize that chronic schizophrenia is only the extreme end of an inherited continuum of psychopathology, yet we find no difference between undiagnosed index and control adoptees. This is even more remarkable when we realize that index adoptees were raised in less healthy environments than controls (see below).

Tienari and associates have concluded that both genes *and* adoptive family rearing environment are "predictor variables" for schizophrenia. Tienari has based this finding on data which he claims show that in seriously disturbed families, index adoptees become schizophrenic more often than control adoptees. Thus,

the results were consistent with the hypothesis that healthy rearing families have possibly protected the vulnerable children, whereas in disturbed rearing families the vulnerable children have been more sensitive to dysfunctional rearing. (Tienari et al., 1994, p. 23)

The correlation between the psychological health of the adoptive family and the rate of index schizophrenia cases was so great, that at least through 1987 Tienari could report that "all adoptees who had been diagnosed either as schizophrenic or paranoid had been reared in seriously disturbed adoptive families" (Tienari, Sorri et al., 1987, p. 482). And in a later report, Tienari et al. wrote that "the individual psychopathology of the adoptive parents, assessed for all subjects in the study, can be used as an environmental predictor variable" (Tienari et al., 1994, p. 23).

As a way of illustrating the problems raised by Tienari's conclusions, we will look briefly at *favism* (G6PD deficiency), which is Gottesman's preferred example of a disease requiring both genetic and environmental factors. Gottesman and Shields described the gene/environment interaction in this condition as follows:

²Non-diagnosed index adoptees: 79/155 [51%] vs. non-diagnosed controls: 106/186 [57%], Fisher's Exact Test, one-tailed, $p = .16$ n.s. Non-diagnosed index adoptees of DSM-III-R mothers: index: 72/136 [53%] vs. non-diagnosed controls: 106/186 [57%], Fisher's Exact Test, one-tailed, $p = .27$ n.s.

Favism, a hemolytic anemia that follows the eating of fava or broadbeans, provides a textbook example of a genotype X environment interaction. Only those persons with the particular X-linked G6PD enzyme variant develop favism, and then, only after eating the bean. Both the gene and the bean are necessary for the disease to appear, neither alone is sufficient, and the disease is *both* a genetic and an environmental one. (1976, p. 447)

Gottesman (1978) has also pointed out, correctly, that a favism adoption study looking at genetically vulnerable children adopted by residents of a “nonbean” community could erroneously conclude that favism lacks a genetic component. It could be said quite accurately that both genetic predisposition and the consumption of fava beans are “predictor variables” of favism — with one or none, there is no favism; with both, there is.

If the principles of this “textbook example” are applied to Tienari’s data, a problem becomes immediately apparent. Tienari has concluded that a genetic predisposition and dysfunctional rearing environments are predictors of schizophrenia, in the same way as “genes and beans” are predictors of favism. The problem arises when we look at index adoptees raised in severely disturbed homes, because in Tienari’s reports they are found to be psychotic only 10–15% of the time. One would think that adoptees exposed to two major theorized predictor variables of schizophrenia would have a much higher rate than that. It can only follow that more than 85% of the prediction variability of schizophrenia remains unknown. What accounts for the fact that about 85% of the biological children of schizophrenics who are raised in severely dysfunctional adoptive homes do not become schizophrenic? Clearly, the possibility exists that a large and unknown factor or set of factors, related to both the biological background and the adoptive family environment of the child, is wholly responsible for an adoptee becoming schizophrenic.

Returning to the problem of favism, what would happen if we were to discover that 85% of people we *believed* to have a genetic predisposition consumed fava beans and did not develop the condition? We would certainly conclude that something in addition to beans and genes contributed to the development of favism. The existence of the genetic factor could only be sustained by the finding of the *physiological* marker for favism in every person who developed the condition. (Such a test does exist.) Likewise, we would have to reconfirm bean consumption in each person found with the disease. With schizophrenia, there are no physiological or genetic markers which distinguish schizophrenics from non-schizophrenics. Therefore, lacking (1) a 100% schizophrenia rate among those having a schizophrenic biological parent and a dysfunctional rearing environment, and lacking (2) biological evidence of schizophrenia; no valid genetic conclusions can be drawn. Lacking physiological markers, the nature of the mysterious 85% predictor

variance must be understood before we can conclude which factors are relevant to schizophrenia.

Evidence of the Selective Placement of Adoptees

Adoption studies are based on the critical theoretical assumption that experimental group adoptees were not systematically placed into rearing environments contributing to a greater prevalence of the trait or condition under study. In other words, index and control adoptees are assumed to have been randomly placed into available adoptive homes. In this section, we will discover that it is unlikely that the Finnish adoption agencies placed prospective adoptees on a random basis, and that the Tienari et al. study is therefore confounded by the reality of selective placement.

In the Finnish Adoptive Family Study, as many as 32% of index adoptees were placed *after* their mother had become psychotic (Tienari, Lahti et al., 1987, p. 44). Because two-thirds were placed before their mothers' psychosis, Tienari could argue that in "most cases . . . it is not likely that information about psychosis could have influenced the placement" (p. 45). This factor was a major methodological problem with the Heston study. Tienari may consider two-thirds of his index adoptees to be "most cases," but this still leaves roughly 35 children who were adopted at a time when their mother's psychiatric status was known by the adoption agency, and probably also by prospective adoptive parents.³

The confounding effect of the adoptive parents' knowledge of a child's biological heritage is evidenced in one of the few case histories presented by the research team. Tienari, Lahti et al. (1987) described the family environment of an adoptee named "P," who was raised in a "severely disturbed" family. We are not told what P's diagnosis was, but the case history suggests that she was not diagnosed as psychotic. Her status, in fact, is of secondary importance to the apparent impact that the knowledge of her mother's psychiatric history had on the adoptive family:

The adoptive father of P, a girl of 19, was a primary school teacher; the mother was a kindergarten teacher. They had been married during the war in 1943 after hardly any contact except by correspondence. The father had threatened to commit suicide unless the mother married him. The parents had been emotionally distant from each other throughout their marriage, devoting themselves to P and four older, biologic children.

³Based on one-third of the 105 index adoptees whose biological mothers had a hospital diagnosis of schizophrenia, through September, 1985 (Tienari, Lahti et al., 1987, p. 40). As of May, 1992, there were 136 index adoptees with schizophrenic biological mothers (Tienari et al., 1994, p. 21), and it was not reported how many additional adoptees, if any, were placed after their mother's schizophrenia diagnosis.

Before P's adoption, the youngest biologic child had started school, and the marriage seemed about to break down. The adoption of P was a conscious effort to save the marriage. Ever since her infancy, P had been the bond between her adoptive parents, understanding both of them and solving their conflicts, while both parents had leaned upon and parentified her. *At the same time, however, both parents attributed to P's biologic background some of the potential madness they had explicitly feared in themselves. They spoke of their strenuous efforts to protect her from becoming crazy, and their relief that they had been able to preserve her sanity for so long [italics added].* (p. 43)

There are several points worth making about P's family. The first is that the parents do not seem like typical candidates for the adoption process; i.e., the possibility exists that their history excluded them from adopting more "normal" children. This means that they probably had to select from a less desirable pool of potential adoptees. It is also possible that this couple intentionally sought out a potentially psychotic child in order to help them resolve their own "explicit fears" of going mad. The fact that they made "strenuous efforts to protect her from becoming crazy" implies that P's family history of schizophrenia was not only a factor in the parent/child relationship, but that it had become a central preoccupation of the family. From a family systems perspective, we might say that P's history was a major factor in her becoming "triangled" into the couple's relationship. A control adoptee, free from the perceived predisposition to madness, would not face this type of potentially "crazy making" family environment.

In fact, there is evidence that P's adoptive parents held views which were widespread among the Finnish for much of the twentieth century. Finland has a long history of eugenics-inspired legislation aimed at curbing the reproduction of people labeled mentally retarded and mentally ill (Hietala, 1996). Eugenic ideas took root in Finland during the 1920s, and a government commission was created in 1926 to look into the desirability of promoting the sterilization of the mentally ill and the mentally retarded — although at that stage, few were calling for mandatory sterilization. By 1935, the Finnish parliament had passed the Sterilization Act, which allowed the *compulsory* sterilization of "idiots," "imbeciles," and the "insane" — which included schizophrenics and manic-depressives. The law stipulated that compulsory sterilizations could be performed on people if there was reason to believe that their "disorders" could be genetically transmitted to their children (Hietala, 1996). The widespread support for this law was evidenced by the fact that only fourteen out of 200 members of parliament voted against it. The year 1950 saw the passage of the Castration Act, which permitted the compulsory castration of criminals, the mentally retarded, and the permanently mentally ill. It was not until the Abortion Act of 1970 that compulsory sterilization was legally abolished in Finland.

The purpose of this discussion is to highlight the conditions under which most of the children in the Finnish Adoptive Family Study were placed. The

adoptees in Tienari's study were born between 1927 and 1979 — and most were therefore placed during a period when eugenic ideas were widespread in Finland and sterilization for eugenic purposes was permitted by law.⁴ A child born to a person with a family history of schizophrenia was seen as a person who should “have never been born,” and as someone who represented a threat to the “purity” of the Finnish gene pool. Those who did make their way into the adoption process were clearly considered to be the carriers of the “hereditary taint” of mental illness. As we have seen, P's parents *expected* her to go mad; for them it was a question of when, not if. Given the prevailing attitudes in Finland, one might ask the same question posed by Kringlen (1987) when discussing Heston's study: What type of parents would want to adopt such a child? As we have seen, up to one-third of Tienari's index adoptees were born to mothers who had already been diagnosed with a psychotic disorder. It is not stated how many of the remaining two-thirds had a known history of mental illness in their biological families, which would likely have also influenced the adoption process.

In an early debate over a proposed sterilization law in Sweden, a farsighted member of parliament described the ultimate logic behind eugenic sterilization. This Socialist party deputy posed the following question to his opponents:

Why shall we only deprive these persons, of no use to society or even for themselves, the ability of reproduction? Is it not even kinder to take their lives? This kind of dubious reasoning will be the outcome of the methods proposed today. (Lindhagen, 1922 quoted in Broberg and Tydén, 1996, p. 104)

This progression was seen in National Socialist Germany, which began with the sterilization of mental patients and culminated in a mass extermination campaign against them, resulting in the murder of approximately 70,000 “mentally ill” and “feeble-minded” persons (Proctor, 1988). To target a group for sterilization carries the implication that its members are not worthy of living, and that the offspring they (“mistakenly”) produce should never have been born. And it is into this type of atmosphere that Finnish children with a known family history of schizophrenia were born.

Manfred Bleuler, who studied the families of 208 of his schizophrenic patients in Switzerland and who was well acquainted with European attitudes toward the “hereditary taint” of schizophrenia, gave the following description of the effect of these attitudes on the afflicted families:

⁴In both Oregon and Denmark, compulsory eugenic sterilization laws aimed at the “mentally ill” and the “feeble-minded” were in force during the period when most of the adoptees in the Heston and Danish/American studies were given up by their natural parents (see Joseph, in press).

If one knows schizophrenics and their families well, it is sometimes a matter for despair to see how much they suffer under the terrible concept of "familial tainting." Like a sinister shadow it darkens the lives of many people and of entire families. The stifling, uncertain fear of coming from an "inferior breed," of carrying within one's self the seeds of something pathological, morbid, and evil (I am speaking in the jargon the afflicted apply to themselves), like a curse that you must pass on to someone else, causes oppressive feeling of inferiority. (Bleuler, 1978, p. 473)

Bleuler's account documents the impact of psychiatric genetic theory and eugenic sterilization laws in strengthening the pre-existing prejudice against families of schizophrenics.

In the Danish/American Adoptees study (Rosenthal et al., 1968), 84% of index parents had given up their child before their first admission to a psychiatric hospital. For this reason, Rosenthal et al. claimed that knowledge of the biological parent's condition could not have played a major role in their study. This figure may seem to have minimized the possibility of selective placement on the basis of a parent's mental health status (in direct contrast to Heston's study, where all of the adopted children were born to institutionalized schizophrenic mothers), but as documented by Mednick and Hutchings (1977), the Danish adoption agencies typically checked the psychiatric records of the *biological families* of potential adoptees, not just the parents.⁵ Therefore, Rosenthal and associates must demonstrate that the existence of mental illness among index biological family members did not create conditions leading to the placement of index adoptees into less qualified adoptive homes. That such conditions likely occurred is based on the fact that — *from either a genetic or environmental perspective* — the biological family of a schizophrenic *or future schizophrenic* would be expected to have more psychiatrically diagnosed members than a control biological family.

⁵The following quotation is taken from Mednick and Hutchings' translation of the 1946–1947 annual report of the Mother's Aid Organization of Copenhagen, which happened to be the largest adoption service in Denmark. As demonstrated by Broberg and Roll-Hansen (1996), these views were typical in Scandinavia during this period:

Before a child is cleared for adoption, it is investigated with respect to health, and an attempt is made to obtain detailed information on the child's family background and to form an impression of its developmental potential. Not only for the adoptive parents, but also for the child itself, these investigations are of great importance for its correct placement. Information is obtained on the child's mother and father; *on whether or not there are serious physical or mental illness in the family background* [italics added]; criminal records are obtained for the biological parents; and in many cases school reports are obtained. By means of personal interview with the mother an impression of her is formed. Where information is uncovered on convicted criminality or on mental retardation, *mental illness, etc. in the family background* [italics added], the case is referred to the Institute of Human Genetics of Copenhagen University, with whom there exists a valuable cooperation for advice on the advisability of adoption. (Mother's Aid Organization for Copenhagen, Copenhagen County and Frederiksberg County. Annual Report for 1946–1947; quoted in Mednick and Hutchings, 1977, p. 163)

This means that in societies (such as Denmark, with its national psychiatric register) where adoption agencies determined placement, in part, on the basis of the mental health status of an adoptee's biological family, the Adoptees method is likely confounded by the reality of selective placement, and therefore constitutes an invalid research model. If adoptees' biological families were similarly checked in Finland, this would constitute a serious problem for Tienari's study as well.

At an earlier point, Tienari had recognized that the adoptive parents' knowledge of the biological mother's psychosis could seriously confound his study. During the period when he was identifying schizophrenic women who had given up a child for adoption, Tienari wrote that separation from the biological mother,

ought to have been complete, in the sense that neither the child nor his adoptive parents should have had any contact with the child's biological mother, *nor should they even have been aware of her psychosis* [italics added]. The group meeting these criteria was still too small. (1975, pp. 34-35)

This passage demonstrates both Tienari's understanding of the significance of the adoptive parents' "awareness," and the likely reason that he was compelled to violate his standard. As in the Danish/American studies, Tienari found it necessary to change his criteria when confronted with the danger of small sample sizes. Critics could sympathize with researchers who have invested enormous amounts of time and effort in their search for the truth. The same critics would be abdicating their responsibility should they also fail to point out the confounding and invalidating nature of such changes in criteria.

The evidence suggests that many of Tienari's index adoptees were placed into inferior homes on the basis of having an actively schizophrenic mother or a family history of psychiatric illness; in other words, a substantial number of index adoptees were not randomly placed into available Finnish adoptive homes. Looking at the distribution of index and control adoptees at the high and low end of Tienari's Family Mental Health Rating scale, an imbalance is clear. According to a table published by Tienari et al. (Tienari, Sorri, Lahti, Naarala, Wahlberg, Rönkkö et al., 1985, p. 231), twice as many index as control adoptees were placed into "severely disturbed" Level 5 families (index 18.2% vs. control 9.1%). On the other end of the scale, twice as many control as index adoptees were placed into "healthy" Level 1 families (control 11.4% vs. index 5.7%). Separately, these differences are not statistically significant for Levels 1 and 5 families. However, if we continue to look at the extreme ends of the rating scale (disregarding Levels 2-4), another picture emerges. If we ask the question "Is there a difference between index and control adoptees placed in either healthy or severely disturbed families?" the result becomes significant. Of the 21 index adoptees placed in Levels 1 or

Level 5 families, 16 are found in Level 5 homes (76%), while of the 18 control adoptees placed in Levels 1 or Level 5 families, only eight are found in Level 5 homes (44%). A Fisher's Exact Test shows this difference to be significant at the .044 level, one-tailed (16/21 index vs. 8/18 control). This distribution is consistent with the operation of selective placement in the Finnish adoption process.⁶

As Tienari has acknowledged, it could be argued that disturbed index children played a role in creating family dysfunction. Kendler (1986) has commented on Tienari's finding that schizophrenic adoptees were raised in disturbed homes:

Most of the adoptive families were evaluated when their adoptees were older than 15. It may have been the disturbed adoptees who affected their adoptive families, not the other way around. Only prospective studies examining families when the adoptees are quite young can hope to disentangle the direction of causality of disturbances in the adoptees and the adoptive family. (pp. 36–37)

Here we have Kendler, who has defended the validity of the twin method on the basis of showing that a child's family rearing environment does not cause "psychopathology," now writing that not only can disturbed parents contribute to the etiology of their child's schizophrenia, but that children can even drive their parents into dysfunction. If in the cases of *adoptees*, Kendler can acknowledge that a particular type of interpersonal relationship can lead to "disturbance," why can he not also recognize this in the case of *twins*? This is a difficult question for someone who has acknowledged on numerous occasions that identical twins are treated much more alike than fraternal twins.

Because the Finnish adoption work is a longitudinal study, Tienari and associates have sometimes had the opportunity to observe family life both before and after an adoptee has had a "schizophrenic break." Yet, there is no indication in the Finnish literature that any of the families' mental health ratings had changed due to life events or any other reason.

It has been argued elsewhere (Joseph, 1998b) that the Danish/American schizophrenia adoption researchers skipped a crucial step in their evaluation of the worthiness of the individual components of their schizophrenia spectrum — which led to the erroneous acceptance of the spectrum's validity. In the case of Tienari, the major error appears to have been *the addition of an unwarranted step* in the determination of the causes of schizophrenia. Tienari

⁶The figures from the Tienari, Sorri, Lahti, Naarala, Wahlberg, Rönkkö et al. 1985 report have been used here due to the fact that Tienari usually combines family types (e.g., 1 and 2, 4 and 5) in tables showing how many index and control adoptees were placed into each type of family.

has failed to see the big picture of the data he has been recording. Instead, he sought to confirm the genetic hypothesis by first concentrating on a narrow set of figures. It has been argued that selective placement of adoptees is a routine practice in the western world (Lewontin et al., 1984) — which suggests that the placement of index adoptees into less healthy adoptive homes, and not the adoptees' genetic background, is largely (or entirely) responsible for the higher rate of psychiatric diagnosis found in the index group. Tienari is also aware of the possible confounding effect of selective placement:

The second major assumption [of adoption studies] is that genetic background and the rearing environment are not correlated, in terms of factors relevant to the development of the specific psychopathology in question. Just as selective placement may invalidate genetic inferences, it may also complicate environmental conclusions. (Tienari, 1992, p. 53).

Let us examine a hypothetical study of non-adoptive populations where non-randomized placement could lead to unwarranted conclusions about the existence of genetic factors. Suppose that we want to study two populations of elementary school children, Group A and Group B. *Group A* consists of the children of affluent, upper middle-class families. These children attend private schools with excellent instruction and small class sizes. Their highly educated parents often help them with their homework and attempt to stimulate their interest in learning. These children are well nourished and receive excellent health care. *Group B* consists of the children of poor, working-class, or unemployed parents. These children attend overcrowded and underfinanced public schools. Their parents are either unavailable, too tired, or academically unable to help them with their schoolwork. These children may also be involved in activities which are helping the family to survive economically. They may be undernourished and the recipients of second-rate or non-existent health care.

Groups A and B will be compared for abilities in reading and mathematics. They are matched for age, sex, and grade level, and are then given achievement tests designed to measure their abilities in the two areas mentioned. After the tests are scored and sorted by group, it is determined that the children from Group A scored significantly higher than Group B children. Using the methodology of Tienari, the significant differences between the two groups would be considered “clear-cut” evidence that Group A children demonstrate an inherited ability to perform better at reading and math than Group B children. *Then* we would study each group's family environment and argue that the child's socioeconomic background “also” predicted achievement test scores. Would such an analysis permit us to conclude that differences in these children's math and reading scores are influenced by both genes and environment?

Clearly, the answer to this question is no, for if we find that both genes and environment predict scores, then we have failed to consider the existence of a third factor confounding the results of the study. By looking at the larger picture of the circumstances of these children's lives, the difference in scores could be completely accounted for by the unfavorable learning environment experienced by Group B children. In this example, it is clear that Group A and B children were not randomly placed into their respective learning environments, but rather were placed as a direct result of the socioeconomic status of their parents. But adoption studies require the random placement of adoptees as a prerequisite to assessing genetic influences for the trait or condition under study. If it is shown that the adopted-away children of schizophrenic mothers are more likely to be placed into chaotic adoptive homes, then this would suggest the same type of environmental placement bias as we saw in the hypothetical example of Groups A and B. Selective placement would now be the confounding variable — just as socioeconomic status was in the study of the two groups of children. The failure to gauge the interaction of all aspects of the Finnish adoption process has led Tienari to the unwarranted conclusion that he has found evidence in favor of the genetic theory of schizophrenia.

Age of Transfer (Abandonment)

Like Rosenthal's Adoptees series, Tienari's study is flawed by the fact that some adoptees lived with their biological parent(s) until a rather late age (up to four years, 11 months [Tienari, Sorri et al., 1987, p. 479]). While Tienari and associates (Tienari, Sorri, Lahti, Naarala, Wahlberg, Moring et al., 1985) have presented evidence that adoptee mental health ratings are not associated with the age of transfer, the trauma of parental abandonment must have been greater for those late-separated adoptees. The fact that a significant number of adoptees spent considerable time with their biological parents is a serious problem with this study (Breggin, 1991; Jacobs, 1994; Lehtonen, 1994). Even Tienari has acknowledged that children adopted away at age four, "were doubtful as to their suitability for a study discriminating between genetics and environment" (Tienari, Sorri et al., 1987, p. 478), and twelve years before that he had written that an adoptee's separation from the biological mother should have occurred "in no case later than the age of 3" (Tienari, 1975, p. 34). Although the number of late-separated adoptees is apparently not large, in adoption studies, every case is important.

Attachment theorists (e.g., Bowlby, 1980) have noted the negative psychological impact of the rupture of the parent/child bond — even in the case of less-than-nurturing parents. One could argue that a child separated from its mother at or near birth would not be greatly damaged in the process, but a child ripped from its caregivers at age one, two, three, or four is truly an

“abandoned child.” The matching of controls on the basis of age-of-transfer cannot eliminate this basic fact.

Report on a Study Using a Rorschach-based “Index of Primitive Thought”

A recent report from the Finnish Adoptive Family Study (Wahlberg et al., 1997) has provided new and interesting information about index and control adoptees. A sample of 58 index adoptees (50 whose mothers were “definite or probable” DSM-III-R schizophrenics, and the remaining eight from “spectrum” mothers), and 96 controls (called “comparison adoptees” in this study) were tested for levels of thought disorder. The comparison group was matched with index participants on the basis of age, sex, SES, and other criteria. None of the comparison adoptees had a biological mother who had been hospitalized for schizophrenia or paranoid psychosis.

Each participant was given a Rorschach test and scored with the Index of Primitive Thought, a system which records certain so-called thought-disordered responses, such as incongruous combination, fabulized combination, contamination, etc. The scoring system formula is as follows: the total number of positive (thought-disordered) responses is divided by the total number of responses (R). The quotient is then multiplied by 100 (Wahlberg et al., 1997). Two examples of how responses would be scored are:

- (1) Participant gives *one* positive response in a 23 response record. (R of 23 reflects the approximate mean for both “normals” and inpatient schizophrenics [Exner, 1995, pp. 167, 207]). $\text{Score} = 1 \div 23 = .043 \times 100 = 4.3$.
- (2) Participant gives *three* positive responses in a 23 response record. $\text{Score} = 3 \div 23 = .13 \times 100 = 13$.

Naturally, a record with zero positive responses would have a score of 0.

The Rorschachs were administered and scored by researchers blind to the status of the adoptees. The results, as reported by Wahlberg et al. (p. 358), showed that there was no difference between the supposedly “high-risk” index adoptees and the comparison group: 41.4% of index and 43.8% of comparison adoptees had a Primitive Thought score of zero, and 36.2% of index adoptees had a score in the 1–10 range, while 30.2% of comparison adoptees fell in this range. At the upper end, 22.4% of index adoptees had a score of 11 or greater, while a remarkable 26% of *controls* scored in this range.

It appears that there is no difference in “primitive thought” between the supposedly high-risk adopted-away children of schizophrenic mothers vs. controls, whose parents had not been hospitalized for schizophrenia. The numbers are very similar in each category and the differences are, of course, statistically non-significant. The authors were surprised “that over one-half (56.2%) of the comparison adoptees showed at least some evidence of this rather extreme form of schizophrenic thought disorder” (Wahlberg et al.,

1997, p. 358). In fact, *both* groups had a surprisingly high number of participants scoring 11 or greater.

These results would seem to suggest that a supposed genetic high risk has no effect on “disordered thought,” but Wahlberg et al. also looked at participants’ Primitive Thought scores as they related to their adoptive parents’ level of “communication deviance.”⁷ The authors produced a graph which showed that as parental communication deviance rises, the “high risk” group becomes more thought disordered, while the comparison group stays the same and drops somewhat at the high communication deviance range. This finding was interpreted as showing that

communication deviance of the adoptive parents does not have any influence on the Index of Primitive Thought scores of the comparison adoptees but does have a strong effect in adoptive families with adoptees at high genetic risk. (Wahlberg et al., 1997, p. 359)

Wahlberg and associates concluded from this finding that the genetic hypothesis was upheld, and that the idea that rearing environment alone could cause schizophrenia had been “refuted” (p. 360). This is a questionable conclusion on several grounds, but the most important is the criteria by which the charted high-risk group was chosen. All participants scoring above zero were compared, meaning that virtually all participants providing *one* or more positive responses were charted together in a single grouping. But this is a highly arbitrary cutoff point, for there is a big difference between someone providing one positive answer and someone providing, for example, four, on this admittedly “fallible vulnerability indicator” (p. 358). It is likely that the group scoring eleven or higher would be far more disturbed than the group scoring one. A score of one would not likely indicate the presence of psychosis; if it did, then this would indicate that 56.2% of *controls* were psychotic. A much better comparison would have been to chart the 38 adoptees who scored 11 or higher. The most important result of this study was a demonstration that there appears to be no difference in levels of “thought disorder” between Tienari’s carefully selected index and control groups.

Summary and Conclusions

The Finnish Adoptive Family Study of Schizophrenia began over a generation ago, and as of this writing, the final results of the study have not yet

⁷This refers to a Rorschach-based scoring method of determining “parental communication deviance,” which has been thought to contribute to the etiology of schizophrenia. See Wynne, Singer, and Toohey (1976).

been published. Unlike the Danish/American adoption series, the Finnish study investigated the family rearing environments of index and control adoptees. Although the research team has designated both genetic background and disturbed family environment as "predictor variables" for schizophrenia, they have not adequately explained why the 85% of adoptees experiencing both of these variables did not become psychotic.

Additionally, about one-third of the index adoptees were placed for adoption after their mothers had been diagnosed as psychotic. It was demonstrated that the biological children of the "mentally ill" were considered "hereditary taint-carriers," whose birth was made possible by virtue of the fact that their parent(s) had not been affected by Finnish eugenic sterilization laws. The case history of "P" demonstrated the confounding effect of being adopted into a family while carrying a perceived predisposition to madness. In addition to the parents, Danish adoption agencies looked at the psychiatric status of a potential adoptee's other biological relatives when making placement decisions (Mednick and Hutchings, 1977). If this occurred in Finland, Tienari's study would be further confounded by the fact that, from either a genetic or environmental perspective, the biological family members of a schizophrenic or *future schizophrenic* would be expected to contain more psychiatrically diagnosed members than the relatives of control biological parents. Some of the adoptees in the Finnish study were placed after their fourth year of life, providing further evidence that this study has not adequately separated rearing environment from biological heritage.

Tienari et al. have created a schizophrenia spectrum whose composition has come to resemble its Danish/American predecessor, but they have failed to demonstrate its validity. Therefore, the Finnish study has been reanalyzed on the basis of the "strict" definition of schizophrenia, for which Tienari has utilized DSM-III-R criteria. While Tienari's index adoptees are significantly more schizophrenic than controls, they do not contain significantly more cases of schizophrenia than the population rate expectation ($p = .060$, n.s.). Therefore, leaving all methodological problems aside, the index/control comparison is not generalizable to the non-adoptee population. As a recent study from the research group has shown, there is no difference in "thought disorder" between index and control adoptees.

Tienari is the creator of the most thoughtful and well-planned schizophrenia adoption study. The Finnish Adoptive Family Study of Schizophrenia has concluded that rearing environment is not only important, but that it is a predictor of which children will become schizophrenic. However, the evidence points to the rejection of the additional finding of a genetic component to schizophrenia.

References

- Blouler, M. (1978). *The schizophrenic disorders: Long-term patient and family disorders*. New Haven: Yale University Press.
- Bowlby, J. (1980). *Attachment and loss: Loss sadness and depression* (Volume III). New York: Basic Books.
- Boyle, M. (1990). *Schizophrenia: A scientific delusion?* New York: Routledge.
- Breggin, P. (1991). *Toxic psychiatry*. New York: St. Martin's Press.
- Broberg, G., and Roll-Hansen, N. (Eds.). (1996). *Eugenics and the welfare state: Sterilization policy in Denmark, Sweden, Norway, and Finland*. East Lansing: Michigan State University Press.
- Broberg, G., and Tydén, M. (1996). Eugenics in Sweden: Efficient care. In G. Broberg and N. Roll-Hansen (Eds.), *Eugenics and the welfare state: Sterilization policy in Denmark, Sweden, Norway, and Finland* (pp. 77-149). East Lansing: Michigan State University Press.
- Cassou, B., Schiff, M., and Stewart, J. (1980). Génétique et schizophrénie: Réévaluation d'un consensus [Genetics and schizophrenia: Reevaluation of a consensus]. *Psychiatrie de l'Enfant*, 23, 87-201.
- Exner, J. (1995). *A Rorschach workbook for the comprehensive system* (fourth edition) Asheville, North Carolina: Rorschach Workshops.
- Gottesman, I. (1991). *Schizophrenia genesis*. New York: W. H. Freeman and Company.
- Gottesman, I. (1978). Schizophrenia and genetics: Where are we? Are you sure? In L. Wynne, R. Cromwell, and S. Matthysse (Eds.), *The nature of schizophrenia: New approaches to research and treatment* (pp. 59-69). New York: John Wiley and Sons.
- Gottesman, I., and Shields, J. (1976). Rejoinder: Toward optimal arousal and away from original din. *Schizophrenia Bulletin*, 2, 447-453.
- Heston, L. (1966). Psychiatric disorders in foster home reared children of schizophrenic mothers. *British Journal of Psychiatry*, 112, 819-825.
- Hietala, M. (1996). From race hygiene to sterilization: The eugenics movement in Finland. In G. Broberg and N. Roll-Hansen (Eds.), *Eugenics and the welfare state: Sterilization policy in Denmark, Sweden, Norway, and Finland* (pp. 195-258). East Lansing: Michigan State University Press.
- Jacobs, D. (1994). Environmental failure-oppression is the only cause of psychopathology. *Journal of Mind and Behavior*, 15, 1-18.
- Joseph, J. (1998a). The equal environment assumption of the classical twin method: A critical analysis. *Journal of Mind and Behavior*, 19, 325-358.
- Joseph, J. (1998b). *Rethinking the genetic theory of schizophrenia: A critical review of schizophrenia twin and adoption studies*. Unpublished manuscript. California School of Professional Psychology, Alameda, California.
- Joseph, J. (in press). The genetic theory of schizophrenia: A critical overview. *Ethical Human Sciences and Services*.
- Kendler, K. (1986). Genetics of schizophrenia. In A. Frances and R. Hales (Eds.), *American psychiatric association annual review* (Volume 5, pp. 25-41). Washington: American Psychiatric Press, Inc.
- Kety, S. (1985). The concept of schizophrenia. In M. Alpert (Ed.), *Controversies in schizophrenia: Changes and constancies — Proceedings of the 74th annual meeting of the psychopathological association, New York, March 1-3, 1984* (pp. 3-11). New York: Guilford Press.
- Kety, S., Rosenthal, D., and Wender, P. (1978). Genetic relationships within the schizophrenia spectrum: Evidence from adoption studies. In R. Spitzer and D. Klein (Eds.), *Critical issues in psychiatric diagnosis* (pp. 213-223). New York: Raven Press.
- Kety, S., Rosenthal, D., Wender, P., and Schulsinger, F. (1968). The types and prevalence of mental illness in the biological and adoptive families of adopted schizophrenics. In D. Rosenthal and S. Kety (Eds.), *The transmission of schizophrenia* (pp. 345-362). New York: Pergamon Press.
- Kety, S., Rosenthal, D., Wender, P., Schulsinger, F., and Jacobsen, B. (1975). Mental illness in the biological and adoptive families of adopted individuals who have become schizophrenic: A preliminary report based on psychiatric interviews. In R. Fieve, D. Rosenthal, and H. Brill (Eds.), *Genetic research in psychiatry* (pp. 147-165). Baltimore: The Johns Hopkins Press.

- Kety, S., Wender, P., Jacobsen, B., Ingraham, L., Jansson, L., Faber, B., and Kinney, D. (1994). Mental illness in the biological and adoptive relatives of schizophrenic adoptees: Replication of the Copenhagen study to the rest of Denmark. *Archives of General Psychiatry*, 51, 442-455.
- Kringlen, E. (1987). Contributions of genetic studies on schizophrenia. In H. Häfner and W. Gattaz (Eds.), *Search for the causes of schizophrenia* (pp. 123-142). New York: Springer Verlag.
- Lehtonen, J. (1994). From dualism to psychobiological interaction: A comment on the study by Tienari and his co-workers. *British Journal of Psychiatry*, 164 (supplement 23), 27-28.
- Lewontin, R., Rose, S., and Kamin, L. (1984). *Not in our genes*. New York: Pantheon.
- Lidz, T. (1976). Commentary on a critical review of recent adoption, twin, and family studies of schizophrenia: Behavioral genetics perspectives. *Schizophrenia Bulletin*, 2, 402-412.
- Lidz, T., and Blatt, S. (1983). Critique of the Danish-American studies of the biological and adoptive relatives of adoptees who became schizophrenic. *American Journal of Psychiatry*, 140, 426-435.
- Lidz, T., Blatt, S., and Cook, B. (1981). Critique of the Danish-American studies of the adopted-away offspring of schizophrenic parents. *American Journal of Psychiatry*, 138, 1063-1068.
- Mednick, S., and Hutchings, B. (1977). Some considerations in the interpretation of the Danish adoption studies in relation to asocial behavior. In S. Mednick and K. Christiansen (Eds.), *Biosocial bases of criminal behavior* (pp. 159-164). New York: Gardner Press.
- Pam, A. (1995). Biological psychiatry: Science or pseudoscience? In C. Ross and A. Pam (Eds.), *Pseudoscience in biological psychiatry: Blaming the body* (pp. 7-84). New York: John Wiley and Sons.
- Proctor, R. (1988). *Racial hygiene: Medicine under the Nazis*. Cambridge: Harvard University Press.
- Rosenthal, D. (1970). *Genetic theory and abnormal behavior*. New York: McGraw-Hill.
- Rosenthal, D., Wender, P., Kety, S., Schulsinger, F., Welner, J., and Østergaard, L. (1968). Schizophrenics' offspring reared in adoptive homes. In D. Rosenthal and S. Kety (Eds.), *The transmission of schizophrenia: Proceedings of the second research conference of the foundations' fund for research in psychiatry, Dorado, Puerto Rico, 26 June to 1 July 1967* (pp. 377-391). New York: Pergamon Press.
- Rosenthal, D., Wender, P., Kety, S., Welner, J., and Schulsinger, F. (1971). The adopted-away offspring of schizophrenics. *American Journal of Psychiatry*, 128, 307-311.
- Slater, E., and Cowie, V. (1971). *The genetics of mental disorders*. London: Oxford University Press.
- Tienari, P. (1963). *Psychiatric illnesses in identical twins*. Copenhagen: Munksgaard.
- Tienari, P. (1975). Schizophrenia in Finnish male twins. *British Journal of Psychiatry Special Publication*, No. 10. M. Lader [Ed.] (pp. 29-35).
- Tienari, P. (1991). Interaction between genetic vulnerability and family environment: The Finnish adoptive family study of schizophrenia. *Acta Psychiatrica Scandinavica*, 84, 460-465.
- Tienari, P. (1992). Implications of adoption studies on schizophrenia. *British Journal of Psychiatry*, 161 (Supplement 18), 52-58.
- Tienari, P., Lahti, I., Sorri, A., Naarala, M., Wahlberg, K., Rönkkö, T., Moring, J., and Wynne, L. (1987). The Finnish adoptive family study of schizophrenia: Possible joint effects of genetic vulnerability and family interaction. In K. Halweg and M. Goldstein (Eds.), *Understanding major mental disorder: The contribution of family interaction research* (pp. 33-54). New York: Family Process Press.
- Tienari, P., Sorri, A., Lahti, I., Naarala, M., Wahlberg, K., Moring, J., and Pohjola, J. (1985). Interaction of genetic and psychosocial factors in schizophrenia. *Acta Psychiatrica Scandinavica* (Supplementum No. 319, 71) 19-30.
- Tienari, P., Sorri, A., Lahti, I., Naarala, M., Wahlberg, K., Moring, J., Pohjola, J., and Wynne, L. (1987). Genetic and psychosocial factors in schizophrenia: The Finnish adoptive family study. *Schizophrenia Bulletin*, 13, 477-484.
- Tienari, P., Sorri, A., Lahti, I., Naarala, M., Wahlberg, K., Rönkkö, T., Pohjola, J., and Moring, J. (1985). The Finnish adoptive family study of schizophrenia. *The Yale Journal of Biology and Medicine*, 58, 227-237.
- Tienari, P., Wynne, L., Läsky, K., Moring, J., Kurki-Suonio, Sorri, A., Lahti, I., Moring, O., and Wahlberg, K. (1997). Schizophrenics and their adopted-away offspring: The Finnish adoptive family study of schizophrenia. *Schizophrenia Research*, 24, [Abstract] 43.

- Tienari, P., Wynne, L., Moring, J., Lahti, I., Naarala, M., Sorri, A., Wahlberg, K., Saarento, O., Seitamaa, M., Kaleva, M., and Läsky, K. (1994). The Finnish adoptive family study of schizophrenia. *British Journal of Psychiatry*, 164 (Supplement 23), 20–26.
- Wahlberg, K., Wynne, L., Oja, H., Kestitalo, P., Pykäläinen, M., Lahti, I., Moring, J., Naarala, M., Sorri, A., Seitamaa, M., Läsky, M., Kolassa, J., and Tienari, P. (1997). Gene–environment interaction in vulnerability to schizophrenia: Findings from the Finnish adoptive family study of schizophrenia. *American Journal of Psychiatry*, 154, 355–362.
- Wender, P., Rosenthal, D., Kety, S., Schulsinger, F., and Welner, J. (1974). Crossfostering: A research strategy for clarifying the role of genetic and experiential factors in the etiology of schizophrenia. *Archives of General Psychiatry*, 30, 121–128.
- Wynne, L., Singer, M., and Toohy, M. (1976). Communication of the adoptive parents of schizophrenics. In J. Jorstad and E. Ugelstad (Eds.), *Schizophrenia 75 — Psychotherapy family studies, research* (pp. 413–451). Oslo: University of Oslo Press.