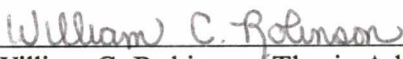
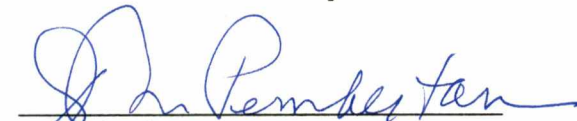


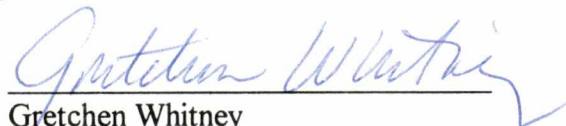
To the Graduate Council:

I am submitting herewith a thesis written by Jeffrey Brian Romanczuk entitled "Autism Etiologies as an Information Mapping Problem." I have examined the final copy of this thesis for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Master of Science in Information Sciences.


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**AUTISM ETIOLOGIES
AS AN INFORMATION MAPPING PROBLEM**

**A Thesis
Presented for the
Master of Science
Degree
The University of Tennessee, Knoxville**

**Jeffrey Brian Romanczuk
May 1998**

DEDICATION

For Luke and Kate, of course,
for whom autism is a universal problem
in a personal way.

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ABSTRACT

Autism is an internationally-occurring, life-long developmental disability for which there is no known cure. There are various treatments for the symptoms of autism, but progress on a cure has been hampered by a lack of consensus on what causes the disability. This thesis uses bibliometric tools of information science to investigate the various etiologies being researched by country, by author, and by publication source from when the term “autism” was first coined in 1943 through sources published in 1996.

There are at least six categories of etiology into which autism causation literature can be arranged: allergy/auto-immunologically induced, biologically based, environmentally caused, genetically determined, neurologically based, or psychological in origin. These six causations were tracked over the 53 years of autism etiology literature published. Abstracts in the following online databases (as well as their print counterparts) were the primary source of data collection: BIOSIS *Previews*, EMBASE, MEDLINE, and PsycINFO.

Although the main research material used for the study is secondary source bibliographic abstracts, the bulk of the literature review broadly investigates primary

source material about autism causes. Since readers of this thesis are likely to be more familiar with information sciences than they would be with autism etiology research, the literature review concentrates on autism literature with bibliometrics sources introduced as they explain and support the methods used, results, and discussion.

This work provides general results from all of the data collected and combines results for certain areas of interest, such as autism causation by decade or the types of causations being investigated from one country to the next. The quantitative results are broken out by type, time period, and location. Other pertinent findings—such as the emphasis on childhood, direct conflicts in the literature, and specific peculiarities of the databases used—are also incorporated.

The final chapter applies bibliometric analyses to the results. Bradford's Law of Scattering (concerning the number of journals that publish articles on autism causation), Goffman's Epidemic Theory (concerning how autism etiology research ideas are generated), and Lotka's Law (concerning author productivity on the specific topic of autism causation) are explored. Some non-bibliometric discussion is also incorporated into the implications, such as autism's evolution and the appearance of "countries" that no longer exist. This chapter finishes this research by suggesting what avenues of further research to pursue and how to continue autism causation research investigations. Suggested methods of data collection and the use of citation analysis were also introduced as possible considerations for a follow-on study of autism causation literature.

Although the initial literature review revealed how multifaceted and distinct are the autism causes under investigation, the final results presented in this research do not bode well for finding a cure for autism, or even reaching consensus on what causes it. Autism's etiology is unclear. A mixture of preconditions could trigger it. It could be biologically based. It could be congenital, or environmental, or none of these. While this chaotic uncertainty is not promising for the future of autism research, it is an ideal framework from which to map a bibliometric research project. The limits placed on the information mapped, however, accounts for some of the non-conformance with expected outcomes. Even so, the results conformed to expected outcomes in more ways than they did not and show that autism etiology research has taken many roads and continues in many divergent directions. As the body of information concerning autism causation continues to grow, the application of bibliometric measures should grow more accurate and more predictive of the future of autism causation research, perhaps showing where all of these paths converge.

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Chapter 1

Introduction

Overview

This introductory chapter includes a rationale for pursuing a bibliometric study of autism causation literature, a statement of the problem being investigated, and objectives of the research described. The rationale includes background on both autism causes and bibliometric studies. The problem statement describes the specific focus of this research, narrowly defining the topic while filtering out closely related issues. The objectives section briefly and broadly lays out what is more fully explained in Chapter 3, “Methods.” The section on objectives also clarifies the hypothesis being tested by this bibliometric investigation of autism cause literature.

Rationale

The *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (*DSM-IV*), lists “autistic disorder” among those conditions “Usually First Diagnosed in Infancy, Childhood, or Adolescence.”¹ Autistic Disorder is the first of the “Pervasive

¹ *Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition, (Washington, DC: American Psychological Association, 1994), 66-71.

Developmental Disorders” discussed. Although links between these disorders and other general medical conditions—chromosomal abnormalities, congenital infections, structural abnormalities of the central nervous system—are mentioned, no causes of autism are supplied. Of course, the *DSM-IV* is principally a diagnostic tool, so causation is not emphasized. However, even *Advocate*—the bimonthly newsletter of the Autism Society of America (ASA)—mentions causes of autism only broadly in its “Definition of Autism” statement that appears in every issue: “The result of a neurological disorder that affects functioning of the brain, autism and its behavioral symptoms. . . .”² Like the *DSM-IV*, *Advocate* stresses the characteristic, treatable symptoms over causation. The definition of autism has been refined often since Kanner first labeled the condition in 1943, after the Greek root “autos,” meaning “self” (highlighting the characteristic self-absorption of autistic individuals.)³ Some put autism’s origin before Kanner’s work. Beauchesne, writing in 1985, claims an origin in psychology dating back to E. Bleuler in 1911 “to describe a secondary dynamic symptom of schizophrenia. . . . Bleuler viewed autism as a normal phenomenon, that of imaginary life. Bleuler’s students further described autistic thinking from the phenomenological perspective. L. Kanner (1943) described infantile autism antinomically, which caused mistakes in the term’s usage.”⁴

² *Advocate* (Bethesda, MD: Autism Society of America, 28, no. 3), 3.

³ Lorna Wing, *Children Apart: Children with Autism and Their Families*, (Bethesda, MD: Autism Society of America, 1993), 1.

⁴ In DIALOG file #11, PsycINFO: Beauchesne, Herve, “L’Origine de la Notion d’Autisme en Psychopathologie. (The Origin of the Concept of Autism in Psychopathology),” *Perspectives Psychiatriques*, 1985, 23 (4, No 103) 273-279.

“Antinomically” means that Kanner was not only the first to describe autism independently of schizophrenia, but also that he allowed for psychological, neurological, or even genetic causations. This opened the way for the biggest problem with autism: that its etiology remains unknown. Bauman and Kemper observe that there is no metabolic, radiographic, or genetic marker yet identified to aid in the diagnosis or prediction of autism.⁵

Most of the causal theories center on some brain defect, either in the development of a specific region or by an abnormal neural cell networking. Although it is not usually diagnosed until early childhood, many sources agree that autism begins during fetal development. Whether this aberrant brain development has a genetic link or could be triggered by environmental factors remains undetermined. Adding to the mystery, in the absence of laboratory tests to diagnose autism, the affliction has always been behaviorally determined. These diagnostic methods have kept open the possibility that autism has a psychological nosology. (Nosology is the branch of medical science that covers the classification of diseases.⁶)

With such a range of possible etiologies to investigate, a bibliometric study of the causes that have been researched bears utility for both the biological and information

⁵ Margaret Bauman and Thomas L. Kemper, *The Neurobiology of Autism*, (Baltimore, MD: The Johns Hopkins University Press, 1994), ix.

⁶ *Webster's Ninth New Collegiate Dictionary*, (Springfield, MA: Merriam-Webster Inc., 1994), 807.

sciences. An overall characterization of literature on the causality of autism would be useful to biological scientists because it could present fresh insights. These scientists tend to specialize in narrowly defined areas. Therefore, a presentation that includes not only how the various characterizations map out—but also how they overlap and intersect—could spark untried methods of research. Those involved in information sciences may gain another method for mapping information growth that is based not on author, journal, or citation usage, but on a specific position held by an author (or characterization of the literature being investigated).

Wallace suggests that “bibliometrics is the application of quantitative methods to the study of information resources.”⁷ He adds that although bibliometric studies have been conducted since early in this century, the term was not proposed until Alan Pritchard coined it in 1969 as an alternative to the unsatisfactory “statistical bibliography.”⁸ Scientometrics (“quantitative analysis of scientific productivity”) and citation analysis (“the practices and patterns of scholarly reference”) are closely related areas of study.⁹ Pao indicates that bibliometrics “uses mathematical and statistical methods to investigate and to quantify the processes of written communication.”

Observable parameters in literature studies include publications, authors, citations, and

⁷ Danny P. Wallace, “Bibliometrics and Citation Analysis,” Chapter 2 in *Principles and Applications of Information Science for Library Professionals*, John Olsgaard, ed., (Chicago, IL: American Library Association, 1989), 10.

⁸ *Ibid.*, 10.

⁹ *Ibid.*, 10.

journals.¹⁰ She offers three reasons for using bibliometrics: to identify literary areas with limited information, to predict trends, and to advance the “knowledge of communication.”¹¹ Nicholas and Ritchie echo these reasons in quoting from Pritchard that bibliometrics’ purpose is “to shed light on the process of written communications and of the nature and course of a discipline. . .by counting and analyzing various facets.”¹² These facets are to be counted in this study: causes being investigated; authors involved; countries publishing this work; publication years, formats, and sources; and the databases indexing these sources. The rationale for this bibliometric count and its objectives are explained in the next two sections.

Statement of the Problem

Autism is a life-long developmental disability for which there is no cure. In fact, research into this condition has not yet reached a primary precondition (if not prerequisite) for finding a cure: consensus among those researching the disability concerning what causes autism. There are various suggestions, but as yet, no clearly supported generalizations.

While much time and effort have been devoted to the treatment of autistic symptoms, or managing autistic behaviors, comparatively little has been written about its

¹⁰ Miranda Lee Pao, *Concepts of Information Retrieval*, (Englewood, CO: Libraries Unlimited, Inc., 1989), 13.

¹¹ *Ibid.*, 13-14.

¹² David Nicholas and Maureen Ritchie, *Literature and Bibliometrics*, (Hamden, CT: Linnet Books, 1978), 9.

cause or causes. Even less bibliometric work has been compiled concerning autism's etiology, and this research has tended toward a narrow focus and year span to highlight a specific autism cause. However, a broader view of autism etiology research reveals many causes under investigation. If autism is caused by genetic, environmental, and brain development malfunctions coming together at some unfortunate opportunity, the research should reflect this mix. On the other hand, if autism is caused by any one of the three (or some other single biological, psychological, or autoimmune cause), this distinction should be borne out in the literature. While clarifying the relationships among suggested causes of autism and tracking their origins and growth will not necessarily lead the way to a cure, it is a fundamental step which could at least ease the way by clarifying the range of what has been said and done. For these reasons, the focus of this research is on the changing nature of autism etiology literature as it has followed the varying research being done into the possible causes of autism.

Objectives of the Study

The researcher intends a bibliometric study of the international research into autism causes over a more than fifty year span, from the condition first being named in 1943 through articles published by the end of 1996. This research will track the growth, migration, and intermixing of various causal theories and test the null hypothesis that no one cause owns a majority percentage (that is, more than half) of the causes given for autism in articles published around the world.

The evolution of causal research literature will be tracked, primarily through secondary biomedical publications and bibliographic sources such as *Index Medicus*, *Biological Abstracts*, *Excerpta Medica*, the National Library of Medicine's *Current Catalog*, and *Psychological Abstracts* (for the abstracts these five sources provide covering the early years of autism etiology research). For post-1965 coverage, the research will emphasize the medical online databases—MEDLINE, BIOSIS, EMBASE, and PsychLIT/PsychINFO. The main intent of using both print and online databases is not so much to expose gaps and confirm duplication as to ensure complete coverage. However, any implications that such gaps and duplication highlight will certainly be incorporated into the overall discussion.

The purpose of this investigation is to map the research into autism causes. Finding a cure for autism has been elusive because there are many probable causes. The hypothesis to be tested is that no one cause will dominate the literature: that is, no single cause will be advanced in the scientific literature statistically more frequently than any other possible causes. There should be much evidence of the four most popular causal theories—neurobiological impairment, genetic influence, psychological causes, or environmentally induced autoimmune malfunctions—with some intermixing of several popular theories, but no clearly identifiable single cause. A bibliometric mapping of the autism cause research results published should demonstrate this multidirectional attack on autism, as well as this lack of causal consensus.

The research will also test the hypothesis that specific causal research categories tend to group together for a certain time span, or by a certain country or journal, before giving way to autism etiology research in a different vein. Goffman's "epidemic theory of communication" is basically that ideas spread like diseases; they are transmitted mainly by direct communication and spread among a susceptible population.¹³ He adds that the journal population is the controlling factor; that is, the number of authors is directly related to the number of journals that publish on a subject. Also, the number of "quality" authors is proportional to the total number of authors in the field. However, the importance of Goffman's work lies in its "predictive power."¹⁴ Just as statistics are predicted for epidemics, as a research topic emerges Goffman's mathematical analysis may be applied to predict controlling conditions, dimensions of growth, and the rate of slow down.¹⁵ Another value of Goffman's work is that it points out the "infectious nature of ideas."¹⁶

Goffman believed, too, that the information retrieval process is more than "strictly a matching procedure"¹⁷ and makes the point that what is learned along the way affects subsequent documents. For him, then, information retrieval becomes a dynamic

¹³ Pao, *Concepts of Information Retrieval*, 34-35.

¹⁴ *Ibid.*, 36.

¹⁵ *Ibid.*, 36.

¹⁶ Joseph C. Donohue, *Understanding Scientific Literatures: A Bibliometric Approach*, (Cambridge, MA: The Massachusetts Institute of Technology Press, 1973), 28.

¹⁷ *Ibid.*, 26.

process of intercommunication and interrelationships. Once all of the data are collected for the fifty plus years of autism cause research, application of Goffman's epidemic theory will provide what should be an interesting framework for some exciting discussion about the direction of autism causation research.

Introduction's Summary

This introductory chapter laid out a rationale justifying the necessity for pursuing a bibliometric study of autism cause literature. The statement of the problem being investigated gave background on both autism causes and bibliometric studies. It also delineated what the issues are and what they are not. The objectives section broadly described the research plan. However, this early indication of how and where the data are to be collected is worthwhile since it is the heart of the research at hand. The section on objectives also clarified the hypothesis being tested by this bibliometric accounting of autism causation literature.

The literature review to be described in the next chapter distinguishes among the three groups of sources necessary for review: secondary biomedical publication's abstracts, bibliometric background literature, and material explaining the various theories about autism's etiology. The latter is the primary focus of Chapter 2, while the former two will be expanded on as needed to clarify points made in Chapter 3, "Methods" and Chapter 4, "Results."

Chapter 2

Review of the Literature

Overview

Although the main research material used for the study is secondary source bibliographic abstracts, the bulk of the literature review conducted for this chapter more broadly investigates primary source material about autism causes. A working understanding of the various causal hypotheses being researched and the rationale behind them is a prerequisite to following the bibliometrics involved. The researcher's assumption is that readers of this thesis are likely to be more familiar with Information Sciences than they would be with autism etiology research. Therefore, this chapter concentrates on autism literature while bibliometrics sources will be introduced as they explain and support the methods used, results, and discussion in the following chapters. This background on the etiology of autism highlights four main causal categories: psychological, neurological, genetic, and environmental, as well as a mix of these causes.

Terminology and Searching

More will be written about this in Chapter 5's "Discussion and Implications," but a technical note regarding the use of "autis?" is useful here. Despite the many names for autism over the years—"early infantile autism," "childhood autism," "late onset autism"—all but "pervasive developmental disorder" use "autism" somewhere in their label. Even "Kanner Syndrome" and "Asperger Syndrome" are generally linked to the keyword "autism."

Investigations of bibliographic references at one point in time are synchronous studies. These contrast with diachronous studies, which follow a pattern of use for specific items over a certain time span. The latter type of study would be more conducive to this work, since autism causation literature over the last fifty years is driving this research.

Not Autism Literature, But Sources About Autism Cause Literature

This thesis focuses not on the autism literature itself, but on the literature about this body of work. That is, online and print medical, psychological, and scientific bibliographies are the primary information base. Most of the sources characterized are from abstracts provided by vendors of biomedical databases. However, a preliminary search was conducted to find if studies similar to the work at hand have already been done.

Although the initial literature search revealed no bibliometric studies of autism, bibliometric research in general (along with nonbibliometric autism research) revealed good ways of addressing the issues involved. A search of *Dissertation Abstracts Online* using “autis?” and “bibliom?” yielded no matches, with 668 autism entries and 105 for bibliometrics. Adding “caus?” to the “autis?” set resulted in 26 hits. Since the search of “autis?” and “bibliom?” found no matches, a broadened query was used next, AND-ing “bibliom?” to “(neuro? or genet? or enviro? or immun?).” This search yielded eight hits, which offered no specific pointers to similar research, but did generate year-span and publication measures that could be incorporated into an autism scientometric, that is, the “quantitative analysis of scientific productivity”¹⁸ in autism research.

A search of *Library and Information Science Abstracts (LISA) Online* under “autis?” revealed a few useful finds, especially an Autism Research Database maintained by Britain’s National Autism Society. The researcher has been granted permission to use this database.

Using “bibliometric? and (study or research)” plus “genetic?” in *LISA* resulted in 12 hits. One citation of note represents a doctoral thesis comparing “[two] ways of measuring obsolescence within the subject of human and medical genetics.” This work compares synchronous studies with diachronous studies. The bibliometric research undertaken here is diachronous, so tracking this type of obsolescence (or the lack of it, that is, the overlap and longevity of etiologies) could be a worthwhile measure.

¹⁸ Wallace, “Bibliometrics and Citation Analysis,” 10.

Although nearly 1,259 abstracts are in the sample, exact or even close matches to the research at hand were limited. Titles such as "Etiology of Autism: A Review of Recent Biogenic Theories," "Autism and Genetics: A Decade of Research," "Autism and Medical Disorders: A Review of the Literature," and "Autism: Fifty Years on from Kanner," were not as useful for this research as expected. What these articles detail is not a broad bibliometric review of autism's etiology over the years but their case for one dominant cause of autism in the present. Unlike the *LISA* and *Dissertation Abstracts Online* searches already mentioned, these articles come from medical journals. Even so, to find only four that come close to this topic highlights the void this research is attempting to fill.

Background on Autism Causes

One problem with attempting such a study is also one of the strongest arguments for undertaking the work. The definition of autism has been refined over the years since 1943, when Kanner first labeled the condition. Even though 1943 may sound recent as a beginning of autism's evolution, Hart points out that only the name is new; "this disability has been part of the human experience for ages."¹⁹ One obvious problem this causes for an inclusive mapping of the literature is what autism was called and what were thought to be its causes before the term was applied.

¹⁹ Charles A. Hart, *A Parent's Guide to Autism: Answers to the Most Common Questions*, (New York, NY: Pocket Books, 1993), 51.

Psychological Basis

The questions about what autism was called and what were thought to be its causes before the label existed can be answered in part by how autism was characterized in its early years, mainly as a psychological affliction. In "Psychoses of Early and Late Childhood," the authors provide excellent background on what those exhibiting psychosis were called in various centuries.²⁰ Until the sixteenth century, some version of "clouding of consciousness" was used. During the seventeenth century, "stupidity" labeled what became "idiocy" in the eighteenth century. "Dementia" began to be used in the nineteenth century, and "schizophrenia" in the early twentieth century. By the middle of this century, clinical descriptions influenced by organic trends and gradual detection of specific symptoms resulted in the "birth" of autism (and a host of other exact names for previously more generalized characterizations of mental illness).²¹

While it is discouraging that little is confirmed about autism in the 1990s, looking at the early years makes clearer the later revelations and breakthroughs. Consider that the disease was labeled during the Freudian heyday of the mid 1940s and that psychologists spent the next twenty years treating autistic individuals (and their parents) with psychotherapy. The "psychogenic" cause stemmed from the belief that parents who were "distant," or at best uneven in their parenting skills, caused autism.²² One early

²⁰ N. Stanojevic, O Antonovic Hadzi, "Psychoses of Early and Late Childhood," *Psihijat Danas*, 10/2, (Yugoslavia: 1978), 141-156.

²¹ *Ibid.*, 156.

²² Isabelle Rapin, "Introduction and Overview," in *Neurobiology of Autism*, 2.

experimenter contrasted the “bright-eyed and alert look” of autistic individuals with the “dull” faces of those similarly mentally handicapped as proof of a psychogenic etiology.²³

The hopeful side of the psychogenic argument is that it implied reachability, that these individuals could be cured. However, this argument began to weaken in Great Britain and the United States when the psychoanalysis and drug treatments successful with mood disorders were largely ineffective in treating autism.²⁴ As the data collected for this research will show, however, many countries still believe and invest much research in confirming a psychological basis for autism. For those countries where this avenue of research had been losing its following, the notion that autism was emotional in origin lost further backing after studies confirming that parents of autistic children exhibit the same range of skills as do any parents.²⁵ Added to these findings were data showing children raised in “old-fashioned institutions” (where they *were* genuinely emotionally deprived) did not develop autism.²⁶

Hart goes into great detail to demarcate the differences between autism and mental illness. Although diseases of the mind and diseases of the body are not always as clearly distinct as once believed, mental illness does not usually occur in children. But the fundamental difference is that the brain’s neurotransmitters work erratically in the mentally ill. In autistic individuals the neurotransmitters work one (wrong) way, or

²³ Rapin, “Introduction and Overview,” in *Neurobiology of Autism*, 2.

²⁴ *Ibid.*, 2.

²⁵ Wing, *Children Apart*, 9.

²⁶ *Ibid.*, 9.

simply do not work at all.²⁷ Rapin adds to this distinction that evidence of a high correlation of autism with rubella and epilepsy indicate a biological, not a psychogenic etiology.²⁸ However, she also points out that—even distinguished from purely psychological disorders—there is still a stigma attached to autism:

Autism is incurable. . . and, of all the developmental disorders of brain function, it is most often associated with behaviors that are extremely difficult to manage and socially unacceptable. The diagnosis of autism is the schizophrenia or cancer of developmental disorders. It is a diagnosis to whisper, avoid altogether, or couch in less-damaging terms.²⁹

In fact, early studies correlated infantile autism to adult schizophrenia, but these results were not replicated by follow-up studies.³⁰

Neurobiological Basis

Rapin makes the point that research into neurobiological causes of autism did not begin until the psychogenic theories about autism's etiology ended (approximately the early 1970s for most countries).³¹ She indicates that the specific deficit areas now being researched include: sensory, cognitive, social, and behavioral.³² Even so, deficits for these diagnostic areas are still assessed by clinical observation, with no dominant theory

²⁷ Hart, *Parent's Guide to Autism*, 44.

²⁸ Rapin, "Introduction and Overview," in *Neurobiology of Autism*, 2.

²⁹ *Ibid.*, 2.

³⁰ Joseph Piven and Susan Folstein, "The Genetics of Autism," in *Neurobiology of Autism*, 25.

³¹ Rapin, "Introduction and Overview," in *Neurobiology of Autism*, 9.

³² *Ibid.*, 9.

as to autism's underlying causes.³³ However, the neurological researchers assert with near certainty that the relationship between the behavioral and cognitive characteristics of autism with those of abnormal brain structure and function implicate the central nervous system (CNS).³⁴ Hart points out that new tests of blood and spinal fluid, as well as recent advances in ways of examining living brains (like magnetic resonance imaging [MRI] and electroencephalographs) are making inroads.³⁵ However, more detailed findings are frustratingly inconsistent. Computer tomographs and MRIs, recent technologies that can "map" a living brain, sometimes indicate physical brain structure differences for autistic individuals and sometimes do not.³⁶ The next obvious step was autistic brain chemistry studies using positron emission tomographs and magnetic resonance spectroscopy. While these results have been "more promising," to date they are also inconclusive.³⁷

Even so, the CNS has been a focus of autism research from the beginning, and more so since technological improvements of the past thirty years have allowed for neurological studies on living brains. Bauman and Kemper preface their 1994 book by stating, "It has become apparent that autism is a developmental disorder of the central

³³ Bauman and Kemper, *Neurobiology of Autism*, 243.

³⁴ *Ibid.*, 244.

³⁵ Hart, *Parent's Guide to Autism*, 42.

³⁶ Bauman and Kemper, *Neurobiology of Autism*, 245.

³⁷ *Ibid.*, 245.

nervous system.”³⁸ Rapin adds that even Kanner and Asperger in the earliest autism research “speculated that early infantile autism was the sign of abnormal brain function.”³⁹ However, Gerlach credits Rimland’s 1964 book, *Infantile Autism: The Syndrome and its Implications for a Neural Theory of Behavior*, with debunking “the assumption that autism was a result of ‘bad’ parenting, and it helped to establish autism as a neurobiological disorder.”⁴⁰

The reason researchers can be so sure autism is neurobiological without knowing its specific causes is that any dysfunction in each brain system produces predictable behavioral deficits. Bauman’s research is guided by the hypothesis that those with similar symptoms may have in common a dysfunction in the same brain region, which may then allow for similar interventions.⁴¹ Bauman and Kemper close their book with a recapitulation of how the previous decade’s epidemiological, clinical, and biologic research has provided insight into “the underlying neurologic substrate” for autism and hypotheses about the characteristic features of autism.⁴²

Autism is characterized by many developmental deficit areas, most notably: sensory, attention, language, cognitive, and social.⁴³ There are many (sometimes

³⁸ Bauman and Kemper, *Neurobiology of Autism*, ix.

³⁹ Rapin, “Introduction and Overview,” in *Neurobiology of Autism*, 1.

⁴⁰ Elizabeth K. Gerlach, *Autism Treatment Guide*, (Eugene, OR: Four Leaf Press, 1993), 3-4.

⁴¹ Rapin, “Introduction and Overview,” in *Neurobiology of Autism*, 8.

⁴² Bauman and Kemper, *Neurobiology of Autism*, 243.

⁴³ Rapin, “Introduction and Overview,” in *Neurobiology of Autism*, 9.

conflicting) theories about the brain area or brain function responsible for each dysfunction. Atypical responses to external stimuli indicate a vestibular dysfunction, often characterized by extremes in auditory responsiveness (either obliviousness to sounds or hypersensitive hearing). Vestibular abnormalities may also contribute to the “savant” traits sometimes noted about those with autism: visual/spatial abilities above the average for their peer group.⁴⁴ The uneven attention spans symptomatic of autism (and many other disorders) are the result of a subcortex dysfunction.⁴⁵ Language deficits, the most frequent complaint attendant to autism, have not been linked to any specific brain region’s abnormalities but may be a cerebellar malfunction or a byproduct of the communicative and social deficits associated with the disorder.⁴⁶ Social deficiencies—the “most salient feature of autistic children”—appear to have a hypothalamic-limbic pathology, though some researchers suspect this atypical behavior is caused by a more general “right hemisphere” dysfunction.⁴⁷

Whether the physical composition of these brain regions is faulty or their transmissions are malfunctioning, the case for most etiologies of autism being neurobiologic is strong. This is especially true considering the frequency with which autism occurs with other CNS disorders.

⁴⁴ Rapin, “Introduction and Overview,” in *Neurobiology of Autism*, 10.

⁴⁵ *Ibid.*, 10.

⁴⁶ Rapin, “Introduction and Overview,” in *Neurobiology of Autism*, 11.

⁴⁷ *Ibid.*, 12.

Especially in children who seem to develop appropriately to a point—only to have their progress curtailed—abnormalities of the limbic system, cerebellum, and cerebellar circuits have been implicated.⁴⁸ Bauman has also noted differences in the neuron size and growth rate in the cerebelli of autistic individuals.⁴⁹ This would suggest abnormal relays between the cerebellum and other regions of the brain.

This brain research also suggests autism is congenital, though the symptoms may not be obvious during the first few years. Also supporting a prenatal etiology is the similarity of autistic traits to “those found in other conditions which have clear physical causes, such as congenital aphasia, or congenital deafness and blindness produced by German measles in the mother during pregnancy.”⁵⁰

Genetic Basis

That autism is a neurobiological disorder does not preclude other etiologies, especially genetic ones. In fact, in the 1940s Asperger noted behavioral similarities between his clients and some of their parents, and speculated about possible genetic factors.⁵¹ However, during the intervening years of belief in the psychological basis of autism, this avenue was not pursued. As late as 1976, researchers were concluding that

⁴⁸ Bauman and Kemper, *Neurobiology of Autism*, 245.

⁴⁹ *Ibid.*, 245.

⁵⁰ Wing, *Children Apart*, 9.

⁵¹ Rapin, “Introduction and Overview,” in *Neurobiology of Autism*, 1.

“the cause was unlikely to be genetic.”⁵² It has been only in the last fifteen years that research into this pathology regained momentum.

In six studies compiled by Smalley in 1988, the prevalence of autism in siblings was found to be 2.7 percent.⁵³ Although this percentage seems small, using 4 or 5 cases of autism in 10,000 births from the general population, this 2.7 percent is about 75 times greater than the norm. Piven and Folstein point out that the risk of autism in siblings could be even higher, since parents of autistic children often limit their plans to have subsequent children.⁵⁴

Not only the siblings, of course, but all relatives of affected individuals have “a higher mean liability” than does the general population.⁵⁵ This was Falconer’s conclusion while researching a “multifactorial polygenic inheritance” model. Hart summarizes a multigenerational study jointly conducted by the University of Utah and the University of California, Los Angeles. This research found not only a statistically significant prevalence of autism in the extended families of autistic members, but also the autism rate for the children of an autistic parent (there were 11 autistic parents in the study) was 46 percent.⁵⁶

⁵² Piven and Folstein, “The Genetics of Autism,” in *Neurobiology of Autism*, 18.

⁵³ *Ibid.*, 20.

⁵⁴ *Ibid.*, 20.

⁵⁵ *Ibid.*, 34.

⁵⁶ Hart, *Parent’s Guide to Autism*, 48.

Other family conditions, such as cognitive deficits (usually in speech and language) and “personality characteristics” showed a higher prevalence in families with an autistic member.⁵⁷ In a 1950s study, Kanner and Eisenberg noted that “a number of parents of these children were serious-minded, perfectionistic individuals with intense interests in abstract ideas, who appeared to lack interest in developing relationships.”⁵⁸ Although parenting style itself is generally no longer believed to cause autism, “autistic traits” are still reported more often in the parents of autistic individuals than in parents in the control groups.⁵⁹

Twin studies in autism to date have produced inconclusive results which serve mainly to show how complex the disability is. While a few studies have found a higher concordance of autism in monozygotic twins than in dizygotic (82 percent versus 10 percent in one study),⁶⁰ the small sample sizes used confound the results. Further, Steffenburg noted “perinatal stress” to be “much more common” in the autistic co-twin of discordant pairs and suggested that complications during birth “increase the risk of autism in individuals with a genetic liability for this disorder.”⁶¹

Hart explains the theory behind the twin studies this way: since identical (monozygotic) twins are “genetic clones,” they have the same neurological structure. If

⁵⁷ Piven and Folstein, “The Genetics of Autism,” in *Neurobiology of Autism*, 22.

⁵⁸ *Ibid.*, 23.

⁵⁹ Piven and Folstein, “The Genetics of Autism,” in *Neurobiology of Autism*, 23.

⁶⁰ *Ibid.*, 21.

⁶¹ Piven and Folstein, “The Genetics of Autism,” in *Neurobiology of Autism*, 21.

autism were purely genetic, either both would have it or neither. Fraternal (dizygotic) twins have “genetic programs” as different as any siblings’ and share only the womb for nine months. So if fraternal twins both develop autism, this would suggest an environmental cause, such as perinatal exposure to some virus or chemical pollutant.⁶² He then relates the results of a twin study conducted by Ritvo which confounds this logic. Ritvo used 23 pairs of identical twins and 17 sets of fraternal twins. The fraternal sets showed few similarities, with several pairs containing one autistic individual and one twin with typical development. While 22 of the monozygotic pairs did develop similar patterns of autism, one pair had an autistic individual and a twin not diagnosed with anything. Hart interprets these results as suggesting that while autism has a strong genetic component, it may not be “purely genetic” but certain preconditions increase susceptibility.⁶³ “Maybe autism cannot be inherited, but the susceptibility to autism might plague some families, just as cancer runs in families.”⁶⁴

Piven and Folstein point out a complication in autism studies not present in cancer studies, though. Autism is a “behavioral syndrome defined by clinical features.”⁶⁵ They cite a Vandenberg report that further complicates the issue: the “search for a single underlying genetic mechanism in autism assumes homogeneity of genetic and

⁶² Hart, *Parent's Guide to Autism*, 43.

⁶³ *Ibid.*, 43.

⁶⁴ *Ibid.*, 43.

⁶⁵ Piven and Folstein, “The Genetics of Autism,” in *Neurobiology of Autism*, 31.

environmental influences.”⁶⁶ Even so, the heritability of autism, after factoring in the variance attributable to the average effects of genes, is estimated to be as high as 90 percent, with the other 10 percent of cases caused by environmental factors.⁶⁷

Environmental Basis

“Environmental” is something of a “catch all” category that includes autoimmune weaknesses (which make an individual more prone to many diseases), allergies, reactions to chemicals (including immunizations), and the presence of other diagnoses. Five disorders often seen individually in connection with autism are Rett Syndrome, phenylketonuria, tuberous sclerosis, neurofibromatosis, and especially Fragile X (or Martin-Bell) Syndrome. Fragile X is a form of mental retardation (MR) linked to the X chromosome and the other three are all presumed to be the result of distinct single gene defects.⁶⁸ Mental retardation generally has a high correlation with autism, with seventy percent of all autistics testing in the MR range.⁶⁹

Both MR and autism are associated with a higher rate of perinatal complications than are experienced by the general population.⁷⁰ This softens the line between genetic and environmental etiologies. However, pregnancy complications (first trimester

⁶⁶ Piven and Folstein, “The Genetics of Autism,” in *Neurobiology of Autism*, 31.

⁶⁷ *Ibid.*, 30.

⁶⁸ Piven and Folstein, “The Genetics of Autism,” in *Neurobiology of Autism*, 29.

⁶⁹ *Ibid.*, 19.

⁷⁰ *Ibid.*, 19.

bleeding, for example) in autism are often “minor” compared to those preceding other MR births.⁷¹ Both MR and autism are associated with infantile spasms and congenital rubella exposure; however, MR is associated with many more disorders that autism is not (like cerebral palsy and Down syndrome).⁷² Hart points out that although autism is considered a birth defect, there are no chemical or chromosomal tests yet to predict it. Although it is considered a developmental disability, we still do not know which effects to label “primary” and which to call “secondary.”⁷³ He adds that although there is a genetic link, we still do not know what that link may be and what added environmental factors, if any, trigger autism’s onset.⁷⁴ He relates the belief that autism is caused by “modern life-styles,” specifically the pollutants of modern times, citing reports that incidents of autism have been increasing.⁷⁵ However, he feels autism is not increasing any more than all birth defects are, and it is very likely that more traits are now being called autistic that were not previously.

ASA’s “How They Grow” indicates some early illnesses—like encephalitis or prolonged high fever—have been shown to produce cases of autism. However, autism is mainly an organic brain defect.⁷⁶ There is some generally abnormal biochemistry and/or

⁷¹ Piven and Folstein, “The Genetics of Autism,” in *Neurobiology of Autism*, 19.

⁷² *Ibid.*, 19.

⁷³ Hart, *Parent’s Guide to Autism*, 44-45.

⁷⁴ *Ibid.*, 49.

⁷⁵ *Ibid.*, 50-51.

⁷⁶ *How They Grow: A Handbook for Parents of Young Children with Autism*, (Silver Spring, MD: Autism Society of America, 1990), 3.

a physical malformation of the brain. Even if the brain areas are of normal structure, their neurotransmitters do not function properly. Hart indicates that autism's broad range of indications and possible etiologies makes the broad term "autistic syndrome" more appropriate. "Researchers call conditions that show hereditary as well as environmental factors syndromes. . . Accordingly, many professionals use the term 'autistic syndrome' when discussing the biology and causes of this disorder."⁷⁷

Mixed Basis

There are many potentially genetic etiologies, but also many non-genetic ones. One school of thought is that exposure in utero or in infancy, to other diseases (like rubella) may trigger autism.⁷⁸ However, this is not supported. Complicating this hypothesis is an aspect unique for autism, but not exclusively the domain of this disease; it is a disability which is congenital, but whose symptoms do not appear (or are not reported) until the toddler years.⁷⁹ As mentioned in the previous chapter, there are no metabolic or chromosomal tests to predict autism. Although there are many nonpsychological theories about what causes autism, the main three are that: (1) it is a neurobiological disorder, (2) it has a genetic component, and (3) because it often occurs in conjunction with other handicapping conditions like epilepsy, there may be an

⁷⁷ Hart, *Parent's Guide to Autism*, 49-50.

⁷⁸ *Ibid.*, 44.

⁷⁹ Hart, *Parent's Guide to Autism*, 44.

autoimmune/environmental etiology. However, none of these causal “theories completely explains autism. At best, some contain hints and clues that may one day answer the questions of how it occurs, how the body’s chemistry is affected, and what parts of the brain are involved.”⁸⁰

Bauman and Kemper posit that part of the trouble with zeroing in on autism is that it is manifested in a spectrum of symptoms that vary in both appearance and severity. There may be many subtypes of autism.⁸¹ ASA’s “How They Grow” booklet calls autism “a class of disabilities,” not one, and adds that it may occur with other brain disturbances. A third of autistic individuals develop epileptic-like seizures in their teen years and a majority of autistics test in the mentally retarded intelligence range.⁸² To use Rapin’s nicely put phrase, one of the “less-damaging terms” for autism is “pervasive developmental disorder” (PDD). The *DSM-IV* lists five PDDs, including the open-ended “PDD-Not Otherwise Specified.”⁸³ Despite the damage-control aspect of this range of labels, it is probably more accurate in that PDD indicates a spectrum of disabilities.⁸⁴

Further complicating the disorder, according to Gerlach, is that there is no test to diagnose autism, only to rule out similar diagnoses (like mental retardation and Fragile X

⁸⁰ *How They Grow*, 4.

⁸¹ Bauman and Kemper, *Neurobiology of Autism*, ix.

⁸² *How They Grow*, 2.

⁸³ *Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition, 77-78.

⁸⁴ Rapin, “Introduction and Overview,” in *Neurobiology of Autism*, 5.

syndrome).⁸⁵ Autism itself is diagnosed behaviorally and is often viewed therefore as a behavioral disorder. However, obvious evidence of genetic predisposition, as well as correlation with some pre- and perinatal infection exposure, introduce biological factors.⁸⁶

Conclusions Drawn from the Literature Review

Autism's etiology is unclear. A mixture of preconditions could trigger it. It could be biologically based. It could be congenital, or environmental, or none of these. While this chaotic uncertainty is not promising for the future of autism research, it is an ideal framework from which to map a bibliometric research project. The range of etiologies being investigated, the international scope and number of countries publishing articles on the topic, and its relatively short evolution over the last half of the twentieth century are promising components for a structured application of bibliometrics.

The next chapter lays out the design of this bibliometric study. From choosing sources, through deciding which facets of the sample would be analyzed, to describing the methods used to count, compare, and contrast the facets, the methods chapter provides the founding structure from which observations can be made and on which results can be built.

⁸⁵ Gerlach, *Autism Treatment Guide*, 5.

⁸⁶ Wing, *Children Apart*, 9.

Chapter 3

Methods

Overview

This chapter describes the design of the study and discusses methods for carrying out this design. The researcher explains how the sample was selected and procedures used to characterize data selected. This chapter closes by acknowledging limitations to the sampling and analysis and suggests how these limitations might be overcome.

Design of the Study

Selection of the Sample

The goal is to map all of the autism causation literature as it has evolved since its origin in the 1940s, specifically from 1943 when the disability was first noted (separately by Kanner and by Asperger) through etiology research published by December 1996 and present in bibliographies published before July 1997. The emphasis is on abstracts of journal articles. The three main sources used will be MEDLINE, EMBASE, BIOSIS *Previews* (and their respective print counterparts *Index Medicus*, *Excerpta Medica*, and *Biological Abstracts*, especially for coverage of the early years of autism, but also for

source comparison research as well). These main sources will be supplemented with the National Library of Medicine's *Current Catalog*, PsycLIT, PsycINFO (and the print version for these two, the American Psychological Association's *Psychological Abstracts*, again for the critical early years of autism causation research).

In her *Autism Treatment Guide*, Gerlach credits the 1964 publication of *Infantile Autism: The Syndrome and its Implications for a Neural Theory of Behavior* with debunking "the assumption that autism was a result of 'bad' parenting."⁸⁷ Therefore, a considerable amount of the literature in the first twenty plus years of autism is in the field of psychology. Although the primary three sources often abstract psychology-related journals, specifically adding psychology abstracts to the search should provide a fairer guarantee of pulling in causal theories from these early years. Even now, autism is behaviorally diagnosed, not by blood tests or brain scans. Therefore, the investigator intends to search widely by concentrating on medical, biological, and psychological databases and bibliographies, and using additional sources—such as the Internet, conference proceedings, and monographs—mainly for confirmation and supplemental information. For example, two sources found not on but through the Internet will be used: "A Collection of Abstracts on Genetic Research of Autism" <http://www.udel.edu/bkirby/asperger/genetic_abstracts.html> and The Autism Research Database's "Titles in Autism" (published by the Autism Research Unit at the University of Sunderland <aru@sunderland.ac.uk>).

⁸⁷ Gerlach, *Autism Treatment Guide*, 3-4.

Procedures

Data Collection

Two data filters are introduced with the selection of the bibliographies for this bibliometric mapping. The first is the researcher-controlled decision of which files to use. The other is the inclusivity of the files themselves, though the four digital bibliographies could be called “exclusive” only in the subjective sense. With coverage back to 1974, EMBASE indexes “over 3,500 biomedical and pharmacological journals published throughout the world.”⁸⁸ Since 1966, MEDLINE has been the digital counterpart not only to *Index Medicus* but also to the *Index to Dental Literature* and the *International Nursing Index*.⁸⁹ It “covers virtually every subject in the broad field of biomedicine, indexing articles from over 3,700 international journals” published in seventy-one countries.⁹⁰ BIOSIS *Previews* began in 1969 and indexes over half a million research accounts yearly from “nearly 7,600 primary journal and monograph titles, . . . notes, letters, . . . government reports, and research communications.”⁹¹ The 7,600 is updated to “nearly 9,000” in the DIALOG *Bluesheets* on the World Wide Web. The site (http://www.krinfo.com/plweb-cgi/idoc.pl?350+unix+_free_user_+web6.dnt.dialog.com..80+dialog+dialog+bluen+bluen++bluesheets#PD) had the July 18, 1997 *Bluesheets* loaded when the researcher visited. The American Psychological

⁸⁸ *Database Catalogue 1994*, (Palo Alto, CA: Knight-Ridder Company, 1994), 65.

⁸⁹ *Ibid.*, 96.

⁹⁰ *Ibid.*, 96.

⁹¹ *Ibid.*, 30-31.

Association's site (<http://www.apa.org/psychnet/>) states that PsycINFO indexes "over 1,350 scholarly journals." PsycINFO covers from 1967 to the present and pulls from the international literature in such fields as "behavioral and social sciences, . . . developmental psychology, educational psychology, experimental human. . . psychology, personality, physical and psychological disorders."⁹² Clearly, once the decision to use a DIALOG file is made, limits on the data sample due to indexing exclusivity are a mild concern, but not one the researcher can control.

Bibliographies are generally used most frequently for bibliometric studies because of their low cost and comprehensiveness. However, because they do not give enough information to stand alone and may not react quickly enough to changes in the subject literature, they provide "no more than a first step toward. . . a literature description."⁹³ Abstracting and indexing services have improved the statistical scope of bibliometric studies. These index down to the article or review level, rather than at the broader journal or book level.⁹⁴ This can be useful or not, depending on the search. However, a clear drawback of the indexing vendors is that completeness is not guaranteed. Despite the improved indexing mentioned above, these services are selective, tending toward the "scholarly" journals, and do not abstract even their core journals from cover to cover.⁹⁵ They do, however, make "simple" statistics available and are more "user-oriented" (if

⁹² *Database Catalogue 1994*, 120.

⁹³ Nicholas and Ritchie, *Literature and Bibliometrics*, 22, 37.

⁹⁴ *Ibid.*, 26.

⁹⁵ Nicholas and Ritchie, *Literature and Bibliometrics*, 127.

not user-friendly) than other secondary services.⁹⁶ Nicholas and Ritchie offer ways to assess coverage patterns, currency, and overlap. They suggest knowing before using the service what its policy on coverage is as to subjects, titles, languages, countries, document forms, and frequency.⁹⁷ While discussing the “currency” issue, Nicholas and Ritchie caution care in filtering out the actual versus “intended” date of publication and state in general terms that journal articles appear quickly, reports/proceedings take a little longer to index, and language translations may take years.⁹⁸ The authors also state the value of checking more than one vendor, even when the subject range appears the same, because the duplication from one service to the next is seldom more than 75 percent.⁹⁹

A search for autism causation literature listed in sources published before January 1997 (and indexed in the bibliographies by June 1997) governs the data collection phase. Since the cut-off date for the publication of the bibliographies is the first half of 1997, nearly all of the material researched will have been published in 1995 and before, with the online databases possibly abstracting some of the early 1996 material, but not the full year. Query construction will use the “autism” root and “pervasive developmental disorder” as well (autism’s “parent” category in the *DSM-IV*). Linked to this set will be the “cause” and “etiology” root words. (A copy of the actual OneSearch query from

⁹⁶ Nicholas and Ritchie, *Literature and Bibliometrics*, 26.

⁹⁷ *Ibid.*, 128.

⁹⁸ *Ibid.*, 131.

⁹⁹ Nicholas and Ritchie, *Literature and Bibliometrics*, 132.

DIALOG is Appendix A.) Since “cause” has many meanings and uses, it could generate many false drops. Rather than construct the query in an attempt to guard against this, the false drops will be filtered during reading of the abstracts to determine whether the sources they represent are truly about autism causation. If the article discusses only an autism treatment or some test of autistic ability without taking a specific stand on the issue of how autism happens, it will be treated as a false drop.

The study does not limit itself to autism causation research within the realm of the four possibilities mentioned. As other causal categories present themselves, additional values can be added to incorporate them. For this study, the operational definition of “autism causation literature” is any document for which the abstract or the source itself uses both “autism” (its root or its “parent” characterization, “pervasive developmental disorder”) and a “cause”/“etiology” derivative which clearly assigns a cause to the defect. For example, the easiest source abstracts to characterize contain comments such as “this research proposes a genetic model for autistic development;” “autism is the result of a CNS [central nervous system] dysfunction, not a hearing deficit;” and “it is clear that autism is a neurologically-based developmental disorder.” Others that are also clearly stated, but require some deduction, include “although autism is associated with a wide variety of genetic conditions, it is a behaviorally defined phenotype arising from CNS damage” [neurological, not genetic] and “there was an extremely high frequency of autism (11.4%) in a sample of 70 children of cocaine mothers” [environmental].

The researcher expected more than half of the sources reviewed to be false drops. Three reasons for this include: (1) "cause" can have a meaning more general than "etiology," (2) "autistic-like" can be used in an abstract which otherwise has nothing to do with autism, and (3) articles may be about autism, but not about autism causes. But the main reason for so many false drops is the researcher's attempt to identify the items first hand and not rely solely on the Boolean logic of query language. All of the synonyms for "autism" were developed by the researcher. (Fortunately, most of these have "autis" somewhere in their name, like *autistic spectrum disorder* and *infantile autism*.)

Nesting (that is, parenthetically pairing off terms to control the order in which they are searched) was used, but there was no NOT-ing out "treatment" or other filtering out of undesired words. Preliminary experimentation with NOT-ing out terms indicated that too much was removed. To test this preliminary finding (and the assumption that more is published about autism treatments than is printed about autism causes), the researcher conducted a MEDLINE search using "autis? and treatment?" and another for "autis? and caus?." The former returned 551 citations; the latter, 135. NOT-ing out "treatments" from the "causes" brought this 135 down to 117 hits. (NOT-ing "causes" out of the "treatments" search similarly reduced the 551 to 533 and hints at further complications to the search for autism causation literature.) The commonality of the words "treatment" and "cause" were the main reason for the researcher adding the more

scientifically precise “etiology” and also his reason for filtering out the false drops manually.

How to harvest and filter the information contained in each file was a concern. Searching the DIALOG files individually, using SearchSave, or using OneSearch seem the obvious options for this autism causation search. The researcher opted for using OneSearch because the query was not so complex that it needed saving. However, the main appeal of DIALOG’s OneSearch option is that it allows for removing duplicate abstracts (the “rd” command).

The next issue for resolution was how to track data collection and enable the sorting necessary to aid in its analysis. To manage this, the researcher set up a simple database with the following categories:

- (1) record number,
- (2) suggested cause/number giving this cause [this number served as a running total],
- (3) author(s),
- (4) title of article,
- (5) country of publication,
- (6) year of publication,
- (7) format,
- (8) abstract source, and
- (9) article source.

The first category, record number, was created mainly to provide a running tally of sources used. However, this field distinguished itself from the second for the many articles that postulate multiple etiologies. That is, the degree to which the total number of etiologies exceeds the number of records used is accounted for by the double and

triple causation sources. There are, then, 1,543 causes attributed in 1,259 database records.

Characterizing articles that deal with more than one causal hypothesis was handled in the following manner: if they merely stated what causes are possible, they were not counted in the research, but treated as false drops. If an article gave equal weight to two or more distinct causal hypotheses, it was count for two or more. If the source leaned in favor of a certain causation, it was considered under this cause only. Obviously, for sources like this, the source itself had to be evaluated, not only its abstract. For a clear majority of articles, however, the researcher assumed that the abstracts accurately reflected the work under analysis for representation. This presented two other problems to work through: (1) deciding the causation when the abstract was not clearly written and (2) deciding what to do about articles whose abstracts only negate a cause without supporting another. This kind of lack of clarity has not been an issue. When an abstract addresses a specific etiology, it is generally to describe that etiology as the main cause of autism.

The bigger problem was abstracts that state only that autism is not caused by genetic or psychological (or other) factors, without saying what the etiology might be instead. It sounds contradictory, but the researcher opted to place these abstracts in the very category from which the authors were removing them as autism cause possibilities. The justification is that the research in each case was investigating the possibility. That the results cleared the category as a possible autism cause did not change the direction of

and reason for each author's research investigation. In any case, all but 14 of the sources that negated a cause offered another either directly or by clear implication. Fourteen is a little more than one tenth of one percent of the 1,259 abstracts in the sample, so it is unlikely that the issue was large enough to skew the results, or even change them.

The database developed documents where the article was published, not necessarily where the research was done or even the authors' native countries. The format indicates whether the source is a journal article, editorial, book, conference proceeding, etc. The abstract source is the online vendor or bibliography in which the item is found, while the article source is the journal (or other source, i.e., the publisher for monographs) in which the item was published.

Another advantage to the use of this project-specific database was that it provided a fourth filter against duplicate entries in addition to (1) OneSearch, (2) the researcher counting the causes as they are characterized, and (3) the cause tally following each source or DIALOG file review. Although the intent of entering sources into this database was for easier manipulation, an immediate benefit was that it provided yet another chance to characterize an article or to filter out sources as duplicates or false drops.

Data Analysis

The intent of this research is to enumerate and map autism causation categories using graphs similar to those used for Bradford's Law of Scattering. However, while

Bradford's Law tracks how the literature on a specific subject is spread over a number of journals,¹⁰⁰ what this investigation seeks to map are the numbers—by cause specifically, but by year, too—for autism causation identified in the research over fifty-three years. The research hypothesis being investigated is that no single cause will dominate the literature (that is, be significantly greater than equal in number to the sum of articles about any other autism etiology). Certainly, no one cause should claim a majority of the abstracts used. What the researcher expects to find, therefore, is a relatively equal distribution of autism causation literature among a handful of possibilities. Preliminary reviews indicated that neurological, genetic, environmental, and psychological causations are the main four under investigation by autism researchers, so the numbers of articles for each of these areas should be nearly equal if no single causation dominates.

A related analysis is tracking the overall growth causal areas and any shifts in the direction of the research as evidenced by journal name changes or tandem year spans that indicate a jump from one specific autism cause to another (which can be a subtle but cogent statement of a shift in thinking). The utility of print versus online databases for this kind of research is also investigated. For example, the researcher makes note of print pointers to autism causation literature, such as those which make “autism” its own heading in cumulative volumes or those which include “autism” under an “etiologies” index (or vice versa). Discussion of Goffman's epidemic theory is also incorporated.

¹⁰⁰ F. Wilfred Lancaster and Amy J. Warner, *Information Retrieval Today* (Arlington, VA: Information Resources Press, 1993), 209.

Limitations of the Study

Sampling Limitations and Their Solutions

Being able to judge the causation category for the abstracts found presents issues more thorny than actually finding them. Two sampling issues to solve before collecting the data were: (1) overlap of the four categories and (2) incorporating other than English-language autism causation research. These issues are addressed in this section. In the interest of clarity and realization of time constraints, the profiles of the research literature and reporting of results were limited to cause suggested, format, author, country of publication, year of publication, article source, and abstract source. Limiting the search to causes—rather than treatments or “cures”—further controlled the sample size.

Overlap of categories was the most unclear issue. Since autism is by definition a brain disorder, even genetic and environmental causations manifest themselves as brain defects. All causes seem to overlap at the neurobiological. This could have been a hair-splitting exercise at best, or (at worst) a judgment call this researcher does not have the qualifications to make. The research analysis avoided this, however, by concentrating not on the nature of autism but on the characterizations offered by the literature about autism causation research. For example, in the abstract mentioned under “Data Collection” that describes the 11.4 percent incidence of autism among the children of 70 cocaine-addicted mothers, the autism etiology was characterized as “environmental”

because the perinatal exposure to cocaine was the emphasis of the abstract.¹⁰¹ Had the authors concentrated on the brain damage involved, the abstract would have been characterized as “neurological.” If the authors had developed both aspects, the abstract would have been classified as both “environmental” and “neurological.”

Since the researcher has no effective secondary languages, this investigation attempts international inclusivity by using foreign articles with abstracts translated into English in the bibliographies already mentioned, mainly those published in the United States of America. There are specific English language versions of *African Index Medicus*, *Index Medicus Israeliticus*, *Index Medicus for WHO [World Health Organization] Eastern Mediterranean Region*, *Index Medicus for WHO South-East Asia Region*, a *South Korean Index Medicus*, and an *Index Medicus Latino-Americano*. However, these are better used for follow-on, expanded research. These are available at the National Library of Medicine, in Bethesda, Maryland, in person (or by the volume through interlibrary loan). Also, since they are not easily accessible, avoiding the use of English language bibliographies published outside of the United States will more readily facilitate replication of this investigation.

¹⁰¹ In DIALOG file #155, MEDLINE: E. Davis, I. Fennoy, D. Laraque, N. Kanem, G. Brown, and J. Mitchell, “Autism and Developmental Abnormalities in Children with Perinatal Cocaine Exposure,” *Journal of the National Medical Association*, April 1992, 84(4): 315-319.

Methodological Limitations and Their Corrections

Limits to the research methods employed included: the uncertainty of “equal” weighting, the issue of validity, and personal “interference.” Deciding what constitutes a single bibliometric unit was one issue to solve. Any document expressing and supporting a view on autism’s cause appears to be the baseline. However, should a book carry no more weight than an article or, even more important, an abstract of an article? Or if the book presents and defends various causes, should it count multiple times in the research? The short answer is “yes,” it should. Each cause presented and/or researched has equal weight with all others. While this thesis is marginally interested in distinguishing among the sources, it is primarily concerned with counting causes, not sources.

Assuming the data collection is straightforward and the research identifies the four causal groups expected—neurobiological, psychological, genetic, and environmental—and that their numbers are relatively easy to tally, will these numbers measure precisely what the research intended? Would the fact that a large percentage of articles point to a genetic cause, for example, mean that this cause of autism is more likely to be the “true” cause? While this outcome would be desirable, it does not account for factors such as the politics of research funding. The bibliometrics may look good on paper, but the research itself would not reveal the politics behind the numbers, making it difficult to draw these kinds of conclusions. However, these issues are beyond

the scope of information sciences, and the research being described here intends to draw bibliometric conclusions only.

The last of the methodological limitations is a personal one. The researcher's two children are autistic, a fact which may have biased him if not in favor of a genetic causation, then at least away from theories his everyday experience has not supported. However, mitigating this bias is the researcher's years of reading what has been written about autism and his personal experience with many individuals with autism, through which he does realize that given the variety of autism's manifestations, anything is possible. That this is a statistical, bibliometric investigation should further mitigate this emotional bias.

Although the issue of what is actually being measured has been sublimated as being beyond the scope of the work undertaken, there are methods to equalize the differences in the amount of information given from one source to the next and to guard against researcher bias. The sources that provide a 100 to 250 word abstract make it easiest to gauge the causes presented. However, there are other cues to use when examining a source or citation. The title of the work, keywords accompanying the citation (useful when the vendor gives little or no abstract), and the title or known focus of the journal which published the article yield reliable measurements. As the researcher could use the combination of this source-surrounding information to characterize the autism causation being presented, the source was added to the data. If not, it was considered a false drop. A danger in this is that sometimes the accompanying information

is misleading, especially titles alone. However, these characterization-supporting factors were not considered in isolation, only in combination with each other, so their potentially misleading information did not influence the majority of the data (that is, the clearly stated autism etiologies). For example, the journal article "Development of Object Relations During the First Year of Life," by K.S. Robson had no accompanying abstract. However, it appeared in *Seminal Psychiatry* (in November, 1992, volume 4, pages 301-16) and the MEDLINE descriptors used include "child development. . .personality disorder—etiology. . .autism, infantile—etiology. . .avoidance learning. . .[and] maternal deprivation."¹⁰² These cues—journal, title, and descriptor taken together—account for the researcher's "psychological" characterization of the article. Another example is "Congenital Rubella and Autistic Behavior" by C.N. Swisher and L. Swisher. The indexing information for this article appeared in EMBASE without an abstract. That the source is the *New England Journal of Medicine* (1975, volume 198, pages 293-4) is not a useful characterization tool, since this journal publishes a wide variety of medical articles. However, the subject headings (Emtags) include "infectious diseases. . .etiology. . .virus infection. . .congenital infection. . .[and] infantile autism." The identifier is "autistic behaviour; relationship with congenital rubella." The section headings include "virology," "epidemiology," and "rubella virus."¹⁰³ These pieces of information taken together are enough to justify an "environmental" characterization. If it were known that

¹⁰² DIALOG file #155, MEDLINE, Knight-Ridder Information, 1997, Accession No. 01621668/75085717.

¹⁰³ DIALOG file #73, EMBASE, Elsevier Science, 1997, Embase No. 76114230.

Robson always wrote about psychological matters or that Swisher and Swisher's work is often associated with congenital defects, these would have aided in the characterization as well.

Three other biases had to be guarded against vigilantly because they could have had a detrimental effect on the numbers. The researcher is predisposed to neurological causes, is biased against psychological causes, and tends to favor works from countries other than the United States. When the cause seemed neurological, or neurological paired with another cause, the researcher read these abstracts more than once to ensure they were indeed presenting causal research and not merely supplying general neurological information. Guarding against the psychological bias required almost the opposite tactic. Especially for the early years of autism research, even if it seemed from the abstract that only general psychological information was being relayed, the researcher read twice to determine if the abstract could be labeled "psychological" as a legitimate presentation of an autism cause. The bias for foreign country inclusion as a subtle way of forcing the issue that autism is an international problem was easier to guard against. The researcher simply began to make the characterization before looking at where the work had been published.

Methods Summary

This study design chapter includes research methods used, describes how the sample was selected and where it was selected from, and lays out the procedures used

for data analysis. Several sampling and methodological limitations to the work at hand are described, along with examples and plans to work around or overcome these obstacles.

The next chapter applies the methods given above to the data collected and describes the results obtained.

Chapter 4

Results

Overview

This chapter provides general results from all of the data collected and combines results for certain areas of interest, such as autism causation by decade or the types of causations being investigated from one country to the next. The quantitative results are broken out by type, time period, and location. Other pertinent findings—such as the emphasis on childhood, direct conflicts in the literature, and specific peculiarities of the databases used—are incorporated at the end of this chapter.

Quantitative Results

Overall Statistics

Besides the four causal categories previously mentioned—environmental, genetic, neurological, and psychological—two other categories emerged during data collection and analysis: allergy/auto-immunological malfunctions and biological influences (i.e., a lack or abundance of some chemical present in autistic individuals). As mentioned under “Environmental Basis” (page 24), immune weaknesses often manifest

themselves as environmental causations (i.e., *in utero* exposure to rubella; ingesting lead or adversely reacting to antibiotics during the toddler years). However, the abstracts are more likely to pair immune system weaknesses with allergies than with environmental factors. Similarly, the biological impacts become evident through brain chemistry unique to autism. Therefore, they appear to be neurological in nature.

Realistically, there could have been more than these six causations. However, the researcher limited the study to these two additions to the four apparent from the literature review since the lines of distinction among the sources blur as their numbers increase. When other causations' investigations were identified, these were mapped to the six using correlations shown in Table 4.1.

Table 4.1. Characterizations Used for the Many Autism Causations in the Literature

if the characterization said—	then the causations was listed as—
autism in conjunction with other afflictions	environmental or immunological
brain chemistry dysfunction	biological or neurological
brain hemispheric (left or right) dysfunction	neurological
chromosomal abnormalities	genetic
cognitive dysfunction	neurological
hereditary problems	genetic
organic deviations	biological or neurological
pre-, peri-, and/or neonatal insults	environmental
social impairments	psychological

Most of the causations labeled “environmental” are in fact related to complications surrounding the pregnancy. Purely environmental causations—such as a chemical factory or nuclear plant in the autistic individual’s home town—have started to be investigated only in the 1990s. However, 8 of the 1,543 causes clearly single out allergic insults (independent of environmental causation) as triggering autism. Ninety-six causes are specifically attributed to autoimmune system deficiencies. Thirty-eight of the 103 biological causes asserted are *neurobiological* (and so counted under *both* causes). Only 47 of the articles—such as those that specifically addressed body chemistry imbalances in the brain like an excess of the chemical serotonin¹⁰⁴—are recorded as solely biological causes. (The remaining 18 biological etiologies are paired with other causations.) Table 4.2 gives the total number of causes counted for all sources. (Appendix B, Figure 1, [page 122] provides a pie chart of this same information to clarify visually the percentages and totals involved.) The 1,543 causes for autism appear in 1,259 abstracts and primary sources. The following sections further break out these totals by decade of publication, by the country in which each document was published, by source (at both the database/bibliography level and at the journal, monograph, conference proceeding, etc. level), and by author.

¹⁰⁴ “A chemical, 5-hydroxytryptamine (5-HT), present in platelets...thought to be involved in neural mechanisms important in sleep and sensory perception.” Clayton L. Thomas, ed., *Taber’s Cyclopedic Medical Dictionary*, Fourteenth Edition, (Philadelphia, PA: F. A. Davis Company, 1981), 1301.

Table 4.2. Total Number and Percentage of Autism Causes

Etiology	Number of Appearances	Percentage
Allergy/Immune	104	6.74
Biological	103	6.68
Environmental	114	7.39
Genetic	305	19.76
Neurological	551	35.71
Psychological	366	23.72
Totals	1,543	100

By Decade

In Appendix C, Table C-1 gives a year-by-year summary of the causes found and Figure 2 provides a decade-by-decade summary of the same information. Table C-1 makes obvious the year-span information for each of the causation. The second page of the table makes clear that once a potential cause surfaces in the literature, it never goes away. Page one of this table (on page 124) shows that a psychological causation was the first proposed, in 1943, with neurological investigations coming soon after. A possible environmental causation first appeared in 1949, while genetic or biological causations were not initially proposed until 1953. Allergy/immunological is the most recent research tack, first mentioned in 1965.

However, psychological causation research accounts for more than half of the pre-1970 writings, with neurological causations appearing about half as frequently, and the other four causations each in single-digit percentages. The issue of whether an

improved understanding of genetics and brain mechanisms contributed to the increase in research into these causations and the declining percentage of psychological etiologies being investigated is addressed in the next chapter under “Bandwagon Appeal.” The following sections provide more specific narrative overviews of the causation data for each decade.

1943 to 1956

For layout convenience, this “decade” includes fourteen inclusive years.

However, this does not skew the overall data for the ten-year decades that follow.

Immediately after autism’s first appearance, follow-up articles were sporadic and sparse.

In fact, the researcher found no articles on autism causation for 1945, 1946, and 1948, and only one source for both 1947 and 1949. Table 4.3 is a by-decade and by-etiology summary of all causes found.

Table 4.3. Instances of Each Autism Etiology by Decade of Publication

Decade	Decade Total	Allergy/ Immune	Biological	Environ- mental	Genetic	Neuro- logical	Psycho- logical
'43-'56	32	0	2	2	1	4	23
'57-'66	60	1	3	2	5	18	31
'67-'76	292	17	13	21	34	102	105
'77-'86	440	28	33	42	89	157	91
'87-'96	719	58	52	47	176	270	116
Totals	1,543	104	103	114	305	551	366

Two articles appeared in 1943, both attributing autism to psychological maladjustments. One of these is Kanner's "Autistic Disturbances of Affective Contact," the seminal article describing autism as "a new form of emotional disorder. . . present from birth."¹⁰⁵ Prior to 1943, autism received passing mention as a substrate of schizophrenia. From 1943 through 1956, 31 more autism causation articles were published. Twenty-two of these attributed autism to psychological disturbances. Four proposed a neurological causation. One author investigated a genetic cause, while two articles offered an environmentally induced causation and two others saw autism's causation as biological.

1957 to 1966

The causal attribution trend from the previous decade continued, with the overall number of articles nearly doubling (from 32 to 60). More than half (31) of these were psychological causation-based, with neurological causes still second in number but with a more compelling 18 instances. The other four causes remained in single digits: genetic (5), environmental (2), biological (3), and allergy/immunological (1).

1967 to 1976

The number of causes given during this decade increased nearly five times from the previous ten years, with 292 autism causation possibilities proposed. Psychological

¹⁰⁵ *Psychological Abstracts*, Volume 17, (Lancaster, PA: American Psychological Association, 1943), 400.

causes still led the numbers, with 105, but neurological causation articles were closer behind at 102 total. This time, the other four causations were all in double-digits with genetic investigations still a distant third at 34 causes given. Environmental causes of autism were written up 21 times; biological causes 13 times. The theory that allergy/auto-immunological malfunctions trigger autism appeared 17 times.

1977 to 1986

The number of autism causation articles continued to grow with the increase in the overall literature on autism, though not at the same rapid pace as in earlier decades. The 440 causes given from 1977 through 1986 represent a little more than one and a half times the total from 1967 through 1976 (the 292 causations covered above). This time neurological causations account for 35.68% (157), while psychological causes dropped to 91 (or 20.68%). Genetic research became a closer third in this decade, with 89 causations so cited. The other causations each remained less than ten percent of the total causes under investigation, with 42 environmental, 33 biological, and 28 allergy/auto-immunological etiologies of autism appeared in published articles.

1987 to 1996

Like the first, the time span for coverage for this last decade is not as exact as for those in between. Perhaps not all of the autism causation articles published in 1996 are represented as yet, especially those from foreign language sources and conference

compilations (an interesting thought considering that 1996 had the second highest total causes for a single year at 95). Seven hundred nineteen of the 1,543 causes found (nearly 47%) come from this last decade. Neurological causations alone number 270. With 176 genetic causes cited, this area of investigation moved ahead of the psychological causations published (116) for the first time. Only 47 environmental causations were offered, with the remaining two etiologies each accounting for about eight percent of the decade's total as well: biological investigations numbered 52 while allergy/immune studies accounted for 58 of the 719 causes addressed.

By Country

Forty-four countries are represented in the autism causation literature appearing between 1943 and 1996. (See Table D-1, Appendix D, for the specific number of sources for all countries.) The United States accounts for nearly half, 621 of the 1,259 sources found (and 746 of the 1,543 causes). There are 121 sources listing the "United Kingdom" as the place of publication, with a different 121 specifying "England." France is the country of origin for 71 of the sources used and Switzerland 47. Forty-two abstracts are of articles published in West Germany, plus two from journals published in the former East Germany and four recent articles from the reunified Germany. There are 32 articles from Japan, 24 from the Netherlands, 22 each from Canada and Italy, 20 from Denmark, 15 from Australia, and 13 from Spain. Eleven articles in this study come from journals published in the Union of Soviet Socialist Republics, not counting a more recent

one from Russia. Norway has 8 sources in the sample. This thesis used six or less articles from each of the remaining 26 countries. (See Table D-2, Appendix D, for the complete by-country rank-order numbers.)

Variance of Cause by Country

Table D-3, Appendix D, provides a by-causation breakout for each country.

Among the ten countries with twenty or more articles published, only three do not have all six causations represented. Italy has no articles attributing autism to biological causes. Canada and France have no allergy/auto-immune-based causations published. That most of the countries frequently publishing autism causation research findings cover the spectrum of causal attributes addresses the international scope of this lack of consensus.

Even for those countries with a moderate amount of articles—like Australia with 15, Spain with 13, or the USSR with 11—the span and the range of causes and years show no clear uniqueness. Other than an interesting aside for the three mentioned—that none of them published any allergy/immune articles—the variety of causes, their mix and span of years, and the percentage for each causation are reasonably consistent with the overall totals. (More discussion on this point appears in the section following.)

The two clearest examples of by-country variance are the slow growth of autism causation literature worldwide and the differences in the number of possible causations researched for each country. Of the thirty articles published before 1960, journals in the United States published twenty of them. Many of the countries with fewer than five

sources used do not enter the debate until after 1970. Only 14 of the 44 countries in which autism causation articles were published have pre-1970 research appearing. Another statistic that can be gleaned from Table D-3's tally by country and by cause is that in only 14 countries were allergy/immunological causation articles published; biological causations articles were published in 19 countries; articles on environmentally induced autism appear in 19 different countries; and 22 of the 44 countries account of all of the genetic causation literature published. The neurological causation is most widely internationally attributed, with 36 countries having articles on this etiology. Sources on psychological causations of autism have been published in 26 countries.

Variance of Cause Within Certain Countries

Even more interesting than the lack of international consensus regarding autism's causation is a comparison of how the percentage for each cause within each country matches up with the overall percentages given in Table 4.2 (page 50). Table 4.4 provides the numbers and percentages for the five countries publishing the most source information on autism's etiology. The issue of how the "United Kingdom" is different from "England" and how "Germany" was counted is discussed in the next chapter along with other geographical issues. For this breakout of the information, the focus is on how these percentages compare to those given in Table 4.2. The relative order of the top three causations is almost the same in all cases while the rank order of the bottom three

is more inconsistent. What is more consistent is a noticeable gap in numbers between the highest three and lowest three causes evident for most countries.

Table 4.4. Number and Percentage of Autism Causes for the Five Most Prolific Countries Compared to the Overall Numbers/Percentages

Etiology	Number of Causes/Percentage of the Total¹					
	All	USA	England/UK	France	Switzerland	Germany
Aller/Imm.	104/6.74	62/8.31	15/4.89	0/0	11/16.92	2/3.51
Bio.	103/6.68	41/5.50	21/6.84	4/4.82	8/12.31	3/5.26
Envir.	114/7.39	69/9.25	16/5.21	1/1.20	4/6.15	1/1.75
Genet.	305/19.76	164/21.98	67/21.82	13/15.66	12/18.46	10/17.55
Neuro.	551/35.71	254/34.05	112/36.48	29/34.94	22/33.85	23/40.35
Psych.	366/23.72	156/20.91	76/24.76	36/43.38	8/12.31	18/31.58
Totals	1,543/100	746/48.35	307/19.90	83/5.38	65/4.21	57/3.69

¹ The total percentage for each causation within each country is based on the overall number of causes for that country. However, the percentages in the bottom row are from the number of causes for each of these 5 countries divided by 1,543—the number of etiologies for all countries.

Some notable exceptions to the neurological, psychological, genetic order for those heavily referenced are: for the United States, genetic causes slightly outnumber psychological causes; for articles published in France, psychological causes emerged as the etiology most written about, with neurological causes distinctly second, and genetic causes a distant third (36, 29, and 13 appearances, respectively). For Switzerland, both genetic and allergy/auto-immunological causations placed ahead of psychological etiology research.

The least referenced causations from the overall list are environmental, biological, and allergy/auto-immunological research articles. This order of incidence is not consistent for many of the individual countries, though. For articles published in the

United States, the allergy/auto-immune etiology appears 62 times versus 41 for biological causations. For England (and the United Kingdom), the bottom three are tightly grouped, with 21 biological cause articles, 16 environmental, and 15 allergy/auto-immune. France's least referenced three appear in this same order with far fewer numbers: 4 biological, 1 environmental, and zero allergy/auto-immune references. Switzerland published 11 allergy/auto-immunological autism causation articles, with only 8 citations each for biological or psychological causes; the environmental etiology appears only 4 times. Only 3 biological, 2 allergy/auto-immune, and 1 environmental causation article were published in Germany. The more inconsistent order of the less frequent causations may be more attributable to their meager numbers than to any lack of causal consensus regarding autism. When a single occurrence represents almost two percent of the total, as is the case with Germany, the utility of these percentages is difficult to gauge. This is why only the top 5 among the 44 countries appearing in the literature are discussed. (Refer to Appendix D, Table D-3 for the countries not discussed.)

By Source

Almost 93 percent of the sources are abstracts of journal articles. Only 21 conference proceedings, 21 book chapters, 15 monographs, 27 letters, 4 editorials, and 2 web site-based articles were found. One thousand one hundred sixty-nine journal articles came from 397 journals. The background information on the databases (see the "Data

Collection” section of the previous chapter) indicates that for most of the databased used, journals are what the database vendors concentrate on. Even so, a credible inference to make from the journal source percentage found is that a statistically significant portion of autism causation research is published in journal article format. Not as much can be inferred from the numbers per database because these numbers were influenced by the order in which the information was collected (see “By Database or Bibliography” beginning on page 64).

By Journal

As stated, 1,169 abstracts came from 397 journals. The 27 letters, 4 editorials, and 11 of the 21 conference proceedings were also published in this same group of journals. (The *Journal of Autism and Developmental Disorders* has a category called “brief reports” and some of the other journals have categories called “research” or “reports,” but this study made no distinction between these categories and journal article types.) The most frequently referenced journals appear in Table E-1, Appendix E. It should be no surprise that journals account for more than 9 of 10 autism causation sources. The timely dissemination of formally published information favors journals, which is why bibliometric studies of scientific or medical information would favor journals.¹⁰⁶ One issue is their currency of information, but other reasons journals are favored in bibliometric studies are easy to reckon: their articles are brief, more narrowly

¹⁰⁶ Nicholas and Ritchie, *Literature and Bibliometrics*, 123.

focused than monographs or even conference proceedings, and usually indexed individually.

The 53 journals having 5 or more articles account for 686 of the 1,211 articles, conference write ups, editorials, and letters counted in the 397 journals, or 56.65 percent. The United States-based *Journal of Autism and Developmental Disorders* and the *Journal of Autism and Childhood Schizophrenia*, its predecessor, account for 155 articles between them, or 12.31 percent of the total sources in this study. This finding has significance in the area of distribution and the next chapter contains a discussion of how Bradford's Law of Scattering applies to the autism causation topic.

Variance of Causation by Journal

All 13 of the early *Excerpta Medica* references came from Section VIII, "Neurology and Psychiatry." This is not too surprising considering this broad combination. (In recent years, the section is called "Neurology and Neurosurgery.") What is surprising is that no autism causations came from the biological or other general medical sections that did exist prior to the creation of EMBASE. What is less surprising—especially considering that the articles were published between 1950 and 1969, with only three published in the 1960s—is that psychological causations dominate the *Excerpta Medica* selections. Nine of the 16 causes given in the 13 volumes used cite a psychological causation. However, all of the other causations except

allergy/auto-immune are represented (neurological 3 times, biological twice, and one instance each of an environmental and a genetic causation).

The same spread of causations was evident across all of the journals with multiple instances of citations. Of course, one would expect a journal such as the *Journal of Autism and Developmental Disorders* and its precursor, the *Journal of Autism and Childhood Schizophrenia*, to reflect the same trends as the whole collection, but it does not (except that all causations are represented). The percentages for allergy/auto-immunological and environmental causations were more than twice as high (25 instances or 13.66%, contrasted with the 104 instances and 6.74% for the overall allergy/immunological causations and 28 environmental causations, or 15.30%, in contrast with the 114 environmental causations representing 7.39% of the overall totals). By contrast, the psychological causations attributed in the *Journal of Autism and Childhood Schizophrenia* and the *Journal of Autism and Developmental Disorders* combined make up less than half of the percentage that psychological causations have of the overall totals (21 for 11.48% of the former versus 366 overall psychological causations, 23.72% of the total—see Table 4.5 following). Part of this discrepancy can be explained by the timing of the *Journal of Autism and Childhood Schizophrenia*. The initial publication of the journal was 1971, nearly thirty years into the evolution of autism causation research.

Table 4.5. Number and Percentage of Autism Causations for Journals With the Highest Number of Autism Etiology Research Items Published

Etiology	Number of Causes/Percentage Per Journal					
	All Sources	JADD/JACS	Devel Med + Child Neuro	JAAC/AP	Amer J of Med Genet	JCPPAD
Aller/Imm.	104/6.74	25/13.66	1/1.92	2/4.17	0/0	0/0
Bio.	103/6.68	13/7.10	5/9.62	2/4.17	0/0	3/8.82
Envir.	114/7.39	28/15.30	2/3.85	4/8.33	1/2.86	1/2.94
Genet.	305/19.76	36/19.67	12/23.08	17/35.41	31/88.57	6/17.65
Neuro.	551/35.71	60/32.79	22/42.30	8/16.67	2/5.71	15/44.12
Psych.	366/23.72	21/11.48	10/19.23	15/31.25	1/2.86	9/26.47
Totals	1,543/100	183/100	52/100	48/100	35/100	34/100

Although the rank order of the causations is different for this primary autism publication, all causes are represented, with biological causations accounting for a little more than seven percent and neurological etiologies claiming nearly a third of the total. One would expect the other top journals cited in this research to favor a specific causation, if only based on their titles: *Developmental Medicine and Child Neurology* (neurological); the *Journal of the American Academy of Child Psychiatry*, the *Journal of the American Academy of Child and Adolescent Psychiatry*, and the *Journal of Childhood Psychology and Psychiatry and Allied Disciplines* (psychological); and the *American Journal of Medical Genetics* (genetic). However, this association holds true only for the last mentioned, in which 31 of its 35 causations are indeed attributed to genetics. Twenty-two of the 52 causations (42.30%) covered in the *Developmental Medicine and Child Neurology* selections are neurological; however, 12 are genetic and

10 are attributed to psychological causations as well (23.08 and 19.23 percent, respectively). The *Journal of the American Academy of Child Psychiatry* and its successor the *Journal of the American Academy of Child and Adolescent Psychiatry* combine for a total of 48 causations. Surprisingly, most of these are genetic, not psychological (17 versus 15, or 35.41% to 31.25%). Eight of the causations mentioned are neurological (16.67%). Similarly, psychological causations are the second most cited etiology in the *Journal of Childhood Psychology and Psychiatry and Allied Disciplines*. This time, though, the most cited causation is not genetic, but neurological (15 of 34, or 44.12%). Psychological causations account for only 9 (26.47%) and the genetic causations are third in number (6, for 17.65%). Again, these peculiarities may be explained at least in part by the time span the journals in question cover. *Developmental Medicine and Child Neurology* began 1965. The *Journal of the American Academy of Child Psychiatry* ran from 1963 to 1986, before being renamed the *Journal of the American Academy of Child and Adolescent Psychiatry* (which is still in publication). The *Journal of Childhood Psychology and Psychiatry and Allied Disciplines* was first published in 1977. Another influencing factor is that three of these most cited journals are published in the United States, and two in England (*Developmental Medicine and Child Neurology* and the *Journal of Childhood Psychology and Psychiatry and Allied Disciplines*). Therefore, the Anglo-American perspective is over-represented. However, this Western influence is mitigated by the fact that all of the most cited journals publish works contributed from research conducted internationally.

Non-Journal Sources

Subtracting the articles, letters, and editorials which came from journals leaves only 48 non-journal sources. Ten of the 21 conference proceedings, 21 book chapters, 15 monographs, and 2 web site-published articles are the sum of nonjournal sources used. The two web-based sources are two of many Internet articles that are repostings of work previously published the traditional way. However, the researcher did not find these two indexed in any of the bibliographic databases searched. The 21 conference write-ups are mainly indexed by BIOSIS *Previews* (9) and PsycLIT/PsycINFO (4). All but 2 of the 21 book chapters are abstracted in PsycLIT, the only database that makes "book chapter" its own index category. Seven of the 15 monographs are indexed in the National Library of Medicine's *Current Catalog*. BIOSIS and the Autism Resource page on the World Wide Web <<http://web.syr.edu/~jmwobus/autism/autism.faq>> each abstracted 3 monographs. The two remaining books are from the researcher's own library.

By Database or Bibliography

Discussion of the numbers per database needs to be prefaced with an explanation of how the researcher treated duplicate appearances of usable articles. Nicholas and Ritchie claim that duplication from one database to the next is no more than 75 percent.¹⁰⁷ In fact the OneSearch (of BIOSIS *Previews*, EMBASE, MEDLINE, and

¹⁰⁷ Nicholas and Ritchie, *Literature and Bibliometrics*, 132.

PsycINFO) run before the “remove duplicates” command was entered returned 2,905 articles; after the “rd” command, 2,086 articles remained (or 71.81%, meaning that nearly 28% of the articles were duplicates). However, the researcher also filtered out many false drops to finish with only 1,259 source items used. This final number is 60.35 percent of the 2,086 abstracts with duplicates removed. But the 1,259 also includes 157 sources not searchable automatically. For example, an April 1964 article appears in both the 1964 and 1965 runs of the *Cumulated Index Medicus*. Where the duplicate is a language translation or a conference report that later became an article or book chapter, these were not always filtered out automatically but manually removed (usually prior to entry in the project-specific database).

But even with those sources for which the duplicates could not be removed automatically, the order in which they were searched controlled the outcome to a degree. The researcher started with the OneSearch, then searched each of these database files’ print-based counterparts for the pre-digital years of bibliographic collection. Other sources—such as the National Library of Medicine’s *Current Catalog*, PsycLIT (to complement *Psychological Abstracts* and DIALOG’s PsycINFO for complete American Psychological Association source coverage), and genetic studies abstracted on the Web—were added next. For each source, more and more usable abstracts were repeats. The researcher noted these repeats, but did not count (or account for) them in the totals used from each source. Repeat occurrences of usable articles were viewed no differently from other false drops. In fact, articles that appeared in several databases were (of

necessity) counted only the first time. The thought going in was that only the final tally matters, not individual source totals. However, since source totals are the subject of this section, this background may help explain Table F-1 (Appendix F).

By Author

Of the 1,259 articles in the study, 632 come from authors appearing only once. Eighty-seven authors have two articles published in this research. Thirty-four authors were published three times each. Twenty-four authors contributed four articles each to the total. Eleven authors have published five articles on autism causation. Five authors have six articles each. Only three authors have had seven articles published on the topic. Five researchers have had their work published eight times. Only two authors were published nine times. One has been published ten times (Sanua). Ritvo has contributed 13 articles to the autism causation debate. Rutter and Wing each have had 15 articles published. Gillberg is clearly the most prolific on the topic with 38 articles published.

An explanation of how the authors were counted is useful here. Especially in the more recent years of autism research, the articles have been published by multi-author teams rather than individual researchers/writers. Therefore, only the lead author is tracked for this tally. While this may miscalculate instances in which a contributing author later became a lead author, the composition of the research teams has generally remained constant enough over the years that correcting for this instance is more time consuming than its benefit warrants.

Part of the reason such an effort would be laborious involves a shortcoming in the researcher's database design. However, another driver behind counting the research teams together was due to an unavoidable lack of information. The database designed for the researcher's data collection allows for only one author(s) field instead of several. The result of this early miscalculation is that the author-counting process is automated only down to the individual article level, not individual coauthors. However, many of the databases and bibliographies do not even include the entire list of authors, but use *et al.* after listing one to three contributors. There can be no work-around for this if the source is cited only in such a database or bibliography (short of the even more time-consuming task of finding the source itself).

A second fault of the "author(s)" field is its not including first names, or at least first initials. The researcher suspected this might be troublesome for common surnames, but did not predict wherein the larger problem would manifest itself. Many of the coauthors are married couples. The DIALOG-based files and their print precursors use only first initials of authors, but PsycLIT (a SilverPlatter database) and Genetic Studies Abstracts supply authors' full names. Of course, using the first initial would not have helped with Christopher and Carol Gillberg, but it would have with the Wings, Ritvos, and many other couples collaborating on autism causation research.

That stated, the method of author collection data is not so flawed as to be useless. More often than not, married researchers collaborate rather than publishing independent of each other. This and other factors make a case for emphasizing the lead

author. The authors, after all, put themselves in a particular (rather than an alphabetical) order usually based on the percentage of their contribution to the published article or research project. The databases use author order as it appears in the source document.

Do the Same Authors Stay with the Same Causes?

Individual authors tend to be consistent in the causes they espouse, but only slightly more consistent than are individual countries. The range of autism causations being investigated, even by the same author, is surprising. Taking the five most published authors, Table 4.6 provides a breakout of the autism causations each has reported. Only Rutter and Wing show clear causal preferences. Perhaps Gillberg and Ritvo, like many of the authors with fewer articles published, are drawn toward different avenues of research depending upon the team with which they are co-investigating. For example, seven of Gillberg's neurological causation articles are written alone. However, articles written with Hagburg and Wahlstrom tend toward genetic causality. The Ritvo articles written alone or with one partner tend to offer neurological causes, while most of those in which he is leading a six (or more) person team tend toward a genetic causation. This explanation does not account for the range given for Sanua's articles, all ten of which are solo efforts. However, as one can judge from the 18 causes in 10 articles, Sanua tends to be multicausal even in each single write-up.

Table 4.6. Number of Articles/Causes for the Most Prolific Authors

Author	# of Articles	# of Causes	Cause(s) and Instances
Gillberg	38	51	neurological (15), genetic (13), environmental (9), biological (7), psychological (5), immunological (2)
Ritvo	13	16	genetic (8), neurological (5), biological (1), psychological (1), immunological (1)
Rutter	15	21	neurological (11), genetic (4), psychological (4), biological (2)
Sanua	10	18	psychological (6), genetic (4), biological (4), environmental (3), neurological (1)
Wing	15	21	neurological (12), genetic (3), psychological (3), biological (2), environmental (1)
	91	127	Total Sources/Causations

Other Findings

Miscellaneous results worth noting include the emphasis on “childhood,” some direct conflicts in the literature, and a few database peculiarities of interest.

Childhood Autism

With the study of autism now more than fifty years old (not including investigations of the disability before it had this label), one would expect to see the emphasis on childhood gradually dissipate. However, despite the assertion from many sources that autism is a life-long disability for which there is no known cure, autism’s emphasis is and remains on childhood. The rationale for this could stem from the

DSM-IV's categorization of autism as a condition "Usually First Diagnosed in Infancy, Childhood, or Adolescence."¹⁰⁸ It should be no surprise, too, that those studying the cause of a condition are going to start at the beginning. Even so, the world of adult autism is under-represented in the literature. Only 9 of the 1,259 articles address adult autism. Twenty-one articles in this study begin with "Childhood Autism. . .," "Childhood Psychosis. . .," or "Childhood Schizophrenia. . ." "Early Infantile Autism" is the subject of 29 articles and "Infantile Autism" appears in 119 titles. Even the titles of the journals in which the autism causation articles appear reflect this childhood emphasis:

Developmental Medicine and Child Neurology, Journal of the American Academy of Child and Adolescent Psychiatry, Journal of Autism and Childhood Schizophrenia, Journal of Childhood Psychology and Psychiatry, Neuropsychiatrie de l'Enfance et de l'Adolescence, and Praxis Kinderpsychologie und Kinderpsychiatrie.

Other "autism" phrases encountered in the literature include "early childhood autism," "autismus infantum," and "infantile dementia." Of course, some of the autism characterizing words were broad enough to incorporate adult autism, like Kanner Syndrome (for "classic" autism) and Asperger Syndrome (for certain high-functioning autism, especially that in which the individual has normal speech). Earlier sources called these primary and secondary autism, or low- and high-functioning autism. The appearance of autistic-like traits in a patient not diagnosed with autism (or diagnosed with something else, like cerebral palsy or mental retardation), is often termed

¹⁰⁸ *Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition, 66-71.

“pseudoautism,” especially in the early years of research into autism. (However, all of the “autistic-like” and “pseudoautism” abstracts are listed as false drops for this research.)

It could be that parents are more likely to have their recently diagnosed children participate in studies, or that adults with autism have settled into the routine of it. Autism could be called something different—like schizophrenia—in adults. Or it could be simply that the formal diagnosis of “late onset autism” is much more rare than “childhood autism” or “infantile autism.”

Direct Conflicts in the Literature

In the previous chapter, the researcher addressed the handling of articles that were negating a cause without offering an alternative hypothesis, for example stating that autism is not genetic. What was not mentioned during that explanation is that at least 42 articles in this study directly oppose positions taken by many of the other articles. Four describe autism as not environmentally induced; one of these four offers no alternate proposal. One article states that autism may or may not be the same as childhood schizophrenia; two others claim that autism is the same as childhood schizophrenia. One article negates the biological causation. Four articles assert that autism is not neurological. Two sources specifically counter the cognitive causal theories and one other, the idea that autism is caused by hearing trouble in childhood. Two articles negate the immunological etiologies. At least five articles counter the psychological causations,

including one that insists autism is not caused by what the parents may have done or not done.

Especially for the genetic, biological, and psychological causations, this opposition demonstrates a voice, if not a majority. Also noticeable in the abstracts is the surety of the neurological proponents matched against the more open-ended causation wording of other proponents (wording such as “autism is a neurologically-based developmental disability” versus “allergies play an important role” or “. . . indicates a genetic component”).

Those proposing a connection between autism and Fragile X Syndrome, Rett’s Syndrome, or tuberous sclerosis have even more opposition in the literature. Two claim there is no link between Fragile X Syndrome and autism. Ten claim that autism is not genetic while four of these assert that Fragile X is genetic. Sometimes when two such articles appear in close proximity, the juxtaposition introduces the appearance of chaos in autism etiology research. All that these direct conflicts clarify is how little is supported about autism’s causation.

Database Peculiarities

The strengths and weaknesses of the online databases became a noticeable side issue during data collection. MEDLINE has a dearth of abstracts for its pre-1975 articles. BIOSIS *Previews* has more conference papers indexed but fewer journal articles than either MEDLINE or EMBASE. Even considering that PsycLIT was run after the

four DIALOG databases used, it yielded the fewest false drops (both clear false drops and unusable items in the researcher's opinion). PsycLIT is also the only source to index chapters of books.

In the on-paper sources, the pointers to autism vary greatly. *Biological Abstracts* is the only one that added key words in context to the subject listing. This proved extremely useful in that the researcher could distinguish immediately those that deal with treatments from those that cover causation. Improving the *Biological Abstracts* search even more is the inclusion of "Autism" as its own index category starting with the 1960 annual. Prior to this, autism fell under various subjects: infancy and childhood, psychiatry, and psychopharmacology. *Psychological Abstracts* characterized autism similarly. Although "autism" can be found alone in the indexes at the back of each annual volume, autism abstracts generally appear under the "Childhood and Adolescence," "Behavior Deviations," or "Receptive and Perceptual Processes" subject headings. In the National Library of Medicine's *Current Catalog* and *Cumulated Index Medicus* "autism" has been its own subject from its beginning (although 1964's is the earliest one used for the former and 1944 for the latter in this research). It has already been noted that all of the *Excerpta Medica* articles came from Section VIII, "Neurology and Psychiatry." However, the subsections used within this section include: "Psycho-pathology" and "Child Psychiatry."

Also noticeable is the progression of euphemisms from the early tomes to the digital databases, and the sensitivity with which disability wording has evolved. The early

abstracts (and by assumption, the articles they index) use wording that would be considered offensive, or at least harsh, in the 1990s: "autists," "retardates," and (for experimental control groups) "normals." Control groups in the 1980s and 1990s began to be called by the more accurate term "neurologically typical." "Handicapped" has all but given way to "disabled." And "subnormal" (mercifully enough) has become "exceptional," "special," and more commonly "individual with autism" in recent years.

The database files that give full journal titles are a boon (EMBASE and especially BIOSIS *Previews*, which makes "full journal title" its own field). However, for certain year-spans in their indexing, all but PsycINFO omit punctuation from their titles. For titles that should include colons or question marks, this could change the meaning, such as in the following examples: Gillberg's "Autism and Anorexia Nervosa: Related Conditions?" and Goodman's "Infantile Autism: A Syndrome of Multiple Primary Deficits?".

BIOSIS *Previews* and MEDLINE use a full list of authors, while EMBASE, PsycINFO, and PsycLIT tend toward *et al.* after listing the first one to three authors. Besides having the fewest multiple authors listed, EMBASE has the most misspellings in its database records. Also, fewer letters and editorials are cited than are used in the other DIALOG database files included.

Summary of Results

This chapter includes general results from all of the data collected and combines results for certain areas of interest, such as autism causation by decade or the types of causations being investigated from one country to the next. The quantitative results are broken out by type, time period, and location. Other pertinent findings—such as the emphasis on childhood, the appearance of direct conflicts in the literature, and specific database peculiarities—close this chapter.

The final chapter applies bibliometric analyses to the results described above. Bradford's Law of Scattering (concerning the number of journals that publish articles on autism causation), Goffman's Epidemic Theory (concerning how autism etiology article ideas are generated), and Lotka's Law (concerning author productivity on the specific topic of autism causation) will be explored. Other implications drawn from the data collected will also be discussed.

Chapter 5

Discussion and Implications

Overview

This chapter discusses results and implications derived during collection of the data. These outcomes are discussed using Bradford's Law of Scattering, Garfield's Law of Concentration, Goffman's Epidemic Theory of Communication, the Bandwagon Appeal in publications, and Lotka's Law of Author Productivity. Some nonbibliometric discussion is also incorporated into the implications, such as the evolution of autism literature and the appearance of "countries" that no longer exist. This chapter concludes by suggesting avenues of further research to pursue and how to continue autism causation research investigations.

General Discussion of Results

The null hypothesis offered in the first chapter is that no one cause will dominate the literature, that is, appear statistically more frequently than any other possible autism etiologies. Given the overall percentages from Table 4.2 (page 50), at 35.71 percent, the neurological causation leads the group, with psychological causations next at 23.72

percent of the sources and genetic causations with 19.71 percent. With more than a third for neurological, and nearly a quarter and one fifth for psychological and genetic, respectively, these etiologies are each more prevalent than the one sixth (or 16.67%) hypothesized. The allergy/immunological, biological, and environmental causations each have less than half of this expected outcome at 6.74 percent, 6.68 percent, and 7.39 percent. The neurological causations number 551 of the 1,543 etiologies found, a plurality but not a majority. However, this 551 is more than twice the 257.17 instances expected for an equal distribution of causation. The 366 psychological and 305 genetic etiologies found are not dramatically greater than the 257.17 expected, but they are when contrasted with the 104, 103, and 114 causations found for allergy/immunologic, biological, and environmental causations respectively. More detailed discussion of this appears in the "Implications for Hypothesis" subsection (starting on page 95).

Bradford's Law of Scattering

Three Zones

Lancaster and Warner provide a compact explanation of Bradford's Law in the midst of their explanation of how to determine the cost-effectiveness of databases.¹⁰⁹ They state that a comprehensive literature search over a specific period will reveal that literature on a specific subject is scattered over a large number of journals. Step one is to arrange these journals by most productive to least, then divide this list into three zones,

¹⁰⁹ Lancaster and Warner, *Information Retrieval Today*, 209.

each with an equal number of journal articles. Although the number of articles in each zone will match, the number of journals yielding these articles will vary greatly. A few journals will have a large majority, while many journals will be represented only once or twice. The utility of these three zones was never pursued or even explained very well in Bradford's work. However, Garfield's "Law of Concentration" finds an application for the three zones in using Bradford's Law specifically for scientific journals. All of the journals on a subject can be divided into multidisciplinary journals, more specialized journals, then "highly specialized" journals.¹¹⁰ The Bradford distribution for the autism causation data collected is shown in Figures 3 and 4, Appendix G, pages 138 and 140.

The difference between the two figures will be explained fully in the next section, but concerns the differences that result when combining journal name changes rather than counting each journal name distinctly. For this data each of the three zones (indicated by the dotted vertical lines in Figures 3 and 4) has approximately 404 of the 1,211 articles, letters, and reports that came from journals. Interestingly, the number in each zone approximates the constant discussed in the next section. (There is no reason this has to be true, but it is so only because of the current totals in the autism causation population. In ten more years the number of items in each zone could well be 500 or 600, but the constant may still be around 400.) In Figure 3, there are 15 highly specialized journals that account for the 404 articles in zone three. For Figure 4, this

¹¹⁰ Wallace, "Bibliometrics and Citation Analysis," 16.

number is 13. The trivial many (zone one) in Figure 3 come from 300 journals: 298 journals for Figure 4.

The Formula

Bradford first published this "Law of Scattering" in 1934 but did not provide a mathematical statement of how and why it works or determine the minimal nucleus for which the law holds.¹¹¹ He left this work to later scholars. In 1948, Vickery showed that this law "implies a J-shaped curve with an initial concave segment leading to a large linear segment and that such a curve gives a good fit to available data."¹¹² That Bradford's Law is constant for a wide range of bibliographic situations supports its utility as a predictive tool. Especially for the linear middle portion of the chart, a constant can be derived using the formula:

$$R(n) = k \times \log(n)$$

Where R is the total number of articles and n is any cumulative number of articles found in the set, one can solve for the constant (k) by multiplying one side of the equation by the logarithm of n and dividing this by the product of $R(n)$ that is on the other side of the equation.

Doing so at two points in each of the three zones in Figure 3 yields the following results (also see Table G-1, Appendix G). Where 223 is the cumulative number of

¹¹¹ Donohue, *Understanding Scientific Literatures*, 17.

¹¹² Ye-Sho Chen, P. Pete Chong, and Morgan Y. Tong, "Dynamic Behavior of Bradford's Law," *Journal of the American Society for Information Science*, 46, no. 5, 370.

articles (all of the journals publishing a single article), the k value is 515.76. Then at data point 377 the “constant” becomes 469.38. In the middle zone, where 526 is the cumulative number of journals $k=445.22$ and where 777 is the journal total, $k=419.03$. At the high end of the cumulative numbers, data point 892 yields a k value of 410.51 and at 1,108, the second to last data point, $k=398.35$.

For Figure 3, instead of remaining constant, the k value in the autism causation Bradford scatter drops as the number of journals increases. This progression is indicated by the slight curvature of the data line, even for the middle of the plot, which would be a straight line under ideal conditions. If the sample size were larger or otherwise less specific and limited, the expected constant might have been closer to a true constant. For example, if the researcher had used all autism literature (or possibly even all 2,086 of the DIALOG abstracts), the k values may have had a tighter grouping than the resulting numbers between 393.18 (the k value at 1,211 articles) and 515.76 (the k value at 223). (See Table 5.1, page 92, for all k values.)

However, if we first combine the numbers of journals that have undergone name changes, the constant is only a little closer to a true constant (see Table G-2, page 141) and the shape of the curve becomes more like the elongated “S” expected of a Bradford scatter when the three regions include the “significant few,” the “middle class,” and the “trivial many.”¹¹³ While Figure 3 favors the J-shaped curve mentioned by Vickery, Figure 4 (Appendix G, page 140) more closely matches the S-shape, with a more

¹¹³ Chen, Chong, and Tong, “Dynamic Behavior of Bradford’s Law,” 371-374.

noticeable “Gross Droop” (or convex curvature)¹¹⁴ at upper right, the area that depicts the most abstracted journals. The “Bradford Restriction” (the concave curvature at the lower left of each graph) is very prominent since the field of autism causation is heavily represented by one-time author contributions. The “significant few” (see the upper right of both Figures 3 and 4) change most dramatically from Figure 3 to Figure 4 since the second most abstracted journal on the subject—the *Journal of Autism and Childhood Schizophrenia*—is the 1971 through 1978 name of the most cited, the *Journal of Autism and Developmental Disorders* (with 52 and 103 articles used, respectively). The other pair of journals that significantly impacts the change in the Bradford distribution is the combination of the *Journal of the American Academy of Child and Adolescent Psychiatry* (with 19 selections), and its predecessor, the *Journal of the American Academy of Child Psychiatry* (with 16 articles used). Three other name changes are reflected in Figure 4. *Current Opinion in Pediatrics* started out as *Current Problems in Pediatrics* in the 1980s. These two combine for 8 articles. The *Japanese Journal of Child Psychiatry* also added *-and Adolescent* after *-Child* (in 1981, approximately). However, this changes the combined total only from four to five. Similarly, the *Psychiatric Clinics of North America* became the *Child and Adolescent Psychiatric Clinics of North America* by 1995. The recent name change, however, adds only one to the combined total (nine instead of eight).

¹¹⁴ Chen, Chong, and Tong, “Dynamic Behavior of Bradford’s Law,” 370.

Solving for the constant by using two data points in each of the three zones of Figure 4 yields the following results (also see Table G-2, Appendix G). Where 221 is the cumulative number of articles (all of the journals publishing a single article), the k value is 517.52. At data point 375, the “constant” becomes 471.21. In the middle zone, where 517 is the cumulative number of journals $k=446.86$ and where 777 is the journal total, $k=419.03$. At the high end of the cumulative numbers, data point 842 yields a k value of 413.31 and at 1,056, the second to last data point, $k=400.99$. Not only is the spread of what should be a constant somewhat wider in Figure 4 than it is in Figure 3 (starting at 517.52 versus 515.76), again instead of remaining constant the k value in the autism causation Bradford scatter drops as the number of journals increases. The only place the two graphs approach a constant is where the cumulative journal totals are in the 800s and the constant differs by less than two at each data point. As mentioned, though, this is a specialized application of Bradford’s Law since we are discussing only autism causation. A truer test would apply Bradford’s Law to all autism literature.

Although Wallace and Bonzi found a correlation between journal productivity (as measured by Bradford’s Law) and the frequency with which these journals were cited in later works,¹¹⁵ Wallace adds an important caveat to the Bradford scatter in stating “this approach requires acceptance of the unproven assumption that frequency of citation can be used as a measure of quality.”¹¹⁶ Donohue attempts to explain this dichotomy by

¹¹⁵ Wallace, “Bibliometrics and Citation Analysis,” 21.

¹¹⁶ *Ibid.*, 21.

stating that Bradford's is a statistical—rather than social—law.¹¹⁷ That a certain journal carries the most articles on autism causation literature does not necessarily make it the voice of authority on the topic. It may be simply the most accessible, or in the case of the *Journal of Autism and Developmental Disorders*, it may be the only journal specific to the topic. Bradford's Law of Scattering is intended to measure only quantity, not quality.

Goffman's Theory

Goffman attempted to get to this next step and incorporate quality into his “epidemic theory of communication.” His theory is that ideas spread like diseases; they are transmitted mainly by direct communication and spread among a susceptible population.¹¹⁸ He adds that the journal population is the controlling factor; that is, the number of authors is directly related to the number of journals that publish on a subject. Also, the number of “quality” authors is proportional to the total number of authors in the field.¹¹⁹ However, the importance of Goffman's theory lies in its “predictive power.” Just as statistics are predicted for epidemics, as a research topic emerges, Goffman's mathematical analysis may be applied to predict controlling conditions, dimensions of

¹¹⁷ Donohue, *Understanding Scientific Literatures*, x.

¹¹⁸ Pao, *Concepts of Information Retrieval*, 34-35.

¹¹⁹ *Ibid.*, 36.

growth, and the rate of slow down.¹²⁰ The other value of Goffman's work is that it points out the "infectious nature of ideas."¹²¹

It may be, however, that Goffman's theory is simply a sophisticated restatement of the "bandwagon appeal" which Price applies to author productivity in scientific writings. He noticed that once a person has successfully published a paper, the likelihood of him or her publishing a second is .33; the likelihood of a third is .50; for a fourth paper the probability goes up to .60 (and so on).¹²² Simply put, it is more likely that prolific authors will continue to write and publish than those who rarely do so.

Bandwagon Appeal

Nicholas and Ritchie note that bibliographic history, especially of journals, can divulge much about shifts in the direction of the subject literature. Changes in a journal's title, or more subtle changes in the terminology it uses, reflect changes in the subject covered.¹²³ For that matter, they indicate that the subject itself is the most important bibliographic characteristic and the most troublesome. While subject-orientation of bibliometric studies is pre-eminent, that there are essentially two ways of thinking about subject literatures complicates matters. One literature is defined by custom, the other by use. "The boundaries of the first are dictated by conventional thought, educational syllabi

¹²⁰ Pao, *Concepts of Information Retrieval*, 36.

¹²¹ Donohue, *Understanding Scientific Literatures*, 28.

¹²² Pao, *Concepts of Information Retrieval*, 34.

¹²³ Nicholas and Ritchie, *Literature and Bibliometrics*, 50.

and classification schemes. The boundaries of the second are much more fluid.”¹²⁴ If the “subject” itself presents a bibliometric problem as a quantitative judgment, subject content is even more troublesome. Most bibliographic attributes are outward features of a work, but to discuss the subject content is to get at the document’s “inner self.”¹²⁵ It is easier to discuss the container but much more interesting and useful to describe its contents.

Wallace, however, points out an even more fundamental problem with bibliometrics; it lacks a well-developed theoretical basis.¹²⁶ He likens it to the “cumulative advantage theory”¹²⁷ of Derek de Solla Price and explains Price’s theory in part by comparison to Saint Matthew’s “For to him who has will more be given, and he will have abundance; but from him who has not, even what he has will be taken away.”¹²⁸

While some evidence of the bandwagon appeal can be found in the growth of allergy, chromosomal, and brain research in recent years, this increase in percentages is more likely attributable to the improved technologies which had made such research impossible or prohibitively time consuming prior to the 1970s. Evidence of the bandwagon appeal in autism causation literature is further weakened by the fact that the

¹²⁴ Nicholas and Ritchie, *Literature and Bibliometrics*, 76.

¹²⁵ *Ibid.*, 83.

¹²⁶ Wallace, “Bibliometrics and Citation Analysis,” 19.

¹²⁷ *Ibid.*, 20.

¹²⁸ *Matthew, Holy Bible*, revised standard version, (Nashville, TN: Thomas Nelson, Inc., 1972), 13:12, 13.

growth in the other areas of research did not eliminate psychological etiology research, only slowed its growth (see Figure 2, page 126). Although technological advances in the other areas may have displaced psychological causations to a degree, even in the most recent decade of data used, psychological causations still account for 116 causations cited, well ahead of allergy-auto-immune (58), biological (52), and environmental (47) research.

Better evidence of the bandwagon appeal can be found not in the causes undergoing investigation, but in the emphasis on childhood and progression of opinion toward autism as a spectrum disorder. Rapin mentions many early deficit areas under research: sensory, cognitive, attention, social, behavioral, and language development.¹²⁹ Hart adds that because autism shows hereditary as well as environmental factors, it is properly characterized as a syndrome.¹³⁰ Even the *DSM-IV* describes autism as a “pervasive developmental disorder.”¹³¹

As mentioned during the discussion of Bradford’s Law, at least five of the most-cited journals in autism etiology have undergone name changes, most to add “and adolescent” to the “child” already in their title. However, the 1978 name shift of the main autism journal from the *Journal of Autism and Childhood Schizophrenia* to the *Journal of Autism and Developmental Disorders* directly addresses what Nicholas and Ritchie

¹²⁹ Rapin, “Introduction and Overview,” in *Neurobiology of Autism*, 9.

¹³⁰ Hart, *Parent’s Guide to Autism*, 49-50.

¹³¹ *Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition, 77-78.

mean by a name change indicating a shift in direction of the subject literature.¹³² In the late 1970s autism became separated from adult schizophrenia and linked with a failure to meet the developmental milestones expected during the toddler years. This shift in thought permeated all or most of the autism causations under investigation.

As for the causations themselves, there is not so much a concentrated adding to the etiology of the moment (characteristic of the bandwagon appeal) as the rapid growth of certain causations (like neurological and genetic) concomitant with the slower growth of other etiologies (as with biological and environmental). Although psychological causations have been declining as an annual percentage, the overall number of psychological etiology articles continues to have up and down years (even into the 1990s) rather than showing a true decline in numbers. The Figure 2 bar chart (page 126) and Table C-1's year by year summary of autism causations (pages 124 and 125) make these progressions obvious.

Epidemic Theory

Evidence of Goffman's epidemic theory at work in autism causation research literature is more difficult to uncover than even the bandwagon appeal. The main evidence of it is that once a possible causation appears in the literature, it never goes away. This is especially surprising when considering that the causation studies are not limited to the six causal areas this researcher decided on. There are organic, cognitive,

¹³² Nicholas and Ritchie, *Literature and Bibliometrics*, 50.

and gestational possibilities, as well as suspected associations with rubella, various viruses, and several other disabilities. All appear in different years, but none fade. Psychological etiologies appeared first, followed by neurological causations. Both continue to appear, along with all four of the other possibilities, including the latest addition to the argument, allergy/autoimmunological causations in 1965.

Even so, there are some hypotheses under investigation about the causation of autism that have been followed up by only a few later scholars. Many of these are narrowly focused studies within the broader psychological, environmental, and genetic fields already discussed. A study of the head circumference of children with autism compared with non-autistic peers is not as odd as it sounds initially. The theory that the brain, or at least certain regions of the brain, are physically larger than usual with autism started being studied post-mortem in the 1980s (mainly by Bauman and Kemper).¹³³ However, some of the physical evidence for autism studies stretch the limits of scientific/medical research. Dermatoglyphics, the study of skin patterns, especially those of the hands and feet, has been useful in identification and genetic studies.¹³⁴ Probably due to the genetic connection, dermatoglyphics has been applied to autism research. Six of the sources used specifically address this area, usually in connection with genetic or environmental causation research. Other genetic and environmental studies less grounded in science include one that investigates clumsiness as a genetic marker for Asperger

¹³³ *Advocate* (Bethesda, MD: Autism Society of America, 24, no. 4), 1, 13-15.

¹³⁴ *Taber's Cyclopedic Medical Dictionary*, 389.

Syndrome¹³⁵ and another testing the prevalence of March birthdays among individuals with autism.¹³⁶

Research that is even more unusual comes from those investigating a psychological etiology for autism. Despite Coleman's assertion in 1990 that "parental culpability. . .is no longer considered a viable hypothesis in an overwhelming majority of cases,"¹³⁷ many countries and individuals continue to investigate psychological causations. As recently as 1994, Frances Tustin claimed autism resulted from a failure of the mother and child to bond. "Autism is a system of protective, but alienating autosensual aberrations . . . developed to deal with an infantile trauma."¹³⁸ As evident from the overall tally, Tustin is not alone. Such causes as a dysfunction of mother-infant metacommunication, nipple separation anxiety, and pre-Oedipal manifestations have been investigated. One study in 1958 had the mothers do Rorschach inkblot tests and concluded that "mothers of children with primary autism have less capacity for establishing social and emotional relationships than do mothers of children with secondary autism."¹³⁹ One study even implicated the father.¹⁴⁰ The personality traits of

¹³⁵ In DIALOG file #73, EMBASE: M. Ghaziuddin, E. Butler, L. Tsai, and N. Ghaziuddin, "Is Clumsiness a Marker for Asperger's Syndrome?" *Journal of Intellectual Disabilities Research*, 1994, 38/5: 519-527.

¹³⁶ In DIALOG file #155, MEDLINE: C. Gillberg, "Do Children with Autism Have March Birthdays?" *Acta Psychiatrica Scandinavica*, 1990, 82(2): 152-156.

¹³⁷ In DIALOG file #5, BIOSIS Previews: M. Coleman, "Delineation of the Subgroups of Autistic Syndrome," *Brain Dysfunction*, 1990, 3(5-6): 208-217.

¹³⁸ In DIALOG file #73, EMBASE: Frances Tustin, "Revised Understandings of Psychogenic Autism," *International Journal of Psycho-Analysis*, 1991, 72/4: 585-591.

¹³⁹ *Biological Abstracts*, Volume 36, Eva Bene, "A Rorschach Investigation into the Mothers of

the parents as a cause of autism has been investigated since the beginning of autism research, not as an epidemic but as an area that some researchers like Tustin keep coming back to. As early as the 1940s Asperger noted behavioral similarities between his clients and some of their parents.¹⁴¹

Team authorship has played a role in the epidemic spread of causations. In the early years, much of the work was being done by individual investigators. In recent years, teams of researchers have broadened the scope of autism etiology possibilities. As mentioned in the "By Author" section of the previous chapter, even authors who show a causation preference while writing alone demonstrate a wider range of etiology investigations in their team summaries. This may be attributable to some group dynamic variant of the bandwagon appeal, but it may be a limited manifestation of the epidemic theory in action. The beliefs and background that each investigator brings to the project can have a mitigating or exacerbating influence on the other members of the research team in ways more subtle and profound than might only the prior publications of each. Of course, it may be simply that no individual or group research results to date have provided a genuine "front-runner" causation convincing enough for researchers in other areas to rally behind.

Autistic Children," *British Journal of Medicine and Psychology*, 1958, 31: 226-227.

¹⁴⁰ In DIALOG file #155, MEDLINE: M.E. Lobascher, P.E. Kingerlee, and S.S. Gubbay, "Childhood Autism: An Investigation of Aetiological Factors in Twenty-Five Cases," *British Journal of Psychiatry*, 1970, Nov 117: 525-529.

¹⁴¹ Rapin, "Introduction and Overview," in *Neurobiology of Autism*, 1.

Lotka's Law

Because of the limited topic, Lotka's Law of Author Productivity does not hold up well in the field of autism causation research publications. Although the parabolic curve of Figure 5 (Appendix H, page 143) roughly matches the data points expected of an author productivity distribution (see Figure 6, also in Appendix H), solving for a constant results in even more disparate numbers than Bradford's Law revealed for the journals. Koenig and Harrell's article summarizes the arithmetic involved in Lotka's Law so: "in a given body of literature, the number of authors contributing n papers is about $1/n^2$ of the number of authors contributing just one paper Thus if two thousand authors have written just one paper, then roughly $1/10^2 \times 2,000$, or only about 20 persons would have written ten papers."¹⁴² An equation to solve for the constant (k) in Lotka's Law is

$$k = n^2 \times p$$

where n is the frequency of occurrence for an author and p is instances of this number of authors being published.

Applying Lotka's Law of Author Productivity to the data given in the "By Author" section of the previous chapter, Table 5.1 supplies the k values found for autism causation literature and what these values should be based on a total of 632 authors appearing once. Notice that the resulting k values are not constant. In fact, the data points do not show a clear progression or regression (as was true for the Bradford data),

¹⁴²Michael Koenig and Toni Harrell, "Lotka's Law, Price's Urn, and Electronic Publishing," *Journal of the American Society for Information Science*, 46, no. 5, 386.

but rise and fall unexpectedly (see the third column of the following table). Modeling an expected constant based on the data having 632 authors who have been published only once clarifies how far from expectations the actual data are.

Table 5.1 Actual and Expected Author Productivity Outcomes

Articles by the Same Author	Instances in Autism Causation Sources	Resulting k value	Expected Instances if the Constant Were Equal to 632
1	632	632	632
2	87	348	158
3	34	306	70
4	24	384	40
5	11	275	25
6	5	216	18
7	3	147	13
8	5	320	10
9	2	162	8
10	1	100	6
13	1	169	4
15	2	450	3
38	1	1,444	1*
Article Totals	1,259		1,989

The decrease from 632 authors being published once to 87 authors being published twice is too rapid to be adequately explained by Lotka's Law. The fourth column depicts the expected representation of authors having two, three, etc. articles published while the second column shows the actual values for single, double, triple, etc. publications by the same author. Either the 632 instances of an author being published only once is too high for a body of only 1,259 sources or the 38 times that Gillberg has

been published is too high for the opposite end of the set. If the constant of 632 were to work out, then the author published most frequently (the asterisk in Table 5.1) should have only 25 or 26 articles published [$632=25.14^2(1)$]. The other indication that the 632 k value is too high is that it would result in a total of 1,989 sources, 730 more than the actual number.

This is not to imply that the predictive value of Lotka's Law is questionable, only that its utility in this specialized application is limited. This investigation addresses only autism causation literature, not the entire body of autism or even autism treatment literature. Perhaps with a broader sample to use, the formula would hold up better. This factor combined with the limits placed on counting authorship (as described in the "By Author" section of Chapter 4) mitigate the usefulness of Lotka's Law as a predictive tool. However, its utility as an analytical tool—demonstrating expected outcomes versus actual—is enhanced by the uniqueness of this data set.

Does the Multidirectional Approach of Autism Causation Research

Imply No Clear Consensus?

An apparent lack of consensus by time span, author, journal, or country prompted this research. The notion of consensus finding quickly gave way to simple counting in an attempt to discover a percentage majority. However, the results of even this exercise show how diverse autism research has been and remains.

In addition to the causations and percentages discussed, the articles collected can be further subdivided into the number of causations that each produced. Of the 1,259 sources in the study, 991 are single-causation articles (78.71%). Two hundred forty-four (19.38%) of the sources present dual causations. There are also 22 articles (1.75%) that posit a triple factor etiology for autism. Two abstracts (0.16%) discuss four etiologies, but these are summary articles: one covering the "biochemistry" and "psychophysiology" of autistic syndromes¹⁴³ and the other tracing autism back to possible genetic, neuronal, and virus developments concomitant with bleeding during the second trimester of gestation.¹⁴⁴ The main point is that 268 of the 1,259 sources could not even limit themselves to a single etiology to support. It is almost the opposite of the epidemic theory or the bandwagon appeal.

Does the multidirectional approach to autism etiology research imply no clear consensus, or is this issue merely a symptom of a problem far worse? If the factional research being done is the result of something completely unrelated to autism (such as funding battles, pride, or politics) this would be unfortunate enough. However, the more likely reason for this lack of consensus is that autism involves such a range of developmental delays and ability levels that the researchers cannot focus on the root of this handicapping condition. High-functioning autism looks completely different from

¹⁴³ In DIALOG file #155, MEDLINE: K. L. Reichelt, "Autistiske Syndromers Biokjemi og Psykosofysiologi (The Biochemistry and Psychophysiology of Autistic Syndromes)," *Tidsskr Norway Laegeforen*, May 10, 1994, 114(12): 1432-4.

¹⁴⁴ In DIALOG file #5, BIOSIS Previews: M. Coleman, "Second Trimester of Gestation: A Time of Risk for Classical Autism?" *Developmental Brain Dysfunction*, 1994, 7(2-3): 104-109.

low-functioning autism. While neurologists and developmental pediatricians have made refinements in labeling a range of behaviors and abilities for each, the fundamental issue remains unresolved: whether autism is truly a spectrum disorder or many distinct kinds of retardation all classified together due to similar diagnostic behavioral traits. As a result, the even more fundamental issue of how autism occurs remains open to speculation and study.

Implications

Implications for Hypothesis

The null hypothesis of this research was that no one cause would claim a majority percentage of the literature. While this is supported, since the most cited causation is neurological (551 of the 1,543 causes collected) with 35.71 percent, the utility of deriving an expected average number of causations per etiology to discuss standard deviation boundaries for the causes found was not pursued. The causations arrived at are inherently contrived, if not arbitrary. A researcher with different thoughts and beliefs about autism could make a cogent case for combining the immunologic and allergy-induced causations with the environmental etiology. The same with biological and neurological causes. Conversely, difficulties during pregnancy (the pre-, peri-, and neonatal causation) could be tracked as its own category, instead of part of the environmental causation. Cognitive and organic could have been tracked individually also. The researcher could have separated social development from the psychological

causation, or have aligned developmental delays with cognitive delays as examples of the neurological etiology. Actions a bit more drastic but justifiable would also result in a shift in the totals. For example, using only the past twenty or twenty-five years and dismissing earlier research as archaic would have greatly reduced the psychological causations.

Even so, to add more etiologies would make the original hypothesis even less likely. If an individual causation did not appear more than half of the time with only six causations, it is not very likely any would with even more causations tracked. Therefore, despite all of the variables and judgments involved, consistent constraints consistently applied should continue to yield consistent results. The causation set is not arbitrary through any fault in the data collection, but reflects the dispersion of autism causation literature. This is the most important finding of this bibliometric research.

Geographical Issues

The United States' apparent dominance of autism causation literature (621 of the 1,259 sources and 746 of the 1,543 etiologies) is mitigated by two factors. The primary autism journal—the *Journal of Autism and Developmental Disorders*—is published in the U.S. but includes foreign authors and research findings. The other mitigating factor is that the data collection focused on English language abstracts. The latter limitation favors American and British journals, but the overall data show only limited indications of being pulled in the Anglo-American direction. The American percentages for

neurological, biological, and psychological causations are lower than the overall percentages, while allergy/immunological, environmental, and genetic etiology percentages are slightly higher for the United States (reference Table 4.4, page 57). The neurological, psychological, genetic, environmental, allergy/immunologic, and biological rank order of the overall causations differs for the U.S. as well, with genetic etiologies second and psychological third. Also, although their order is the same, there is a larger gap between the allergy/immunological causations and the biological ones than is evident in the overall numbers. For causation articles published in England combined with those attributed to the "United Kingdom," the percentages of each causation are one to two points higher for the neurological, psychological, and genetic etiologies, nearly the same for biological (6.84% versus the overall 6.68%) and about two points lower for environmental and allergy/immunologic causations. When the publications attributed to the United Kingdom are added to those attributed to England, the rank order of the top three is the same but the order of the bottom three causations is completely different. Interestingly, although the overall biological percentage is nearly the same as the British percentage, this etiology ranks last overall but fourth for England/United Kingdom. Allergy/immunologic etiologies place last in British research with environmental causations second to last. The 21 biological causations attributed to the England and U.K. articles combined is considerably higher than the 16 environmental and 15 allergy/auto-immunological etiologies found. As represented in Table D-3, Appendix D,

this is an accurate representation that the belief in a biological basis for autism is stronger in Europe than it is in the United States.

How the United Kingdom is distinguished from England in the databases brings to the fore another geographical issue worth noting: the geo-political lessons underlying this data collection. Some of the sources attribute the place of publication to the United Kingdom, others to England, the end result being 121 articles for the U.K. (and 155 causations) and 121 articles from England (152 causations). While articles listed as coming from England can be comfortably ascribed to just this country, the United Kingdom articles are not so clear. Especially with Ireland (there are no Northern Ireland items in this research, however) and Scotland appearing separately in the study (as well as Australia and Canada, which are part of the British Empire, though independent of the U.K.), how synonymously “United Kingdom” and “England” are being used from one database to the next is not clear.

The reason behind the division of certain other countries is more clearly a matter of when individual articles were published. Germany was partitioned into East and West for most of the 1943 through 1996 data span. The numbers found reflect this, with 42 of the 48 “Germany” sources coming from West Germany and only four (post 1990 ones) from the reunified Germany. Similarly, eleven articles are from the Union of Soviet Socialist Republics, with only one 1994 publication from Russia (and none from the other former Soviet Republics). Czechoslovakia has three sources included in the research; the recently created Czech Republic has one. The Yugoslavia that appears

three times in this data set is the one that existed from 1943 to 1992, roughly the period of this research. (The present-day Federal Republic of Yugoslavia was created in 1992 from parts of the former country.)

Implications for Further Research

Short of some breakthrough in one causal area of research, clarification of what is and is not autism (then, possibly, what causes it) should become clear by further tracking of the growing body of research. Following up on the numbers and percentages as they were tracked herein will allow replication researchers to add later author, journal, country, and database statistics to these results and make the same comparisons.

Further research might do more with Goffman's Epidemic Theory of Communication also. This research concluded only that since new causes surface and never disappear, the theory that specific causal categories will group together for a certain time span before giving way to autism etiology research in a different vein is partially true. Adding a comparison of the various names for autism at specific times and matching this to the causation statistics for these time spans may provide a correlation only touched upon in this research. In the previous chapter, the discussion of this aspect of autism focused on only how difficult the various labels make tracking autism etiologies. The more prominent result showing that every country has been investigating every cause in almost every decade reveals that instances of every etiology have always dominated the discussion. This point was the focus of this research. But investigating

the divergence and uniqueness in autism's labels could be a fruitful exercise if combined with grouping the causations popular when each term for autism was at its peak use. For example, one might expect to find that the psychological causation was the most investigated autism etiology when autism and childhood schizophrenia were considered synonymous.

In addition to clarifying what is and is not autism, another benefit of collecting the data for a few more years is that this would demonstrate the direction and influence of the most recent autism causation research. Along with advice to do this and create new Bradford and Lotka graphs, this researcher makes the following suggestions to improve the data collection phase.

Separate the country wherein the research was done (usually the native country of the authors) from the country that published the work. Most databases allow for this, or at least supply enough information for reasonable surmises. For example, after listing the authors, MEDLINE gives the university or institute and its location. After the journal title, the country of publication is parenthetically supplied. EMBASE similarly separates the place of scholarly research from the place of publication. BIOSIS *Previews* and PsycINFO supply the place the research was conducted but do not provide the country in which the named journal is published. The latter, however, does indicate in what language the article is written. Another advantage to this avenue of database information tracking is that it may clarify how having the majority of the research conducted in an individual country or by an individual university may be constricting an international

consensus on what causes autism. However, conclusions could be difficult to draw since the print bibliographic sources are not as good about separating the place of the research from the place of publication. Only *Excerpta Medica* does; the rest make getting even this basic information challenging. However, as Goffman indicated, the information retrieval itself is a dynamic process,¹⁴⁵ and source knowledge gained from data collection will allow the researcher to fill in most gaps. Perhaps the language in which a piece is written does not always provide a valid approximation of where the work occurred, but taken in conjunction with other abstract fields, this information could provide better accuracy.

Another suggestion for the data collection phase is that of designing the database to search multiple authors individually, since only 525 of the 1,259 pieces are by individual authors. Even before adding more years to the research, it would be interesting to contrast what such data might reveal through Lotka's Law versus what was shown using the primary author method this research employed.

Similarly, a follow-on study should allow for rechecking the United States' dominance of the publications by introducing true foreign medical and psychological abstract collections. Using not just translated sources but the National Library of Medicine's sources as well (such as the African, Israeli, Korean, and Latin-American versions of *Index Medicus*) may supply a compelling contrast with the international data presented here.

¹⁴⁵ Donohue, *Understanding Scientific Literatures*, 28.

Finally, pursuing other measures in addition to those of Bradford, Goffman, and Lotka might place more emphasis on the quality of the sources rather than their quantity. Another area of bibliometrics is citation analysis, which investigates the “practices and patterns of scholarly references.”¹⁴⁶ The point is not merely to enumerate from bibliographic listings but that (as Nicholas and Ritchie elegantly put it) “[d]escriptive studies may seek to show how individual members of a literature possess common features and may be grouped according to these affinities.”¹⁴⁷ As early as 1927, Gross and Gross conducted a study which looked at not only citation relationships and emerging patterns, but also each author’s possible motivation for citing a particular work.¹⁴⁸ Other researchers have continued this, exploring the arithmetic of the “critical measure” of “coupling units.”¹⁴⁹

While research dynamics are important to keep in mind, the method for showing the “interconnectedness of citations”¹⁵⁰ is the main thrust of citation analysis. Price claimed that each year there are 7 new papers published for every 100 previously published in that same subdiscipline. An average of about 15 references in each of the 7 papers will yield 105 references back to the original 100, for a citation average of a little

¹⁴⁶ Wallace, “Bibliometrics and Citation Analysis,” 10.

¹⁴⁷ Nicholas and Ritchie, *Literature and Bibliometrics*, 27.

¹⁴⁸ Wallace, “Bibliometrics and Citation Analysis,” 18.

¹⁴⁹ *Ibid.*, 27.

¹⁵⁰ Donohue, *Understanding Scientific Literatures*, 3.

over one each.¹⁵¹ In reality, though, some are never cited, a small group is heavily cited for a few years, and a small percent (the “citation classics”) continue to be cited year after year. Among the works cited heavily for a few years, there is a balance between the value of the information increasing with usage and the “half life” of the topic. “[H]alf life has been interpreted as the time during which half of the total use of individual items has been or is expected to be made.”¹⁵² However, these numbers do not account for re-citations; that is, newer works using the ideas first presented in works which are now obsolete.

Problems with measuring obsolescence stem from how this term—as well as the related term “half-life”—are defined. As mentioned above, the ideas from some obsolete works are reused in newer articles. Lancaster and Warner, however, describe the law of obsolescence from a database user’s perspective. They explain that, especially in science and technology, the probability of demand for materials declines as the materials age.¹⁵³ However, they add that median use, or half-life is the number of years that are needed to satisfy half of the requests. That is, if fifty percent of the hits come from the last three years, this is the “half-life” for the subject being searched.

Two other problems in defining and using obsolescence are both related to quality. Frequency of citation is not necessarily a measure of quality. Related to this is

¹⁵¹ *Ibid.*, 24.

¹⁵² Pao, *Concepts of Information Retrieval*, 18.

¹⁵³ Lancaster and Warner, *Information Retrieval Today*, 211.

that—while Bradford's Law has limited utility as a collection development tool—most collection development is done on a qualitative basis, not the quantitative basis on which Bradford's Law centers.¹⁵⁴ Therefore, the main value of adding obsolescence or citation analysis to the discussion of autism causation literature is to introduce a look at quality that was not included in this thesis.

Summary of Discussion and Implications

In this final chapter, the outcomes from the previous chapter and their implications were discussed in the context of bibliometric information mapping using traditional information sciences methods. This application of bibliometric analyses involved a nonstandard use of standard information science tools such as Bradford's Law of Scattering, Goffman's Epidemic Theory, and Lotka's Law of Author Productivity. Suggested methods of data collection and the use of citation analysis were also introduced as possible considerations for a follow-on study of autism causation literature.

The limits placed on the information mapped account for some of the nonconformance with expected outcomes. Even so, the results conformed to expected outcomes in more ways than they did not, and they show that autism etiology research has taken many roads and continues in many divergent directions. As the body of information concerning autism causation continues to grow, the application of

¹⁵⁴ Wallace, "Bibliometrics and Citation Analysis," 25.

bibliometric measures should grow more accurate and more predictive of the future of autism causation research, perhaps showing where all of these paths converge.

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APPENDICES

Appendix A

b5,11,73,155

10jul97 04:54:40 User077446 Session D203.1

\$0.50 0.033 Hrs FileHomeBase

\$0.50 Estimated cost FileHomeBase

\$0.50 Estimated cost this search

\$0.50 Estimated total session cost 0.033 Hrs.

SYSTEM:OS - DIALOG OneSearch

File 5:BIOSIS PREVIEWS(R) 1969-1997/Jul W2

(c) 1997 BIOSIS

File 11:PsycINFO(R) 1967-1997/Aug

(c) 1997 Amer. Psychological Asso.

File 73:EMBASE 1974-1997/Jun W5

(c) 1997 Elsevier Science B.V.

File 155:MEDLINE(R) 1966-1997/Aug W4

(c) format only 1997 Knight-Ridder Info

Set Items Description

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?ss autism?

5: BIOSIS PREVIEWS(R)_1969-1997/Jul W2

3598 AUTIS?

11: PsycINFO(R)_1967-1997/Aug

5345 AUTIS?

73: EMBASE_1974-1997/Jun W5

3884 AUTIS?

155: MEDLINE(R)_1966-1997/Aug W4

4845 AUTIS?

TOTAL: FILES 5,11,73 and 155

S1 17672 AUTIS?

?ss pdd or PDD or pervasive(w)developmental(w)disorder

5: BIOSIS PREVIEWS(R)_1969-1997/Jul W2

401 PDD

852 PERVASIVE

73450 DEVELOPMENTAL

84898 DISORDER

117 PERVASIVE(W)DEVELOPMENTAL(W)DISORDER

401 PDD

497 PDD OR PDD OR
PERVASIVE(W)DEVELOPMENTAL(W)DISORDER

11: PsycINFO(R)_1967-1997/Aug

115 PDD

1442 PERVASIVE

33625 DEVELOPMENTAL

37083 DISORDER

161 PERVASIVE(W)DEVELOPMENTAL(W)DISORDER

115 PDD

236 PDD OR PDD OR
PERVASIVE(W)DEVELOPMENTAL(W)DISORDER

73: EMBASE_1974-1997/Jun W5

381 PDD

1080 PERVASIVE

122343 DEVELOPMENTAL

283049 DISORDER

110 PERVASIVE(W)DEVELOPMENTAL(W)DISORDER

381 PDD

467 PDD OR PDD OR
PERVASIVE(W)DEVELOPMENTAL(W)DISORDER

155: MEDLINE(R)_1966-1997/Aug W4

410 PDD

1283 PERVASIVE

57344 DEVELOPMENTAL

110698 DISORDER

119 PERVASIVE(W)DEVELOPMENTAL(W)DISORDER

410 PDD

507 PDD OR PDD OR
PERVASIVE(W)DEVELOPMENTAL(W)DISORDER

TOTAL: FILES 5,11,73 and 155

S2 1307 PDD

S3 1307 PDD

S4 4657 PERVASIVE

S5 286762 DEVELOPMENTAL

S6 515728 DISORDER

S7 507 PERVASIVE(W)DEVELOPMENTAL(W)DISORDER

S8 1707 PDD OR PDD OR
PERVASIVE(W)DEVELOPMENTAL(W)DISORDER

?ss s1 or s8

5: BIOSIS PREVIEWS(R)_1969-1997/Jul W2

497 S8

3598 S1

4002 S1 OR S8

11: PsycINFO(R)_1967-1997/Aug

236 S8

5345 S1

5469 S1 OR S8

73: EMBASE_1974-1997/Jun W5

467 S8

3884 S1

4257 S1 OR S8

155: MEDLINE(R)_1966-1997/Aug W4

507 S8

4845 S1

5245 S1 OR S8

TOTAL: FILES 5,11,73 and 155

17672 S1

1707 S8

S9 18973 S1 OR S8

?ss s9 and caus?

5: BIOSIS PREVIEWS(R)_1969-1997/Jul W2

4002 S9

518449 CAUS?

206 S9 AND CAUS?

11: PsycINFO(R)_1967-1997/Aug

5469 S9

38896 CAUS?

266 S9 AND CAUS?

73: EMBASE_1974-1997/Jun W5

4257 S9

526198 CAUS?

271 S9 AND CAUS?

155: MEDLINE(R)_1966-1997/Aug W4

5245 S9

533879 CAUS?

285 S9 AND CAUS?

TOTAL: FILES 5,11,73 and 155

18973 S9

S10 1617422 CAUS?

S11 1028 S9 AND CAUS?

?ss s9 and etiol?

5: BIOSIS PREVIEWS(R)_1969-1997/Jul W2

4002 S9

56323 ETIOL?

163 S9 AND ETIOL?

11: PsycINFO(R)_1967-1997/Aug

13248 ETIOL?

5469 S9

382 S9 AND ETIOL?

73: EMBASE_1974-1997/Jun W5

4257 S9

761704 ETIOL?

811 S9 AND ETIOL?

155: MEDLINE(R)_1966-1997/Aug W4

5245 S9

996423 ETIOL?

802 S9 AND ETIOL?

TOTAL: FILES 5,11,73 and ...

18973 S9

S12 1827698 ETIOL?

S13 2158 S9 AND ETIOL?

?ss s11 or s13

5: BIOSIS PREVIEWS(R)_1969-1997/Jul W2

163 S13

206 S11

344 S11 OR S13

11: PsycINFO(R)_1967-1997/Aug

266 S11

382 S13

579 S11 OR S13

73: EMBASE_1974-1997/Jun W5

271 S11

811 S13

985 S11 OR S13

155: MEDLINE(R)_1966-1997/Aug W4

285 S11

802 S13

997 S11 OR S13

TOTAL: FILES 5,11,73 and 155

1028 S11

2158 S13

S14 2905 S11 OR S13

?rd s14

...examined 50 records (50)

...examined 50 records (100)

...examined 50 records (150)

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...completed examining records

Processing

S15 2086 RD S14 (unique items)

Appendix B

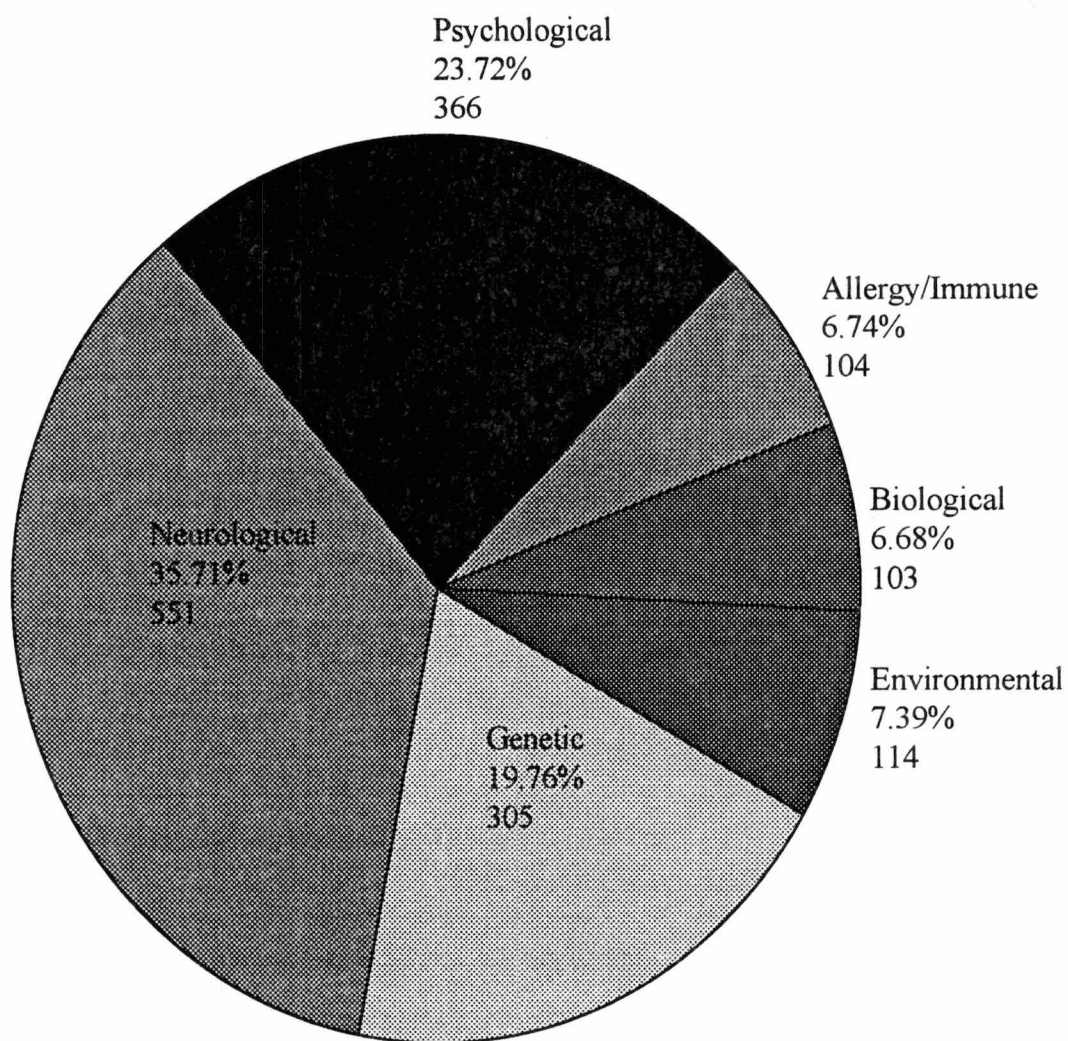


Figure 1. Pie Chart of All Causations (1943 to 1996)

Appendix C

Table C-1. Instances of Each Autism Etiology by Year of Publication

	Total for Year	Allergy/ Immune	Biological	Environ- mental	Genetic	Neuro- logical	Psycho- logical
'43	2						2
'44	3					1	2
'45	0						
'46	0						
'47	1						1
'48	0						
'49	2			1			1
'50	6					1	5
'51	4			1			3
'52	2						2
'53	3		1		1		1
'54	5					1	4
'55	3		1			1	1
'56	1						1
'57	2			1			1
'58	3		1			1	1
'59	1						1
'60	4				1	1	2
'61	0						
'62	3					1	2
'63	9				1	3	5
'64	15			1	1	4	9
'65	19	1	2		2	7	7
'66	4					1	3
'67	6				2	1	3
'68	17	1			2	8	6
'69	22	2			2	8	10
'70	20	2	1	2	2	5	8
Pg.	157	6	6	6	14	44	81

Table C-1. Instances of Each Autism Etiology by Year of Publication
(continued)

	Total for Year	Allergy/ Immune	Biological	Environ- mental	Genetic	Neuro- logical	Psycho- logical
'71	37	3	2	6	3	11	12
'72	25	3	1	1	3	13	14
'73	27	2	3	2	1	8	11
'74	41	1	1	3	5	16	15
'75	53	1	4	3	10	18	17
'76	34	2	1	4	4	14	9
'77	39	2		3	10	14	10
'78	37	2	2	4	7	14	8
'79	48	6	4	10	6	10	12
'80	23	3	2	5	2	6	5
'81	35	1	2	3	4	19	6
'82	31	2	3	2	4	14	6
'83	53	2	4	5	7	18	17
'84	52	2	3	4	12	24	7
'85	51	4	5	2	17	15	8
'86	71	4	8	4	20	23	12
'87	42	5	7	4	5	13	8
'88	48	5	4	3	14	16	6
'89	56	3	4	3	13	21	12
'90	79	9	9	6	17	32	6
'91	82	5	5	9	23	27	13
'92	74	4	2	5	20	30	13
'93	72	5	3	4	19	26	15
'94	99	6	10	4	32	36	11
'95	72	5	3	2	15	32	15
'96	95	11	5	7	18	37	17
All	1,543	104	103	114	305	551	366

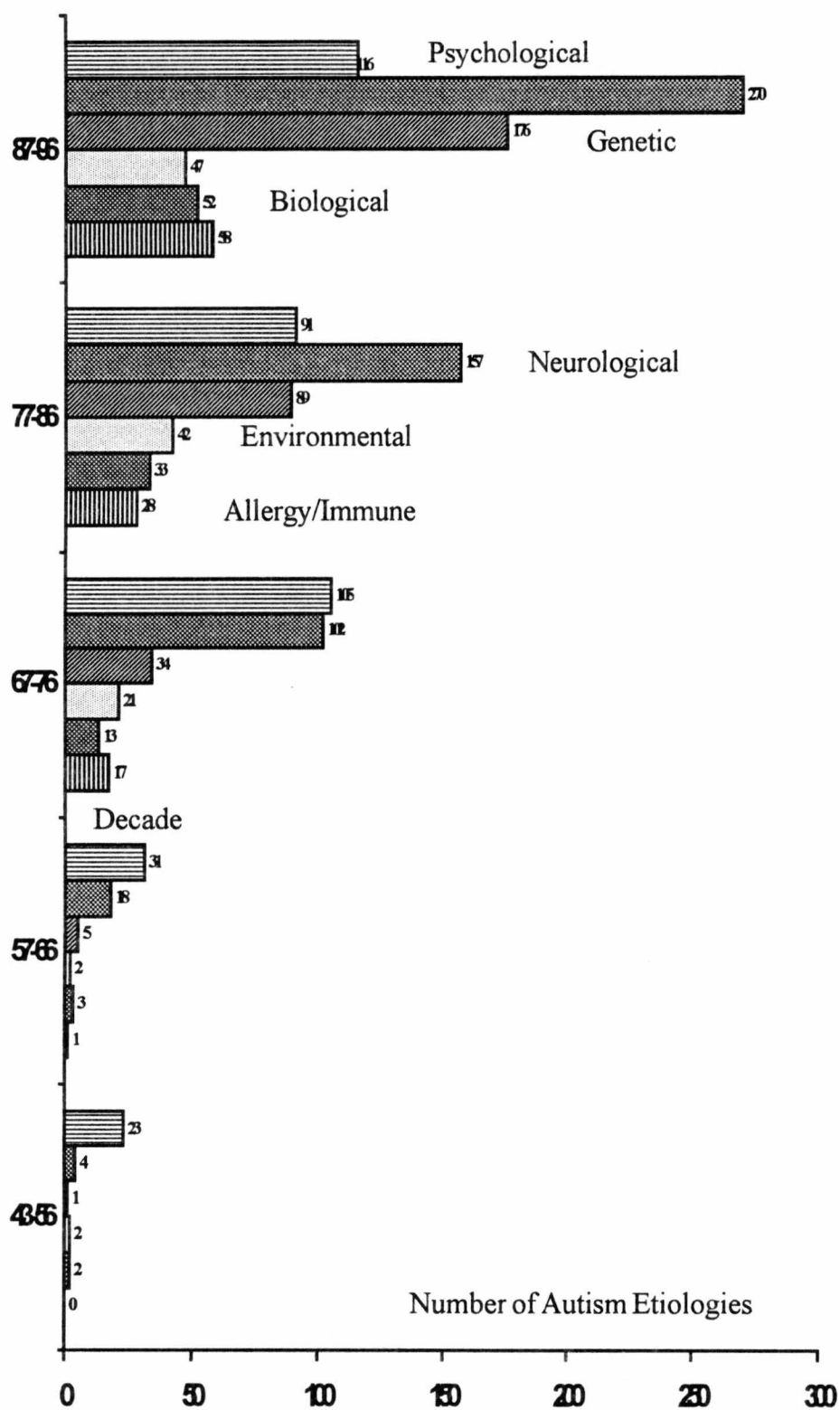


Figure 2. Decade Breakdown of Causations

Appendix D

Table D-1. Number of Sources by Publication Country (1943 through 1996)

Country	#		Country	#
Argentina	1		Ireland	5
Australia	15		Israel	2
Austria	5		Italy	22
Belgium	5		Japan	32
Brazil	4		Korea (South)	1
Bulgaria	1		Mexico	1
Canada	22		Netherlands	24
Colombia	1		Norway	8
Croatia	2		Poland	4
Cuba	2		Romania	6
Czech Republic	1		Russia	1
Czechoslovakia	3		Scotland	2
Denmark	20		Singapore	1
England	121		South Africa	1
Ethiopia	1		Spain	13
Finland	1		Sweden	5
France	71		Switzerland	47
Germany (reunified)	4		United Kingdom	121
Germany (East)	2		Union of Soviet Socialist Reps.	11
Germany (West)	42		United States of America	621
Hungary	1		Venezuela	1
India	1		Yugoslavia	3

Table D-2. Number of Sources Rank Ordered by Publication Country
(1943 through 1996)

Country	#		Country	#
United States of America	621		Poland	4
England	121		Czechoslovakia	3
United Kingdom	121		Yugoslavia	3
France	71		Croatia	2
Switzerland	47		Cuba	2
Germany (West)	42		Germany (East)	2
Japan	32		Israel	2
Netherlands	24		Scotland	2
Canada	22		Argentina	1
Italy	22		Bulgaria	1
Denmark	20		Colombia	1
Australia	15		Czech Republic	1
Spain	13		Ethiopia	1
Union of Soviet Socialist Reps.	11		Finland	1
Norway	8		Hungary	1
Romania	6		India	1
Austria	5		Korea (South)	1
Belgium	5		Mexico	1
Ireland	5		Russia	1
Sweden	5		Singapore	1
Brazil	4		South Africa	1
Germany (reunified)	4		Venezuela	1

Table D-3. Number of Autism Etiologies by Publication Country
(1943 through 1996)

Country	Doc.#	Etiol. #	Aller.	Bio.	Envir.	Genet.	Neuro.	Psych.
Argentina	1	1						1
Australia	15	18		4	3	2	5	4
Austria	5	8	1		1	2	2	2
Belgium	6	7					4	3
Brazil	4	5			1		2	2
Bulgaria	1	2			1	1		
Canada	22	26		5	4	1	13	3
Colombia	1	2		1				1
Croatia	2	2	1				1	
Cuba	2	2		1			1	
Czech Republic	1	1					1	
Czechoslovakia	3	3					1	2
Denmark	20	27	1	3	1	6	9	7
England	121	152	14	6	8	32	52	40
Ethiopia	1	1					1	
Finland	1	1	1					
France	71	83		4	1	13	29	36
Germany (united)	4	5	1			2	2	
Germany (East)	2	3		1			2	
Germany (West)	42	49	1	2	1	8	19	18
Hungary	1	2			1	1		
India	1	1						1
Page Total	327	401	20	27	22	68	144	120

Table D-3. Number of Autism Etiologies by Publication Country
(continued)

Country	Doc.#	Etiol. #	Aller.	Bio.	Envir.	Genet.	Neuro.	Psych.
Ireland	5	5			1		4	
Israel	2	4				1	1	2
Italy	22	26	3		1	4	5	13
Japan	32	40	1	4	2	5	21	7
Korea (South)	1	1					1	
Mexico	1	1					1	
Netherlands	24	30	4	2	1	2	14	7
Norway	8	13	2	1	1	4	4	1
Poland	4	6			1		3	2
Romania	6	7		2			5	
Russia	1	1					1	
Scotland	2	2					2	
Singapore	1	1					1	
South Africa	1	1						1
Spain	13	14		1	1	3	4	5
Sweden	5	6		1	1	3	1	
Switzerland	47	65	11	8	4	12	22	8
United Kingdom	121	155	1	15	8	35	60	36
USSR	11	14		1	1	3	2	7
USA	621	746	62	41	69	164	254	156
Venezuela	1	1				1		
Yugoslavia	3	3			1		1	1
Page Total	932	1,142	84	76	92	237	407	246
Grand Total	1,259	1,543	104	103	114	305	551	366

Appendix E

Table E-1. Year Spans of Journals with Five or More Articles (from First Autism Causation Appearance Through Last [or 1996])

# of Instances	Years	Journal Name
13	'60-'92	Acta Paedopsychiatrica
7	'67-'90	Acta Psychiatrica Scandanavia
5	'60-'80	American Journal of Disabled Children
5	'91-'96	American Journal of Human Genetics
32	'86-'96	American Journal of Medical Genetics
7	'51-'87	American Journal of Orthopsychiatry
22	'47-'95	American Journal of Psychiatry
22	'63-'96	Archives of General Psychiatry
7	'78-'96	Archives of Neurology
11	'75-'96	Biological Psychiatry
5	'82-'95	Brain Development
13	'90-'92	Brain Dysfunction
6	'68-'91	British Journal of Disorders of Communication
14	'70-'91	British Journal of Psychiatry
5	'68-'72	British Medical Journal
6	'81-'92	Canadian Journal of Psychiatry
9	'74-'94	Child Psychiatry and Human Development
7	'78-'93	Clinical Genetics
5	'90-'96	Current Opinion in Pediatrics
5	'93-'96	Current Opinion in Psychiatry
8	'94-'96	Developmental Brain Dysfunction
40	'65-'96	Developmental Medicine and Child Neurology
6	'69-'94	Electroencephalography and Clinical Neurophysiology
5	'84-'95	Encephale
6	'74-'96	International Journal of Psycho-Analysis
7	'88-'95	International Pediatrics
19	'87-'94	Journal of the American Academy of Child and Adolescent Psychiatry

Table E-1. Year Spans of Journals with Five or More Articles (continued)

# of Instances	Years	Journal Name
16	'63-'86	Journal of the American Academy of Child Psychiatry
52	'71-'78	Journal of Autism and Childhood Schizophrenia
103	'79-'96	Journal of Autism and Developmental Disorders
9	'89-'96	Journal of Child Neurology
22	'62-'96	Journal of Childhood Psychology and Psychiatry
26	'77-'96	Journal of Childhood Psychology and Psychiatry and Allied Disciplines
7	'83-'92	Journal of Developmental and Behavioral Pediatrics
8	'92-'96	Journal of Intellectual Disability Research
5	'90-'95	Journal of Neuropsychiatry and Clinical Neurosciences
9	'44-'95	Journal of Pediatrics
5	'69-'82	Journal of Speech and Hearing Disorders
9	'71-'94	Lancet
12	'76-'96	Medical Hypotheses
5	'64-'90	Nervenartz
5	'85-'96	Neurology
13	'83-'94	Neuropsychiatrie de l'Enfance et de l'Adolescence
5	'89-'95	Neuropsychobiology
5	'70-'88	New England Journal of Medicine
9	'84-'94	No To Hattatsu
15	'71-'92	Pediatrics
5	'53-'90	Praxis Kinderpsychologie und Kinderpsychiatrie
8	'84-'91	Psychiatric Clinics of North America
9	'93-'95	Psychiatrie Infant
11	'74-'95	Psychological Medicine
5	'76-'86	Schizophrenia Bulletin
11	'72-'94	Zhournal Nevropatol Im SS Korsakova
686	'44-'96	top 53 journals

Appendix F

Table F-1. Number of Articles per Source

# of Articles	Source
61	Autism Genetic Studies (Web-based collection)
3	<i>Biological Abstracts</i>
41	BIOSIS <i>Previews</i>
42	<i>Cumulated Index Medicus</i>
374	EMBASE
13	<i>Excerpta Medica</i>
521	MEDLINE
7	National Autism Society's <i>Titles in Autism</i>
12	National Library of Medicine's <i>Current Catalog</i>
80	PsycINFO
85	PsycLIT
9	<i>Psychological Abstracts</i>
8	Source in Hand (or Personal Library)
1	Wilson's General Science Index
2	World Wide Web
1,259	Total Source Articles

Appendix G

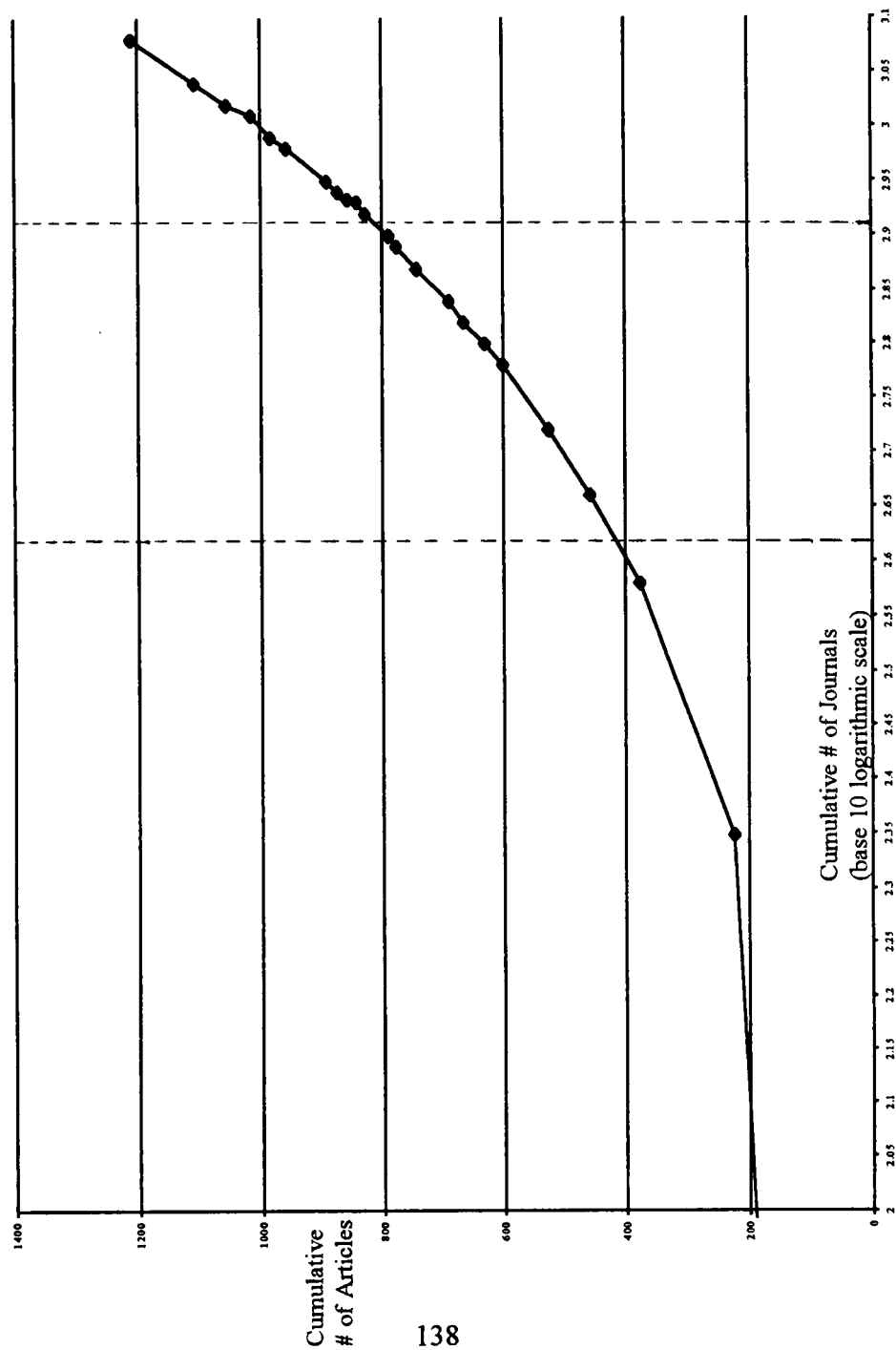


Figure 3. Bradford's Law of Scattering Applied to Autism Causation Research (distinct journal names)

Table G-1. Logarithmic Calculations for Journal Totals (Matching Figure 3)

Journ. Use #	# of Times	Running # of Times	Each Journ. Total	Cum. Journ. Total	Log of Cum. Journ. Total	k Value
1	223	223	223	223	2.35	515.76
2	77	300	154	377	2.58	469.38
3	27	327	81	458	2.66	455.26
4	17	344	68	526	2.72	445.22
5	15	359	75	601	2.78	435.61
6	5	364	30	631	2.8	432.5
7	5	369	35	666	2.82	429.43
8	3	372	24	690	2.84	426.41
9	6	378	54	744	2.87	421.95
11	3	381	33	777	2.89	419.03
12	1	382	12	789	2.9	417.59
13	3	385	39	828	2.92	414.73
14	1	386	14	842	2.93	413.31
15	1	387	15	857	2.93	413.31
16	1	388	16	873	2.94	411.9
19	1	389	19	892	2.95	410.51
22	3	392	66	958	2.98	406.38
26	1	393	26	984	2.99	405.02
32	1	394	32	1,016	3.01	402.33
40	1	395	40	1,056	3.02	400.99
52	1	396	52	1,108	3.04	398.35
103	1	397	103	1,211	3.08	393.18

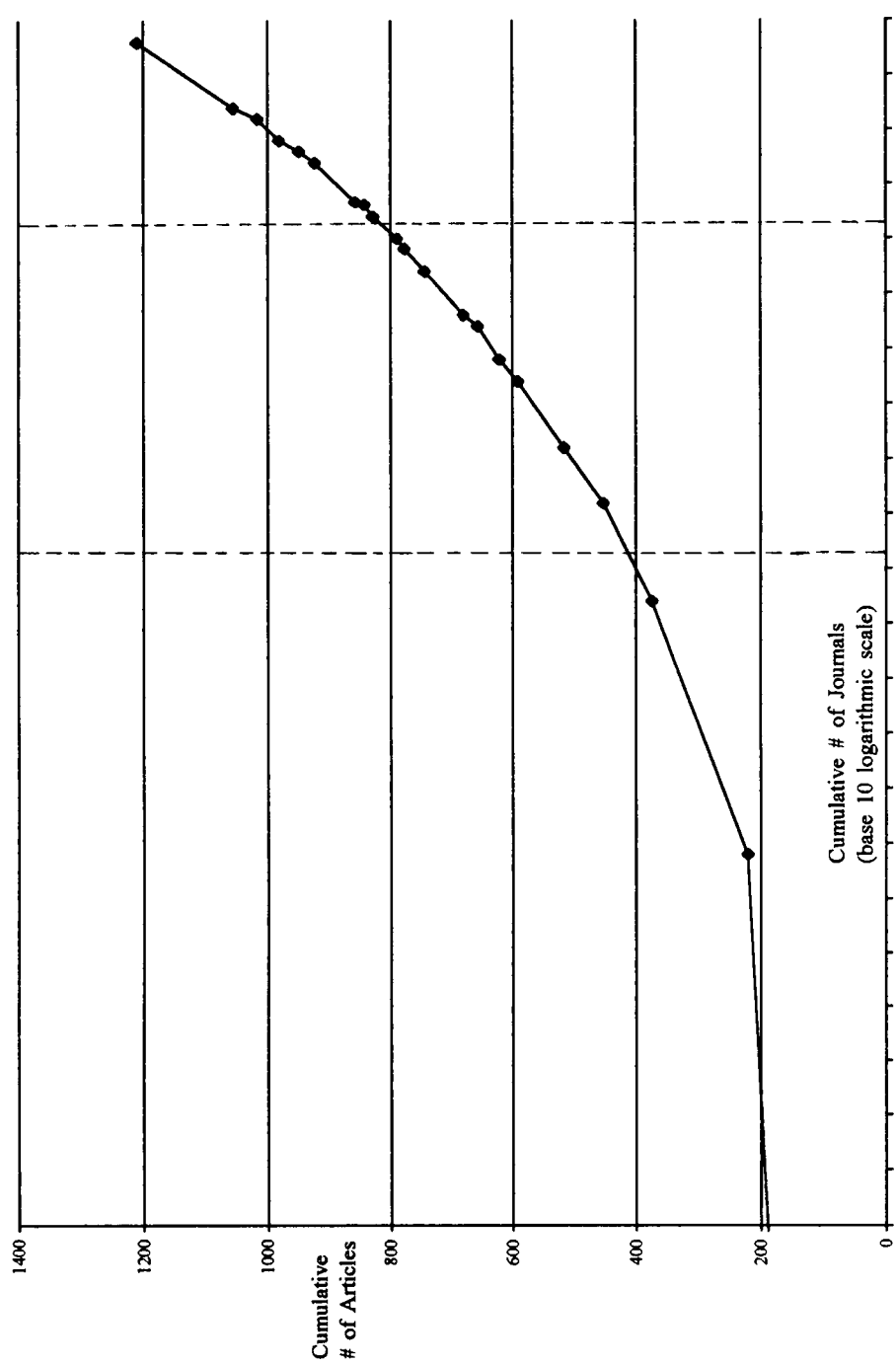


Figure 4. Bradford's Law of Scattering Applied to Autism Causation Research (combined journal names)

Table G-2. Logarithmic Calculations for Journal Totals (Matching Figure 4)

Journ. Use #	# of Times	Running # of Times	Each Journ. Total	Cum. Journ. Total	Log of Cum. Journ. Total	k Value
1	221	221	221	221	2.34	517.52
2	77	298	154	375	2.57	471.21
3	26	324	78	453	2.66	455.26
4	16	340	64	517	2.71	446.86
5	15	355	75	592	2.77	437.4
6	5	360	30	622	2.79	434.05
7	5	365	35	657	2.82	429.43
8	3	368	24	681	2.83	427.92
9	7	375	63	744	2.87	421.95
11	3	378	33	777	2.89	419.03
12	1	379	12	789	2.9	417.59
13	3	382	13	828	2.92	414.73
14	1	383	14	842	2.93	413.31
15	1	384	15	857	2.93	413.31
22	3	387	66	923	2.97	407.74
26	1	388	26	949	2.98	406.38
32	1	389	32	981	2.99	405.02
35	1	390	35	1,016	3.01	402.33
40	1	391	40	1,056	3.02	400.99
155	1	392	155	1,211	3.08	393.18

Appendix H

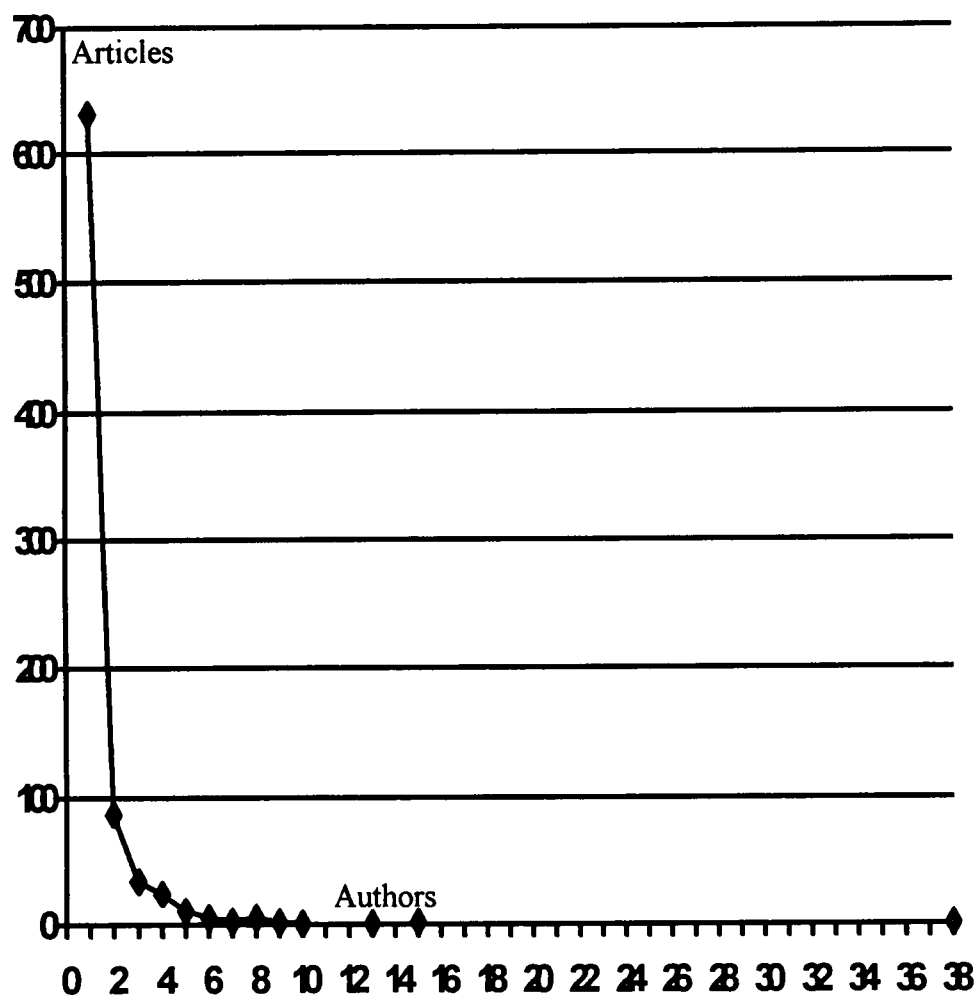


Figure 5. Lotka's Law Applied to Autism Causation Literature
(actual outcome)

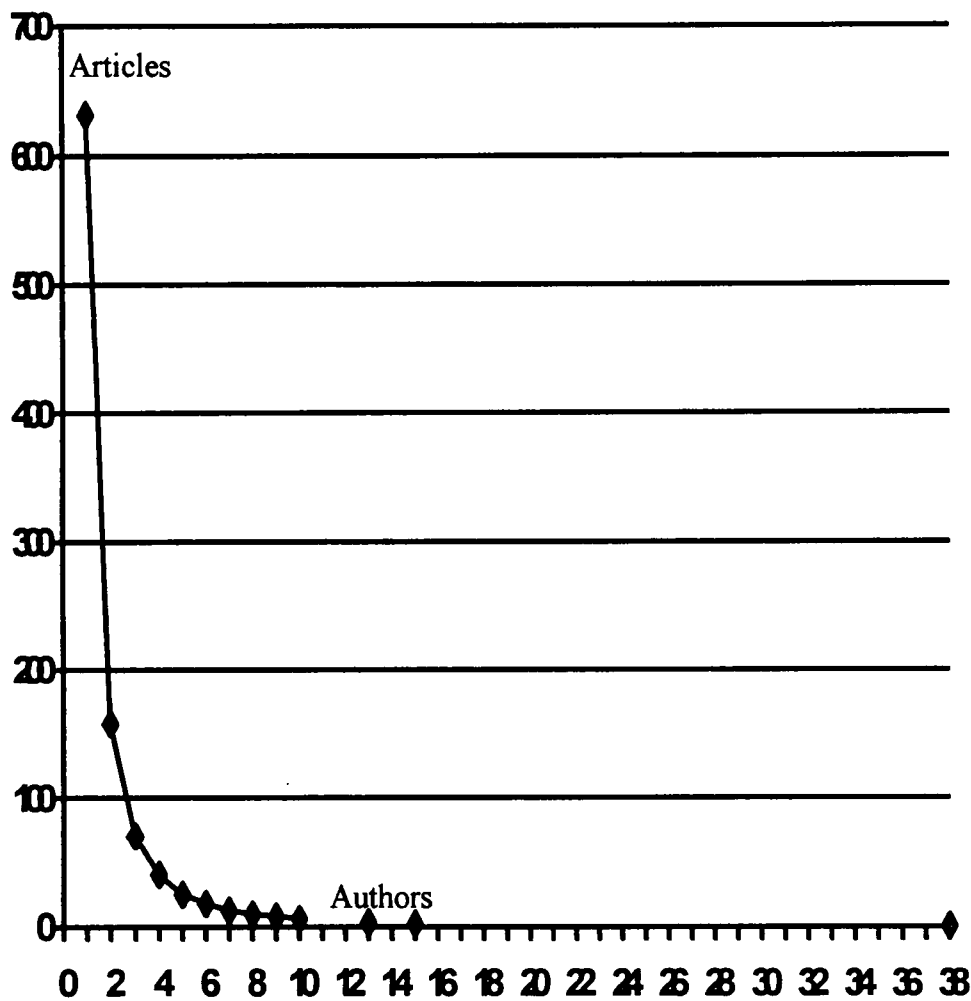


Figure 6. Lotka's Law Applied to Autism Causation Literature
(expected outcome)

VITA

Jeffrey Brian Romanczuk was born in Philadelphia, Pennsylvania. He graduated from the Northeast Catholic High School for Boys in Philadelphia and received his Bachelor of Science degree in Secondary Education from the Pennsylvania State University.

Working experience has included two years of teaching high school English, seven years as an officer in the United States Air Force, and research associate/training project manager positions with the Oak Ridge Associated Universities since 1993.

Jeff is a member of the Special Libraries Association and the Project Management Institute. He is a life member of both the Autism Society of America and the Air Force Association. His work has been published in the Philadelphia Inquirer's *Today* magazine, *New Voices in American Poetry*, the newsletter of the Southern Appalachian Chapter of the Special Libraries Association, and *Records Management Quarterly*.

He is married to the former Cathy Jean Webb, of Maryville, Tennessee. They have two children, Luke and Kate.