Forced Drugging Defense Package

Background

On March 4, 2008, in connection with the MindFreedom Shield Program, PsychRights and MindFreedom International announced a Task Force on Mental Health Legal Advocacy & Activism to help people facing the horrors of forced psychiatric drugging and electroshock. As set forth in the law review article, Involuntary Commitment and Forced Psychiatric Drugging in the Trial Courts: Rights Violations as a Matter of Course, by Jim Gottstein of PsychRights, 25 *Alaska L. Rev.* 51 (2008), "lawyers representing psychiatric respondents interpose little, if any, defense and are not discovering and presenting to judges the evidence of the harm to their clients." In addition to lawyer indifference, most appointed lawyers do not have funding to obtain expert testimony even when they might want to do a good job for their clients.

In trying to address this problem, PsychRights has agreed to make available certified copies of written testimony (affidavits) of Robert Whitaker[1] and Grace E. Jackson, MD., PsychRights filed in a couple of forced drugging cases, and has developed a generic set of pleadings wrapped around this testimony.[2] **In order to use this, certified copies of the Whitaker and Jackson testimony must be obtained from MindFreedom.** Ideally, this testimony will be used by attorneys representing people facing forced drugging petitions and, in order to facilitate adaptation of the generic pleadings to include state law and people's specific circumstances, a Microsoft Word version of the pleadings is available at http://www.mindfreedom.org/shield/forced-drug-defense-pkg/ForcedDruggingDefensePleadings.doc.

The Written Testimony

As mentioned, but it bears repeating, in order for the written testimony (Affidavits) to be considered testimony, people need to obtain **certified copies** from MindFreedom. Certified copies of the Whitaker and Jackson written testimony may be requested by e-mailing office@MindFreedom.org; writing MindFreedom International, at P.O. Box 11284, Eugene, OR 97440-3484 USA; calling (541) 345-9106, or faxing (480) 287-8833. MindFreedom members or people who have signed up for the MindFreedom Shield Program will not be asked to contribute anything for the certified copies. People who are not MindFreedom members or have not signed up for the MindFreedom Shield Program will be asked to make at least a $25 donation. If people don't have the money it will be waived, or they will be asked to pay what they can afford. In all events, only those who have a current need for the testimony should ask for it.

The Generic Pleadings

There are three generic pleadings that have been prepared, plus a Certificate of Service as part of this package:

1. Certificate of Service
2. Motion and Memorandum for Summary Judgment
3. Motion for Stay Pending Appeal

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[1] A version of Robert Whitaker's affidavit with hyperlinks to all of the references (except books) is available at http://psychrights.org/Litigation/WhitakerAffidavit.pdf.

[2] Neither the Law Project for Psychiatric Rights, nor Mr. Gottstein are acting as anyone's attorney with respect to this Forced Drugging Self-Help Defense Package and are not providing legal advice to anyone through it.

a. Order Granting Stay Pending Appeal

4. Motion for Appointment of Psychopharmacology Expert

Each Court tends to have a different way of setting up the "Case Caption," which is the name of the case. The main thing is to have it be exactly the way it is on the forced drugging petition. This needs to be done for each one of the pleadings. For the substantive ones, there is also a blank for the "Movant," which would be the name of the person facing the forced drugging petition.

The best thing is to have your lawyer, if you have one, take these generic pleadings and include citations to the law in your state and also adapt the pleadings to fit the specific facts in your case. Again, for that reason, a Microsoft Word version of the pleadings is available at http://www.mindfreedom.org/shield/forced-drug-defense-pkg/ForcedDruggingDefensePleadings.doc .

(A) Certificate of Service

Copies of everything that is filed needs to be given to the other party(ies) in a case, which is called being "served". The Certificate of Service lets the court know who has been "served" with the documents and is required. The name(s) and address(es) of the other side's attorney should be filled in (along with all the caption information).

(B)Motion and Memorandum for Summary Judgment

As a general rule, one is entitled to "summary judgment," if based on written testimony, "there is no genuine issue as to any material fact and that the moving party is entitled to judgment as a matter of law." Once a summary judgment motion is filed with supporting written testimony, the opposing party has to submit sufficient written testimony to create a "genuine" factual dispute(s) that needs to be resolved in order to defeat the summary judgment motion. In other words, if the other side doesn't present sufficient testimony in opposition to the Whitaker and Jackson testimony, the summary judgment motion should be granted. However, the judges in these types of cases tend to ignore the law so don't be surprised if the summary judgment motion isn't granted, even if the other side doesn't come in with any or enough competent testimony. That's the reason for the next pleading, the motion for stay pending appeal.

(C) Motion for Stay Pending Appeal

The motion for stay pending appeal is to try and keep a forced drugging order from going into effect while an appeal is being taken in the event the force drugging petition is granted, ie, the motion for summary judgment fails and the person also loses after the hearing. The grounds for the motion is that the person will face irreparable harm. As with all three pleadings, the Whitaker and Jackson written testimony provide the factual basis for this. A copy of a recent Alaska Supreme Court order granting a stay pending appeal based at least in part on the Whitaker and Jackson written testimony is attached to this motion to try and get the trial court to take it seriously.[3]

[3] Dr. Jackson also testified telephonically at the hearing. This testimony can be found at http://psychrights.org/States/Alaska/CaseXX/3AN-08-493PS/14may08bigley.pdf. There is also a discussion of Mr. Whitaker being an expert at the analysis of clinical trials and Dr. Jackson testifying that Mr. Whitaker's testimony is "a very clear and accurate presentation" (page 4/112) Submitting this transcript could also be useful.

There is also a proposed order, which many courts require to be filed with a motion. This order provides that the stay will terminate if no appeal is filed. PsychRights has informed MindFreedom that it may be able to help in prosecuting such an appeal. No guarantees, though, because PsychRights has limited resources, but it is a possibility.

There are circumstances where a stay pending appeal may not make sense. One of those is an outpatient commitment continuation petition in New York under what is popularly known as "Kendra's Law." In that circumstance, the current outpatient commitment order stays in place while the continuation petition is pending, so there is really nothing to stay.

(D) Motion for Appointment of Psychopharmacology Expert

The third pleading is also designed to address the situation if the motion for summary judgment is not granted. One of the problems people facing these forced drugging orders have is that they virtually never have access to any expert testimony on their behalf. The motion for summary judgment presents such testimony, but if it is unsuccessful, one needs to have someone testify at the hearing/trial. The Whitaker and Jackson written testimony demonstrates there are serious problems with the forced drugging petition, both as to best interests and that there are less intrusive/less harmful ways to help people. The grounds for the motion for the appointment of a psychopharmacology expert (chosen by the person facing the forced drugging petition) is that without such an expert, the trial can not possibly be a fair one.

IN THE _____ COURT, STATE OF _____

_____)
_____) Case No. _____
_____)
)
_____)

Certificate of Service

_____ (Movant) hereby certifies that on this date, the following were mailed or hand delivered to _____ _____.

1. This Certificate of Service.
2. Motion and Memorandum for Summary Judgment;
3. Motion for Stay Pending Appeal;
4. Proposed form of Order for Stay Pending Appeal;
5. Motion for Appointment of Psychopharmacology Expert;
6. Certified Copy of Affidavit of Robert Whitaker; and
7. Certified Copy of written testimony of Grace E. Jackson, MD.

Dated: _____ By: _____

IN THE _____ COURT, STATE OF _____

_____)
_____) Case No. _____
_____)
)
_____)

MOTION & MEMORANDUM FOR SUMMARY JUDGMENT

_____ (Movant) hereby moves for summary judgment against being forced to take psychotropic medication(s) against Movant's will.

In support of this motion, filed contemporaneously herewith, is the written testimony of Robert Whitaker, and Grace E. Jackson, MD.

Robert Whitaker's written testimony establishes that:

(a) Neuroleptics, also called antipsychotics, increase the likelihood that a person will become chronically ill.

(b) Long-term recovery rates are much higher for unmedicated patients than for those who are maintained on neuroleptic drugs.

(c) Neuroleptics cause a host of debilitating physical, emotional and cognitive side effects, and lead to early death.

(d) The new “atypical” neuroleptics are not better than the old ones in terms of their safety and tolerability, and quality of life may even be worse on the new drugs than on the old ones.

(e) Non-medication approaches have been proven far more effective.

Dr. Jackson's written testimony confirms the Whitaker testimony, and describes in some detail the brain damage caused by neuroleptics, summarizing it as follows:

> Evidence from neuroimaging studies reveals that ***old and new*** neuroleptics contribute to the progressive shrinkage and/or loss of brain tissue. Atrophy is especially prominent in the frontal lobes which control decision making, intention, and judgment. These

> changes are consistent with *cortical* dementia, such as Niemann-Pick's or Alzheimer's disease.
>
> Evidence from postmortem analyses in lab animals reveals that ***old and new*** neuroleptics induce a significant reduction in total brain weight and volume, with prominent changes in the frontal and parietal lobes.
>
> Evidence from biological measurements suggests that ***old and new*** neuroleptics increase the concentrations of tTG (a marker of programmed cell death) in the central nervous system of living humans.
>
> Evidence from *in vitro* studies reveals that haloperidol reduces the viability of hippocampal neurons when cells are exposed to clinically relevant concentrations. (Other experiments have documented similar findings with the second-generation antipsychotics.)
>
> Shortly after their introduction, neuroleptic drugs were identified as chemical lobotomizers. Although this terminology was originally metaphorical, subsequent technologies have demonstrated the scientific reality behind this designation.
>
> Neuroleptics are associated with the destruction of brain tissue in humans, in animals, and in tissue cultures. Not surprisingly, this damage has been found to contribute to the induction or worsening of psychiatric symptoms, and to the acceleration of cognitive and neurobehavioral decline.

(boldfacing in original, underlining added)

This testimony establishes that (1) forcing psychotropic drugs on Movant is not in Movant's best interests and (2) there are less intrusive alternatives.

There being no genuine factual dispute over these dispositive issues, Movant is entitled to a decision in Movant's favor as a matter of law.

Dated: _____ By: _____

IN THE _____ COURT, STATE OF _____

_____)
_____) Case No. _____
_____)
)
_____)

MOTION FOR STAY PENDING APPEAL

_____ (Movant), in order to avoid irreparable harm should the court issue an order requiring Movant to take psychotropic medication(s) against Movant's will (Forced Drugging Order), hereby prophylactically moves for a stay pending appeal. The reason this motion is made in advance of such a ruling is Movant anticipates that should this court issue a Forced Drugging Order, Movant would otherwise immediately be subjected to such forced drugging and effectively denied his right to seek a stay pending appeal.

This motion should be granted because Movant faces irreparable harm should the stay be denied as shown by the written testimony of Robert Whitaker, and Grace E. Jackson, MD, establishing:

(a) Neuroleptics, also called antipsychotics, increase the likelihood that a person will become chronically ill.

(b) Long-term recovery rates are much higher for unmedicated patients than for those who are maintained on neuroleptic drugs.

(c) Neuroleptics cause a host of debilitating physical, emotional and cognitive side effects, and lead to early death.

(d) The new “atypical” neuroleptics are not better than the old ones in terms of their safety and tolerability, and quality of life may even be worse on the new drugs than on the old ones.

and

> Evidence from neuroimaging studies reveals that ***old and new*** neuroleptics contribute to the progressive shrinkage and/or loss of brain tissue. Atrophy is especially prominent in the frontal lobes which control decision making, intention, and judgment. These changes are consistent with *cortical* dementia, such as Niemann-Pick's or Alzheimer's disease.
>
> Evidence from postmortem analyses in lab animals reveals that ***old and new*** neuroleptics induce a significant reduction in total brain weight and volume, with prominent changes in the frontal and parietal lobes.
>
> Evidence from biological measurements suggests that ***old and new*** neuroleptics increase the concentrations of tTG (a marker of programmed cell death) in the central nervous system of living humans.
>
> Evidence from *in vitro* studies reveals that haloperidol reduces the viability of hippocampal neurons when cells are exposed to clinically relevant concentrations. (Other experiments have documented similar findings with the second-generation antipsychotics.)
>
> Shortly after their introduction, neuroleptic drugs were identified as chemical lobotomizers. Although this terminology was originally metaphorical, subsequent technologies have demonstrated the scientific reality behind this designation.
>
> Neuroleptics are associated with the destruction of brain tissue in humans, in animals, and in tissue cultures. Not surprisingly, <u>this damage has been found to contribute to the induction or worsening of psychiatric symptoms, and to the acceleration of cognitive and neurobehavioral decline</u>.

(boldfacing in original, underlining added)

This written testimony was the fundamental basis for the Alaska Supreme Court granting a Stay Pending appeal in *Bigley v. Alaska Psychiatric Institute*, Case No. S-13116, Alaska Supreme Court, a copy of which is attached hereto as Exhibit A.[1]

Dated: _____ By: _____

[1] *See*, also, the cross-examination of Dr. Jackson on her written testimony and redirect, available on the Internet at http://psychrights.org/States/Alaska/CaseXX/3AN-08-493PS/14may08bigley.pdf.

In the Supreme Court of the State of Alaska

William S. Bigley,	)	
	)	Supreme Court No. **S-13116**
Appellant,	)	
v.	)	**Order**
	)	
Alaska Psychiatric Institute,	)	
	)	
Appellee.	)	Date of Order: **5/23/08**
	)	

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MAY 27 2008

Trial Court Case # **3AN-08-00493PR**

By motion of 5/20/08 (updated 5/21/08), appellant has moved on an emergency basis for a stay of the superior court's findings and order of 5/19/08 granting API's petition to administer psychotropic medication during appellant's period of commitment. The order limits the medication to Risperadone in an amount not to exceed fifty milligrams per two weeks. On 5/19/08 12:30 p.m. the superior court also entered a forty-eight hour stay to allow appellant to seek a stay in this court. API has opposed appellant's stay motion. API has also moved to strike an affidavit executed 5/20/08 by Grace E. Jackson, MD and submitted with appellant's 5/20 stay motion. Appellant has responded, at the court's request, to the motion to strike, and has requested alternative stay relief. Upon consideration of the stay motion and opposition, and the motion to strike and the response to that motion,

IT IS ORDERED:

1. It is first necessary to identify the standard for deciding whether a stay is appropriate. The standard depends on the nature of the threatened injury and the adequacy of protection for the opposing party. Thus, if the movant faces a danger of

irreparable harm and the opposing party is adequately protected, the "balance of hardships" approach applies. Under that approach, the movant "must raise 'serious' and substantial questions going to the merits of the case; that is, the issues raised cannot be 'frivolous or obviously without merit.' " *State, Div. of Elections v. Metcalfe*, 110 P.3d 976, 978 (Alaska 2005). On the other hand, if the movant's threatened harm is less than irreparable or if the opposing party cannot be adequately protected, the movant must demonstrate a "clear showing of probable success on the merits." *Id.* The latter standard is proposed here by API. Appellant has not clearly identified the standard he thinks controls. He does, however, assert that he will suffer irreparable harm if he must undergo involuntary medication.

There is at least implicit disagreement in this case about whether administration of psychotropic medication causes medical health problems that are potentially grave or whether it may even contribute to mental illness. At least by implication, the involuntary administration of medication against appellant's fervent wishes may cause psychic harm. Whether long-term administration of such medication causes irreparable harm is an issue that implicates the merits of this appeal. The evidence appellant produced at the mid-May hearing permits a conclusion long-term medication will cause him irreparable harm. It also appears to imply that even the administration of a single dose, or an additional dose, intravenously may contribute to irreparable harm. The 5/20 affidavit of Dr. Jackson does not seem to expressly address the harm that might result from a single fifty-milligram intravenous injection of Risperadone. But it also appears that the likelihood the medication will end with the proposed injection authorized 5/19/08 by the superior court is small. Appellant has been admitted seventy-five times to API. It is

likely that if he is released with or without medication (his thirty-day commitment order was entered 5/5/08), he will be readmitted to API in the future and that API staff will again seek a medication order. Thus, if the medication is administered as presently authorized, it seems likely that he will sooner or later following return to the community decline to voluntarily accept medication and that API will seek permission to administer additional doses. In other words, whether irreparable harm will result from the medication authorized by the 5/19 order necessarily raises longer-term questions.

API asserts that its interests cannot be adequately protected. It certainly has an important interest in fulfilling its duty to patients and in satisfying its charter obligations to the public. But the evidence to date does not establish that medication is necessary to protect appellant from self-inflicted harm or from retaliatory harm in response to his behavior, threatening as it may seem to others. Nor has API identified any need to protect others from him, including API staff during his commitment or the public upon his release. This is not to minimize API's interest both in doing what it believes best for appellant and in carrying out its responsibilities. But it does not appear that API cannot adequately protect those interests. API's interest in protecting appellant does not dramatically outweigh his desire to make treatment decisions for himself. It therefore appears that the appropriate standard for a stay pending appeal is whether appellant has raised serious and substantial questions going to the merits of the case. He does not have to demonstrate a clear showing of probable success on the merits.

2. Applying that standard, the court concludes that a stay of the 5/19 order is appropriate. The evidence presented at the mid-May hearing supports appellant's contentions, but does not necessarily foreclose API's contentions. Because the findings

of fact of the superior court are reviewed under a clearly erroneous standard, and because necessary conclusions of law are considered de novo, this court cannot now conclude on the basis of the evidence review conducted in context of the stay motion that appellant's appellate issues are all frivolous or obviously without merit. The court cannot say that appellant has clearly demonstrated probable success on the merits. But he is not required to do so in this case to obtain a stay. His motion for stay is therefore **GRANTED**.

3. API's motion to strike the 5/20 affidavit of Dr. Jackson is **DENIED**. The affidavit appears to largely summarize other evidence offered at the May hearing. But the only alternative to striking or accepting the affidavit would be remand to the superior court for reconsideration of appellant's stay motion. The superior court, as a fact-finding court, is in a superior position to weigh Dr. Jackson's most recent statements and determine whether appellant has demonstrated irreparable harm. But doing so will simply delay the ultimate resolution of the medication issue. Unless a stay were granted in the superior court, it is probable appellant would renew his stay motion in this court, and then, if that motion were denied, seek full-court reconsideration. In the meantime, the thirty-day commitment period is running. In any event, the 5/20/08 affidavit is not the evidentiary basis for this stay order.

4. This appeal was filed 5/20/08, and the appellant characterized it as a Rule 204 appeal in his notice of appeal and docketing statement. Even if appellate briefing is expedited, it is highly likely the present commitment order will have expired before briefing is complete, and therefore before this court can rule on the merits. The possibility of technical mootness is substantial. The parties should anticipate this issue

in their briefing and discuss whether the court should nonetheless reach the merits of the 5/19/08 order permitting administration of Risperadone.

Entered at the direction of an individual justice.

Clerk of the Appellate Courts

Lori A. Wade, Chief Deputy Clerk

cc: Supreme Court Justices
Judge Gleason by fax
Trial Court Clerk by fax

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In the Supreme Court of the State of Alaska

William S. Bigley, Appellant, v. **Alaska Psychiatric Institute,** Appellee.	Supreme Court No. **S-13116**
	Order
	Date of Order: **6/25/08**

Trial Court Case # **3AN-08-00493PR**

Before: Fabe, Chief Justice, and Matthews, Eastaugh, Carpeneti, and Winfree, Justices.

On consideration of appellee's 5/28/08 motion to reconsider the 5/23/08 individual justice order granting appellant's emergency motion to stay the 5/19/08 superior court order granting API's petition to administer psychotropic medication during appellant's period of commitment, and the 6/9/08 opposition,

IT IS ORDERED: the motion is **DENIED**.

Entered by direction of the court.

Clerk of the Appellate Courts

Lori A. Wade, Chief Deputy Clerk

cc: Supreme Court Justices

IN THE _____ COURT, STATE OF _____

_____	)	
_____	)	Case No. _____
_____	)	
	)	
_____	)	

ORDER GRANTING MOTION FOR STAY PENDING APPEAL

Upon consideration of the motion for stay pending appeal in this matter, and any oppositions thereto, it is hereby ORDERED, the motion for stay pending appeal is GRANTED.

IT IS FURTHER ORDERED, should no appeal be filed within the time allowed to file such an appeal, the stay shall terminate.

Dated: _____ By: _____

IN THE _____ COURT, STATE OF _____

_____)
_____) Case No. _____
_____)
)
_____)

MOTION FOR APPOINTMENT OF PSYCHOPHARMACOLOGY EXPERT

_____ (Movant) hereby moves the court for the appointment of a qualified expert in psychopharmacology acceptable to Movant, such as Grace E. Jackson, MD, to assist the court in this matter. Movant has filed a motion for summary judgment based on the written testimony of Robert Whitaker and Grace E Jackson, establishing

> (j) Neuroleptics, also called antipsychotics, increase the likelihood that a person will become chronically ill.
>
> (k) Long-term recovery rates are much higher for unmedicated patients than for those who are maintained on neuroleptic drugs.
>
> (l) Neuroleptics cause a host of debilitating physical, emotional and cognitive side effects, and lead to early death.
>
> (m) The new "atypical" neuroleptics are not better than the old ones in terms of their safety and tolerability, and quality of life may even be worse on the new drugs than on the old ones.
>
> (n) Non-medication approaches have been proven far more effective.

and

> Evidence from neuroimaging studies reveals that ***old and new*** neuroleptics contribute to the progressive shrinkage and/or loss of brain tissue. Atrophy is especially prominent in the frontal lobes which control decision making, intention, and judgment. These changes are consistent with *cortical* dementia, such as Niemann-Pick's or Alzheimer's disease.

> Evidence from postmortem analyses in lab animals reveals that ***old and new*** neuroleptics induce a significant reduction in total brain weight and volume, with prominent changes in the frontal and parietal lobes.
>
> Evidence from biological measurements suggests that ***old and new*** neuroleptics increase the concentrations of tTG (a marker of programmed cell death) in the central nervous system of living humans.
>
> Evidence from *in vitro* studies reveals that haloperidol reduces the viability of hippocampal neurons when cells are exposed to clinically relevant concentrations. (Other experiments have documented similar findings with the second-generation antipsychotics.)
>
> Shortly after their introduction, neuroleptic drugs were identified as chemical lobotomizers. Although this terminology was originally metaphorical, subsequent technologies have demonstrated the scientific reality behind this designation.
>
> Neuroleptics are associated with the destruction of brain tissue in humans, in animals, and in tissue cultures. Not surprisingly, this damage has been found to contribute to the induction or worsening of psychiatric symptoms, and to the acceleration of cognitive and neurobehavioral decline.

(boldfacing in original, underlining added)

In going to a hearing in spite of the motion for summary judgment, Movant needs to be able to present live testimony in opposition to being ordered to take psychotropic drugs against his wishes.

Forced psychiatric drugging has been equated with the intrusiveness of electroshock and lobotomy,[1] and a deprivation of Movant's fundamental constitutional

[1] *Myers v. Alaska Psychiatric Institute*, 138 P. 3d 238, 242 (Ak. 2006); *Jarvis v. Levine*, 418 N.W.2d 139, 146 (Mn. 1988); *In re K.K.B.*, 609 P.2d 747, 749 (Ok. 1980).

rights.[2] Without access to such expert testimony, Movant will be denied the right to due process of law.

Dated: _____ By: _____

[2] *See*, *Mills v. Rogers*, 457 U.S. 291, n16, 102 S.Ct. 2442 (1982). *See*, also, *Sell v. United States*, 539 U.S. 166, 178, 123 S.Ct. 2174 (2003), which while involving forcing someone to take psychotropic drugs to make him competent to stand trial, re-affirmed, "an individual has a 'significant' constitutionally protected 'liberty interest' in 'avoiding the unwanted administration of antipsychotic drugs,'" necessitating the same sort of due process analysis as applies to the deprivation of fundamental constitutional rights.

IN THE SUPERIOR COURT FOR THE STATE OF ALASKA
THIRD JUDICIAL DISTRICT, AT ANCHORAGE

In The Matter of the Necessity for the)
Hospitalization of W[redacted] S. B[redacted])
Respondent,)
[redacted])
[redacted])
Case No. 3AN 07-1064 P/S

AFFIDAVIT OF ROBERT WHITAKER

STATE OF MASSACHUSETTS)
) ss.
SUFFOLK COUNTY)

By Robert Whitaker

I. Personal Background

1. As a journalist, I have been writing about science and medicine, in a variety of forums, for about 20 years. My relevant experience is as follows:

a) From 1989 to 1994, I was the science and medical writer for the *Albany Times Union* in Albany, New York.

b) During 1992-1993, I was a fellow in the Knight Fellowship for Science Writers at the Massachusetts Institute of Technology.

c) From 1994-1995, I was director of publications at Harvard Medical School.

d) In 1994, I co-founded a publishing company, CenterWatch, that reported on the clinical development of new drugs. I directed the company's editorial operations until late 1998, when we sold the company. I continued to write freelance articles for the *Boston Globe* and various magazines during this period.

e) Articles that I wrote on the pharmaceutical industry and psychiatry for the *Boston Globe* and *Fortune* magazine won several national awards, including the George Polk Award for medical writing in 1999, and the National Association of Science Writers award for best magazine article that same year. A series I wrote for the *Boston Globe* on problems in psychiatric research was a finalist for the Pulitzer Prize in Public Service in 1999.

f) Since 1999, I have focused on writing books. My first book, *Mad in America*, reported on our country's treatment of the mentally ill throughout its history, and explored in particular why schizophrenia patients fare so much worse in the United States and other developed countries than in the poor countries of the world. The book was picked by *Discover* magazine as one of the best science books of 2002; the American Library Association named it as one of the best histories of 2002.

2. Prior to writing *Mad in America*, I shared conventional beliefs about the nature of schizophrenia and the need for patients so diagnosed to be on antipsychotic medications for life. I had interviewed many psychiatric experts who told me that the drugs were like "insulin for diabetes" and corrected a chemical imbalance in the brain.

3. However, while writing a series for the *Boston Globe* during the summer of 1998, I came upon two studies that looked at long-term outcomes for schizophrenia patients that raised questions about this model of care. First, in 1994, Harvard researchers reported that outcomes for schizophrenia patients in the United States had declined in the past 20 years and were now no better than they had been in 1900.[1] Second, the World Health Organization twice found that schizophrenia patients in the poor countries of the world fare much better than in the U.S. and other "developed" countries, so much so that they concluded that living in a developed country was a

[1] Hegarty, J, et al. "One hundred years of schizophrenia: a meta-analysis of the outcome literature." *American Journal of Psychiatry* 151 (1994):1409-16.

"strong predictor" that a person so diagnosed would never recover.[2,3] Although the WHO didn't identify a reason for that disparity in outcomes, it did note a difference in the use of antipsychotic medications between the two groups. In the poor countries, only 16% of patients were regularly maintained on antipsychotic medications, whereas in the U.S. and other rich countries, this was the standard of care, with 61% of schizophrenia patients staying on the drugs continuously. (Exhibit 1)

4. I wrote *Mad in America*, in large part, to investigate why schizophrenia patients in the U.S. and other developed countries fare so poorly. A primary part of that task was researching the scientific literature on schizophrenia and antipsychotic drugs.

II. Overview of Research Literature on Schizophrenia and Standard Antipsychotic Medications

5. Although the public has often been told that people with schizophrenia suffer from too much "dopamine" in the brain, researchers who investigated this hypothesis during the 1970s and 1980s were unable to find evidence that people so diagnosed have, in fact, overactive dopamine systems. Within the psychiatric research community, this was widely acknowledged in the late 1980s and early 1990s. As Pierre Deniker, who was one of the founding fathers of psychopharmacology, confessed in 1990: "The dopaminergic theory of schizophrenia retains little credibility for psychiatrists."[4]

6. Since people with schizophrenia have no known "chemical imbalance" in the brain, antipsychotic drugs cannot be said to work by "balancing" brain chemistry. These drugs are not like "insulin for diabetes." They do not serve as a corrective to a known biological abnormality. Instead, Thorazine and other standard antipsychotics (also known as

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[2] Leff, J, et al. "The international pilot study of schizophrenia: five-year follow-up findings." *Psychological Medicine* 22 (1992):131-45.

[3] Jablensky, A, et al. "Schizophrenia: manifestations, incidence and course in different cultures, a World Health Organization ten-country study." *Psychological Medicine* 20, monograph supplement, (1992):1-95.

[4] Deniker, P. "The neuroleptics: a historical survey." *Acta Psychiatrica Scandinavica* 82, supplement 358 (1990):83-87.

neuroleptics) work by powerfully blocking dopamine transmission in the brain. Specifically, these drugs block 70% to 90% of a particular group of dopamine receptors known as D2 receptors. This thwarting of normal dopamine transmission is what causes the drugs to be so problematic in terms of their side effects.

8. Psychiatry's belief in the necessity of using the drugs on a continual basis stems from two types of studies.

a) First, research by the NIMH has shown that the drugs are more effective than placebo in curbing psychotic symptoms over the short term (six weeks).[5]

b) Second, researchers have found that if patients abruptly quit taking antipsychotic medications, they are at high risk of relapsing. [6]

9. Although the studies cited above provide a rationale for continual drug use, there is a long line of evidence in the research literature, one that is not generally known by the public or even by most psychiatrists, that shows that these drugs, over time, produce these results:

a) They increase the likelihood that a person will become chronically ill.
b) They cause a host of debilitating side effects.
c) They lead to early death.

III. Evidence Revealing Increased Chronicity of Psychotic Symptoms

10. In the early 1960s, the NIMH conducted a six-week study of 344 patients at nine hospitals that documented the efficacy of antipsychotics in knocking down psychosis

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[5] Cole, J, et al. "Phenothiazine treatment in acute schizophrenia." *Archives of General Psychiatry* 10 (1964):246-61.
[6] Gilbert, P, et al. "Neuroleptic withdrawal in schizophrenic patients." *Archives of General Psychiatry* 52 (1995):173-188.

over a short term. (See footnote five, above). The drug-treated patients fared better than the placebo patients over the short term. However, when the NIMH investigators followed up on the patients one year later, they found, much to their surprise, that it was the drug-treated patients who were more likely to have relapsed/ This was the first evidence of a paradox: Drugs that were effective in curbing psychosis over the short term were making patients more likely to become psychotic over the long term.[7]

11. In the 1970s, the NIMH conducted three studies that compared antipsychotic treatment with "environmental" care that minimized use of the drugs. In each instance, patients treated without drugs did better over the long term than those treated in a conventional manner.[8, 9, 10] Those findings led NIMH scientist William Carpenter to conclude that "antipsychotic medication may make some schizophrenic patients more vulnerable to future relapse than would be the case in the natural course of the illness."

12. In the 1970s, two physicians at McGill University, Guy Chouinard and Barry Jones, offered a biological explanation for why this is so. The brain responds to neuroleptics and their blocking of dopamine receptors as though they are a pathological insult. To compensate, dopaminergic brain cells increase the density of their D2 receptors by 40% or more. The brain is now "supersensitive" to dopamine, and as a result, the person has become more *biologically* vulnerable to psychosis than he or she would be naturally. The two Canadian researchers wrote: "Neuroleptics can produce a dopamine supersensitivity that leads to both dyskinetic and psychotic symptoms. An implication is that the tendency

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[7] Schooler, N, et al. "One year after discharge: community adjustment of schizophrenic patients." *American Journal of Psychiatry* 123 (1967):986-95.

[8] Rappaport, M, et al. "Are there schizophrenics for whom drugs may be unnecessary or contraindicated?" *Int Pharmacopsychiatry* 13 (1978):100-11.

[9] Carpenter, W, et al. "The treatment of acute schizophrenia without drugs." *American Journal of Psychiatry* 134 (1977):14-20.

[10] Bola J, et al. "Treatment of acute psychosis without neuroleptics: two-year outcomes from the Soteria project." *Journal of Nervous Mental Disease* 191 (2003):219-29.

toward psychotic relapse in a patient who had developed such a supersensitivity is determined by more than just the normal course of the illness. [11]

13. MRI-imaging studies have powerfully confirmed this hypothesis. During the 1990s, several research teams reported that antipsychotic drugs cause atrophy of the cerebral cortex and an enlargement of the basal ganglia.[12, 13, 14] In 1998, investigators at the University of Pennsylvania reported that the drug-induced enlargement of the basal ganglia is "associated with greater severity of both negative and positive symptoms." In other words, they found that the drugs cause morphological changes in the brain that are associated with a worsening of the very symptoms the drugs are supposed to alleviate.[15]

IV. Research Showing that Recovery Rates are Higher for Non-Medicated Patients than for Medicated Patients.

14. The studies cited above show that the drugs increase the chronicity of psychotic symptoms over the long term. There are also now a number of studies documenting that long-term recovery rates are much higher for patients off antipsychotic medications. Specifically:

a) In 1994, Courtenay Harding at Boston University reported on the long-term outcomes of 82 chronic schizophrenics discharged from Vermont State Hospital in the late 1950s. She found that one-third of this cohort had recovered

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[11] Chouinard, G, et al. "Neuroleptic-induced supersensitivity psychosis." *American Journal of Psychiatry* 135 (1978):1409-10. Also see Chouinard, G, et al. "Neuroleptic-induced supersensitivity psychosis: clinical and pharmacologic characteristics." *American Journal of Psychiatry* 137(1980):16-20.

[12] Gur, R, et al. "A follow-up magnetic resonance imaging study of schizophrenia." *Archives of General Psychiatry* 55 (1998):142-152.

[13] Chakos M, et al. "Increase in caudate nuclei volumes of first-episode schizophrenic patients taking antipsychotic drugs." *American Journal of Psychiatry* 151 (1994):1430-6.

[14] Madsen A, et al. "Neuroleptics in progressive structural brain abnormalities in psychiatric illness." *The Lancet* 352 (1998): 784-5.

[15] Gur, R, et al. "Subcortical MRI volumes in neuroleptic-naive and treated patients with schizophrenia." *American Journal of Psychiatry* 155 (1998):1711-17.

completely, and that all who did shared one characteristic: They had all stopped taking antipsychotic medication. The notion that schizophrenics needed to stay on antipsychotics all their lives was a "myth," Harding said.[16, 17, 18]

b) In the World Health Organization studies, 63% of patients in the poor countries had good outcomes, and only one-third became chronically ill. In the U.S. countries and other developed countries, only 37% of patients had good outcomes, and the remaining patients did not fare so well. In the undeveloped countries, only 16% of patients were regularly maintained on antipsychotics, versus 61% of patients in the developed countries.

c) In response to this body of literature, physicians in Switzerland, Sweden and Finland have developed programs that involve minimizing use of antipsychotic drugs, and they are reporting much better results than what we see in the United States.[19, 20, 21, 22] In particular, Jaako Seikkula recently reported that five years after initial diagnosis, 82% of his psychotic patients are symptom-free, 86% have returned to their jobs or to school, and only 14% of his patients are on antipsychotic medications.[23]

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[16] Harding, C. "The Vermont longitudinal study of persons with severe mental illness," *American Journal of Psychiatry* 144 (1987):727-34.

[17] Harding, C. "Empirical correction of seven myths about schizophrenia with implications for treatment." *Acta Psychiatrica Scandinavica* 90, suppl. 384 (1994):140-6.

[18] McGuire, P. "New hope for people with schizophrenia," *APA Monitor* 31 (February 2000).

[19] Ciompi, L, et al. "The pilot project Soteria Berne." *British Journal of Psychiatry* 161, supplement 18 (1992):145-53.

[20] Cullberg J. "Integrating psychosocial therapy and low dose medical treatment in a total material of first-episode psychotic patients compared to treatment as usual." *Medical Archives* 53 (199):167-70.

[21] Cullberg J. "One-year outcome in first episode psychosis patients in the Swedish Parachute Project. *Acta Psychiatrica Scandinavica* 106 (2002):276-85.

[22] Lehtinen V, et al. "Two-year outcome in first-episode psychosis according to an integrated model. *European Psychiatry* 15 (2000):312-320.

[23] Seikkula J, et al. Five-year experience of first-episode nonaffective psychosis in open-dialogue approach. *Psychotherapy Research* 16/2 (2006): 214-228.

d) This spring, researchers at the University of Illinois Medical School reported on the long-term outcomes of schizophrenia patients in the Chicago area since 1990. They found that 40% of those who refused to take their antipsychotic medications were recovered at five-year and 15-year followup exams, versus five percent of the medicated patients.[24]

V. Harmful Side Effects from Antipsychotic Medications

15. In addition to making patients chronically ill, standard antipsychotics cause a wide range of debilitating side effects. Specifically:

a) Tardive dyskinesia. The most visible sign of tardive dyskinesia is a rhythmic movement of the tongue, which is the result of permanent damage to the basal ganglia, which controls motor movement. People suffering from tardive dyskinesia may have trouble walking, sitting still, eating, and speaking. In addition, people with tardive dyskinesia show accelerated cognitive decline. NIMH researcher George Crane said that tardive dyskinesia resembles "in every respect known neurological diseases, such as Huntington's disease, dystonia musculorum deformans, and postencephalitic brain damage."[25] Tardive dyskinesia appears in five percent of patients treated with standard neuroleptics in one year, with the percentage so afflicted increasing an additional five percent with each additional year of exposure.

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[24] Harrow M, et al. "Factors involved in outcome and recovery in schizophrenia patients not on antipsychotic medications." *Journal of Nervous and Mental Disease* 195 (2007): 406-414.

[25] Crane, G. "Clinical psychopharmacology in its 20th year," *Science* 181 (1973):124-128. Also see American Psychiatric Association, *Tardive Dyskinesia: A Task Force Report* (1992).

b) Akathisia. This is an inner restlessness and anxiety that many patients describe as the worst sort of torment. This side effect has been linked to assaultive, murderous behavior.[26, 27, 28, 29, 30]

c) Emotional impairment. Many patients describe feeling like "zombies" on the drugs. In 1979, UCLA psychiatrist Theodore van Putten reported that most patients on antipsychotics were spending their lives in "virtual solitude, either staring vacantly at television, or wandering aimlessly around the neighborhood, sometimes stopping for a nap on a lawn or a park bench . . . they are bland, passive, lack initiative, have blunted affect, make short, laconic replies to direct questions, and do not volunteer symptoms . . . there is a lack not only of interaction and initiative, but of any activity whatsoever.[31] The quality of life on conventional neuroleptics, researchers agreed, is "very poor." [32]

d) Cognitive impairment. Various studies have found that neuroleptics reduce one's capacity to learn and retain information. As Duke University scientist Richard Keefe said in 1999, these drugs may "actually prevent adequate learning effects and worsen motor skills, memory function, and executive abilities, such as problem solving and performance assessment."[33]

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[26] Shear, K et al. "Suicide associated with akathisia and deport fluphenazine treatment," *Journal of Clinical Psychopharmacology* 3 (1982):235-6.
[27] Van Putten, T. "Behavioral toxicity of antipsychotic drugs." *Journal of Clinical Psychiatry* 48 (1987):13-19.
[28] Van Putten, T. "The many faces of akathisia," *Comprehensive Psychiatry* 16 91975):43-46.
[29] Herrera, J. "High-potency neuroleptics and violence in schizophrenia," *Journal of Nervous and Mental Disease* 176 (1988):558-561.
[30] Galynker, I. "Akathisia as violence." *Journal of Clinical Psychiatry* 58 (1997):16-24.
[31] Van Putten, T. "The board and care home." *Hospital and Community Psychiatry* 30 (1979):461-464.
[32] Weiden P. "Atypical antipsychotic drugs and long-term outcome in schizophrenia." *Journal of Clinical Psychiatry* 57, supplement 11 (1996):53-60.
[33] Keefe, R. "Do novel antipsychotics improve cognition?" *Psychiatric Annals* 29 (1999):623-629.

d) Other side effects of standard neuroleptics include an increased incidence of blindness, fatal blood clots, arrhythmia, heat stroke, swollen breasts, leaking breasts, obesity, sexual dysfunction, skin rashes and seizures, and early death.[34, 35, 36] Schizophrenia patients now commit suicide at 20 times the rate they did prior to the use of neuroleptics.[37]

VI. The Research Literature on Atypical Antipsychotics

16. The conventional wisdom today is that the "atypical" antipsychotics that have been brought to market—Risperdal, Zyprexa, and Seroquel, to name three—are much better and safer than Haldol, Thorazine and the other older drugs. However, it is now clear that the new drugs have no such advantage, and there is even evidence suggesting that they are worse than the old ones.

17. Risperdal, which is manufactured by Janssen, was approved in 1994. Although it was hailed in the press as a "breakthrough "medication, the FDA, in its review of the clinical trial data, concluded that there was no evidence that this drug was better or safer than Haldol (haloperidol.) The FDA told Janssen: "We would consider any advertisement or promotion labeling for RISPERDAL false, misleading, or lacking fair balance under section 501 (a) and 502 (n) of the ACT if there is presentation of data that conveys the impression that risperidone is superior to haloperidol or any other marketed antipsychotic drug product with regard to safety or effectiveness."[38]

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[34] Arana, G. "An overview of side effects caused by typical antipsychotics." *Journal of Clinical Psychiatry* 61, supplement 8 (2000):5-13.
[35] Waddington, J. "Mortality in schizophrenia." *British Journal of Psychiatry* 173 (1998):325-329.
[36] Joukamaa, M, et al. Schizophrenia, neuroleptic medication and mortality. *British Journal of Psychiatry* 188 (2006):122-127.
[37] Healy, D et al. "Lifetime suicide rates in treated schizophrenia." *British Journal of Psychiatry* 188 (2006):223-228.
[38] FDA approval letter from Robert Temple to Janssen Research Foundation, December 21, 1993.

18. After Risperdal (risperidone) was approved, physicians who weren't funded by Janssen were able were able to conduct independent studies of the drug. They concluded that risperidone, in comparison to Haldol, caused a higher incidence of Parkinsonian symptoms; that it was more likely to stir akathisia; and that many patients had to quit taking the drug because it didn't knock down their psychotic symptoms.[39, 40, 41, 42, 43] Jeffrey Mattes, director of the Psychopharmacology Research Association, concluded in 1997: "It is possible, based on the available studies, that risperidone is not as effective as standard neuroleptics for typical positive symptoms."[44] Letters also poured into medical journals linking risperidone to neuroleptic malignant syndrome, tardive dyskinesia, tardive dystonia, liver toxicity, mania, and an unusual disorder of the mouth called "rabbit syndrome."

19. Zyprexa, which is manufactured by Eli Lilly, was approved by the FDA in 1996. This drug, the public was told, worked in a more "comprehensive" manner than either risperidone or haloperidol, and was much "safer and more effective" than the standard neuroleptics. However, the FDA, in its review of the trial data for Zyprexa, noted that Eli Lilly had designed its studies in ways that were "biased against haloperidol." In fact, 20 of the 2500 patients treated with Zyprexa in the trials died. Twenty-two percent of the Zyprexa patients suffered a "serious" adverse event, compared to 18 percent of the Haldol patients. There was also evidence that Zyprexa caused some sort of metabolic dysfunction, as patients gained nearly a pound per week. Other problems that showed up in Zyprexa patients included Parkinsonian symptoms, akathisia, dystonia, hypotension,

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[39] Rosebush, P. "Neurologic side effects in neuroleptic-naïve patients treated with haloperidol or risperidone." *Neurology* 52 (1999):782-785.
[40] Knable, M. "Extrapyramidal side effects with risperidone and haloperidol at comparable D2 receptor levels." *Psychiatry Research: Neuroimaging Section* 75 (1997):91-101.
[41] Sweeney, J. "Adverse effects of risperidone on eye movement activity." *Neuropsychopharmacology* 16 (1997):217-228.
[42] Carter, C. "Risperidone use in a teaching hospital during its first year after market approval." *Psychopharmacology Bulletin* 31 (1995):719-725.
[43] Binder, R. "A naturalistic study of clinical use of risperidone." *Psychiatric Services* 49 (1998):524-6.
[44] Mattes, J. "Risperidone: How good is the evidence for efficacy?" *Schizophrenia Bulletin* 23 (1997):155-161.

constipation, tachycardia, seizures, liver abnormalities, white blood cell disorders, and diabetic complications. Moreover, two-thirds of the Zyprexa patients were unable to complete the trials either because the drugs didn't work or because of intolerable side effects.[45]

20. There is now increasing recognition in scientific circles that the atypical antipsychotics are no better than the old drugs, and may in fact be worse. Specifically:

a) In 2000, a team of English researchers led by John Geddes at the University of Oxford reviewed results from 52 studies, involving 12,649 patients. They concluded: "There is no clear evidence that atypicals are more effective or are better tolerated than conventional antipsychotics." The English researchers noted that Janssen, Eli Lilly and other manufacturers of atypicals had used various ruses in their clinical trials to make their new drugs look better than the old ones. In particular, the drug companies had used "excessive doses of the comparator drug."[46]

b) In 2005, a National Institute of Mental Health study found that that were "no significant differences" between the old drugs and the atypicals in terms of their efficacy or how well patients tolerated them. Seventy-five percent of the 1432 patients in the study were unable to stay on antipsychotics owing to the drugs' "inefficacy or intolerable side effects," or for other reasons.[47]

c) In 2007, a study by the British government found that schizophrenia patients had better "quality of life" on the old drugs than on the new ones.[48] This finding was

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[45] See Whitaker, R. *Mad in America*. New York: Perseus Press (2002):279-281.
[46] Geddes, J. "Atypical antipsychotics in the treatment of schizophrenia." *British Medical Journal* 321 (2000):1371-76.
[47] Lieberman, J, et al. "Effectiveness of antipsychotic drugs in patients with schizophrenia." *New England Journal of Medicine* 353 (2005):1209-1233.
[48] Davies, L, et al. "Cost-effectiveness of first- v. second-generation antipsychotic drugs." *The British Journal of Psychiatry* 191 (2007):14-22.

quite startling given that researchers had previously determined that patients medicated with the old drugs had a "very poor" quality of life.

20. There is also growing evidence that the atypicals may be exacerbating the problem of early death. Although the atypicals may not clamp down on dopamine transmission quite as powerfully as the old standard neuroleptics, they also block a number of other neurotransmitter systems, most notably serotonin and glutamate. As a result, they may cause a broader range of physical ailments, with diabetes and metabolic dysfunction particularly common for patients treated with Zyprexa. In a 2003 study of Irish patients, 25 of 72 patients (35%) died over a period of 7.5 years, leading the researchers to conclude that the risk of death for schizophrenics had "doubled" since the introduction of the atypical antipsychotics.[49]

VII. Conclusion

21. In summary, the research literature reveals the following:

a) Antipsychotics increase the likelihood that a person will become chronically ill.

b) Long-term recovery rates are much higher for unmedicated patients than for those who are maintained on antipsychotic drugs.

c) Antipsychotics cause a host of debilitating physical, emotional and cognitive side effects, and lead to early death.

[49] Morgan, M, et al. "Prospective analysis of premature morbidity in schizophrenia in relation to health service engagement." *Psychiatry Research* 117 (2003):127-35.

d) The new "atypical" antipsychotics are not better than the old ones in terms of their safety and tolerability, and quality of life may even be worse on the new drugs than on the old ones.

DATED this 4 day of September, 2007, in Cambridge, Massachusetts.

Robert B. Whitaker

Robert Whitaker

SUBSCRIBED AND SWORN TO before me this 4th day of September, 2007.

Notary Public in and for Massachusetts
My Commission Expires: August 6, 2013

TENZIN DEMAY
Notary Public
COMMONWEALTH OF MASSACHUSETTS
My Commission Expires
August 6, 2013

State of Alaska)
)ss
Third Judicial District)

I, James B. Gottstein, hereby affirm that this reproduction of Affidavit of Robert Whitaker, to which this is appended, is a true, correct and, except for removal of identifying information, complete photocopy of the original filed in 3AN 07-1064PR, Superior Court for the State of Alaska, Third Judicial District at Anchorage.

Dated: _____ _____
James B. Gottstein

SUBSCRIBED AND SWORN TO before me this ____day of _____, 20___.

_____
Notary Public in and for Alaska
My Commission expires: _____

STATE OF NORTH CAROLINA)
) ss.
_____COUNTY_____)

Appendix A

Evidence for the Neurotoxicity of Antipsychotic Drugs

The History of Neuroleptics

The modern history of psychiatric drugs dates back to the early 1950s, when derivatives of the synthetic dye and rocket fuel industries were found to have medicinal properties. Following World War II, a wide variety of compounds came to be tested in humans. The antihistamine known as chlorpromazine (Thorazine) is generally regarded as the first "anti-psychotic" drug, responsible for igniting the psychopharmacology revolution. As Thorazine grew in popularity, medications replaced neurosurgery and shock therapies as the favored treatments for the institutionalized mentally ill. (For three excellent reviews on this subject, see Cohen, Healy, and Valenstein).[1-3]

When, in 1955, Drs. Jean Delay and Pierre Deniker coined the term "neuroleptic" to describe Thorazine, they identified five defining properties of this prototype: the gradual reduction of psychotic symptoms, the induction of psychic indifference, sedation, movement abnormalities (parkinsonism), and predominant subcortical effects.[4] At its inception, Thorazine was celebrated as a *chemical lobotomizer* due to behavioral effects which paralleled those associated with the removal of brain tissue.[5] As the concept of lobotomy fell into disfavor, the alleged antipsychotic features of the neuroleptics came to be emphasized. Ultimately, the two terms became synonymous.

Ignorant of the historical definition of neuroleptics as ***chemical lobotomizers***, members of the psychiatric profession have only rarely acknowledged the fact that these dopamine blocking compounds have been, and continue to be, a major cause of brain injury and dementia. Nevertheless, the emergence of improved technologies and epidemiological investigations have made it possible to demonstrate why these medications should be characterized as neurotoxins, rather than neurotherapies.

Evidence for Neuroleptic (Antipsychotic) Induced Brain Injury

Proof of neuroleptic toxicity can be drawn from five major lines of evidence:

1) postmortem studies of human brain tissue
2) neuroimaging studies of living humans
3) postmortem studies of lab animal brain tissue
4) biological markers of cell damage in living humans
5) lab studies of cell cultures/chemical systems following drug exposure

Line of Evidence #1: Postmortem Studies in Humans

In 1977, Jellinger published his findings of neuropathological changes in the brain tissue of twenty-eight patients who had been exposed to neuroleptics for an average of four to five years.[6] In most cases, the periods of drug treatment had been intermittent. At autopsy, 46% of the subjects were found to have significant tissue damage in the movement centers (basal ganglia) of the brain, including swelling of the large neurons in the caudate nucleus, proliferation of astrocytes and other glial cells, and occasional degeneration of neurons. Three patients exposed to chronic neuroleptic therapy also demonstrated inflammation of the cerebral veins (phlebitis). An example of the abnormalities is shown below:

This photo demonstrates reactive gliosis (black dots represent scar tissue) in the caudate of a patient who had received neuroleptic therapy. Patients in this study had received the following drug treatments: chlorpromazine (Thorazine), reserpine, haloperidol (Haldol), trifluoperazine (Stelazine), chlorprothixen (Taractan), thioridazine (Mellaril), tricyclic antidepressants, and/or minor tranquilizers.

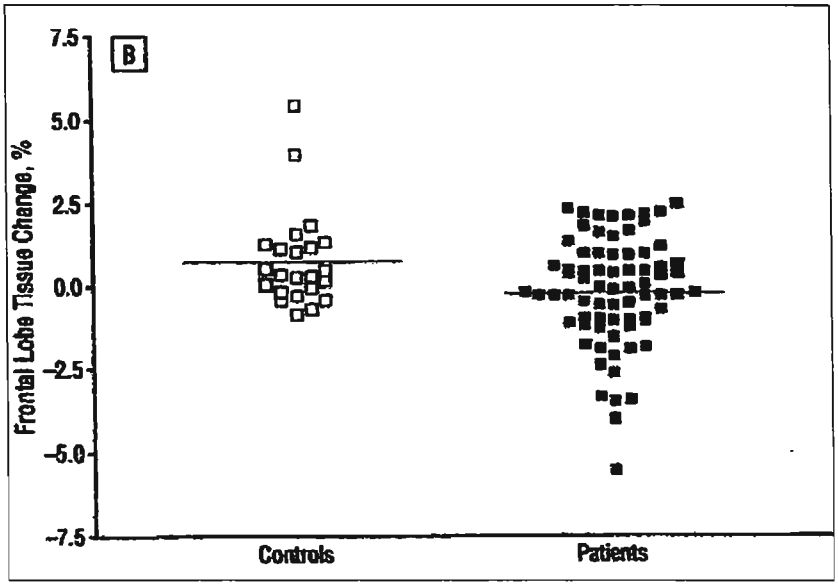
The Jellinger study is historically important because it included two comparison or control groups, allowing for the determination of treatment-related vs. illness-related changes. Damage to the basal ganglia was seen in only 4% of an age-matched group of psychotic patients who had *avoided* long-term therapy with neuroleptics; and in only 2% of a group of patients with routine neurological disease. Based upon the anatomic evidence, Jellinger referred to the abnormal findings as ***human neuroleptic encephalopathy*** (meaning: a drug-induced, degenerative brain process).

Line of Evidence #2: Neuroimaging Studies of Living Human Subjects

Several groups of researchers have documented a progressive reduction of frontal lobe tissue in patients treated with neuroleptics. Madsen et al. performed serial C.T. scans on thirty-one previously unmedicated psychotic patients and nine healthy controls. Imaging was performed at baseline and again after five years.[7-8] During this time, the patients received neuroleptic therapy in the form of traditional antipsychotics (such as Thorazine) and/or clozapine. Findings were remarkable for a significant progression of frontal lobe atrophy in all of the patients, relative to the controls. ***The researchers detected a dose-dependent link to brain shrinkage, estimating the risk of frontal degeneration to be 6% for every 10 grams of cumulative Thorazine (or equivalent) exposure.***

Similar findings have been documented with newer technologies, such as magnetic resonance imaging (MRI). In 1998, Gur et al. published the results of a study which followed forty psychotic patients prospectively for 2 ½ years.[9] At entry, half of these individuals had received previous treatment with neuroleptics, and half were neuroleptic naïve. All patients subsequently received treatment with antipsychotic medications. ***At the end of thirty months, the patients displayed a significant loss of brain volume (4 to 9%) in the frontal and temporal lobes***. For both patient groups, this volume loss was associated with unimpressive changes in target symptoms (e.g., the inability to experience pleasure, restricted affect, and limited speech) and ***with significant deteriorations in cognitive functioning*** (such as attention, verbal memory, and abstract thought).

Researchers at the University of Iowa began a longitudinal investigation of psychotic patients between 1991 and 2001.[10] Enrolling 23 healthy controls, and 73 patients recently diagnosed with schizophrenia, the study design called for a series of MRI exams to be conducted at various intervals (planned for 2, 5, 9, and 12 years). In 2003, the research team published the results from the first interval. Head scans and neuropsychological testing were repeated on all patients after a period of three years of neuroleptic treatment. Several findings were remarkable. ***First, patients demonstrated statistically significant reductions in frontal lobe volume (0.2% decrease per year) compared to the healthy controls***:



These changes were associated with more severe negative symptoms of schizophrenia (alogia, anhedonia, avolition, affective flattening), and with impairments in executive functioning (e.g., planning, organizing, switching). ***Second, almost 40% of the patients failed to experience a remission***, defined by the investigators as eight consecutive weeks with nothing more than mild positive symptoms (delusions, hallucinations, bizarre behavior, inappropriate affect, formal thought disorder). In other words, *almost half of the patients remained floridly psychotic*. ***Third, these poor outcomes occurred despite the fact that the patients had been maintained on neuroleptics*** for 84% of the inter-MRI duration, and ***despite the fact that the newest therapies had been favored***: atypical antipsychotics had been given for 62% of the treatment period. Reflecting upon these disappointing results, the research team conceded:

> "...the medications currently used cannot modify an injurious process occurring in the brain, which is the underlying basis of symptoms...We found that progressive volumetric brain changes were occurring despite ongoing antipsychotic drug treatment." [11]

In 2005, Lieberman et al. published the results of their international study involving serial MRI scans of 58 healthy controls and 161 patients experiencing a first episode of psychosis.[12] Most patients (67-77%) had received prior treatment with antipsychotics for a cumulative duration of at least four months. Throughout the two-year period of follow-up, patients were randomized to double-blind treatment with olanzapine (5 to 20 mg per day) or haloperidol (2 to 20 mg per day). The study protocol permitted the use of concomitant medications, such as minor tranquilizers (up to 21 days of cumulative therapy). Mood stabilizers and antidepressants other than Prozac (which could be used at any time) were allowed only after the first three months of the study. The primary outcome analysis involved a comparison of MRI changes from baseline, focusing upon seven regions of interest: whole brain, whole brain gray matter, whole brain white matter, lateral ventricles, 3rd ventricle, and caudate. ***Haloperidol recipients experienced persistent gray matter reductions throughout the brain***. These abnormalities emerged as early as twelve weeks. ***For olanzapine recipients, significant brain atrophy (loss of gray matter) was detected in the frontal, parietal, and occipital lobes following one year of drug exposure***:

Average change in tissue volume (cubic centimeter) by week 52

	olanzapine	haloperidol	controls
frontal gray	- 3.16	- 7.56	+ 0.54
parietal gray	- 0.86	- 1.71	+ 0.70
occipital gray	- 1.49	- 1.50	+ 0.99
whole brain gray	- 3.70	- 11.69	+ 4.12

In addition to these changes, both groups of patients experienced enlargements in whole brain fluid and lateral ventricle volumes. These disturbances in brain morphology (structure) were associated with retarded improvement in symptoms and neurocognitive functioning.

Line of Evidence #3: Postmortem Animal Studies

Acknowledging the longstanding problem in medicine of distinguishing the effects of treatment from underlying disease processes, scientists at the University of Pittsburgh have advocated the use of animal research involving monkeys (non-human primates). In one such study, the researchers attempted to identify the effects of lab procedures upon brain samples prepared for biochemical and microscopic analyses.[13] Eighteen adult male macaques (aged 4.5 to 5.3 years) were divided into three groups and were trained to self-administer drug treatments. *Monkeys received oral doses of haloperidol, placebo (sham pellets), or olanzapine for a period of 17 to 27 months*. During this time, blood samples were taken periodically and drug doses were adjusted in order to achieve plasma levels identical to those which occur in clinical practice (1 to 1.5 ng/mL for haloperidol; 10-25 ng/mL for olanzapine). At the end of the treatment period, the animals were euthanized. Brains were removed, and brain size was quantified using two different experimental procedures.

A variety of behavioral and anatomical effects were noted. ***First, all animals appeared to develop an aversion to the taste and/or subjective effects of the medications***. This required creative changes in the methods which were used to administer the drug treatments. ***Second, a significant number of monkeys became aggressive during the period of study*** (four of the six monkeys exposed to olanzapine; two of the six monkeys exposed to haloperidol). One monkey, originally placed in the sham treatment group, engaged in self-mutilatory behaviors. A switch to olanzapine resulted in no improvement. However, when the animal was provided with increasing human contact, a doubling of cage space, a decrease in environmental stimuli, and enhanced enrichment, his behavior stabilized. ***Third, the chronic exposure to neuroleptics resulted in significant reductions in total brain weight compared to controls (8% lower weight for haloperidol, 10% lower weight for olanzapine)***. Regional changes in weight and volume were also significant, with the largest changes identified in the frontal and parietal lobes:

	volume reduction in brain weight (relative to sham controls)	
	olanzapine	haloperidol
frontal lobe	10.4%	10.1%
parietal lobe	13.6%	11.2%

Based upon these results, the researchers concluded that the progressive reductions in brain volume which have been reported in many studies on schizophrenia may reflect the effects of drug treatment. They proposed that further studies be undertaken to characterize the mechanisms responsible for these changes and to identify the precise targets (neurons, glia) of these effects.

Line of Evidence #4: Biological Markers of Cell Damage

Researchers in Austria have been interested in identifying a biological marker which can be used to diagnose Alzheimer's dementia or other forms of degenerative disease prior to death. In 2005, Bonelli et al. published the results of an investigation which involved the retrospective analysis of the cerebrospinal fluid (CSF) from 84 patients who had been hospitalized for the treatment of neurological conditions.[14] Hospital diagnoses included two forms of dementia (33 cases of Alzheimer's dementia, 18 cases of vascular dementia), low back pain (9 patients), headache (5 patients), and neuropathy (4 patients). Researchers evaluated the fluid samples for tTG (tissue transglutaminase), an enzyme which is activated during the process of apoptosis or programmed cell death. Medical histories were also reviewed in order to identify pharmaceuticals consumed within 24 hours of the fluid collection via lumbar puncture.

Findings were remarkable for significant relationships between treatment with neuroleptics and elevations in tTG, particularly for females and patients with Alzheimer's dementia. When specific medications were reviewed, five antipsychotics (***including three of the so-called atypicals: melperone, olanzapine and zotepine***) were associated with above average levels of tTG:

tTG levels for patients receiving antipsychotic medications	
melperone	14.95 ng/dL
zotepine	8.78 ng/dL
olanzapine	8.50 ng/dL
flupentixol	7.86 ng/dL
haloperidol	7.30 ng/dL
average tTG for entire patient group:	4.78 ng/dL

Based upon these results, the research team drew the following conclusions:

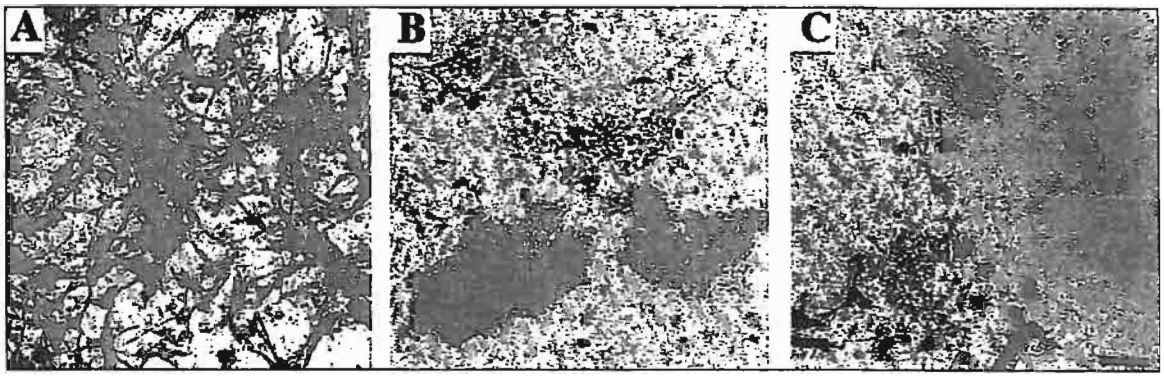
> "...our study failed to show a difference in neurotoxicity between atypical and typical neuroleptics, and we should be careful when using neuroleptics as first-line drugs in Alzheimer's dementia patients...Because the level of cerebral apoptosis of non-demented patients on antipsychotics appears to be indistinguishable to [sic] Alzheimer's dementia patients without this medication, the question might arise as to whether neuroleptics actually induce some degenerative process...In conclusion, we suggest that typical and atypical neuroleptics should be strictly limited in all elderly patients, especially in females and all patients with Alzheimer's dementia." [15]

While there were limitations to the Austrian study, it remains the only existing investigation of cell death in living subjects – none of whom received neuroleptics for mental illness. Furthermore, although the study failed to address possible relationships between apoptosis and antipsychotic exposure in terms of *dose* and *duration of treatment*, the implications extend far beyond the geriatric population. In fact, the finding that neuroleptic medications (and other psychiatric drugs) induce the process of apoptosis has inspired the oncology community to research these chemicals as adjuvant treatments for cancer. In other words, many psychiatric drugs are lethal to rapidly proliferating cells. To the extent that these chemotherapies are lethal to normal as well as cancerous tissues, there exists an urgent need for medical professionals and regulatory authorities to properly characterize the full effects of these toxins.

Line of Evidence #5: Lab Studies of Isolated Cells or Tissues

In vitro studies refer to research conducted upon tissue samples or isolated chemical systems obtained from lab animals or humans. In one such project, researchers in Germany exposed cell cultures to varying concentrations of haloperidol (Haldol).[16] The experiment involved the removal of hippocampal neurons from embryonic rats. Some of these neurons were then incubated with the neuroleptic and or its active metabolite (reduced haloperidol), while a control group of neurons remained drug free. Following a twenty-four hour period of incubation, neurons exhibited a dose-related reduction in viability, relative to the control:

drug concentration	Haldol	Reduced Haldol (drug metabolite)
1 uM	27% cell death	13% cell death
10 uM	35% cell death	29% cell death
100 uM	96% cell death	95% cell death



Examples of neuronal cell loss (death) following incubation with Haldol

A: normal neurons (dark) from unmedicated hippocampal brain tissue
B: 100 uM of Haldol: severe loss of cell bodies and neuron extensions.
Note: Dark patches at bottom of slide represent abnormal cells which have rounded up and detached from the culture dish.
C: 10 uM of Haldol: moderate loss of neurons and neuronal extensions.

Although this particular investigation involved a non-human species (rats), its results were medically concerning. First, the study employed Haldol concentrations which are clinically relevant to humans. In common medical practice, psychiatric patients are exposed to doses of Haldol which produce blood levels of 4 to 26 ng/mL. Brain levels are five to forty times higher. This means that psychiatric patients are indeed exposed to Haldol concentrations (1.4 to 2.8 uM) identical to the low levels that were tested in the German study. Second, the potential toxicity of Haldol in humans may be far greater than that revealed here, based upon the fact that this experiment was time limited (24 hour incubation only). Third, the neurons sampled in this experiment were taken from the key brain structure (hippocampus) associated with learning and memory. The possibility that Haldol kills neurons in this area (even if limited to 30%) provides a mechanism of action which accounts for the cognitive deterioration that is frequently observed in patients who receive this neuroleptic.

Dementia

Several teams of investigators have documented the problems associated with the use of neuroleptics in patients with pre-existing dementia. In a study which enrolled 179 individuals diagnosed with probable Alzheimer's disease, subjects were followed prospectively for an average of four years (range: 0.2 to 14 years).[17] Symptoms were evaluated on an annual basis, and changes in medication were carefully observed. Over the course of the investigation, 41% of the subjected progressed to severe dementia, and 56% of the patients died. Using a statistical procedure called proportional hazards modeling, the ***researchers documented a statistically significant relationship between exposure to neuroleptics and a two-fold higher likelihood of severe neurobehavioral decline***.

In England, a longitudinal investigation followed 71 demented patients (mean age: 72.6 years) over the course of two years.[18] Interviews were conducted at four-month intervals, and autopsy analyses of brain tissue were performed on 42 patients who expired. Main outcomes in this study were changes in cognitive functioning, behavioral difficulties, and (where applicable) postmortem neuropathology. ***The research team discovered that the initiation of neuroleptic therapy was associated with a doubling of the speed of cognitive decline***. This relationship was independent of the degree of dementia or the severity of behavioral symptoms for which the medications may have been prescribed.

While the methodology could not definitively prove that the drugs were the cause of mental deterioration, the study clearly demonstrated their inability to prevent it. The researchers concluded that:

> "an appropriate response at present would be to undertake regular review of the need for patients to continue taking neuroleptic drugs, pursuing trials without medication where possible. This study highlights the importance of understanding the neurological basis of behavioural changes in dementia so that less toxic drugs can be developed for their treatment." [19]

In 2005, an United Kingdom team of investigators performed autopsies on forty patients who had suffered from dementia (mean duration: four years) and Parkinsonian symptoms (mean duration: three years) prior to death.[20] Based upon a postmortem tissue analysis of the brain, exposure to neuroleptics (***old and new***) was associated with a four-fold increase in neurofibrillary tangles, and a 30% increase in amyloid plaques in the cortex of the frontal lobes. Due to the fact that the prevalence of symptoms did not vary between patients who received neuroleptics and those who remained neuroleptic free, the abnormalities detected appeared to be a result of the pharmaceutical agents, rather than a pre-existing disease. Most importantly, the findings suggest that all of the antipsychotics (***old and new***) are capable of inducing or accelerating the pathological changes (plaques and tangles) which are the defining features of Alzheimer's disease.

To review:

> Evidence from postmortem human analyses reveals that older neuroleptics create scarring and neuronal loss in the movement centers of the brain. These changes are an example of *subcortical* dementia, such as Parkinson's or Huntington's disease.
>
> Evidence from neuroimaging studies reveals that ***old and new*** neuroleptics contribute to the progressive shrinkage and/or loss of brain tissue. Atrophy is especially prominent in the frontal lobes which control decision making, intention, and judgment. These changes are consistent with *cortical* dementia, such as Niemann-Pick's or Alzheimer's disease.
>
> Evidence from postmortem analyses in lab animals reveals that ***old and new*** neuroleptics induce a significant reduction in total brain weight and volume, with prominent changes in the frontal and parietal lobes.
>
> Evidence from biological measurements suggests that ***old and new*** neuroleptics increase the concentrations of tTG (a marker of programmed cell death) in the central nervous system of living humans.
>
> Evidence from *in vitro* studies reveals that haloperidol reduces the viability of hippocampal neurons when cells are exposed to clinically relevant concentrations. (Other experiments have documented similar findings with the second-generation antipsychotics.)

Shortly after their introduction, neuroleptic drugs were identified as chemical lobotomizers. Although this terminology was originally metaphorical, subsequent technologies have demonstrated the scientific reality behind this designation. Neuroleptics are associated with the destruction of brain tissue in humans, in animals, and in tissue cultures. Not surprisingly, this damage has been found to contribute to the induction or worsening of psychiatric symptoms, and to the acceleration of cognitive and neurobehavioral decline.

Appendix B

Successful Alternatives to Antipsychotic Drug Therapy [21-22]

In a paper entitled "The Tragedy of Schizophrenia," psychologist and psychotherapist, Dr. Bert Karon, challenges the prevailing notion that psychosis remains a largely incurable brain disease which is best modified by pharmacotherapy. Mindful of the fact that "there has never been a lack of treatments which do more harm than good," Karon explicitly contends that humane psychotherapy remains the treatment of choice for schizophrenia, and he understands why this has always been so.

Karon reminds his readers that history provides important lessons for contemporary practitioners. The Moral Treatment Movement in the late 18th century emphasized four essential elements in the care of the mentally ill:

- respect for the patient (no humiliation or cruelty)
- the encouragement of work and social relations
- the collection of accurate life histories
- the attempt to understand each person as an individual

When these imperatives were applied in the asylums of America and Europe, the rates of discharge reached 60-80%. This was far better than the 30% recovery rate which occurred about a century later, in the era of pharmacotherapy.

Although the Moral Treatment Movement was replaced by the tenets of biological psychiatry in the late 1800s, its elements were incorporated in the theory and practice of various psychosocial therapies. For reasons which were largely political and economic, however, the consensus in American psychiatry came to denigrate the use of these Moral Treatment offshoots – particularly, in the treatment of psychosis.

Academic opinion leaders in the field of psychiatry now contend that there is insufficient evidence to support the use of psychotherapy as a major or independent intervention for psychosis. This perspective is contradicted by a rich (but suppressed) history in the published literature, and by the success of many ongoing programs, some of which are summarized below.

The Bockoven Study

This study compared the prognoses of 100 patients who were treated at Boston Psychopathic Hospital between 1947 and 1952; and 100 patients who were treated at the Solomon Mental Health Center between 1967 and 1972. Patients were similar in the severity of their symptoms, but the earlier cohort received treatment that was limited to psychosocial therapies. In contrast, the 1967 cohort received medication, including neuroleptics. Five-year outcomes were superior for the earlier cohort: 76% return to community and a 44% relapse in terms of re-hospitalization. In comparison, the 1967 cohort experienced an 87% return to the community, but a 66% rate of rehospitalization. The investigators concluded that medications were associated with higher numbers of relapsing patients, and a higher number of relapses per patient.

The Vermont Longitudinal Study of Persons With Severe Mental Illness

In 1955, a multidisciplinary team of mental health care professionals developed a program of comprehensive rehabilitation and community placement for 269 severely disabled, back wards patients at the Vermont State Hospital. When none of these patients improve sufficiently through two or more years of neuroleptic therapy, they were offered a revised plan of treatment. The intensive rehabilitation program was offered between 1955 and 1960. Subsequently, patients were released to the community as they became eligible for discharge, receiving a variety of services that emphasized continuity of care. At a long-term follow-up performed between 1980 and 1982, 68% of patients exhibited no signs of schizophrenia, and 45% displayed no psychiatric symptoms at all. Most patients had stopped using medication (16% not receiving, 34% not using, and 25% using only sporadically). A subsequent analysis revealed that all of the patients with full recoveries had stopped pharmacotherapy completely. (In other words, compliance with antipsychotic drug treatment was neither necessary, nor sufficient, for recovery.)

The Michigan State Psychotherapy Project

Between 1966 and 1981, Drs. Bert Karon and Gary VandenBos supervised the Michigan State Psychotherapy Project in Lansing, Michigan. Patients were randomly assigned to receive about 70 sessions of psychoanalytically informed psychotherapy, medication, or both over a period of 20 months. By the end of treatment, the psychotherapy group had experienced earlier hospital discharge, fewer readmissions (30-50% fewer days of hospitalization), and superior improvement in the quality of symptoms and overall functioning. The poorest outcomes occurred among the chronically medicated, even when drugs were combined with psychotherapy.

The Colorado Experiment

In 1970, Drs. Arthur Deikman and Leighton Whitaker presided over an innovative treatment ward at the University of Colorado. Occurring just 20 years after the advent of the neuroleptics, the Colorado experiment attached a priority to psychosocial interventions during the inpatient care of 51 patients diagnosed with severe mental illness. Individual and group psychotherapies were delivered in the spirit of the Moral Treatment Movement, motivated by a spirit of collaboration, respect, and a desire to understand behaviors as expressive of meaning. Furthermore, psychotherapies were used with the goal of restoring pre-psychotic abilities and independent functioning, rather than with the more limited goal of blunting symptoms in order to justify rapid discharge. *Medications were used as interventions of last resort.* After ten months of experimentation, the researchers made the following discovery: compared to "treatment as usual" (neuroleptics and supportive therapy), the recipients of intensive psychotherapy experienced lower recidivism (fewer readmissions after discharge) and lower mortality.

The Soteria Project

Between 1973 and 1981, Dr. Loren Mosher (then Director of Schizophrenia Research at the National Institute of Mental Health) presided over an investigational program in Northern California. Over the course of nine years, the Soteria project involved the treatment of 179 young psychotic subjects, newly diagnosed with schizophrenia or schizophrenia-like conditions. A control group consisted of consecutive patients arriving at a conventional medical facility, who were assigned to receive care at a nearby psychiatric hospital. Soteria was distinguished by an attitude of hopefulness; a treatment philosophy which de-emphasized biology and medicalization; a care setting marked by involvement and spontaneity; and a therapeutic component which placed a priority upon human relationship. Most significantly, Soteria involved the minimal use of neuroleptics or other drug therapies. Two-year outcomes demonstrated superior efficacy for the Soteria approach. Although 76% of the Soteria patients remained free of antipsychotics in the early stages of treatment; and although 42% remained free of antipsychotics throughout the entire two-year period, the Soteria cohort outperformed the hospital control group (94% of whom received continuous neuroleptic therapy) by achieving superior outcomes in terms of residual symptoms, the need for rehospitalization, and the ability to return to work.

The Agnews State Hospital Experiment

In 1978, Rappoport et al. summarized the clinical outcomes of 80 young males (aged 16-40) who had been hospitalized in San Jose at Agnews State Hospital for the treatment of early schizophrenia. Following acceptance into a double-blind, randomized controlled study, subjects were assigned to receive placebo or neuroleptic therapy (chlorpromazine). Treatment effectiveness was evaluated using various rating scales for as long as 36 months after hospital discharge. The best outcomes, in terms of severity of illness, were found among the patients who avoided neuroleptic therapy both during and after hospitalization. Patients who received placebo during hospitalization, with little or no antipsychotic exposure afterward, experienced the greatest symptomatic improvement; the lowest number of hospital readmissions (8% vs. 16-53% for the other treatment groups); and the fewest overall functional disturbances.

Finland – Acute Psychosis Integrated Treatment (Needs Adapted Approach)

In 1992, clinicians in Finland launched a multi-center research project using Acute Psychosis Integrated (API) Treatment. Keenly aware of the problems associated with antipsychotic drug therapy, the research team adopted a model of care which emphasized four features: family collaboration, teamwork, a basic therapeutic attitude, and adaptation to the specific needs of each patient. The initial phase of the project enrolled 135 subjects (aged 25-34) experiencing a first episode of psychosis. All were neuroleptic naïve, and all had limited or no previous exposure to psychotherapy. Three of the six participating treatment facilities agreed to use antipsychotic medications sparingly. The experimental protocol assigned patients to two groups with 84 receiving the Needs Adapted Approach, and 51 receiving treatment as usual. Two-year outcomes favored the experimental treatment group: fewer days of hospitalization, more patients without psychosis, and more patients with higher functioning. These outcomes occurred despite the fact that the Needs Adapted group consisted of more patients with severe illness (diagnosed schizophrenia) and longer durations of untreated psychosis, and despite the fact that 43% of the Needs Adapted subjects avoided antipsychotics altogether (vs. 6% of the controls).

Subsequent refinements to the Needs Adapted Approach have expanded upon these initial successes.[23-25] In a series of papers describing outcomes for what has evolved to be known as the Open Dialogue Approach, the Finnish clinicians have achieved the following five-year outcomes for first-episode, non-affective psychosis:

82% rate of full remission of psychotic symptoms
86% rate of return to studies of full-time employment
14% rate of disability (based upon need for disability allowance)

The results of the Finnish experiment stand in stark contrast to the results of the prevailing American standard of care, which currently features a 33% rate of lasting symptom reduction or remission; and, at most, a 40% rate of social or vocational recovery.[26]

Pre-Therapy: A Client-Centered Approach [27]

It has been suggested by many professionals that it is not possible to conduct meaningful psychotherapy with any individual who is deep in the throes of a psychotic process. Pre-Therapy refers to a client-centered form of psychotherapy which reaches through psychosis and/or other difficulties (such as cognitive limitations, autism, and dementia) in order to make contact with the pre-verbal or pre-expressive Self. Drawing upon the principles of the late Carl Rogers and developed by American psychologist, Dr. Garry Prouty, Pre-Therapy emphasizes the following treatment philosophy and techniques:

unconditional positive regard for the client:
"the warm acceptance of each aspect of the client's world"

empathy: "sensing the client's private world as if it were your own"

congruence: "within the relationship, the therapist is freely and deeply himself or herself"

non-directiveness: "a surrendering of the therapist to the client's own intent, directionality, and process"

psychological contact: exemplified by the therapist's use of contact reflections, an understanding of the client's psychological or contact functions, and the interpretation of the client's contact behaviors

Although Pre-Therapy has not been promoted or publicized within the United States, it has been used successfully around the world to assist regressed or language-impaired individuals in regaining or improving their capacity for verbal expression. (It has even been used to resolve catatonia successfully, without the use of drug therapy.) [28]

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DATED this 16th day of May, 2008, in WILMINGTON, North Carolina.

Grace E. Jackson MD

Grace E. Jackson, MD

SUBSCRIBED AND SWORN TO before me this 16th day of May, 2008.

Notary Public in and for North Carolina

State of Alaska)
)ss
Third Judicial District)

I, James B. Gottstein, hereby swears that this reproduction of the written testimony of Grace E. Jackson, MD, to which this is appended, is a true, correct and complete photocopy of the original filed in 3AN 08-493PR, Superior Court for the State of Alaska, Third Judicial District at Anchorage.

Dated: _____ _____
James B. Gottstein

SUBSCRIBED AND SWORN TO before me this ____day of _____, 20___.

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Notary Public in and for Alaska
My Commission expires: _____