IN THE SUPERIOR COURT FOR THE STATE OF ALASKA
AT Anchorage

In the Matter of the Necessity) for the Hospitalization of:)	
William Bigley	) Case No. 3AN 08 493 P/R
Respondent.	)
	) PETITION FOR COURT APPROVAL OF
	) ADMINISTRATION OF PSYCHOTROPIC
	) MEDICATION [AS 47.30.839]

Lawrence J. MAIUE Ph.D. petitioner, requests a hearing on the respondent's capacity to give or withhold informed consent to the use of psychotropic medication, and alleges that:

☒ There have been, or it appears that there will be, repeated crisis situations requiring the immediate use of medication to preserve the life of, or prevent significant physical harm to, the patient or another person. The facility wishes to use psychotropic medication in future crisis situations.

☒ Petitioner has reason to believe the patient is incapable of giving or withholding informed consent. The facility wishes to use psychotropic medication in a noncrisis situation.

☐ Court approval has been granted during a previous commitment period, and the facility wishes to continue medication during the subsequent commitment period. A 90/180 day petition is being filed. The patient continues to be incapable of giving or withholding informed consent.

The patient ☒ has refused ☐ has not refused the medication.

4/28/08
Date

L. J. MAIUE Ph.D.
Signature
(Representative of evaluation or designated treatment facility)

L. J. MAIUE Ph.D.
Printed Name

Licensed Psychologist
Title

Verification

Petitioner says on oath or affirms that petitioner has read this petition and believes all statements made in the petition are true.

Subscribed and sworn or affirmed before me at Anchorage Alaska on 4/29/08 (date).

Mary C. Martinez
Clerk of Court, Notary Public, or other person authorized to administer oaths..
My commission expires: with office

MARY C. MARTINEZ NOTARY PUBLIC STATE OF ALASKA My Commission Expires With Office

Exhibit A

IN THE SUPERIOR COURT FOR THE STATE OF ALASKA
AT ANCHORAGE

In the Matter of the Necessity for the Hospitalization of:	)	
WILLIAM BIGLEY, Respondent.	)	Case No. 3AN-08-00493 P/R
	)	FINDINGS AND ORDER CONCERNING COURT-ORDERED ADMINISTRATION OF MEDICATION

FINDINGS AND ORDER

A petition for the court approval of administration of psychotropic medication was filed on April 28, 2008.

Respondent was committed on May 5, 2008 for a period of time not to exceed 30 days in an order signed by Judge Rindner on that date.

Hearings were held on May 12, May 14 and May 15, 2008, to inquire into respondent's capacity to give or withhold informed consent to the use of psychotropic medication, and to determine whether administration of psychotropic medication is in the respondent's best interested considered in light of any available less intrusive treatments. *See Myers v. API*, 138 P.3d 238, 252 (Alaska 2006).

Having considered the allegations of the petition, the evidence presented and the arguments of counsel, the court finds:

1. The evidence is clear and convincing evidence that the respondent is not competent to provide informed consent concerning the administration of psychotropic medication. The evidence presented was clear and convincing that Mr. Bigley lacks the

capacity to assimilate relevant facts about his current mental health condition. This finding is supported not only by the testimony of the health care professionals from API, the court visitor, and Mr. Cornils, but by Mr. Bigley's own demeanor during the course of the court proceedings. Mr. Bigley's demeanor in the courtroom was indicative of some limited understanding by him that the court proceedings were to address API's request for an order to administer psychotropic medication without his consent. But he was quite agitated and maintained a running monologue throughout most of the court proceedings. The evidence was clear and convincing, particularly the testimony of Dr. Maile, that Mr. Bigley denies the existence of a mental illness and is unwilling to confer with either the court visitor or API staff in an effort to assimilate relevant facts about his mental health. The evidence was also clear and convincing that Mr. Bigley is unwilling to participate in treatment decisions at all because he is unwilling to communicate or cooperate at all with API staff or with the court visitor regarding any such proposed treatment. The court visitor attempted to assess Mr. Bigley's capacity to give or withhold informed consent, but was unable to do so because of Mr. Bigley's complete refusal to cooperate with her. Mr. Bigley has indicated that he believes the hospital staff is poisoning him, both as to the food and drink he was provided as well as any medication. Counsel for Mr. Bigley asserted that Mr. Bigley's belief that the medication could poison him was a reasonable objection to the medication, given the medication's side effects. But the evidence was clear and convincing that Mr. Bigley's concern of being poisoned is not due to any potential side effect of the proposed medication; rather, it constitutes a delusional belief that API would attempt to administer a substance that is poison in the strictest sense of that term --rather than an antipsychotic medication with potentially significant side effects. The evidence is clear and convincing that Mr. Bigley does not have the capacity to participate in treatment decisions by means of a rational thought process, and is not able to articulate reasonable objections to using the proposed medication.

2. The evidence is clear and convincing that Mr. Bigley has never previously made a statement while competent that reliably expressed a desire to refuse future treatment with psychotropic medication. The court visitor testified she was unaware of any such statement. Mr. Bigley did not introduce any evidence of such a statement. Through his counsel, Mr. Bigley asserted that the fact that Mr. Bigley promptly ceased taking antipsychotic medication after his prior releases from API is demonstrative of such a statement to refuse future treatment. But this court finds that the fact that Mr. Bigley has ceased taking antipsychotic medication in the past does not, in itself, reliably express a desire to refuse such medication in the future.

3. The evidence is clear and convincing that the proposed course of treatment is in Mr. Bigley's best interest. API has proposed to administer one medication to Mr. Bigley at this time – risperadone. The proposed dosage is up to 50 mgs. every two weeks. API presented clear and convincing evidence that the administration of this medication to Mr. Bigley meets the standard of medical care in Alaska for individuals with Mr. Bigley's medical condition. The evidence is clear and convincing that Mr. Bigley is unable at the present time to obtain any housing or mental health services outside of API because of his current aggressive and angry behavior. He is not welcome at the Brother Francis Shelter or in any assisted living home at the present time. The option that Mr. Bigley simply be permitted to come and go from API as he chooses is not a realistic alternative for two reasons – first, it is inconsistent with API's role as an acute care facility for individuals throughout the state that are in need of acute mental health care, and second, the evidence is clear and convincing that Mr. Bigley would not avail himself of this option even if it were available to him. As such, it is not a less intrusive treatment at all. When medication has been administered in the past to Mr. Bigley, his behavior has improved to such an extent that he has been able to successfully reside in the community, albeit for short periods of time. Without the administration of medication at this time, the evidence is clear and convincing that there will not be any improvement in Mr.

Bigley's mental functioning. And this particular medication has not caused severe side effects to Mr. Bigley in the past. Evidence was introduced that Mr. Bigley has had tardive dyskinesia as a result of the long term administration of antipsychotic medication to him over a period of many years, but the risk of that condition is considerable less with risperadone that with some other medications. [See Transcript of 2003 proceedings at 42-45; 3AN-02-00277 CI] Although CHOICES has provided valuable assistance to Mr. Bigley in the recent past that has enabled Mr. Bigley to function outside of API, the testimony of Paul Cornils constitutes clear and convincing evidence that that entity is not able to provide assistance to Mr. Bigley to enable him to live in the community at the present time because Mr. Bigley is not following treatment advice to receive medication. Although Mr. Bigley presented evidence as to the potential side effects of risperadone, both long term and short term, he presented no viable alternative to such treatment at the present time. In short, the evidence is clear and convincing that in order for Mr. Bigley to be most likely to achieve a less restrictive alternative than his current placement at API, the involuntary administration of risperadone is needed. In reaching this conclusion, this court has considered that the involuntary administration of risperadone to Mr. Bigley by injection is highly intrusive, and that there is a certain degree of pain associated with the receipt of an injection, particularly if it is to be administered to a patient that is strongly opposed to its administration. And the court has considered the adverse side effects of risperadone that were presented in court, and the fact that Mr. Bigley has not experienced some of those side effects, such as diabetes or undesirable weight gain when the drug has been administered to him in the past. The drug has been in use since the early 1990's, and, as noted above, falls within the standard of care in Alaska at the present time. The risk to Mr. Bigley of nontreatment is very high- the evidence is clear and convincing that Mr. Bigley will continue to be unable to function in the community unless he receives this treatment - the only form of treatment that is available to him at the current time. As such, although highly

intrusive to Mr. Bigley in the short term, this court finds that the proposed treatment is the least intrusive means of protecting Mr. Bigley's constitutional right to individual choice in his mental health treatment over the long term.

ORDER

For the foregoing reasons, API's petition for the administration of psychotropic medication is GRANTED, solely with respect to the use of risperadone in an amount not to exceed 50 mg per two weeks during the respondent's period of commitment. If API seeks to use additional or other medication during the period of commitment, it may file a motion to amend this order. If API seeks to continue the use of psychotropic medication without the patient's consent during a period of commitment that occurs after the period in which the court's approval was obtained, the facility shall file a request to continue the medication when it files the petition to continue the patient's commitment.

Pursuant to Mr. Bigley's request at the close of the evidence in this proceeding, this decision is STAYED for a period of 48 hours so as to permit Mr. Bigley to seek a stay of this order from the Alaska Supreme Court.

5-19-08
DATE
12:30 p.m.

Sharon Gleason
SHARON L. GLEASON
Judge of the Superior Court

I certify that on 5/19/08
a copy of this order was sent to:

respondent's attorney
attorney general
treatment facility
court visitor
guardian

Clerk: A. Stanley

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

In The Matter of the Necessity for the Hospitalization of William Bigley, Respondent	)
Case No. 3AN 08-00493PR	

LIMITED ENTRY OF APPEARANCE

Pursuant to Civil Rule 81(d), the Law Project for Psychiatric Rights (PsychRights) hereby enters its appearance on behalf of William Bigley, the Respondent in this matter, limited only to any forced drugging under AS 47.30.838 or AS 47.30.839. All papers filed in this proceeding should be served on the undersigned at 406 G Street, Suite 206, Anchorage, Alaska 99501. Attached hereto are the Submission for Representation Hearing[1] and the affidavits of Robert Whitaker, Ronald Bassman and Paul Cornils, and Motion for a Less Restrictive Alternative, filed in 3AN 08-247PR, pertaining to the Respondent, of which this Court may take Judicial Notice, and a copy of the April 26-29, 2007, e-mail thread advising the petitioner of PsychRights' representation of Respondent.

DATED: April 29, 2008.

Law Project for Psychiatric Rights

By: _____
James B. Gottstein
ABA # 7811100

[1] Counsel was notified at 4:37 pm April 29, 2008, of the hearing to be held in this matter at 8:30 a.m., the next morning, necessitating the attachment of prior pleadings rather than drafting new ones. If counsel had had a chance to draft new pleadings he would have substantially changed his characterization of the Public Defender Agency's performance based on more recent information.

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Attorney for Respondent

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

In The Matter of the Necessity for the)
Hospitalization of William S. Bigley,)
Respondent.)
Case No. 3AN 08-00247 P/S

MOTION FOR LESS INTRUSIVE ALTERNATIVE

COMES NOW, Respondent William S. Bigley (Mr. Bigley), pursuant to *Myers v. Alaska Psychiatric Institute*,[1] and moves for an order requiring API to provide the following less intrusive alternative:[2]

1. Mr. Bigley be allowed to come and go from API as he wishes, including being given food, good sleeping conditions, laundry and toiletry items as reasonably requested by Mr. Bigley.

2. If involuntarily in a treatment facility in the future, Mr. Bigley be allowed out on passes at least once each day for four hours with escort by staff members who like him, or some other party willing and able to do so.

3. API shall procure and pay for a reasonably nice apartment that is available to Mr. Bigley should he choose it.[3] API shall first attempt to negotiate an acceptable abode, and failing that procure it and make it available to Mr. Bigley.

---

[1] 138 P.3d 238 (Alaska 2006).

[2] In his Submission for Representation Hearing, Mr. Bigley pointed out that the AS 47.30.839 forced drugging petition is premature under *Myers*, 138 P.3d at 242-3, and *Wetherhorn v. Alaska Psychiatric Institute*, 156 P.3d 371, 382 (Alaska 2007). Thus, this motion is technically premature as well. However, this motion is being made in the event the Court disagrees the forced drugging petition is premature.

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4. At API's expense, make sufficient staff available to be with Mr. Bigley to enable him to be successful in the community.

5. The foregoing may be contracted for from an outpatient provider.

This motion is supported by Submission For Representation Hearing, Affidavit of Paul Cornils, Affidavit of Ronald Bassman, PhD., and Affidavit of Robert Whitaker, all filed March 6, 2008.

DATED: March 10, 2008.

Law Project for Psychiatric Rights

By: _____
James B. Gottstein
ABA # 7811100

The foregoing and proposed form or order, was hand delivered to Timothy Twomley of the Attorney General's Office and Elizabeth Brennan/Kelly Gibson of the Alaska Public Defender Agency and faxed to the Court Visitor on March 10, 2008.

_____
James B. Gottstein

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[3] API may seek to obtain a housing subsidy from another source, but such source may not be his Social Security Disability income.

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Attorney for Respondent

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

In The Matter of the Necessity for the Hospitalization of William S. Bigley,

Respondent

Case No. 3AN 08-00247 PR

SUBMISSION FOR REPRESENTATION HEARING

In the afternoon of March 5, 2008, I received a call from the Court advising me that Mr. Bigley informed the Court earlier that afternoon that he desired me to represent him in the above captioned matter and that a representation hearing was set for 3:00 pm today.

I. Background

The Law Project for Psychiatric Rights (PsychRights®) with whom I work, is a public interest law firm whose mission is to mount a strategic litigation campaign against unwarranted forced psychiatric drugging and electroshock around the country.[1] A key component of this strategic campaign is to rectify that judges ordering people to take these

[1] Forced electroshock is not administered in Alaska to my knowledge.

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drugs are being misled about them.[2] Psychiatric respondents are particularly vulnerable because what they say is characterized as symptoms of mental illness, *ie.*, that they are delusional. In other words, judges (usually Probate Masters in Anchorage) and even the lawyers assigned to represent them, exhibit an attitude of "if he wasn't crazy, he would know this is good for him," and therefore don't engage in the required adversary process that make judicial proceedings legitimate. If a proper adversarial process were to occur, the courts would be presented with the truth about these drugs, or at least closer to the truth about them,[3] which reveals they are far less effective and far more harmful than the courts are being told and that the ubiquitous use of these drugs is at least halving the number of people who would fully recover after experiencing a psychotic episode(s) and finding themselves subject to involuntary commitment and forced drugging proceedings.[4]

The failure of the Alaska Public Defender Agency to do any investigation of this,[5] nor present any evidence on their clients behalf with respect thereto has led to the current

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[2] Because judges tend to reflect the larger society's views, and because the public should also be told the truth about these drugs, another key component of PsychRights strategic campaign is public education.

[3] Drug manufacturers hide negative data regarding their drugs, claiming they are "trade secrets" and not even the Food and Drug Administration (FDA) is provided with this important data. In my most recent representation of Mr. Bigley, I subpoenaed this secret material from the drug manufacturers involved on the grounds that the court can not possibly properly find Mr. Bigley should be drugged against his will for it being in his best interests under *Myers v. Alaska Psychiatric Institute*, 138 P.3d 238 (Alaska 2006) when critical efficacy and safety data is being hidden. These subpoenas became moot when API abandoned its forced drugging petition.

[4] This will be discussed below.

[5] In fact, they fail to present this evidence even though I have given it to them.

situation where the courts are unknowingly ordering massive amounts of harm on society's most vulnerable people.

As mentioned above, PsychRights seeks to mount strategic litigation and selects which cases it will take based on an evaluation of its potential for achieving PsychRights' strategic objectives.[6] It will also only take cases in which it believes it can provide zealous representation through adequate preparation, and presentation to the court, including appropriate motions. This is the context in which this representation hearing is taking place.

In the instant case, when Mr. Bigley implored me to represent him, I decided I was simply not in a position at that time to zealously represent him because of impending deadlines. However, I am prepared to represent Mr. Bigley with respect to the forced drugging petition only upon the considerations and motions which follow.[7]

II. Mr. Bigley's History and Previous Proceedings

(A) Respondent's History

Prior to 1980, Respondent was successful in the community, he had long-term employment in a good job, was married with two daughters.[8]

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[6] Of course, once a case is taken, the client is entitled to zealous representation with respect to all of the client's issues in the case and PsychRights' strategic objectives are subordinated to the client's interests.

[7] Mr. Bigley, of course, is entitled to the lawyer of his choice, if he can obtain such representation.

[8] Appendix 1-8.

In 1980, Respondent's wife divorced him, took his two daughters and saddled him with high child support and house (trailer) payments, resulting in his first hospitalization at the Alaska Psychiatric Institute (API).[9]

When asked at the time what the problem was Respondent said "he had just gotten divorced and consequently had a nervous breakdown."[10] He was cooperative with staff throughout that first admission.[11]

At discharge, his treating psychiatrist indicated that his prognosis was "somewhat guarded depending upon the type of follow- up treatment patient will receive in dealing with his recent divorce."[12]

Instead of giving him help in dealing with his recent divorce and other problems, API's approach was to lock him up and force him to take drugs that, for him at least, do not work, are intolerable, and have harmful mental and physical effects.[13]

This pattern was set by his third admission to API as described in the Discharge Summery for that admission:" The medication seemed not to have noticeable favorable effects throughout the first several hospital weeks, despite the fact that there were a

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[9] Appendix 1.
[10] Appendix 1.
[11] Appendix 5.
[12] Appendix 8.
[13] The Affidavit of Robert Whitaker, the substance of which is set forth below, describes what the scientific research reveals regarding the lack of effectiveness of these drugs for many, if not most, the way they dramatically increase the likelihood of relapses and prevent recovery, and the extreme physical harm caused by these drugs.

variety of unpleasant Extra Pyramidal Symptoms (EPS)."[14] The Discharge Summary of this admission also states:

> On 3/26/81, a judicial hearing determined that there would be granted a 30 day extension during which time treatment efforts would continue, following which there would be a further hearing concerning the possibility of judicial commitment. Mr. Bigley was furiously angry that he was deprived of his right to freedom outside the hospital, but despite his persistent anger and occasional verbal threats, he never became physically assaultive, nor did he abuse limited privileges away from the locked unit.
>
> After the first six hospital weeks he continued to believe that he had some special mission involving Easter Island - drug addicts and alien visitors to the Earth. When these views were gently challenged he became extremely angry, usually walking away from whoever questioned his obviously disordered thoughts.[15]

Twenty-Three years and over Fifty admissions later, the Visitor's Report of May 25, 2004 in his guardianship case, reports, "when hospitalized and on medications, [Respondent's] behaviors don't appear to change much . . . . Hospitalization and psychotropic medication have not helped stabilize him."[16]

On March 23, 2007, at discharge from his 68th admission to API, Dr. Worrall, summarized his condition after having "potentially reached the maximum benefits from hospital care," by which, he has consistently testified solely means forcing Respondent to take psychiatric drugs against his will, that Respondent was "delusional" had "no insight

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[14] Appendix 11. Extra Pyramidal Symptoms, are involuntary movements resulting from the brain damage caused by these drugs. In the early 1980's, the standard of care was that the "therapeutic dose" had been achieved when Extra Pyramidal Symptoms appeared.
[15] Appendix 11.
[16] 3AN-99-1108. The Court may take judicial notice of this and other filings in this and other proceedings. *Drake v. Wickwire*, 795 P.2d 195, n1 (Alaska 1990).

and poor judgment, . . . paranoid and guarded." [17] In other words, even after he had been given the drugs against his will and achieved "maximum benefit" therefrom, he was still "delusional" had "no insight and poor judgment, . . . paranoid and guarded."

Prior to the Alaska Supreme Court's ruling in *Wetherhorn*, API's plan was to have Mr. Bigley continuously on an involuntary commitment under the unconstitutional "gravely disabled" standard definition contained in AS 47.30.915(7)(B), pump him full of long-acting Risperdal Consta, administer other psychotropic drugs, such as Seroquel and Depakote, give him an "Early Release" under AS 47.30.795(a), knowing he would quit them once discharged and then order him returned pursuant to AS 47.30.795(c) when he wasn't drugged to their liking.[18]

The Office of Public Advocacy (OPA) was appointed Mr. Bigley's conservator in 1996 or so in Case No. 3AN-99-1108.

On April 14, 2004, API filed a petition for temporary and permanent guardianship. On June 30, 2004, OPA was appointed Mr. Bigley's temporary full guardian and on December 26, 2004, permanent full guardian.

After being appointed, the Guardian unilaterally, without consultation with Mr. Bigley, decided he should become Medicaid eligible even though Mr. Bigley did not want Medicaid Services.[19]

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[17] Appendix 15.
[18] Tr. 4/3/07:275 (3AN 07-247 PR). This is an illegal use of AS 47.30.795(c) because it only allows an order to return if the outpatient provider "determines" the person is a harm to self or others or gravely disabled.
[19] Tr. 4/3/07:216 *et. seq.* (3AN 07-247 PR).

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Because Mr. Bigley's income was above the Medicaid limit, the Guardian established an irrevocable trust, known as a "Miller Trust," with the Guardian as trustee without discussing this with Mr. Bigley or certainly obtaining his consent.[20]

This removed a substantial percentage of Mr. Bigley's income as available for general financial support.[21] Mr. Bigley is eligible for free medical care as an Alaska Native and doesn't need Medicaid to be eligible for such services.[22]

The Guardian has filed a number of *ex parte* petitions to have Mr. Bigley committed in order to have him forcibly drugged against his will.[23]

This includes "insisting" Respondent is gravely disabled under the "unable to survive safely in freedom" standard recently enunciated in *Wetherhorn v. API*, 156 P.3d 371, 379 (Alaska 2007), when his treating psychiatrist did not believe his survival was in jeopardy as required by *Wetherhorn*.[24]

(B)2007 Involuntary Commitment and Forced Drugging Proceedings

30-Day petitions for commitment and forced drugging were filed on February 23, 2007 under Case No. 3AN-07-274 P/S, a hearing held before the Probate Master on February 27, 2007, and approved by the Superior Court on March 2, 2007.

Mr. Bigley was given an "early release" under AS 47.30.795(a), and then illegally "ordered to return," under AS 47.30.795(c), prior to the expiration of the 30-day

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[20] *Id.*
[21] *Id.*
[22] Tr. 4/3/07:208. (3AN 07-247 PR).
[23] *See*, e.g., Tr. 4/3/07:202 (3AN 07-247 PR).
[24] Appendix 19.

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commitment for not taking Depakote as prescribed.[25] This put Respondent back in API before the expiration of the 30-Day commitment order and on March 21, 2007, a 90-day continuation petition was filed.

On March 22, 2007, PsychRights, which had not represented Respondent at the 30-Day Petition hearing, filed an entry of appearance on behalf of Respondent, electing, among other things, a jury trial.

Respondent won the jury trial when the jury found API had not met its burden of proving Respondent's mental condition would be improved by the course of treatment, and he was released on April 4, 2007.

Yet another 30-day commitment petition was filed on May 14, 2007, and a forced drugging petition on May 15th, both of which were granted. PsychRights did not represent Respondent. In due course, API filed 90-day petitions for commitment and forced drugging petition. PsychRights did not represent Respondent with respect to those petitions, but I testified as a fact witness on his behalf in the public jury trial elected by Respondent. On June 26, 2007, the jury found API had not met its burden of proving Respondent was gravely disabled and he was released.[26]

On August 29, 2007, Mr. Bigley was brought in on an *Ex Parte* Order,[27] and I subsequently filed an entry of appearance on his behalf for the forced drugging petition

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[25] Appendix 20-24. The order to return was illegal because it was based solely on Respondent failing to take Depakote and AS 47.30.795(c) only allows someone to be ordered to return if it is determined, the person is a danger to self or others or gravely disabled.

[26] Appendix 25-26.

[27] 3AN 07-1064PR.

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only. I mounted a serious defense and filed for a specific less intrusive alternative which was available, essentially what is presented here, and before the court could consider the less intrusive alternative, API abandoned the forced drugging petition, discharging him to the street knowing full well that he was likely to be arrested because he was bothering Senator Murkowski's staff. This exactly what happened.

Then when I was on an extended trip outside of the State, API filed a new set of involuntary commitment and forced drugging petitions. I came back before the hearing, but did not represent Mr. Bigley and he was involuntarily committed for 30 days and subjected to a forced drugging order, which was subsequently extended for 90 days. Mr. Bigley was then placed in an assisted living home outside of Houston, Alaska, called the "Country Club," which required him to take his prescribed medications. After living there for over a month, he quit taking his medications and left, whereupon he was picked up and delivered to API, which resulted in these proceedings.

(C) CHOICES, Inc.'s Involvement with Respondent.

Paul Cornils of CHOICES, Inc., an independent case management agency, first began working with Respondent Bill Bigley in January of 2007, under contract with PsychRights, but when the cost of services exceeded $5,000 PsychRights said it could not afford to continue paying and Mr. Bigley informed Mr. Cornils he did not want to work with him any more so services were discontinued.[28]

[28] ¶B of Paul Cornils Affidavit.

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CHOICES began working with Mr. Bigley again in July of that year at the request of the Office of Public Advocacy (OPA), Mr. Bigley's Guardian, and has continued to do so.[29]

According to Mr. Cornils, Respondent is so angry at being put under a guardianship that he takes extreme measures to try to get rid of his guardianship, and as a result, he is mostly refusing to cooperate in virtually any way with the Guardian.[30]

Mr. Cornils cites as an example that Respondent rips up checks from the Guardian made out to Vendors on his behalf, trying to force the Guardian to give him his money directly and as part of his effort to eliminate the guardianship.[31]

According to Mr. Cornils, Respondent has also refused various offers of "help" from the Guardian, such as grocery shopping in a similar attempt to get out from under the guardianship.[32]

Mr. Cornils further testified that Respondent exhibits the same types of behavior to him, but CHOICES/Mr. Cornils have a different approach, which involves negotiation and discussion, does not involve coercion and where the natural consequences of Respondent's actions are allowed to occur.[33]

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[29] ¶C of Paul Cornils Affidavit.
[30] ¶D of Paul Cornils Affidavit.
[31] ¶E of Paul Cornils Affidavit.
[32] ¶F of Paul Cornils Affidavit.
[33] ¶G of Paul Cornils Affidavit.

(D) 2006/2007 Guardianship Proceedings

In late November, 2006, I was invited to subpoena documents pursuant to a protective order in the *Zyprexa Products Liability Litigation*,[34] that had been culled from some 15 million pages of documents produced by Eli Lilly, the manufacturer, by an expert retained in that case. Getting such information legally out to the public would advance PsychRights strategic goals so I looked for an appropriate case from which to subpoena the documents. On December 5, 2006, I met with Mr. Bigley at API and determined his was a suitable case.[35]

On December 6, 2006, I filed a petition in the guardianship proceeding, Case No. 3AN 04-545 PG, to:

(1) Terminate the Guardianship.

(2) Remove the Guardian and appoint a successor of Respondent's choice.

(3) Amend the powers of the Guardian under the Guardianship Plan to the least restrictive necessary to meet Respondent's essential requirements for physical health and safety.

(4) Review and reverse the decision of the guardian to consent to the administration of psychotropic medication against the wishes of Respondent.

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[34] MDL 1596, United States District Court for the Eastern District of New York.

[35] Great consternation has ensued over my subpoenaing and releasing these documents to the New York Times and other persons, but I am not otherwise addressing it here. However, all of the court documents and related material are available on the Internet at http://psychrights.org/States/Alaska/CaseXX.htm. Because of how much Zyprexa is prescribed, I was pretty sure when I subpoenaed the documents that Mr. Bigley had been prescribed it pursuant to a forced drugging order. He had. Appendix 28. He was also later "taken down" with a Zypexa injection, in what is known as an "IM Backup." Appendix 29. To me the opportunity to subpoena an expert who had already combed the documents and could testify to them was "low hanging fruit." In contrast, I think it is fair to characterize Eli Lilly's view of how the events ended up transpiring as a "drive by shooting."

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(5) Amend the powers of the Guardian to eliminate the authority to consent to mental health treatment.

After numerous proceedings, this resulted in a settlement agreement on July 20, 2007, which (a) established some parameters for the administration of the guardianship and (b) provided Respondent with a clear path towards terminating his guardianship (Guardianship Settlement Agreement). As relevant here, the Guardianship Settlement Agreement provides:

> 4.2. Increase of Discretionary Funds. It is recognized the amounts available for food and spending money (Discretionary Funds) are low and efforts will be made to find housing acceptable to Respondent which will increase the amount of Discretionary Funds. To that end, the Guardian shall make its best efforts to obtain subsidized housing for Respondent that will allow an increase in Respondent's Discretionary Funds. ...
>
> 6. Mental Health Services. Respondent has largely been unwilling to accept mental health services. Some services that Respondent may hereafter, from time to time, desire are identified in the subsections that follow. Others may be identified later. To the extent Respondent, from time to time, desires such services, the Guardian and API will support the provision of such services, including taking such steps as may be required of them to facilitate the acquisition thereof to the best of their ability.[36]
>
> 6.2. Extended Services. Extended services, such as Case Management, Rehabilitation, Socialization, Chores, etc., beyond the standard limits for such services.
>
> 6.3. Other Services. Additional "wrap-around" or other types of services Respondent, from time to time, desires.
>
> 7. Involuntary Commitment Proceedings. The Guardian will make a good faith effort to (a) avoid filing any initiation of involuntary commitment petitions against Respondent under AS 47.30.700. In making such efforts,

[36] A footnote here, states: "By agreeing to this stipulation API is not making any judgment regarding eligibility standards under Medicaid regulations."

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the Guardian will explore all available alternatives, including notifying and requesting the assistance of Respondent's counsel herein, James B. Gottstein.

7.2. Unless the Guardian determines it is highly probable that serious illness, injury or death is imminent, in the event the Guardian believes a petition to initiate involuntary commitment might be warranted, rather than the Guardian filing such a petition, the Guardian shall relay its concerns to another appropriate party for evaluation. Without in any way limiting the generality of the foregoing, appropriate parties, might be Respondent's outpatient provider, if any; other people working with him; or other people who know him.

8. Psychotropic Medications. API shall not accept a consent by the Guardian to the administration of psychotropic medication, while Respondent is committed to API to which Respondent objects.

III. Substantive and Procedural Matters

The core holding of the Alaska Supreme Court in *Myers* is:

> [A] court may not permit a treatment facility to administer psychotropic drugs unless the court makes findings that comply with all applicable statutory requirements and, in addition, expressly finds by clear and convincing evidence that the proposed treatment is in the patient's *best interests* and that *no less intrusive alternative is available*.[37]

(A) Best Interests

In addressing the required *Myers* requirements, API must rebut the following, which is taken from the Affidavit of Robert Whitaker filed in the forced drugging proceeding API abandoned last September, a certified copy of which is filed herewith.[38]

> **II. Overview of Research Literature on Schizophrenia and Standard Antipsychotic Medication**
>
> 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

[37] 38 P.3d at 254, emphasis added.
[38] 3AN 08-1064PR

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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."[39]

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 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).[40]

b) Second, researchers have found that if patients abruptly quit taking antipsychotic medications, they are at high risk of relapsing. [41]

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.

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

[40] Cole, J, et al. "Phenothiazine treatment in acute schizophrenia." *Archives of General Psychiatry* 10 (1964):246-61.

[41] Gilbert, P, et al. "Neuroleptic withdrawal in schizophrenic patients." *Archives of General Psychiatry* 52 (1995):173-188.

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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 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.[42]

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.[43, 44, 45] 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 toward psychotic relapse in

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

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

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

[45] 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.

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a patient who had developed such a supersensitivity is determined by more than just the normal course of the illness. [46]

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.[47, 48, 49] 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.[50]

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 completely, and that all who did shared one characteristic: They had all stopped taking antipsychotic medication.

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[46] 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.

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

[48] 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.

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

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

The notion that schizophrenics needed to stay on antipsychotics all their lives was a "myth," Harding said.[51, 52, 53]

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.[54, 55, 56, 57] 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.[58]

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

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[51] Harding, C. "The Vermont longitudinal study of persons with severe mental illness," *American Journal of Psychiatry* 144 (1987):727-34.
[52] Harding, C. "Empirical correction of seven myths about schizophrenia with implications for treatment." *Acta Psychiatrica Scandinavica* 90, suppl. 384 (1994):140-6.
[53] McGuire, P. "New hope for people with schizophrenia," *APA Monitor* 31 (February 2000).
[54] Ciompi, L, et al. "The pilot project Soteria Berne." *British Journal of Psychiatry* 161, supplement 18 (1992):145-53.
[55] 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.
[56] Cullberg J. "One-year outcome in first episode psychosis patients in the Swedish Parachute Project. *Acta Psychiatrica Scandinavica* 106 (2002):276-85.
[57] Lehtinen V, et al. "Two-year outcome in first-episode psychosis according to an integrated model. *European Psychiatry* 15 (2000):312-320.
[58] Seikkula J, et al. Five-year experience of first-episode nonaffective psychosis in open-dialogue approach. *Psychotherapy Research* 16/2 (2006): 214-228.

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15-year followup exams, versus five percent of the medicated patients.[59]

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."[60] 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.

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.[61, 62, 63, 64, 65]

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[59] 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.

[60] 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).

[61] Shear, K et al. "Suicide associated with akathisia and deport fluphenazine treatment," *Journal of Clinical Psychopharmacology* 3 (1982):235-6.

[62] Van Putten, T. "Behavioral toxicity of antipsychotic drugs." *Journal of Clinical Psychiatry* 48 (1987):13-19.

[63] Van Putten, T. "The many faces of akathisia," *Comprehensive Psychiatry* 16 91975):43-46.

[64] Herrera, J. "High-potency neuroleptics and violence in schizophrenia," *Journal of Nervous and Mental Disease* 176 (1988):558-561.

[65] Galynker, I. "Akathisia as violence." *Journal of Clinical Psychiatry* 58 (1997):16-24.

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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.[66] The quality of life on conventional neuroleptics, researchers agreed, is "very poor." [67]

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."[68]

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.[69, 70, 71] Schizophrenia patients now commit suicide at 20 times the rate they did prior to the use of neuroleptics.[72]

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[66] Van Putten, T. "The board and care home." *Hospital and Community Psychiatry* 30 (1979):461-464.
[67] Weiden P. "Atypical antipsychotic drugs and long-term outcome in schizophrenia." *Journal of Clinical Psychiatry* 57, supplement 11 (1996):53-60.
[68] Keefe, R. "Do novel antipsychotics improve cognition?" *Psychiatric Annals* 29 (1999):623-629.
[69] Arana, G. "An overview of side effects caused by typical antipsychotics." *Journal of Clinical Psychiatry* 61, supplement 8 (2000):5-13.
[70] Waddington, J. "Mortality in schizophrenia." *British Journal of Psychiatry* 173 (1998):325-329.
[71] Joukamaa, M, et al. Schizophrenia, neuroleptic medication and mortality. *British Journal of Psychiatry* 188 (2006):122-127.
[72] Healy, D et al. "Lifetime suicide rates in treated schizophrenia." *British Journal of Psychiatry* 188 (2006):223-228.

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."[73]

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.[74, 75, 76, 77, 78] Jeffrey Mattes, director of the Psychopharmacology Research Association, concluded in 1997: "It is possible, based on the available studies, that risperidone is not

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[73] FDA approval letter from Robert Temple to Janssen Research Foundation, December 21, 1993.

[74] Rosebush, P. "Neurologic side effects in neuroleptic-naïve patients treated with haloperidol or risperidone." *Neurology* 52 (1999):782-785.

[75] Knable, M. "Extrapyramidal side effects with risperidone and haloperidol at comparable D2 receptor levels." *Psychiatry Research: Neuroimaging Section* 75 (1997):91-101.

[76] Sweeney, J. "Adverse effects of risperidone on eye movement activity." *Neuropsychopharmacology* 16 (1997):217-228.

[77] Carter, C. "Risperidone use in a teaching hospital during its first year after market approval." *Psychopharmacology Bulletin* 31 (1995):719-725.

[78] Binder, R. "A naturalistic study of clinical use of risperidone." *Psychiatric Services* 49 (1998):524-6.

as effective as standard neuroleptics for typical positive symptoms."[79] 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, 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.[80]

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."[81]

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[79] Mattes, J. "Risperidone: How good is the evidence for efficacy?" *Schizophrenia Bulletin* 23 (1997):155-161.
[80] See Whitaker, R. *Mad in America*. New York: Perseus Press (2002):279-281.
[81] Geddes, J. "Atypical antipsychotics in the treatment of schizophrenia." *British Medical Journal* 321 (2000):1371-76.

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.[82]

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.[83] This finding was 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.[84]

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.

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[82] Lieberman, J, et al. "Effectiveness of antipsychotic drugs in patients with schizophrenia." *New England Journal of Medicine* 353 (2005):1209-1233.
[83] Davies, L, et al. "Cost-effectiveness of first- v. second-generation antipsychotic drugs." *The British Journal of Psychiatry* 191 (2007):14-22.
[84] Morgan, M, et al. "Prospective analysis of premature morbidity in schizophrenia in relation to health service engagement." *Psychiatry Research* 117 (2003):127-35.

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

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.

The foregoing makes clear that the continued forced drugging of Mr. Bigley is not in his best interests.

(B)There is a Less Intrusive Alternative Available

Mr. Whitaker's Affidavit discusses successful less intrusive alternatives. In addition, the affidavit of Ronald Bassman, PhD filed in the same case, a certified copy of which is filed herewith, testifies to less intrusive alternatives, and included citations to the scientific literature. In particular, Dr. Bassman testifies:

> In the above concepts promoting recovery there is a conspicuous absence of psychiatric medication. Psychologist Courtenay Harding, principal researcher of the "Vermont Longitudinal Study," has empirically demonstrated that people do recover from long-term chronic disorders such as schizophrenia at a minimum rate of 32 % and as high as 60%. These studies have consistently found that half to two thirds of patients significantly improved or recovered, including some cohorts of very chronic cases. The 32 % for full recovery is with one of the five criteria being *no longer taking any psychiatric medication*. Dr. Harding in delineating the seven myths of schizophrenia, addresses the myth about psychiatric medication. Myth number 5. Myth: Patients must be on medication all their lives. Reality: It may be a small percentage who need medication indefinitely. According to Harding and Zahniser, the myths limit the scope and effectiveness of treatments available to patients.

(citations omitted, italics in original, underlining added)

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Sarah Porter, who happened to be in Anchorage, was qualified as an expert in the area of alternative treatments and testified to the following:[85]

> A. I've worked in the mental health [field] in New Zealand for the last 15 years in a variety of roles. I'm currently employed as a strategic advisor by the Capital and Coast District Health Board. I'm currently doing a course of study called the Advanced Leadership and Management in Mental Health Program in New Zealand. And, in fact, the reason I'm here is, I won a scholarship through that program to study innovative programs that are going on in other parts of the world so that I could bring some of that information back to New Zealand. I also have personal experience of using mental health services which dates back to 1976 when I was a relatively young child. . . . set up and run a program in New Zealand which operates as an alternative to acute mental health services. It's called the KEYWA Program. That's spelled K-E-Y-W-A. Because it was developed and designed to operate as an alternative to the hospital program that currently is provided in New Zealand. That's been operating since December last year, so it's a relatively new program, but our outcomes to date have been outstanding, and the funding body that provided with the resources to do the program is extremely excited about the results that we've been able to achieve, with people receiving the service and helping us to assist and [starting] out more similar programs in New Zealand.
>
> Q You're a member of the organization called INTAR, is that correct?
>
> A I am a member of INTAR, which is the International Network of Treatment Alternatives for Recovery. And I'm also a member of the New Zealand Mental Health Foundation, which is an organization in New Zealand that's charged with the responsibility for promotion of mental health and prevention of mental disability in New Zealand.
>
> Q Okay. Are there -- can you describe a little bit what INTAR is about?
>
> A INTAR is an international network of people who are interested in promoting the knowledge about, and availability of access to alternatives to traditional and mainstream approaches to treating mental distress. And INTAR is really interested in identifying successful methods of working with people experiencing distress to promote mental well being, and, in particular,

[85] Tr. 9/5/2007:73-81.

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alternatives to the use of mainstream medical model or medication type treatments.

Q And are there people in INTAR that are actually running those kind of programs?

A There are. There's a wide variety of people doing that. And some of them are, also, themselves, interestingly, have backgrounds in psychiatry and psychology.

Q . . . Are there members of INTAR who are psychiatrists?

A There are. Indeed. Yes, indeed.

Q Do you know -- do you remember any of their names?

A Dr. Peter Stastny is a psychiatrist, Dr. Pat [Bracken], who manages the mental health services in West Cork, Ireland, and also in parts of England, as a psychiatrist. . .

Q Okay. Is it fair to say that all these people believe that there should be other methods of treating people who are diagnosed with mental illness than insisting on medication?

A Absolutely, there are. And that's quite a strong theme, in fact, for -- for that group, and I believe that it's based on the fact that there is now growing recognition that medication is not a satisfactory answer for a significant proportion of the people who experience mental distress, and that for some people...it creates more problems than solutions. . . .

Q. Now, I believe you testified that you have experience dealing with those sorts of people as well, is that correct?

A I do.

Q And would that include someone who has been in the system for a long time, who is on and off drugs, and who might refuse them?

A Yes. Absolutely. We've worked with people in our services across the spectrum. People who have had long term experience of using services and others for whom it's their first presentation.

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Q And when you say "long term use of services," does that include -- does that mean they need medication?

A Unfortunately, in New Zealand the primary form of treatment, until very recent times, has been medication, through the lack of alternatives. . . . And we're just now beginning to develop alternatives. They'd offer people real choice and options in terms of what is available instead of medication that might enable people to further address the issues which are raised by the concerns related to their mental state.

Q And I think I understood you to say that the program that you run along that line has had very good outcomes, is that correct?

A It has. The outcomes to date have been outstanding. The feedback from services users and from other people working with the services -- both, peoples families and the clinical personnel working with those people has supported the approach that we have taken.

Q And is -- and I think you said that, in fact, it's been so impressive that the government is looking at expanding that program with more funding?

A Indeed. And, in fact, right across New Zealand they are now looking at what can be done to create -- make resources available to set up...more such services in New Zealand. . .

Q Is there a philosophy that you might describe in terms of how -- that would go along with this kind of alternative approach?

A The way that I would describe that is that it's -- it's really about relationships. It's about building a good therapeutic relationship with the person in distress and supporting that person to recognize and come to terms with the issues that are going on in their life, in such a way that builds a therapeutic alliance and is based on negotiation, rather than the use of force or coercion, primarily...

A ...because we recognize that the use of force and coercion actually undermines the therapeutic relationship and decreases the likelihood of compliance in the long term with whatever kinds of treatment or support has been implicated for the person. So we have created and set up our service along the lines of making relationship and negotiation the primary basis for working with the person and supporting the person to reflect on and reconsider what's going on to create what might be defined as a crisis, and to

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devise strategies and plans for how the person might be with the issues and challenges that they face in their life. . . .

Q Now, you mentioned -- I think you said that coercion creates problems. Could you describe those kind of problems?

A Well, that's really about the fact that [there is] growing recognition -- I think worldwide, but particularly in New Zealand, that coercion, itself, creates trauma and further distress for the person, and that that, in itself, actually undermines the benefits of the treatment that is being provided in a forced context. And so our aiming and teaching is to be able to support the person to resolve the issues without actually having to trample...on the person's autonomy, or hound them physically or emotionally in doing so.

Q And I think you testified that would be --include people who have been in the system for a long time, right?

A It does, indeed. Yes.

Q And would that include people who have been coerced for a long time?

A In many cases, yes. . . .

Q And -- and have you seen success in that approach?

A We have. It's been phenomenal, actually. Jim, I've been -- personally, I -- I had high hopes that it would work, but I've...been really impressed how well, in fact, it has worked.

The affidavit of Paul Cornils, a certified copy of which is filed herewith shows a less intrusive alternative is available.

It is expected Mr. Whitaker, Ms. Porter and Dr. Bassman can be available for further testimony and cross-examination by telephone and Paul Cornils in person.

API may not avoid its obligation to provide a less intrusive alternative by choosing to not make it available. *Wyatt v. Stickney*, 344 F.Supp. 387, 392 (M.D.Ala.1972) ("no default can be justified by a want of operating funds."), affirmed, *Wyatt v. Anderholt*, 503

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F.2d 1305, 1315 (5th Cir. 1974)(state legislature is not free to provide social service in a way that denies constitutional right). In *Wyatt* the federal courts required the State of Alabama to spend funds in specific ways to provide constitutionally adequate services.

Having invoked its awesome power to confine Respondent and having sought to exercise its similarly awesome power to forcibly medicate him against his will "for his own good," Respondent's constitutional right to a less intrusive alternative has sprung into being. This is what *Myers* holds. *Wyatt* holds that API may not avoid its obligation to do so merely by choosing not to provide the less intrusive alternative, *i.e.*, providing a social service that denies Respondent's right to a less intrusive alternative.

Neither should API be allowed to again discharge its obligation to provide a less intrusive alternative by discharging Mr. Bigley from the hospital so it can pick him up at a later point when PsychRights is not available to represent him.

IV. Procedural Issues

In addition to the substantive issues of *best interests* and *less intrusive alternative*, there are a some procedural issues which are hereby raised at this time.

(A) Objection to Referral to the Probate Master.

First, Mr. Bigley objects to the referral of the forced drugging petition to the Probate Master pursuant to Probate Rule 2(c). There are many reasons why the referral to the Probate Master should not be maintained.

(1) Objections to an Unfavorable Recommendation Will Be Filed

For the substantive reasons that (i) the forced drugging is not in Mr. Bigley's best interests, and (ii) there is a less intrusive alternative available, objections under Probate

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Rule 2(f) will be filed to an unfavorable recommendation. Mr. Bigley respectfully suggests both practicality and the Superior Court taking its obligations to consider both of these *Myers* requirements seriously, dictate that it handle the case directly.

(2) Probate Rule 2(b)(3)(D) is Invalid

Another reason why the referral to the Probate Master should not be maintained is that Probate Rule 2(b)(3)(D), providing that the master's recommendation to grant the forced drugging petition is effective pending superior court review is invalid.

In *Myers v. Alaska Psychiatric Institute*, 138 P.3d 238, 254 (Alaska 2006), the Alaska Supreme Court held:

> [A] *court* may not permit a treatment facility to administer psychotropic drugs unless the *court* makes findings that comply with all applicable statutory requirements and, in addition, expressly finds by clear and convincing evidence that the proposed treatment is in the patient's best interests and that no less intrusive alternative is available.

(emphasis added).

Probate Rule 2(b)(3)(D) making the Probate Master's recommendation to approve the forced drugging petition effective before Superior Court approval is therefore invalid.

In *Wetherhorn v. Alaska Psychiatric Institute*, 156 P.3d 371, 381 (Alaska 2007), the Alaska Supreme Court held:

> The expedited process required for involuntary commitment proceedings is aimed at mitigating the infringement of the respondent's liberty rights that begins the moment the respondent is detained involuntarily. In contrast, so long as no drugs have been administered, the rights to liberty and privacy implicated by the right to refuse psychotropic medications remain intact. Therefore, in the absence of an emergency, there is no reason why the statutory protections should be neglected in the interests of speed.

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Probate Rule 2(b)(3)(D) impermissibly dispenses with statutory protections as well as the constitutional protections *Wetherhorn* requires.[86] Because these proceedings are normally conducted in a *pro forma* manner, with respondents immediately forcibly drugged, which the Alaska Supreme Court has equated with electroshock and lobotomy,[87] without a meaningful opportunity to present a defense, and before even the Superior Court has approved it, as required by Alaska Statutes, let alone given a chance for Supreme Court review, Mr. Bigley feels he must make his objection to the employment of Probate Rule 2(b)(3)(D) prophylactically now in the event the referral to the Probate Master is maintained and he recommends approval of the forced drugging petition.

If the referral to the Probate Master is maintained, and the Probate Master recommends granting the forced drugging petition, in the alternative, Mr. Bigley prophylactically moves for a stay pursuant to Probate Rule 2(f)(2), pending Superior Court review.

In the alternative to that, Mr. Bigley prophylactically moves for a one week stay to seek relief in the Supreme Court. This motion is supported by the foregoing discussion and evidence regarding best interests and a less intrusive alternative.

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[86] Moreover, because Probate Rule 2(b)(3)(D) only makes the Probate Master's determinations as to capacity to give informed consent effective pending Superior Court Review and does not make the Probate Master's recommendations as to best interests and less intrusive alternatives required by *Myers* effective pending Superior Court review, it does not authorize the hospital to forcibly drug Respondent before Superior Court review after *Myers*.

[87] *See*, *Myers* 138 P3d at 242; *Wetherhorn*, 156 P.3d at 382.

(3) Civil Rule 53(d)(1)'s Requirement of a Transcript is Violated As a Matter of Course

Civil Rule 53(d)(1) requires a transcript accompany the Probate Master's report. This requirement is routinely ignored. Mr. Bigley is entitled to have this rule followed and referral should not be maintained when this Court expects the Probate Master to violate the rule.[88]

(B)The Forced Drugging Petition is Premature

In *Myers v. Alaska Psychiatric Institute,* the Alaska Supreme Court explained involuntary commitments and forced drugging involve two separate steps:[89]

> To treat an unwilling and involuntarily *committed mental patient* with psychotropic medication, the state must initiate the second step of the process by filing a second petition, asking the court to approve the treatment it proposes to give.

This was reiterated in *Wetherhorn v. Alaska Psychiatric Institute,*[90]:

> Unlike involuntary commitment petitions, there is no statutory requirement that a hearing be held on a petition for the involuntary administration of psychotropic drugs within seventy-two hours of a respondent's initial detention. The expedited process required for involuntary commitment proceedings is aimed at mitigating the infringement of the respondent's liberty rights that begins the moment the respondent is detained involuntarily. In contrast, so long as no drugs have been administered, the rights to liberty and privacy implicated by the right to refuse psychotropic medications remain intact. Therefore, in the absence of an emergency, there is no reason why the statutory protections should be neglected in the interests of speed.

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[88] The failure of the Probate Masters to comply with Civil Rule 53(d)(1) being fatal to a superior court approval without a transcript is on appeal in S-12677.
[89] 138 P.2d 238, 242-3 (Alaska 2006), emphasis added.
[90] 156 P.3d 371, 382 (Alaska 2007), footnotes omitted.

The Alaska Supreme Court thus specifically held it is a two-step process wherein the forced drugging petition cannot proceed before the involuntary commitment process has been completed:

> Alaska requires a two-step process before psychotropic drugs may be administered involuntarily in a non-crisis situation: the State must first petition for the respondent's commitment to a treatment facility, and then petition the court to approve the medication it proposes to administer. The second step requires that the State prove by clear and convincing evidence that: (1) the *committed patient* is currently unable to give or withhold informed consent;[91]

Both *Myers* and *Wetherhorn* specifically referred to these two steps and to a "committed" patient. In *Myers* this Court held the Forced Drugging Petition is filed *after* a commitment has been granted.[92] Thus, only after a commitment order has been signed by the *Superior Court Judge* may a forced drugging petition be filed.

(C) The Forced Drugging Petition Is Defective and at a Minimum, API should Be Ordered to Conform it to the Requirements of *Myers*

In *Myers* 138 P.3d at 254, with respect to the required best interest element the Alaska Supreme Court held:

> At a minimum, we think that courts should consider the information that our statutes direct the treatment facility to give to its patients in order to ensure the patient's ability to make an informed treatment choice. As codified in AS 47.30.837(d)(2), these items include:
>
> * * *
>
> (B) information about *the proposed medication*, its purpose, the method of its administration, the recommended ranges of dosages, possible side effects and benefits, ways to treat side effects, and risks of other conditions, such as tardive dyskinesia;

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[91] 156 P.3d at 382, emphasis added.
[92] 138 P.3d at 242-3.

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(C) a review of the patient's history, including medication history and previous side effects from medication;

(D) *an explanation of interactions with other drugs*, including over-the-counter drugs, street drugs, and alcohol; . . .[93]

The Alaska Supreme Court also cited with approval the Supreme Court of Minnesota's requirement considering the following factors:

(1) the extent and duration of changes in behavior patterns and mental activity effected by the treatment;

(2) the risks of adverse side effects;

. . . ; and

(5) the extent of intrusion into the patient's body and the pain connected with the treatment.[94]

All of these factors are drug and dose dependent and the last one relates to the manner of administration. Thus, *Myers* specifically requires a drug by drug, dose by dose, and manner of administration determination by the Court.

Sell v. United States, 539 U.S. 166, 123 S.Ct. 2174 (2003), a forced drugging to make one competent to stand trial case, based on the requirements of the United States Constitution, also requires a drug by drug analysis ("The specific kinds of drugs at issue may matter here as elsewhere. Different kinds of antipsychotic drugs may produce different side effects and enjoy different levels of success."). [95]

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[93] 138 P.3d 252, emphasis added.
[94] *Id.*
[95] While *Sell* is a competence to stand trial case, the U.S. Supreme Court used the same sort of standard constitutional law compelling state interest, further state interest and least intrusive alternative analysis the Alaska Supreme Court employed in *Myers* and is fully applicable here with respect to this issue.

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API has not changed its forced drugging petition form to comply with *Myers*. It is therefore defective and should be dismissed for that reason. In the alternative, API should be required to file an amended petition comporting with the requirements of *Myers*. A failure to do so is a violation of Mr. Bigley's due process rights.

V. Motion for Settlement Conference

Mr. Bigley has been abused enough. What API has done to him for 28 years and some 75 admissions should not be allowed to continue. What API has done to Mr. Bigley for 28 years and some 75 admissions is not working and something different should be tried. Mr. Bigley hereby moves the Court to order a settlement conference to discuss a better approach for Mr. Bigley. Mr. Cornils affidavit describes a less intrusive alternative and it seems preferable for the parties to get together to try and work something out before the forced medication petition is heard.

DATED: March 6, 2008.

Law Project for Psychiatric Rights

By: _____
James B. Gottstein, Esq.
ABA # 7811100

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Curriculum Vitae

Grace E. Jackson, MD

1201 Clipper Lane
Wilmington, NC 28405
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Email Address:
grace.e.jackson@att.net

Education:

University of Colorado Health Sciences Center - School of Medicine, M.D.
Graduated 5/96.

California Lutheran University, B.S. Major: Biology. Summa cum laude.
Graduated 5/92.

California Lutheran University, MPA. Major: Public Administration. GPA: 4.00
Graduated 8/87.

California Lutheran University, B.A. Major: Political Science. Summa cum laude.
Graduated 5/86.

Current and Past Certifications:

Board Certified Psychiatrist (Diplomate, American Board of Psychiatry and Neurology), 2004 – 2014.

Basic Life Support: expires 4/2008.

Past Certifications: Advanced Cardiac Life Support, Advanced Trauma Life Support, Pediatric Advanced Cardiac Life Support.

Honors and Awards:

Esprit de Corps Award (awarded by fellow residents - 6/00). Hippocrates Award (5/96). Richard C. Hardin Award (5/95). Honors in Surgery, Family Practice, Psychiatry clinical rotations (UCHSC School of Medicine). Scholastic Honor Society (CLU equivalent of Phi Beta Kappa). Alpha Mu Gamma (foreign language honor society). Kwan Fong Institute Scholarship in East Asian Studies. Most Inspirational Runner, Cross Country.

Medical Training:
Psychiatry Residency, National Capital Area Consortium - Malcolm Grow Medical Center, National Naval Medical Center, Walter Reed Army Medical Center - JUL 1997 - JUN 2000. Graduated 6/00.

Psychiatry Internship, Naval Medical Center San Diego, San Diego, CA
JUN 1996 - JUL 1997
Including Combat Casualty Care Course and ATLS, San Antonio TX (February 1997).

Psychiatric Experience: Clinical, Forensic, and Research

Clinical and Forensic Consultant – 1201 Clipper Lane – Wilmington, NC 28450
February 2008 through present
Contract consultant for clinicians, patients, and attorneys specializing in review of records, preparation of treatment plans, neurotoxicology research, lecturing, and writing.

Private practice – 1213 Culberth Drive – Ste. 139, Wilmington, NC 28405
May 2007 through January 2008
Clinical psychiatrist specializing in forensic consultation, psychotherapy, medication management (detox/neurorehabilitation), neurotoxicology, lecturing, and writing.

Forensic Consultant – 4021 Brookstone Drive – Winterville, NC 28450
October 2006 through April 2007
Contract consultant for forensic cases involving psychiatric rights, medical negligence, product liability, and neurotoxicology.

Veterans Administration Mental Health Clinic – Locum Tenens Psychiatrist, Eugene OR
July 2006 – September 2006
Clinical psychiatrist assigned to outpatient psychiatric clinic. Responsible for psychiatric evaluations, medication management, medical workups, and monitoring. Updated metabolic profiles in accordance with Veterans Administration IG guidelines. Ordered and read EKGs where indicated. Close collaboration with social workers, nursing staff, and community caregivers in the case management of patients with severe and chronic mental illness. Assignment required adjustment of complex polypharmacy regimens in order to minimize metabolic and neurobehavioral toxicities of previous and continuing treatments. Caseload: 200+ patients ranging in age from 20s to 80s.

Forensic Consultant - 4021 Brookstone Drive – Winterville, NC 28450
March 2004 through June 2006
Contract consultant for forensic cases involving psychiatric rights, medical negligence, product liability, and neurotoxicology.

NC Department of Corrections – Locum Tenens Psychiatrist, Eastern NC
August 2003 – March 2004
Clinical psychiatrist assigned to misdemeanor in-processing camp, low custody camp (outpatient), and long term residential facility (housing chronically mentally ill prisoners). Responsible for evaluations, medication management, psychotherapy, discharge summaries, and treatment planning with multidisciplinary team.

Independent forensic consultant, researcher, author, lecturer –
4003 Gaston Court - New Bern NC 28562
April 2002 – June 2003
Expert witness with Law Project for Psychiatric Rights. Initial stages of background research preparatory for writing of first book (*Rethinking Psychiatric Drugs: A Guide for Informed Consent*) published in July 2005.

Staff Psychiatrist, National Naval Medical Center, Bethesda, MD
July 2000 - March 2002
Assigned to adult outpatient clinic at Bethesda Naval Hospital and US Naval Academy. Evaluated and treated active duty military members, dependents, and retirees. Responsible for thorough medical workups and consultation with all relevant specialty clinics. Prepared variety of administrative documents, including medical boards, TDRL (Temporary Disability Retirement List) reports, memoranda for administrative separations, letters for insurers or employers. Devised and delivered comprehensive treatment plans, incorporating supportive, cognitive / behavioral, and psychodynamic psychotherapy; pharmacotherapy; and referrals to outside providers (nutritional, exercise, relaxation, energy-based, music, and/or art therapies). Supervised residents as attending physician on-call, assisting with emergency room assessments and dispositions, adolescent admissions, and surgical/medical ward consultations. Supervised psychiatry interns during their weekly continuity clinic, including pre-clinic viewing and discussion of pertinent films (humanities/literature). Back-filled for staff psychiatrist / department head in Corpus Christi, TX, performing leadership role as only staff psychiatrist on site (October 2000). Assisted Bethesda Chief of Clinical Staff in preparation of Command Provider Morale Survey (August 2001).

Internship and Residency Rotations - 1996 - 2000:

PGY-1 rotating internship, including two months of inpatient psychiatry; two months of neurology; one month each of C/L psychiatry, emergency medicine, family practice, pediatrics, ambulatory care, OB/GYN, general surgery, CCU, internal medicine.

PGY-2 Seven months inpatient adult psychiatry at Walter Reed Army medical center (54 bed locked psych/med ward), 1 month inpatient addictions (Malcolm Grow), 1 month adult Partial Psychiatric Hospitalization program (Walter Reed), 1 month inpatient child/adolescent psychiatry, 1 month emergency psychiatry / night float, 1 month NOVA (Northern Virginia State Hospital) chronically mentally ill

PGY-3 dedicated year of outpatient psychiatry, including long-term and short-term psychotherapy: two long-term psychodynamic cases, two CBT cases, one short-term psychodynamic case, two family therapy cases, one marital psychotherapy case, one short-term psychotherapy group, one long-term psychotherapy group, > 100 active medication management cases (active duty members, dependents, retirees)

PGY-4 Two months inpatient adult psychiatry as subattending (Walter Reed Army Medical Center), two months intensive outpatient treatment (Partial Hospitalization Program - Walter Reed), 4 months electives (neurology consult, child /adolescent outpatient, research, outpatient addictions), 3 months emergency/consult-liaison psychiatry (Walter Reed), 1 month community psychiatry (including forensic psychiatry at Clifton T. Perkins maximum security hospital in Jessup, MD and care of indigent at Montgomery County Crisis Center, Rockville, MD)

Personal Training Psychotherapy:

Psychodynamic/Psychoanalytic training therapy: 3 1/2 yrs. with Dr. Ann-Louise Silver, a former analysand of Harold Searles. Intermittent psychotherapy with Dr. Alexander Lowen, founder of Bioenergetic Analysis. Additional experience with energy modalities, music therapy, deep tissue massage, and Jungian / trance work.

Governmental Testimony:

Florida State Legislature in support of H.B. 1213 and S.B. 2286,
Informed Consent in Education (12 April 2006) – written testimony

Food and Drug Administration, Psychopharmacologic Drug Advisory Committee,
Open Public Hearing, Gaithersburg, MD (23 March 2006) – oral testimony

Food and Drug Administration, Pediatric Advisory Committee,
Open Public Hearing, Gaithersburg, MD (22 March 2006) – oral testimony

Lecturing Experience:

"The Role of Psychiatric Drugs in the Treatment of Addiction," presented at the 58th Annual Conference of the National Catholic Council on Alcoholism and other related drug problems (NCCA), New Orleans, LA (23 January 2008)

"Chemo Brain: A psychiatric drug phenomenon," presented at the 10th Annual Conference of the International Center for the Study of Psychiatry and Psychology, Arlington, VA (13 October 2007)

"Parens Patriae, Parens Inscius: Beware the Dangers of the Incompetent State," presented at the 9th Annual Conference of the International Center for the Study of Psychiatry and Psychology, Bethesda, MD (09 October 2006)

"Addiction and Stimulants," presented at ICSPP Press Conference, Gaithersburg, MD (22 March 2006)

"Ritalin vs. Jiminy Cricket: The Suppression of Human Intention (Are Psychiatrists Medicating Can't or Won't?)," presented at the 5th Annual Conference of the New Jersey Institute for Training in Psychoanalysis, Inc., Teaneck, NJ (12 March 2006)
"Risk Assessment and the Challenge of Neurotechnologies: When Do Treatments Become Toxins to the Self ?" presented before the Novel Tech Ethics Research Team of Dalhousie University, Halifax, Nova Scotia (06 February 2006)

"Rethinking Psychiatric Drugs," presented before the Committee for Public Counsel Services / Continuing Legal Education for attorneys, Boston MA (14 November 2005)

"*Parens patriae*, *Parens inscius*: The Problem of the Incompetent State," presented at the 7th Annual Conference of ISPS-US (International Society for the Psychosocial Treatments of Schizophrenia and Other Psychoses), Boston MA (12 November 2005)

"Allostatic Load: How Psychiatric Drugs Stress the Brain and Body," presented at the 8th Annual Conference of the International Center for the Study of Psychiatry and Psychology, New York City (09 October 2005)

"Rethinking Psychiatric Drugs," presented at META Services, Phoenix, AZ (18 May 2005)

"What Doctors May Not Tell You About Psychiatric Drugs," presented at University of Central England, Birmingham, UK (09 June 2004)

"Psychiatric Drugs: What We All Need to Know," presented to community health centers in Shropshire County UK (07 and 08 June 2004)

"Cybernetic Children," presented for the British Psychological Society/Psychotherapy Section at the Tavistock Clinic, London UK (05 June 2004)

"SOS: The Current Crisis in Psychiatric Drugs," presented for Global Opportunities, Inc. and Children's Development Council. Palm Beach, FL (17 April 2004)

"Gulf War Syndrome: Then and Now," presented for the New Bern Coalition for Peace and Justice New Bern, NC (20 May 2003)

"Be Careful What You Fish For: An Introduction to Pre-Psychosis Screening Programs," presented at the Columbia Academy of Psychodynamics, Columbia, MD (19 March 2003)

"The Limitations of Biological Psychiatry," and "Recognizing the Drug-Induced Crisis," plenary lecture and individual workshop presented at the annual conference of ICSPP (International Center for the Study of Psychiatry and Psychology), Newark, NJ (11-13 OCT 2002)

"A Plea for Psyche," and "Postmodern Psychiatry," presented at Mental Health in the 21st Century Conference, Teesside University, Middlesbrough UK (06 and 13 SEP 2002)

"The Promise of Biotechnology: Unintended Consequences in the Posthuman Era," presented at 7th annual Women in Technology International Conference, Santa Clara, CA (20 JUN 2002)

"The Meaning of ADD/ADHD," presented at 1st Steven Baldwin Memorial Conference, Teesside University, Middlesbrough UK (28 FEB 2002)

"Beyond Reductionism - One Resident's Search for Mind," Chief Resident Research Project, presented at Walter Reed Army Medical Center (14 JUN 2000)

Teaching Experience:

Expert panelist/contributor to "A Critical Skills Curriculum on Psychiatric Medications for Mental Health Professionals" (Florida International University, Miami, FL - 2007).

Chief Resident in Psychiatry (Walter Reed Army Medical Center - 1999 - 2000):
Supervised junior residents, interns, and medical students on various rotations, including inpatient, partial hospitalization program, addictions medicine, and consult-liaison service. Organized and led morning report on inpatient ward, selecting daily case presentations as subattending. Delivered lectures on case formulation, psychotherapies, psychiatric history, and biopsychosocial model of illness. Assisted consult-liaison service chief with hypnotherapy interventions in pain and rehab/physiatry clinics.

Instructor, Political Science (California Lutheran University, Thousand Oaks, CA – 1986 - 1988):
Prepared and delivered original curriculum in American government. Advised, tested, and evaluated students. Assisted students with career development planning. Prepared grant proposals for tenured faculty members and Dean for International Affairs. Completed advanced degree in Public Administration, including community service project (library site selection assessment) for city of Thousand Oaks.

Forensic Experience:

Expert Witness
in re: Thomsen vs. Thomsen
Morristown, NJ (April – May 2008)

Professional Consultant:
Vickery, Waldner, & Mallia
(November 2006 through February 2008)

Expert Witness
in re: Rogers vs. Ulmer's Drug
Homer, AK (April – May 2007)

Expert Witness
in re: L. Welch
Nampa, ID (March – April 2007)

Expert Witness
in re: J. Freeman
Springfield, Massachusetts (June 2006)

Expert Witness
in re: G. Daniels
Melbourne Australia (December 2005 – present)

Expert Witness in guardianship case
in re: A. Braman
Columbia Circuit Court, OR (July 15, 2005)

Expert Witness in foster care case
Witness for Attorney Ad Litem – Pasco County FL
Juvenile Dependency Division Case No. 96-01158DPAES (August 4, 2004)

Forensic consultant re:
State of Utah vs. Leon Gall (April 30, 2004)

Expert Witness and Professional Consultant - Law Project for Psychiatric Rights
March 2003 - Present
Ad hoc forensic assistant for Alaska attorney specializing in rights of mentally ill. Activities have included professional testimony and affidavits, retrieval and analysis of medical research, and assistance with development of publicly accessible computer database.

Creighton in re: Office of Hearings and Appeals (August 26, 2004)
Bavilla vs. Department of Corrections (April 4, 2004)
Myers vs. Alaska Psychiatric Institute (February 2003)

Other Employment:

Rapid City Regional Hospital – Family Practice Residency Rapid City, SD
June 2003 - July 2003
First year resident in family practice, responsible for inpatient treatment of medical patients, consultations, and outpatient clinic (children and adults). Responsibilities included EKG stress tests, Intensive Care Unit / Cardiac Care Unit (patient management). Left residency in good standing to resume work as mental health specialist due to concerns about continuing crisis in "evidence based medicine" and drug safety.

Secretary / Receptionist , Kamiya Biomedical Company
June 1992 - August 1992
Temporary assistant for independent biomedical firm in Westlake Village, CA. Responsible for preparing all shipping documents, updating mail and invoice computer database, processing incoming orders, and interacting with large domestic and international customer network, correspondence, phones.

Administrative Assistant, Pepperdine University
June 1991 - August 1991
Temporary assistant in Insurance and Risk Management Department. Adjusted student athletic claims, property floater, employee and student insurance database.

Treasury Analyst, Pepperdine University
April 1989 - August 1989
Administered living trusts. Fulfilled debt compliance and daily cash management requirements for University. Executed wire transfers, foreign currency transactions, and various custodial duties for University accounts and securities. Generated financial reports, correspondence. Systematized procedures of this position prior to transition back to school for premedical studies.

Administrative Assistant, Pepperdine University
January 1989 - April 1989
Assistant to VP for Finance, overseeing payments of taxes and expenses for University-managed property. Maintained investment and real estate files. Regulated access to off-site safekeeping vault. Generated correspondence and reports. Supervised student workers. Ordered department supplies, routed mail, scheduled appointments, and screened incoming calls for office personnel.

Administrative Assistant, Pepperdine University
November 1988 - January 1989
Temporary assistant in Insurance and Risk Management Department. Adjusted student athletic claims, updated University property floater and driver records, edited and prepared University Safety Manual, supervised athletic policy changeover.

Publications:

"A Critical Analysis of the Neurogenesis Theory of Antidepressant Efficacy," (April 2008) – under peer review.

"Chemo Brain: A Psychiatric Drug Phenomenon ?" *Medical Hypotheses* 70:3 (2008): 572-577.

"The Case Against Stimulants," contributed chapter, in S. Timimi and J. Leo, *Rethinking ADHD* (Hampshire, UK: Palgrave Macmillan, expected 2008).

"Mental Health Screening in Schools: Essentials of Informed Consent." *Ethical Human Psychology and Psychiatry* 8 (2006): 217-225**.**

"A Curious Consensus: Brain Scans Prove Disease?" *Ethical Human Psychology and Psychiatry* 8 (2006): 55-60.

Rethinking Psychiatric Drugs – A Guide for Informed Consent (Bloomington, IN: Author House, 2005).

"Cybernetic Children," contributed chapter, in C. Newnes and N. Radcliffe, *Making and Breaking Children's Lives* (Ross on Wye: PCCS Books, 2005).

Contributor to "The Myth of the Magic Pill" in B. Duncan, S. Miller, and J. Sparks. *The Heroic Client*, 2nd ed. (San Francisco: Jossey Bass, 2004).

"A Plea for Psyche." *Review of Existential Psychology & Psychiatry* XXVI (2003): 97-100.

"The Dilemma of Early Intervention: Some Problems with Mental Health Screening and Labeling." *Ethical Human Sciences and Services* 5 (2003): 35-40.

"Rethinking the Finnish Adoption Studies: A Challenge to the Doctrine of Genetic Determinism." *Journal of Critical Psychology, Counselling, and Psychotherapy* 3 (2003): 129-138.

Other Independent Research:

"Aerospace Medicine: A Review of Major Responses to Space Flight" - Aerospace Medicine Clerkship at Johnson Space Center, Houston TX (spring 1996)

"Psychobiology: Mind/Body Communication in the Manifestation and Mitigation of Illness" (spring 1992)

Volunteer Activities:

Member, Board of Directors - ICSPP January 2001- present
As active member of International Center for the Study of Psychiatry and Psychology, have participated in lectures, research, and communiques with fellow health care professionals, policy makers, and public. Contributed to position paper on ADHD as part of Task Force on Child/Adolescent Mental Health Care. Frequent consultant on risks associated with use of mind-altering drugs and alternatives to same.

US Navy June 1996 - March 2002
As psychiatry intern, prepared and distributed intern directory; assisted with annual beach picnic, and coordinated purchase and distribution of discount lab coats. As resident: facilitated small group discussions of Uniformed Services 2nd yr. medical student course in psychiatry; instructor at Operational Medicine Course (Bushmaster) at Camp Bullis, TX (November 1988). Member of Call Committee, responsible for preparation and distribution of call schedule for over 40 interns and residents covering three separate emergency rooms / hospitals. Pioneered night float system for PGY2s.

University of Colorado School of Medicine 1992 - 1996
Class Secretary / Treasurer (1992 - 1996). Responsible for student administered accounts, fundraising activities, and minutes of all class government meetings. Student Council Secretary (1992-1993). Co-President, AMSA (American Medical Student Association) - University of Colorado Chapter (1993-1994): donated medical books to Romania, oversaw fundraising efforts, supervised Medicine Wheel alternative medicine lecture series. Course Representative, Microbiology and Immunology (1993 - 1994). Co-editor, Medical Examiner, medical school newspaper (1993-1994). National Editor, AMSA Medical Education Task Force Quarterly Newsletter (1993 - 1994). Sports: class softball and soccer teams (1993 - 1994). Senior Class Co-President (1995-1996). Coordinated Match Day celebration, co-wrote Senior Skit, recruited and hosted Graduation speaker.

Professional Memberships:

International Center for the Study of Psychiatry and Psychology (member, Board of Directors), International Society for the Psychosocial Treatment of Schizophrenia and Other Psychoses.

Personal Facts:

Facile writer and speaker. Well travelled (East Asia, Europe, USA). Hobbies include medical research, movies, poetry, music, physical fitness, time in nature, foreign languages, literature.

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.

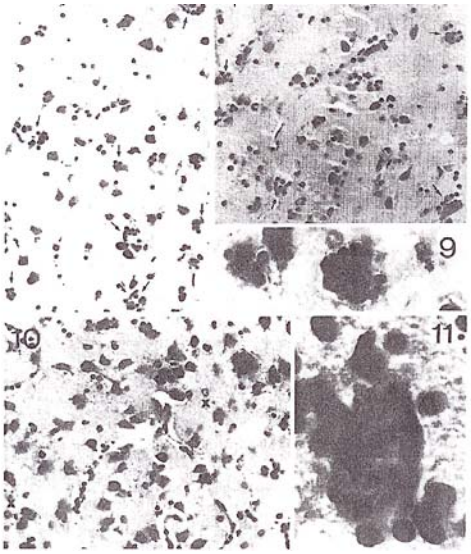
Evidence for Neuroleptic (Antipsychotic) Induced Brain Injury

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.

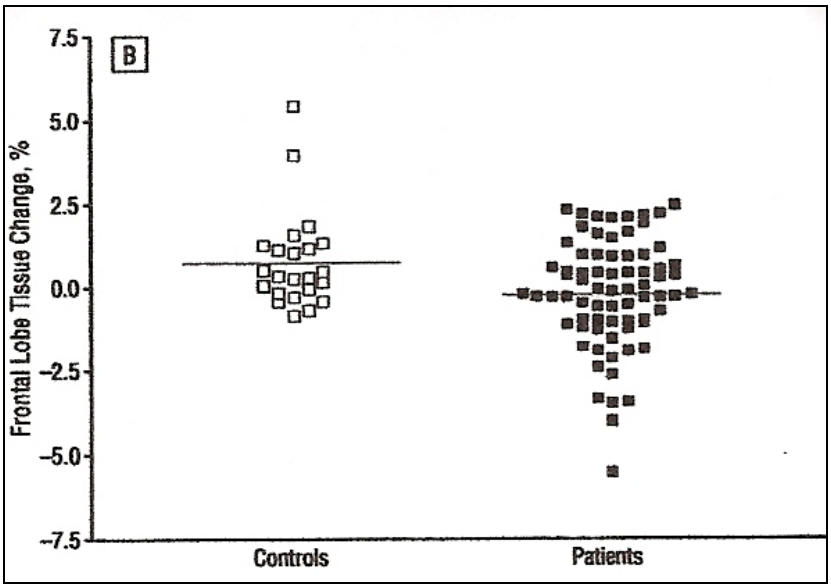
The Jellinger study is historically important because it included two comparison or control groups, allowing for the determination of treatment-related vs. illness-related changes. Damage to the basal ganglia was seen in only 4% of an age-matched group of psychotic patients who had *avoided* long-term 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:

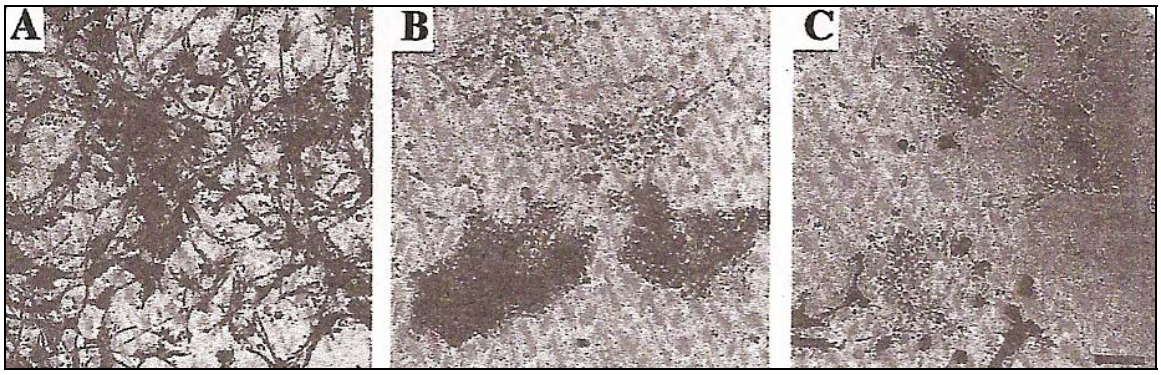
> "…our study failed to show a difference in neurotoxicity between atypical and typical neuroleptics, and we should be careful when using neuroleptics as first-line drugs in Alzheimer's dementia patients…Because the level of cerebral apoptosis of non-demented patients on antipsychotics appears to be 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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Respectfully Submitted,

_____	14 May 2008 _____
Grace E. Jackson, MD	Date of Submission

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

In The Matter of the Necessity for the)
Hospitalization of William Bigley,)
)
Respondent)
Case No. 3AN 08-00493PS

NOTICE OF FILING TESTIMONY

The following prior testimony is hereby filed by Respondent in connection with consideration of the current AS 47.30.839 forced drugging petition:

1. Transcript of the March 5, 2003, testimony of Loren Mosher, in 3AN 03-00277 CI;
2. Affidavit of Loren Mosher in 3AN 03-00277 CI; and
3. Transcript of the September 5, 2007, testimony of Sarah Porter in Pages in 3AN 07-1064 PS.

All of this testimony is admissible pursuant to Evidence Rule 804(b)(1). Dr. Mosher is now deceased and therefore unavailable, and the Petitioner not only had the opportunity and similar motive to develop the testimony by direct, cross, or redirect, the Petitioner, it self, had such an opportunity and similar motive.

Ms. Porter lives in New Zealand and is unavailable for that reason. Not only, as with Dr. Mosher, did the Petitioner have the opportunity and similar motive to develop the testimony by direct, cross, or redirect, the testimony was with respect to a previous forced drugging petition against Respondent, which Petitioner abandoned.

DATED: May 13, 2008. Law Project for Psychiatric Rights

By: _____
James B. Gottstein, ABA # 7811100

LAW PROJECT FOR PSYCHIATRIC RIGHTS, INC.
406 G Street, Suite 206
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IN THE SUPERIOR COURT FOR THE STATE OF ALASKA

THIRD JUDICIAL DISTRICT

IN THE MATTER OF F.M.

3AN-02-00277 CI

_____/

VOLUME I

TRANSCRIPT OF PROCEEDINGS

March 5, 2003 -- Pages 1 through 198

March 10, 2003 -- Pages 198 through 223

HEARING REGARDING BURDEN OF PROOF THAT DEFENDANT IS MENTALLY ILL AND REGARDING ADMINISTRATION OF MEDICATION

BEFORE THE HONORABLE MORGAN CHRISTEN

Anchorage, Alaska
March 5, 2003

APPEARANCES:

FOR THE PLAINTIFF: Jeff Killip
Assistant Attorney General
State of Alaska
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FOR THE DEFENDANT: James B. Gottstein
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P R O C E E D I N G S

4403-41

8:52:51 AM

THE COURT: We're on record in Case No. 3AN-03-277. It's a case regarding Faith Myers. Mr. Gottstein, before I go any further, I'll just state your appearance. Mr. Gottstein is present, for the record, as is Mr. Killip for the State. Your client requested this be an open hearing, is that correct?

MR. GOTTSTEIN: That's correct. She's not here yet, though, and she's supposed to be here. So, I don't know what the hang-up is. Dr. Kletti, wasn't she --?

THE COURT: Right. She has the right to be present.

DR. KLETTI: Right. She was scheduled for transportation to court this morning.

THE COURT: I was told that you all were ready. I didn't realize that you weren't. We need to wait for her. So we'll go ahead and go back off record and do that. Well, actually, maybe I'll take up some housekeeping, first, but we're not going to proceed in substance with her, certainly.

I just have the one exhibit list. Counselor, do you have --

MR. GOTTSTEIN: The respondent's?

THE COURT: Yes. Do you have an exhibit list, Mr.

Killip?

MR. KILLIP: Your Honor, given the accelerated pace, the witnesses just showed up. I had a chance to speak with one for almost an hour yesterday, but there are two more I haven't had a chance to talk with and one of them presented me with some photographs. I don't have an exhibit list that I've generated yet, but I can do it right now.

THE COURT: Okay, that's fine. We can do it when we go off record for a minute. As long as Mr. Gottstein has it and has a chance to take a look, that's fine.

MR. GOTTSTEIN: Your Honor, I would note under AS 47.37.30(a)(6) that the petition must list the prospective witnesses who will testify in support of commitment or involuntary treatment, and only Dr. Hanowell was listed. And I would object to any witness other than the one specifically listed testifying.

THE COURT: All right. The objection is noted, but again, I'm not going to make any substantive ruling until your client gets here. My intention is to stay on record just to get some housekeeping taken care of.

MR. GOTTSTEIN: Can I respond to that, Your Honor?

THE COURT: No, not yet.

MR. GOTTSTEIN: Okay.

THE COURT: Because we're not going to get into

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THE COURT: Mr. Gottstein?
DIRECT EXAMINATION (continued)
BY MR. GOTTSTEIN:
Q Yeah. Dr. Jackson, can you explain why you failed the exam? Or, you were failed, I guess I should say.
A Well, the Board of Examiners does not send you any kind of feedback, but I was subjected to quite intense cross-examination as to why I would not give a patient with psychotic symptoms medication for life. And I had done extensive research up to that point to prepare myself for -- for my philosophy of treatment. And I was not willing to purger myself in the cross-examination process of board certification exam, so I did not pass that exam.
Q What do you mean by that? You were not prepared to purger yourself?
A I could have lied. I could have told the examiners that the woman in the videotaped interview, who had previously had a case of schizophrenia, needed to be on medication for life, which is what they were attempting to get out of me. Because they kept saying, well, she told you that she had previously been on these medicines. Why won't you give them to her now? And I had done a great deal of research and had very good reasons why I would not continue a person, necessarily on life-long medication. But that, apparently, was not the answer that they were

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looking for.
I should say that my passed portion of the exam, which was based on a live patient interview in the morning, was based -- I passed that exam, and the reason for that or the tone of that was actually quite different. My examiners were more psycho-dynamically oriented individuals, and they accepted the fact that a life-long medication strategy was not necessarily in the best interest of all patients.
So, the board certification process, itself, is extremely relative. I would expect to encounter the exact difficulties when I sit for the examination again and I will give the same answers, based on the same scientifically-based knowledge.
THE COURT: I'll accept this witness as an expert and weigh her testimony accordingly.
Q Dr. Jackson, did you prepare a report and sign an affidavit -- well -- excuse me, Your Honor.
THE COURT: That's okay. But could you get closer to the microphone?
Q Yes. Did you notarize a statement -- have notarized a statement in preparation for this hearing?
A Yes, I did.
THE COURT: Mr. Gottstein, I'm sorry to do this to you, but I just got the email that Dr. Mosher is on the

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phone. Do you want me to have him call back in 10 minutes, or what do you want to do?
MR. GOTTSTEIN: Grace, can you? Let's take Dr. Mosher.
THE COURT: That's your preference?
MR. GOTTSTEIN: Yes.
THE COURT: Ma'am, I'm very sorry to do this. We've been trying to get Dr. Mosher on the line, and the witnesses we typically go in order. And he was not available by phone. I've just received an email that he's called back in.
DR. JACKSON: That's absolutely fine.
THE COURT: All right. I appreciate it very much.
DR. JACKSON: Would you like me -- you'll call me back?
THE COURT: Yes.
DR. JACKSON: Okay. Thank you.
THE COURT: You bet. Dr. Mosher, can you hear me?
DR. MOSHER: Yes. Long distant, but I can hear you.
THE COURT: All right. I'll try to speak into the microphone more clearly. My name is Morgan Christen. I'm a superior court judge and I'm assigned to this case. I have you on a speaker phone on an overhead in the courtroom, sir. And Mr. Gottstein has asked that you testify. Are you able to do that at this time?

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DR. MOSHER: Well, I guess. I didn't prepare must, but anyway, I'll do my best.
THE COURT: All right. That's fine. I need to have the oath administered to you. Could you please raise your right hand?
DR. MOSHER: Okay.
THE CLERK: Do you swear or affirm that the information you are about to give in this matter before the court is the truth, the whole truth, and nothing but the truth?
DR. MOSHER: I do.
THE COURT: Sir, could you please state your full name and spell your last name?
DR. MOSHER: It's Loren Mosher, M-O-S-H-E-R-.
THE COURT: All right. Thank you. Mr. Gottstein, you may inquire.
DR. LOREN MOSHER
testified as follows on:
DIRECT EXAMINATION
BY MR. GOTTSTEIN:
Q Dr. Mosher, I can't express my appreciation enough for your willingness to testify after just getting back from Germany yesterday, and I just felt like I wanted to express that.
Your affidavit has just been admitted. And I

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represented that you would have it notarized and send it. Is that true?
A I just did that. It should be there tomorrow afternoon.
Q Thank you. Could you briefly -- because we've got a total of, I think 28 minutes left in this whole hearing, including to hear from Dr. Jackson -- discuss your credentials, please?
A I graduated from Stanford as an undergraduate, Harvard Medical School, Harvard psychiatric training, more training at the National Institute of Mental Health, post-doctoral fellowship in England, professor -- assistant professor of psychiatry at Yale -- I'm sort of going chronologically -- from '68 to '80 I was the chief for the Center for Studies of Schizophrenia, at the National Institute of Mental Health from 1980 to '88 I was professor of psychiatry at the Uniform Services University of the Health Sciences in Bethesda, Maryland. That's a full-time, tenured, academic position. '88 to '96 I was the chief medical director of the Montgomery County Maryland Public Mental Health System. That's a bedroom community to Washington, D.C. From '96 to '98 I was clinical director of the San Diego County Public Mental Health System. Since November of '98 I have been the director and principle in Satiria (ph) Associates, a

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private consulting firm that I formed, and I also hold clinical professorships at the University of California San Diego School of Medicine, and at the Uniform Services University of the Health Sciences in Bethesda, Maryland. So that's briefly my credentials.
Q Dr. Mosher, did you mention being head of schizophrenia research at the National Institute of Mental Health?
A Yeah, I said I was the head of the Center for Studies of Schizophrenia from 1968 until 1980.
Q Okay. I move to qualify Dr. Mosher as an expert psychiatrist, especially in schizophrenia.
MR. KILLIP: Your Honor, just a couple questions.
VOIR DIRE EXAMINATION
BY MR. KILLIP:
Q Dr. Mosher, Jeff Killip with the Alaska Attorney General's Office. I just want to ask you if you are currently board certified in psychiatry?
A I've been board certified since 1969.
Q Okay. And are you currently a member in good standing with the American Psychiatric Association?
A No, I am not. I resigned from the American Psychiatric Association.
Q And do you have a reason for that?
A Yes, I have a reason for it. I felt like they no

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longer represented my interested and the $1,000 a year that I was paying for them was just basically a waste of money, while they pursued their own interests to the detriment of what I consider to be the people they should be pursuing an interest for, and that's their patients. So anyway, I'm not a member. I resigned in December of 1998.
Q So, is it fair to say that you have a philosophical disagreement with their approach, presently?
A Well, yeah. I don't like how they do business.
Q When you say do business, you mean practice psychiatry in the United States?
A Well, we could take up the next half hour on that subject, but basically I feel that they have taken the person out of psychiatry and psychiatry has -- is now a dehumanizing, impersonal, non-individualized specialty that is interested purely in pharmical therapy now. That's big, broad brush strokes, but that's -- obviously that's not true of every single one, but that's my complaint about the organization.
Q Okay.
A There's a -- if you want to read my letter of resignation, you can look on my web site.
Q Okay, thank you.
THE COURT: Any objection?

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MR. KILLIP: No.
THE COURT: All right. This witness will be qualified
Q Thank you, Dr. Mosher. In the first sentence of the introduce of your affidavit on page two, you talk about the biomedical model. I was going to ask you what you mean by that. Have you already answered that, or would you like to expand on that?
A Well, you know, what I mean by that is the phrase is currently being used that, let's take, for example, schizophrenia is a brain disease. Well, that's a perfect example of the medical model -- of the biomedical model. When -- whereas, there is no evidence that schizophrenia is, in fact, a brain disease. And so a hypothesis that schizophrenia is a brain disease, has been converted into a biomedical fact. And I disagree with converting hypotheses into beliefs in the absence of supporting evidence.
Q Okay, thank you. Now, in your opinion, is medication the only viable treatment for schizophrenia paranoid type?
A Well, no, it's not the only viable treatment. It is one that will reduce the so-called positive symptoms, the symptoms that are expressed outwardly for those kinds of folks. And that way they may seem better, but in the long

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run, the drugs have so many problems, that in my view, if you have to use them, you should use them in as small a dose for as short a period of time as possible. And if you can supply some other form of social environmental treatment -- family therapy, psychotherapy, and a bunch of other things, then you can probably get along without using them at all, or, if at all, for a very brief period of time. But you have to be able to provide the other things. You know, it's like, if you don't have the other things, then your hand is forced.

MR. KILLIP: Excuse me, Your Honor. I just would renew our continuing objection about offering test on medical practice in the context of this hearing.

THE COURT: This hearing is going to last 20 more minutes, and I'm going to let Mr. Gottstein use the time.

Q Now, as a hypothetical question, if a woman who had managed -- who has over a 25 year experience with medications and has -- including navaine, paxil, risperdal and zyprexa -- and then has managed to not -- to wean herself from those for a year, would your recommendation be that she be placed back on them, particularly against her will?

A Well, I think she is an absolute saint if she was able to get off of those drugs. Those drugs are extraordinarily difficult to get off of, especially

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zyprexa, which is a thienobenzodiazepine derivative and the thienobenzodiazepine valium-type drugs are very addictive. And so, zyprexa, in particular, is difficult to get off. And if she got off herself -- got herself off of zyprexa, that's quite a remarkable feat in my clinical experience. So I would be loath to put her back onto, especially zyprexa. But, you know, the other -- risperdal is also problematic for getting off. Actually, they all are, it's just a matter of degree. And if she got off for a year, then I would certainly try to do whatever I can to avoid putting her back on. And if she doesn't want them, then that's even -- you know, if you can't negotiate some drug that she may calm down on, like, for example, if she if kind of agitated and anxious -- I don't know this woman. I've never seen her face-to-face, so I can't really speak to her particular problem without having seen her, but if she is, let's say, unhappy, agitated, and so forth, then sometimes short-term use of drugs like valium is quite helpful and it get's people through a crisis without getting them back onto the neuroleptics drugs, the anti-psychotic drugs.

Q Okay, thank you. Now, in your affidavit, you say involuntary treatment should be difficult to implement and used only in the direst of circumstances. Could you explain why you have that opinion?

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A Well, it's just, you know, the degree to which you have to force people to do anything.....

MR. KILLIP: Your Honor, I'm going to object.

A .....is the degree to which it's going to be very difficult to forge a good therapeutic relationship. And in the field of psychiatry, it is the therapeutic relationship which is the single most important thing. And if you have been a cop, you know, that is, some kind of a social controller and using force, then it becomes nearly impossible to change roles into the role -- the traditional role of the physician as healer advocate for his or her patient. And so I think that that -- we should stay out of the job of being police. That's why we have police. So they can do that job, and it's not our job.

Now, if because of some altered state of consciousness, somebody is about to do themselves grievous harm or someone else grievous harm, well then, I would stop them in whatever way I needed to. I would probably prefer to do it with the police, but if it came to it, I guess I would do it. In my career I have never committed anyone. It just is -- I make it my business to form the kind of relationship that the person will -- that we can establish a ongoing treatment plan that is acceptable to both of us. And that may you avoid getting into the fight around whatever. And, you know, our job is to be healers,

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not fighters.

THE COURT: There's an objection to that question. The objection was relevance?

MR. KILLIP: Yes.

THE COURT: Overruled.

Q Now, you say you've never committed anybody. But you've had a lot of experience with -- or, I should say, have you had a lot of experience with people with schizophrenia?

A Oh, dear. I probably am the person on the planet who has seen more acutely psychotic people off of medication, without any medications, than anyone else on the face of the planet today.

Q Thank you.

A Because of the Satiria Project that we did for 12 years where I would sit with people who were not on medications for hours on end. And I've seen them in my private practice, and I see them to this day in my now, very small, private practice. But --

THE COURT: Sir, I think I understand the answer.

A I find that people who are psychotic and not medicated are among the most interesting of all the customers one finds.

Q Thank you, Dr. Mosher.

THE COURT: That's a yes.

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Q Dr you know Dr. Grace Jackson?
A I do.
Q Do you have an opinion on her knowledge of psychopharmacology?
A I think she knows more about the mechanisms of actions of the various psychotropic agents than anyone who is a clinician, that I'm aware of. Now, there may be, you know, basic psychopharmacologists, you know, who do lab work who know more, but as far as a clinician, a practitioner, I don't know anyone who is better-versed in the mechanisms, the actions, the effects and the adverse effects of the various psychotropic drugs.
Q Thank you, Dr. Mosher. I have no questions, but perhaps the State will have some.
MR. KILLIP: Yes, thank you.
DR. LOREN MOSHER
testified as follows on:
CROSS-EXAMINATION
BY MR. KILLIP:
Q Dr. Mosher, is it not your understanding that the use of anti-psychotic medications is the standard of care for treatment of psychosis in the United States, presently?
A Yes, that's true.
Q Okay, so is it fair to say that your viewpoint --

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MR. GOTTSTEIN: Objection, relevance.
THE COURT: Overruled.
Q Would you say that your viewpoint presented today falls within the minority of the psychiatric community?
A Yes, but I would just like to say that my viewpoint is supported by research evidence. And so, that being the case, it's a matter of who judges the evidence as being stronger, or whatever. So, I'm not speaking just opinion, I'm speaking from a body of evidence.
Q Thank you, Dr. Mosher.
THE COURT: Nothing further?
MR. KILLIP: Nothing.
MR. GOTTSTEIN: No, Your Honor.
THE COURT: All right. Sir, I appreciate your testimony very much and want to thank you. It sounds like the lawyers are done with you, so you can hang up.
DR. MOSHER: Okay. Well, good luck and I hope -- what's her name, Ms. Myers?
THE COURT: Faith Myers.
DR. MOSHER: Gets out and without drugs. Thank you.
THE COURT: Thank you, sir. All right. Do you want to try to call Dr. Jackson back?
MR. GOTTSTEIN: Yes, Your Honor.
THE COURT: All right. Dr. Jackson?
DR. JACKSON: Yes?

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THE COURT: Great. We're back on record. This is Morgan Christen again. I have you back on the same overhead speaker.
DR. JACKSON: Yes, ma'am.
THE COURT: What I'm going to do, I think, to save time, is to just remind you that you remain under oath and allow Mr. Gottstein to ask his questions.
DR. JACKSON: Um-hmm. Yes, ma'am.
DR. GRACE JACKSON
testified as follows on:
DIRECT EXAMINATION (continued)
BY MR. GOTTSTEIN:
Q Thank you, Dr. Jackson. Obviously we're down to 10 minutes now, and I appreciate you waiting all day. And I'm going to have to be, obviously, a little bit -- or more than a little bit brief.
Did you -- we were just talking about an affidavit, I think, that you signed, or a report that you swore. Did you do so?
A Yes, that is correct. Yup.
Q And is it -- can I --?
THE COURT: Do I have this? Oh, you're just handing it to me now, okay.
MR. GOTTSTEIN: I was in the middle of that.
THE COURT: I see. I beg your pardon.

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MR. GOTTSTEIN: Exhibit D.
THE COURT: Thank you, sir.
Q What's the title of that?
A This is an analysis of the olanzapine that is zyprexa, the clinical trials, and I've called this A Dangerous Drug with Dubious Efficacy.
Q Okay.
MR. KILLIP: Excuse me, Your Honor. I just wanted to note for the record that we've got about 20+ pages, half of them are stapled upside down. We're probably not going to have a meaningful opportunity to look at this before cross-examination. I just want to make that record.
THE COURT: Yes, I have the same exhibit.
MR. KILLIP: Thank you.
MR. GOTTSTEIN: And I would note that I received nothing from them before anything.
Q I think what I -- does this accurately -- well, obviously it accurately describes the results of your research into the drug olanzapine. Is that correct?
A Yes, that's right.
Q Okay. Have you -- I'm going to try -- I'm trying to get some stuff into the record here, Your Honor. And so -- and then we'll get to more substantive.
Did you send me some information regarding the

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MR. GOTTSTEIN: .....if that's what our decision is.
THE COURT: If you could let me know, I'd sure appreciate it, because I'm --
MR. GOTTSTEIN: Absolutely, Your Honor. I included you in that.
THE COURT: Yeah, I appreciate it. Because, as I said, I'm -- I have a personal appointment out of the office that's actually a medical appointment I scheduled for some months and moved several times, myself, so I'd like to know as soon as I can, so that I can know how to handle that.
And I appreciate what you're both doing, which strikes me as you're both being very, very cooperative and trying your level best to get this done in a timely manner that jumps through all the hoops required by the statute and make sure that I have the information that I need to make the decision.
Is there anything further I can take up today, productively? No?
MR. KILLIP: I don't think so, Your Honor.
THE COURT: All right. Well then, I'll let you both ring off. It's after 5:00 and I've kept you. Thanks very much for your help. I'll have Hilary confirm tomorrow morning about that time, but that should be at least in pencil on your calendars. And I'll let you know if I need

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to speak to you sooner, after I get the report from the court-appointed visitor.
MR. KILLIP: Okay.
THE COURT: Thank you both very much.
MR. KILLIP: Thank you.
MR. GOTTSTEIN: Thank you.
THE COURT: Off record.
(Off record.)
5:03:47

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TRANSCRIBER'S CERTIFICATE

I, Joanne Kearse, hereby certify that the foregoing pages numbered 1 through 222 are a true, accurate, and complete transcript of the hearings that took place on March 5, 2003 and March 10, 2003, In the Matter of F.M., Superior Ct. No. 3AN-03-277 PR, transcribed by me from a copy of the electronic sound recording to the best of my knowledge and ability.
Dated this 7th day of April, 2003.

JOANNE KEARSE

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

In The Matter of the Necessity for the)
Hospitalization of William S. Bigley,)
Respondent,)
William Worral, MD,)
Petitioner)
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

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[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.

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[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 complete photocopy of the original filed in 3AN 07-1064PR.

Dated: May 13, 2008 _____
James B. Gottstein

SUBSCRIBED AND SWORN TO before me this 13th day of May, 2008.

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

STATE OF NORTH CAROLINA)
) ss.
New Hanover COUNTY)

To: Mr. Jim Gottstein, Esq.
Law Project for Psychiatric Rights
406 G. Street – Suite 206
Anchorage, AK 99501

From: Grace E. Jackson, MD
1201 Clipper Lane
Wilmington NC 28405

Date: 20 May 2008

Re: William Bigley
Case # 3AN-08-00493 P/R
API Petition for Court Ordered Administration of Medication

I. Introduction

Educational and Professional Background

I am a Board Certified psychiatrist residing in North Carolina where I specialize as a clinical psychiatrist, an independent researcher in the areas of neuropharmacology and neurotoxicology, and a writer and lecturer.

I hold a B.A. in political science, a B.S. in Biology, and a Master's degree in Public Administration. I received my medical degree from the University of Colorado School of Medicine in May of 1996. Following medical school, I was commissioned in the U.S. Navy with orders for post-graduate training in psychiatry: internship at San Diego Naval Medical Center (Balboa Hospital - graduating in 1997); residency in Washington, D.C. in the National Capital consortium (a tri-service training program performed at Walter Reed Army Hospital, Bethesda Naval Hospital, and Malcolm Grow Hospital at Andrews Air Force Base). Subsequent to the successful completion of my residency in June 2000, I was assigned as a staff psychiatrist to Bethesda Naval Hospital, where I supervised the work of trainees and provided care to active duty personnel, their dependents, and retirees. Since transitioning out of the military in spring 2002, I have pursued work as a private consultant, and have worked as a clinician within the North Carolina Department of Corrections and the Veterans Administration health care system.

II. Testimony as an Expert in Psychopharmacology

In spring of 2003, I participated as an expert witness in the case of *Myers vs. Alaska Psychiatric Institute* (API). The case was important because of its consideration of my testimony about the efficacy and safety of antipsychotic drugs. Special emphasis was placed upon the FDA's analysis and approval of olanzapine (Zyprexa) as a primary example of the newer therapies. Interestingly, on March 1, 2004, the FDA announced its requirement for warnings about health risks associated with olanzapine and similar chemicals. This FDA alert was consistent with many of the concerns which I had expressed in my affidavit. In considering my testimony in the Myers case, the Alaska Superior Court, and the former Director of Schizophrenia Research at NIMH (National Institute of Mental Health) qualified me as an expert in the area of psychopharmacology. Subsequent forensic experience and independent research have been preparatory for peer reviewed journal articles and book chapters explaining the mechanisms through which psychiatric medications often prevent or delay recovery. For the past six years, I have lectured locally, nationally, and internationally on the subject of psychiatric drug toxicity. My first book (*Rethinking Psychiatric Drugs: A Guide for Informed Consent*) has been adopted by several professors nationwide as a required text for students in sociology, psychology, psychotherapy, and social work. Most recently, I have accepted an invitation from Florida International University to join a panel of independent experts in preparing a website-based "Critical Skills Curriculum on Psychiatric Medications for Mental Health Professionals."

III. Sources of Information

In preparing this report, I have relied upon the following materials:

1) Motion for Less Intrusive Alternative, dated 10 March 2008

2) Submission for Representation Hearing, dated 06 March 2008
pages 1-13, 23-28, 32-34

3) Selected Medical Records
API admission note of 4/18/80 by Annie Bowen, MSW
API discharge note of 4/30/08 by Robert Alberts, MD
API discharge summary from 5/4/81 by Robert Marshall, MD
API admission note of 2/22/07 by William Worrall, MD
API discharge summary of 3/14/07 by William A. Worrall, MD
API report contact of 3/19/07 re: Depakote, by L. Silberschimidt, LCSW

4) Affidavit of Ronald Bassman, PhD, dated 04 SEP 2007

5) Affidavit of Paul A. Cornils, dated 12 SEP 2007

6) Affidavit of Robert Whitaker, undated (? SEP 2007)

7) log notes from Superior Court at Anchorage, AK dated 12 May 2008

8) Exhibit E: my affidavit prepared for hearing of 14 May 2008

9) product labels for Risperidone tablet, Risperidone liquid, Risperidone Consta

10) findings and Order of Superior Court in Anchorage, AK, dated 19 May 2008

11) consultation with pertinent articles in peer reviewed literature (etc)

IV. Purpose of This Affidavit

This affidavit is written for the express purpose of responding to the Findings and Order of the Superior Court of Anchorage, AK (Judge Sharon L. Gleason) as rendered on 19 May 2008 in the aforementioned case. Specifically, this affidavit presents the reasons why a failure to grant a stay of the Superior Court's order will most likely result in irreparable and (ultimately) lethal harm.

V. Limitations of Current Report

The content of the current report is limited by the following factors:

1) lack of face-to-face or telephonic interview with the patient

2) lack of access to *all* medical records, including:

   all admission and discharge summaries from hospitalizations
   all outpatient provider notes (from birth to present)
   all pharmacy records

3) lack of access to collateral sources of information (e.g., interviews with immediate and extended family, friends of patient, etc.).

4) apparent failure of past and present providers to obtain up-to-date diagnostic tests, including but not limited to: EKG, MRI of brain, EEG, heavy metal toxicity screens, tests of renal/thyroid/liver/heme/pancreatic function, tests of metabolic and dietary abnormalities (e.g., vitamins, electrolytes, lipids, glucose), tests for infectious disease, consultations with pertinent specialists

These limitations are mentioned, not as a disqualification of the remarks which follow, but as a reminder of the crucial pre-requisites for the rendering of appropriate diagnoses and treatments.

VI. Failure to Grant Stay of Order Will Result in Irreparable Harm

The failure of the Higher Court(s) to grant a Stay of Order will result in irreparable harm. Commensurate with the *Myers vs. API* decision of 2003 ("best interest" standard), there are three reasons why the proposed intervention of the Alaska Psychiatric Institute should now be rejected: a) misdiagnosis; b) failure to perform essential baseline assessments; and c) failure to act in the patient's best interests.

Misdiagnosis

Beginning with the respondent's very first API hospitalization at the age of 27 (4/15/80 through 4/30/80), Mr. Bigley was subjected to a dose of Haldol (10 mg po bid) which was 4 times higher than today's therapeutic dose ["therapeutic" as defined by those physicians who believe that antipsychotic effects arise from the blockade of 60-80% of the D2 receptors in the striatum]. Mr. Bigley's initial dose of Haldol guaranteed the induction of Parkinsonian symptoms by day #3 of treatment (4/17/80). Furthermore, the continued administration of Haldol -- a chemical which replicates the mitochondrial effects of rat poison and insecticide -- guaranteed the rapid deterioration of his condition. By killing brain cells, Haldol converted a possibly transient and reversible episode of psychosis or psychotic depression into a case of tardive dysmentia.

For example, the discharge summary from hospitalization #3 (2/27/81 through 5/4/81) reveals continuing problems with paranoia and disorganized speech; frontal lobe damage (several frontal release reflexes were noted on physical exam); and possible signs of tardive dystonia ("sitting in stiff fashion with head and neck markedly extended as he gazes at the ceiling"). Unfortunately, Mr. Bigley was not only continued on Haldol at that time, but the dose was raised to 20 mg po tid (60 mg per day). This was a dose which was 12 times higher than recommended, according to the theory of D2 receptor blockade.

Although the time constraints of this case have, thus far, limited my ability to review all pertinent records, the materials which I have reviewed (see Section III, #3 above) demonstrate a persistent and continuing failure of API clinicians to consider the most likely diagnosis in the case at hand. In all probability, Mr. Bigley now suffers from a chemical brain injury (CBI). This development should preclude the attachment of any and all psychiatric labels at this time. It should also trigger the legal and medical systems to prioritize the delivery of interventions which promote neuro-rehabilitation, rather than neurodegeneration.

Failure to Perform Essential Baseline Assessments

Prior to administering risperidone (or any other neuroleptic), the current recommendations of the drug manufacturers and professional organizations (such as the American Psychiatric Association) call for the performance of certain "baseline" evaluations of physical health. These assessments are crucial, in order to prevent sudden death arising from adverse cardiac events (e.g., tachycardia, QT prolongation, torsades, or other arrhythmia), endocrine disease (e.g., diabetic ketoacidosis or non-ketotic hyperosmolar coma), and/or other potential emergencies (e.g., infection due to low white blood cell count; liver failure; or neuroleptic malignant syndrome).

Especially before initiating risperidone, it is essential for providers at API to establish the presence or absence of pre-existing dysfunctions as described above (see Section V, #4). Moreover, given Mr. Bigley's 28-year history of exposure to various neurotoxicants, the differential diagnosis must now include several varieties of dementia (such as Lewy Body dementia and Alzheimer's disease), *for which the use of risperidone is specifically not advised.*

To put it simply, even if the Higher Court(s) were to agree with the Order of the Superior Court, the form of that order as presently written contradicts the recommendations of the medical profession, the Food and Drug Administration, and the manufacturers of the antipsychotic drugs.

Failure to Act in the Patient's Best Interests

Alaska Psychiatric Institute has proposed the immediate use of injectable risperidone (Consta) up to the maximal dose of 50 mg (IM) every two weeks. There are four chief problems with this treatment plan.

1) the manufacturer of risperidone specifically recommends a trial period of the short-acting preparation of the drug, prior to initiating Consta, in order to rule out a hypersensitivity reaction which might be fatal

[i.e., one does not begin with the injectable form of the drug and hope for the best]

2) the injectable form of risperidone (Consta) takes three weeks to take effect

From the available records, it does not appear that API has requested a court order for additional medication (such as oral risperidone) to cover the initial three week interval. To the extent that API would consider a three-week period of psychosocial supports to be adequate treatment during this interval, one must seriously question API's objections to the even more rigorous plan which has been outlined as the "less intrusive alternative" to pharmacotherapy.

3) the injectable form of risperidone (Consta) persists in the bloodstream for a period of seven weeks (and persists in the brain for at least one week longer)

It is because of the enduring effects of injectable forms of neuroleptics, such as Consta, that many concerned physicians oppose their use. Should Mr. Bigley develop neuroleptic malignant syndrome, cardiac defects, constipation and bowel obstruction, and/or a variety of tardive phenomena (such as respiratory dyskinesia), it will not be possible to eliminate the source of these events for up to two months.

4) risperidone (Consta or oral forms) will potentially kill Mr. Bigley while offering no significant prospect of improvement, and zero probability of recovery

Risperidone is an inhibitor of mitochondrial function and an inducer of oxidative stress. Through these cellular effects, risperidone then disrupts the structure and function of the cardiac, endocrine, hepatic, and neurological systems. It possesses some features which make it particularly undesirable, even among drug enthusiasts.

First, risperidone is unique among the newer "antipsychotic" drugs in terms of its potential to elevate prolactin. In some studies, hyperprolactinemia has occurred in as many as 90% of the risperidone patients. This is more than a trifling occurrence, due to the fact that hyperprolactinemia has been repeatedly linked to cardiac disease (e.g., via platelet aggregation, cardiomegaly, and heart failure).

Second, even at typical or "ordinary" doses (D2 blockade of 60-80%), risperidone induces Parkinsonian side effects at a rate which equals or surpasses the so-called traditional or conventional neuroleptics (e.g., in 30-50% of the patients).

Third, the real-world risk of tardive dyskinesia due to risperidone is significant and far more prominent than API's spokesmen have presumably opined. In Jose de Leon's recent study of patients who began treatment with the newer therapies (65% receiving risperidone), more than 60% of the subjects with treatment histories similar to Mr. Bigley's developed tardive dyskinesia despite the use of these "safer" drugs.

Fourth, given Mr. Bigley's advancing age (55 considered "elderly" in at least one published study); the early onset of Parkinsonian side effects (EPS at age 27); and a pre-existing organic brain syndrome (i.e., chemical brain injury), he is at high risk for tardive dyskinesia. In light of the fact that tardive dyskinesia (TD) reflects extensive damage to the brain – including impairments of judgment and insight, as much as impairment of movement – it is essential to avoid the use of any chemical intervention which might accelerate the emergence of this condition.

Fifth, commensurate with the affidavits, exhibits, and testimony on behalf of the respondent, it is extremely improbable that risperidone will do anything but aggravate the effects of the dysmentia (chemical brain injury) from which Mr. Bigley continues to suffer. To the contrary, risperidone will compound that condition with real and substantial risks of sudden death from stroke, heart attack, pulmonary embolism, diabetes, falls, accidents, pneumonia, NMS, and – ultimately – dementia.

For the aforementioned reasons, a Failure to Grant a Stay of the Superior Court's Order will result in irreparable harm.

DATED this 20th day of May, 2008, in WILMINGTON, North Carolina.

Grace E. Jackson MD

Grace E. Jackson, MD

SUBSCRIBED AND SWORN TO before me this 20 day of May, 2008.

"OFFICIAL SEAL"
NOTARY PUBLIC, NORTH CAROLINA
COUNTY OF WAKE
ERIN JENKINS
My Commission Expires:3-23-2009

Notary Public in and for North Carolina
My Commission Expires: 3/23/09