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

[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 swear 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 Case No. 3AN 07-1064PR, Superior Court, Third Judicial District, State of Alaska.

Dated: 10/27/2008

James B. Gottstein

SUBSCRIBED AND SWORN TO before me this 27th day of October, 2008.

STATE OF ALASKA
NOTARY PUBLIC
Lisa E. Smith
My Commission Expires April 23, 2011

Lisa E. Smith

Notary Public in and for Alaska
My Commission expires: 4/23/2011