Research

About the Intrinsic Suicidal Effects of Neuroleptics: Towards breaking the taboo and fighting therapeutic recklessness

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Abstract

Suicide is a frequent cause of death in people diagnosed with “schizophrenia.” These patients generally receive neuroleptics, which have an intrinsic suicidal effect. There are placebo studies, epidemiological surveys, first-hand reports, self-experiments and clinical research providing evidence about neuroleptic-caused depression and suicidality. Publications about the suicidal effects of neuroleptics – currently taboo – and suicide registers might reduce suicidality in “schizophrenics.”

Current suicide registers have been shown to have limited effectiveness, and meaningful programs to prevent suicide due to psychiatric treatment are needed. Effective suicide registers involving users and survivors of psychiatry would gather findings that could be used to warn the public, consumers, and caregivers. As long as there are so few alternatives beyond neuroleptic-based psychiatry, people have to protect themselves with advance directives and criminal charges.

Key Words: neuroleptic, antipsychotic, depression, schizophrenia, intrinsic effect, side-effect, suicide, suicidality

Über die suizidale Eigenwirkung von Neuroleptika – Das Tabu brechen, therapeutische Rücksichtslosigkeit bekämpfen

Zusammenfassung:


Wie sich zeigt, haben gegenwärtige Suizidregister eine begrenzte Wirkung. Man braucht wirkungsvolle Programme, um behandlungsbedingten Suiziden vorzubeugen. Wirksame Suizidregister unter Beteiligung von Psychiatriebetroffenen würden Erkenntnisse sammeln, mit denen man die Öffentlichkeit, die Betroffenen und ihre Betreuer warnen könnte.
ABOUT THE INTRINSIC SUICIDAL EFFECTS OF NEUROLEPTICS

Solange es so wenige Alternativen jenseits der neuroleptikabasierten Psychiatrie gibt, müssen die Betroffenen selbst für ihren Schutz sorgen, und zwar mit Vorausverfügungen und Strafanträgen.

Schlüsselwörter: Neuroleptika, Eigenwirkung, Nebenwirkung, Depression, Suizid, Suizidalität.

Le rapport entre le suicide et la prise de neuroleptiques : faire tomber les tabous et contrer la ‘légèreté thérapeutique’

Résumé:

Les suicides sont fréquents chez les personnes qui ont reçu un diagnostic de « schizophrénie ». En règle générale, on prescrit à ces patients des neuroleptiques qui ont un effet suicidaire intrinsèque. Différentes études de type placébo ou épidémiologique, des témoignages directs, des tests auto-expérimentaux entrepris, ainsi que la recherche clinique ont mis en évidence la manière dont les neuroleptiques peuvent provoquer des dépressions et des tendances suicidaires. Les publications sur les effets des neuroleptiques demeurent tabou alors que des registres détaillant les suicides permettraient de diminuer le taux de suicides chez les personnes dites ‘schizophrènes’.

Les registres actuels ne sont cependant pas très effectifs et il vaudrait mieux mettre en place des programmes adéquats de prévention du suicide du à un traitement psychiatrique. Pour que les registres soient effectifs, il faudrait inclure dans le processus les usagers et rescapés de la psychiatrie ; ils collecteraient des données qui pourraient être utilisées pour mettre en garde la collectivité, les usagers et les soignants. Tant que les traitements psychiatriques seront centrés essentiellement sur la prise de neuroleptiques, les usagers devront se protéger à l’aide de directives anticipées et d’actions en justice.

Mots-clés: neuroleptiques, antipsychotiques, schizophrénie, effet intrinsèque, effets secondaires, suicide, tendances suicidaires.

О суицидальном воздействии нейролептиксов: к отмене табу и борьбе с терапевтическим безрассудством

Резюме:

Суицид является частой причиной смерти людей с диагнозом «шизофрения». Таким пациентам как правило назначают нейролептиков, которые обладают свойством провоцирования суицидального поведения. Существуют данные, полученные в результате использования плацебо, эпидемиологических опросов, отчетов «из первых рук», отчетов испытуемых лиц и клинических исследований, свидетельствующие что склонность пациентов к депрессии и суициду вызвана приемом нейролептиков. Публикации (которые в настоящее время табуированы) о суицидальном воздействии нейролептиксов, а также регистрация суицидальных случаев могут снизить общую суицидальность «шизофреников».

Установлено, что существующая в настоящее время простая регистрация случаев суицидов недостаточно эффективна, поэтому необходимы обоснованные программы предотвращения попыток самоубийства вследствие психиатрического лечения. Более эффективный учет, охватывающий всех пользователей психиатрических услуг, должен включать все сведения, которые могли бы быть полезны для информирования общественности, больных и ухаживающих за ними людей. До тех пор, пока в психиатрическом лечении существует так мало альтернатив кроме нейролептиксов,
In patients with one or more filled prescription for an antipsychotic drug, an inverse relation between mortality and duration of cumulative use was noted…” (Tiihonen et al., 2009, p. 1). “Our results suggest that usage of antipsychotic medication has a beneficial effect on all-cause mortality, and also to some degree on suicide mortality." (Haukka et al., 2008, pp. 691–692).

Introduction

In a long-term Finish study, Tiihonen and colleagues write that the poor life expectancy of psychiatric patients (22.5 to 25 years less than the general population) is not caused by the toxicity of psychiatric drugs. Other authors argue that the cumulative administration of neuroleptic drugs could even have a beneficial effect on mortality (Tiihonen et al., 2009), including suicide prevention (Haukka et al., 2008).

Neuroleptics, also known as “major tranquilizers” and “antipsychotic drugs,” are synthetic drugs capable of affecting the brain, especially by reducing the intensity of nerve functioning, mainly the transmission of nerve impulses with dopamine. The main effect is the pharmacodynamic blockade of the autonomous nerve system and the state of a “retarded life,” as described by Meyer (1953).

Neuroleptics are used in the general medicine, in psychiatry and in veterinary medicine. In some totalitarian countries, political prisoners have been both tortured and calmed down with these substances.

Typical indications for use in psychiatric medicine are diagnoses like psychoses, schizophrenia, schizoaffective disorders, Tourette’s syndrome, psychosomatic disorders (like allergic reactions, nausea, vomiting, profuse hyperemesis gravidarum), etc. In humans and animals, neuroleptics are used for anaesthesia preparation and for tranquillisation in case of aggressivity. By medical definition, a drug can only be called “neuroleptic” when it has the power to cross the neuroleptic threshold; that is, to trigger (more or less subtle) Parkinsonian symptoms. The potency of a neuroleptic is measured in its quantitative relation to the neuroleptic prototype chlorpromazine (marketed as Largactil, etc.), which has the potency “1.”

Sometimes neuroleptics are combined with anti-Parkinsonian drugs to mask the Parkinsonian symptoms. Newer neuroleptics, the so-called atypical ones, have a broadband effect and work like the combination of neuroleptic and anti-Parkinsonian drugs: they make these substances at least, in the short run, subjectively more bearable, but they also have  

1 Neuroleptics developed over the years include amisulpride (marketed as Solian, etc.), aripiprazole (marketed as Abilify), asenapine (marketed as Saphris, etc.), clozapine (marketed as Clozaril, etc.), haloperidol (marketed as Haldol, etc.), olanzapine (marketed as Zympexa, etc.), paliperidone (marketed as Invega), risperidone (marketed as Risperdal, etc.), sertindole (marketed as Serdolect), sulpiride (marketed as Dolmatil, etc.), thioridazine (marketed as Mellaril, etc.), ziprasidone (marketed as Geodon, etc.), zuclopenthixol (marketed as Clopimexil, etc.) and many more.
dangerous risks (Ebner, 2003; Saha et al., 2007). Some psychiatric research shows that the life expectancy of psychiatric patients is lowered by two to three decades, compared with the general population (Ösby et al., 2000; Colton & Manderscheid, 2006). Some psychiatrists surely doubt the connection of this outcome with the effects of the administered drugs, and some may see even a reduced mortality, especially as a result of drug treatment.

Writing about the influence of neuroleptics on mortality in “schizophrenics,” Weinmann and colleagues (2009) present compelling evidence that counters Tiihonen’s argument that neuroleptics enhance life expectancy. Aderhold discusses many drug-related causes of death (cardio- and cerebrovascular, digestive, endocrine, respiratory, infectious, genitourinary, neoplastic and nervous diseases including tardive dyskinesia and malign neuroleptic syndrome). This contradictory information is echoed in the authors’ declarations of conflicts of interest. Aderhold and colleagues deny any conflict, whereas Tiihonen – like Haukka – have connections to major pharmaceutical companies (like Lundbeck, Organon, Janssen-Cilag, Eli Lilly, AstraZeneca, Hoffman-La Roche, and Bristol-Myers Squibb) and deliver “expert opinions” and lectures for them (Tiihonen et al., 2011).

“Therapeutic” Diseases and Mortality Registers

Since the 1950s, the standard biological psychiatric treatment for people with diagnoses like “schizophrenia” is a range of neuroleptic drugs, the so-called antipsychotics. These psychotropic drugs intervene in the patients’ (natural) metabolic systems and produce a “secondary illness” (Haddenbrock, 1964, p. 63) by administering these neuroleptics to cover the supposed primary illness. Dörner, a “progressive” German psychiatrist, and the psychologist Plog explain the modern treatment principle of trying to make “mental diseases” disappear:

“We temporarily turn the mentally suffering patient into a person with an organic brain disease, with ECT it happens in a more global way, but for a substantially shorter period of time than with pharmacological therapy.” (1992, p. 545).

The newer generation of neuroleptics, the so-called atypicals, are widely suspected of causing increased circulatory problems, abnormal blood cell counts, obesity, diabetes and receptor changes that can, in time, also lead to chronic illnesses, including psychoses: so the problem is not cured, it is compounded. In 2003, Ebner admitted that there were severe risks and injuries caused by “atypical” neuroleptics:

“It is not a case of fewer side-effects, but of different ones which can be just as debilitating even if the patient isn’t immediately aware of them. Therefore, patients can be more easily motivated to take these drugs because they no longer suffer instantly and as much from the excruciating dyskinesias/extrapyramidal side-effects.” (p. 30).

Beside the high risk of being damaged physically, often with chronic consequences, users and survivors of psychiatry in Europe generally are systematically discriminated against in the medical and psychiatric sector. This was documented by an action project: “Harassment and Discrimination Faced by People with Psycho-Social Disability in Health Services – A European Survey,” with support from the European Union. There is no reason to believe that psychiatric patients outside of Europe are treated any better.

One of many proposed measures to combat discrimination was the recommendation

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that laws espousing equality of treatment should be adopted and funds provided to enforce these protections. These laws should guarantee respect for human rights in a proactive way, and focus on the protection of human dignity, the right not to be violated, the right to self-determination, the right to privacy and the right to respect – for example, through the legal protection of advance directives, or through the introduction of a suicide register (see ENSP, 2005).

Mortality registers are not unusual in the medical field to identify connections between reduced life-expectancy, lethal outcomes of medical treatments, and risk factors. Barreira, for example, wrote about patterns in the causes of suicides, mortality and reduced life expectancy of psychiatric patients:

"From the standpoint of public policy, it is essential to conduct further research with databases from across mental health systems and different states to explain the differences in life expectancy and causes of death." (1999).

According to Müller (1989), suicide in people diagnosed as “schizophrenics” is about 50 times more frequent than in the general population. New psychiatric data show a consensus on the lifetime risk of suicide in people diagnosed as “schizophrenics,” a rate of approximately 5%, which is ten times higher than in the general population (Hor & Taylor, 2010; Nordentoft et al., 2004; Heilä et al., 2005; Qin & Nordentoft, 2005). Schneider calls suicide the most frequent cause of premature death in “schizophrenics” (2003). But while mainstream “neuromythological psychiatry” has a biochemical explanation for all human emotions, even for love or being moved by looking into the sunset ("Alles," 2000), when it comes to the explanation of suicides of “schizophrenics,” psychiatry explains this only by emotional and socio-economical factors. In a review of the literature about mortality and causes of death in “schizophrenics,” Tabbane and colleagues refer to many possible causes of suicide, but do not mention any pharmacological factors:

"Premature death is highly linked to suicide … Suicide risk factors are numerous. Some of them are accepted as valid and others are still discussed. The former are: male, young and at least ten years since onset, associated depressive symptoms, past history of suicide attempts, iterative relapses and post-hospital discharge period. The latter are: social isolation, celibacy, unemployment, high level of education, delusional and hallucinatory activity, and familial rejection." (Tabbane et al., 1993).

Hor and Taylor (2010) undertook a systematic review of all original studies concerning suicide in people diagnosed as “schizophrenics” published since 2004. To the causes reported by Tabbane and colleagues, they add that comorbid substance misuse is associated with later suicide. But, of course, by “substance misuse” they do not mean neuroleptics prescribed by a doctor.

And all these studies hold that the delivery of the “best available treatment for psychotic symptoms” is the only consistent protective factor for suicide: By this, they mean treatment with “atypical” neuroleptics. So it is no surprise that Tiihonen and colleagues’ study of “first episode” patients found that the suicide risk of those not currently taking neuroleptics was 37 times higher than in compliant patients (Tiihonen et al., 2006). The database of the U.S.

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3 For many years, Rufer has criticized the insolence and scientific almightiness of modern psychiatry:

"Neurobiology is booming, governments and industry are investing billions. The media have blown up the findings of brain research into a huge success – brain research, the 'science of the century,' is in the process of becoming the new social science. A new mythology has emerged – neuro-mythology" (Rufer, 2007, p. 383).
Food and Drug Administration, evaluated by Khan and colleagues, showed no difference in the suicide risk of people taking placebos or modern neuroleptics (Khan et al., 2001).4

Risk Factors for Depression and Suicidality

There are many well-known factors that can trigger depression and suicidal behaviour: political, social and economic, emotional and physical factors (Lehmann, 2010b).

There can be psychiatric factors: Unhappiness, depression, and suicidal ideation can each arbitrarily be called a psychiatric disease; fear of forced admission (“Angst,” 1988), or desperation about the stigmatisation and discrimination that goes along with diagnoses like “schizophrenia” (Rufer, 1988). Desperation about an “incurable” psychiatric diagnosis can trigger suicide, especially combined with discrimination, self-stigmatisation and social decline (Hentschel et al., 1987), or traumatization by inhumane treatment (e.g. by combined insulin- and electroshock, plus administration of all kind of psychiatric drugs [see Kempker, 2000 and Lehmann, 2010a]).

There can be medical diseases and disorders: Neurological diseases like cerebrovascular diseases, tumours, Parkinson’s disease; infections like AIDS or hepatitis; endocrinological diseases like morbus Cushing; metabolic disorders like dehydration; cancer; alcohol dependence; or genetic abnormalities in the serotonin system – have all been found to trigger depression (Härter et al., 2007).

Depression, leading to suicidality, can be caused by illegal drugs like mephedrone (Rehfeld, 2011) and by prescribed drugs (Jain, 2012, p. 62) including psychiatric drugs like tranquilizers (i.e., benzodiazepines [Hall & Joffe, 1972; Remschmidt, 1980; Van der Kroef, 1979; Lydiard et al., 1987; Lehmann, 1996b, p. 361]; mood stabilizers [Patorno et al., 2010]; antidepressants [Arzneimittelkommission, 2004; Fergusson et al., 2005; Olsson et al., 2006; U.S. Food and Drug Administration, 2007]5; and neuroleptics (see below). “Suicidality can evidently also have chemical-biological causes in the brain” (cited in Schmalenberg, 2010, p. 21), Müller-Oerlinghausen says about the possibility of brain malfunction, which can also trigger suicidality. Compared to warnings about the suicide risks of medical and other psychiatric drugs, the amount of warning about suicide risks of neuroleptics is significantly silent.

Depression, Suicidality and Neuroleptics

As previously stated, when psychiatrists give the diagnosis “schizophrenia” or similar ones like “psychosis,” the standard treatment is the administration of neuroleptic drugs. Discussing studies on suicides in patients with the diagnosis “schizophrenia” and comparing suicide rates in different time periods, Healy and colleagues plead for an explanation of the “excess of suicides among patients receiving treatment” (2006, p. 227). For people with a little medical knowledge, the reason seems obvious: Neuroleptics have a blockading effect, primarily against the transmitter dopamine, resulting in subtle Parkinsonian-type syndromes. This is a complex of symptoms, characterized by walking with a stoop, muscle tremor, blurred speech

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4 Taking Tiihonen and colleagues’ data seriously, you could argue that the authors ignore the fact, that, in general, people who refuse psychiatric drugs are punished by denial of all forms of psychotherapeutic and social support. In the cited study, this denial, which might enhance the suicide risk, is totally ignored, as are all kinds of social-economical factors, although the authors admit the latter deficit in their study. Conversely, Khan and colleagues’ study could be interpreted as proof that the effects of psychotherapeutic and social support that people receive–together with placebos, prevent suicide.

5 See also the many reports on the anonymous website SSRI Stories – Antidepressant Nightmares – http://ssristories.com
and – on the mental level – the “Parkinson psyche” (a defined personality change specific to Parkinson disease: which includes the development of apathy, depression, desperation, hopelessness, suicidality, and disturbances up to psychoses). Parkinson's disease seems to have a very close connection with dopamine blockage (Gerlach et al., 2003). The potency of neuroleptics was defined by their power to create Parkinsonian symptoms. This is not an unwanted side effect; this is the therapeutic main effect as defined by psychiatrists.

Neuroleptics can produce akathisia (increased motor activity), an akinetic syndrome (muscle rigidity, bradykinesia [diminished movement of body musculature] or akinesia [loss of normal motor function]). Both are forms of Parkinson's disease, which in turn can produce torturing sleeplessness and promote suicide (Wolfersdorf & Etzersberger, 2011, pp. 150/173). Parkinson's disease involves mental and emotional alterations, too. Neurologists define this as Parkinson personality. The symptomatology includes apathy, loss of willpower, depression and suicidality and states of confusion and delirium (Fünfgeld, 1967, pp. 3-25). In 1955, after the first administrations of Megaphen (chlorpromazine) the German psychiatrist Von Ditfurth pointed to the parallels between the emotional Parkinsonian deadening after a brain disease and the emotional deadening after neuroleptic treatment:

“As we may believe, it looks like as if the psychic alterations provoked by Megaphen especially on the emotional level are from the same nature as the 'affective deadening and restriction,' which is registered so often at post-encephalitic parkinsonists.” (p. 56).

Depression and suicidality are common effects of neuroleptics, as shown above, and thus psychiatrists accept them seemingly without question. Ayd wrote in 1975:

“There is now general agreement that mild to severe depressions that may lead to suicide may happen during treatment with any depot neuroleptic, just as they may occur during treatment with any oral neuroleptic. These depressive mood changes may transpire at any time during depot neuroleptic therapy. Some clinicians have noted depressions shortly after the initiation of treatment; others have observed this months or years after treatment was started.” (p. 497).

Benkert and Hippius (1980) found that:

“Depression, suicidality, states of excitement and delirium under the influence of drugs generally occur during doses prescribed by the treating physician.” (p. 258).

De Alarcon and Carney studied depressive mood changes after administration of neuroleptics with other variables staying the same. They reported on suicides under the influence of fluphenazine and described a fluphenazine trial with a 39 year-old man who already had tried to kill himself under the influence of this drug. When the psychiatrists realized that this man had regularly developed suicidal intentions some days after the two-week depot-injections, they wanted to witness the mood-worsening effect of the neuroleptic with their own eyes. In the psychiatric institution, the man was observed over four weeks, without being treated with neuroleptics, and without displaying anything remarkable mood fluctuations. They then injected him intramuscularly with 25 mg of fluphenazine:

“He was given the trial injection on a Wednesday at 3 p.m.; by mid-afternoon on the following day he felt low, wanted to be left on his own, and had no desire to talk to anyone, read, or

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6 In Parkinson’s disease, the dopamine-transmitting neurons in that area of the mid-brain called the substantia negra die off. As a result, the brains of people with Parkinson’s disease contain almost no dopamine. Dopamine-agonist drugs, like L-DOPA, a drug that can be converted to simulate dopamine (in that it binds to dopamine receptors in place of dopamine) are thus often used for Parkinson’s disease to relieve the symptoms. Adapted from University of Texas website: http://www.utexas.edu/research/asrec/dopamine.html, accessed on 4th Dec, 2011.
watch television. He took to his bed at about 4 p.m. In the opinion of the charge nurse he was a suicidal risk. When interviewed on the Friday the change in external appearance was striking – he looked gloomy, he did not respond with a smile to a joke, and there was no spontaneous conversation. His answers were limited to what was strictly necessary. He denied any paranoid or hypochondriacal ideas or any feelings of guilt. He simply said that he felt very low and if he were alone in digs he would take his life. By Friday evening there was some improvement, and when he was interviewed again on Saturday he had returned to his usual normal self.” (1969, pp. 565-566).

In a placebo-controlled study, Müller (1981) found that a much higher percentage of people treated with neuroleptics had depressive symptoms than people treated with placebos.

“From 47 cases, the depressive mood lifted in 41 cases, in only two cases there was no change, and in four cases the effect was dubious. It was very surprising to see that in the predominant number of cases the reduction of the doses alone (normally to half of the former dose) lead to an improvement of the depressive symptoms. Often it was only a partial improvement, but even this brought clear relief to the patient. On the other hand, in other patients, or in the same ones whose situation improved only slightly when taking lower doses, complete withdrawal made them feel much better. Some patients reported that only now did they feel completely healthy again, as they had long before their depressions. The depressive symptoms, which were seen to be unchangeable by some psychiatrists, and which could possibly have been taken to be a start of organic disorder, vanished completely. The possible argument that these could be psycho-reactive effects caused by the patients’ relief about the withdrawal of the psychiatric drug is refutable, because nearly all patients received depot-injections and were not informed about their doses or got placebo-injections. (...) Their change was quite impressive to themselves, their relatives and their medical examiners in some cases. The patients reported that now they felt completely healthy again. In the group of people still treated with psychiatric drugs, this was mostly not the case. These results quite definitely speak for pharmacogenic influences and against psychiatric morbidity developments.” (pp. 52-53 / 64).

Müller continued:

“Depressive syndromes after the remission of the psychoses and under treatment with psychiatric drugs are not rare, but occur in about two-thirds of the patients, and sometimes even more frequently, especially when depot-drugs are given. Without treatment with psychiatric drugs, depressive syndromes after a complete remission are only found in exceptional cases.” (p. 72).

Müller’s reports are supported by many of his colleagues (Lehmann, 1996, pp. 57-87, 109-115). Battegay and Gehring (1968) warned after a comparison of treatment courses before and after the era of psychiatric drugs:

“During the last years, a shifting of the schizophrenic syndromes to a depressive syndrome was repeatedly described. More and more schizophrenias show a depressive-apathetic course. It became clear that often exactly what develops under psychiatric drugs, what should be avoided with their help and what is called a defect.” (pp. 107-108).

Pöldinger and Siebern wrote:

“It is not unusual that depressions caused by medication are marked by a frequent occurrence of suicidal ideation” (1983, p. 131).
According to Scharfetter, who emphasized the effective time of the maximal neuroleptic effect at the point of suicide (1986, p. 89), Rufer warned: “Schizophrenics, who receive neuroleptics in high dosages, kill themselves in increased numbers” (1988). In 1976, Haase reported that the number of perilous depressive occurrences after treatment with psychiatric drugs increased at least ten times (Haase, 1976). The increase in the suicide rate is “alarming and worrying,” said Armbruster in 1986 Nervenarzt – without, nevertheless, alerting psychiatric patients, their relatives and carers, or even the public. Hessö wrote, in 1977, that it seemed to be clear: “...that the increased incidence of suicide, both absolutely and relatively, started in the year 1955. This was the year that neuroleptics were introduced in Scandinavian psychiatric hospitals.” (p. 122).

In 1982, Modestin reported his findings at two Swiss institutions:
“Our results show a dramatic increase of the suicide frequency among the patients in Berne and Münsingen in the last years.” (p. 258).

**Firsthand Reports about Neuroleptic-caused Suicidlity**

In Coming off Psychiatric Drugs (Lehmann, 1998, 2004), Bellion gave a report about her psychic condition under Haldol administered by a community psychiatrist:

“I vegetate behind my neuroleptic wall and I am locked out of the world and out of life. The real world is further from me than Pluto is from the sun. My own secret world is also gone – my last refuge and I had destroyed it with Haldol. This is not my life. This is not me. I may as well be dead. An idea has begun to take shape. Before winter comes I will hang myself. But before that I want to try and see if my life would be different without Haldol. I reduce the number of drops. I take less and less until I arrive at zero. After one month I am clean. Then I begin to notice how unkempt I am. I wash my hair, make the bed, clean the apartment. I prepare a warm meal. I even enjoy doing this. I can think again.” (2004, p. 280).

Another psychiatric patient was given a prescription of Haldol and the antidepressant doxepin. Under the influence of this combination, she tried to end her suffering by committing suicide:

“When I got out again I would sit in my kitchen in front of the water-faucet, thirsty but yet unable to pour myself a glass of water or to bite into the bread that had become stale and hard. The supermarket was not far away, but I couldn't manage to get up and so I wished that I were simply dead so that I would have some peace at last. I was broken by my illness. I saw it as a punishment for two dark points in my life. Worst of all was the vicious circle of endlessly recurring psychotic patterns of thought. I tried again and again to think of something else even just for a moment – but it didn't work. My thoughts always revolved in the same circles, a hundred times a day, sometimes at a time-loop tempo in slow motion, other times constantly accelerating until my brain was spinning. And that was hell for me, the devil's game. I felt damned and abandoned by God with no hope of salvation. I could do nothing but suffer through this film, my life, lying down. I knew that I had to learn to have faith again, but I couldn't, and so I tried to end my life.” (Marmotte, 2004, p. 114).

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Fortunately, she decided to withdraw from her psychiatric drugs; she did not kill herself. Leponex (clozapine), the prototype of “atypical” neuroleptics, seems to have suicidal effects, too, as reported by Fröhlich:

“Since I began taking Leponex I do not want sex anymore, did not feel like moving and had no joy in life. A life without joy is, however, worse than death. All that remained with me is watching TV, where I have watched others living for seven years. I am still alive biologically, but my senses are long since dead, everything that I former enjoyed I am not able to do anymore. In a way, my life does not exist anymore, I feel so empty and unimportant. In the morning, the feeling is the worst. Every day I intend to start a healthy life the following day, to throw away the drugs, to drink many vitamins and fruit juices and to start with a daily fitness routine. The psychiatric drugs cause a feeling as if it was possible for me to start with a completely different, a new life the following day. But when I wake up in the morning I feel like smashed, and I never come out of bed before 9 o'clock, my depressions are so extreme that I think of suicide every day.” (cited in Lehmann, 1996, pp. 70-71).

Psychiatrists who have ingested these drugs have had similar experiences. In 1955, Heimann and Witt published their experiences after once taking chlorpromazine. They experimented with spiders and control subjects; they conducted three self-experiments and nine with psychiatrists and pharmacologists. The marked feelings of inferiority and powerlessness (typical elements of Parkinsonian symptoms), after taking the neuroleptic became very clear:

“I felt physically and mentally ill. Suddenly my whole situation appeared hopeless and difficult. Above all, the fact was torturing that one can be so miserable and exposed, so empty and superfluous, neither filled by wishes nor by something else ... (After finishing the examinations, P.L.): The tasks of life grew immense in front of me: dinner, go to the other building, come back – and all of that by foot. With that this state reached its maximum of uncomfortable emotions: The experience of a passive existence with clear knowledge of the other possibilities ...” (p. 113).

Heimann and Witt’s 1955 publication demonstrates the extreme depressive effect of neuroleptics. While they are aware of the theoretic possibility of living an active life, the experienced apathy caused by neuroleptics makes them feel as if they no longer have that possibility (and will never again have that possibility), and they may react with depressive desperation. Placebo studies, epidemiological surveys, first-hand reports, self-experiments and even clinical research show coinciding results. Benkert and Hippius demonstrated that the problem, in general, is independent from the dose. They wrote:

“But also small doses can trigger depressive moods, especially in elder patients. The danger of suicide in a pharmacogene depression is just as big as in a depression of another genesis and though has to be taken absolutely seriously” (1980, p. 257).

Newer studies show that there has been a trend over the years that people diagnosed with schizophrenia who kill themselves shortly after discharge from psychiatric inpatient treatment are doing so at increasingly younger ages (Wolfersdorf, 1996). In general, this immediate post-discharge period involves the intensive administration of neuroleptics, such as in the context of the Assertive Community Treatment model.
Suicide Registers and Psychiatric Drugs

For different reasons, as we will see, suicide registers have shown limited effectiveness, whether they are run by former psychiatric patients, by psychiatrists and by governmental administrations.

By Survivors of Psychiatry

In early 1983, a Berlin organisation of survivors of psychiatry publicly warned of suicides caused by neuroleptics, after they received information about people who had hung themselves, gassed or poisoned themselves, jumped to death, or thrown themselves in front of subway trains (“Psychopharmaka,” 1983). A public call to support the establishment and funding of a suicide register bore no results, and the initiative eventually came to an end. But the demand for a public suicide register was born.

By Psychiatrists

Another type of suicide register was developed in the form of the “Arzneimittelüberwachung in der Psychiatrie” (AMÜP – a drug monitoring system in the psychiatric field) in Germany, founded in 1979 and supported by the National Health Administration. Since the 1990s, psychiatric hospitals in this region have gathered data on complications that may have resulted from treatment, including the registration of drug-triggered suicide attempts and suicides by drugs, in order to make public information on risks and develop programs for prevention and early detection (Haen et al., 1999, p. 93). “Findings are discussed within the psychiatric community without prejudice...” (ibid., p. 94). If a psychiatrist identifies a drug as potentially suicide-triggering, they report to the National Institute for the Safety of Drugs, the Drug Commission of the German Medical Association and the Drug Producers. This article did not include information on how many reports were sent after the reported 89 registered suicide attempts and suicides prior to January 1998.

In a 2002 review, Bavarian psychiatrists reflected on their results from 1991 through 1999 and the methodological problems that arose from registering suicides and identifying the one exclusive cause that triggers suicidality. They mention, for example, problems with the definition of suicidality, if no overt suicidal act is committed, and plead for the further development of questionnaires and registration cards (France et al., 2002).

Repeated offers by the author, as a board member of ENUSP, to discuss the possibility of including users and survivors of psychiatry in the Bavarian suicide register and to help make the registration criteria sharper and more effective, met with no response or result – like the Bavarian suicide register itself.

By Governmental Administrations

Larsson described a Swedish suicide register in October 2009. Referring to regulations in The Act on Professional Activity in Health and Medical Services, since February 2006, all suicides committed in health care and within four weeks after the last health care visit must be reported for investigation to the National Board of Health and Welfare. Larsson’s data about various neuroleptics (fig. 1), antidepressants (fig. 2) and tranquilizers (fig. 3) administered within four weeks of the suicide shows that:

“According to the data received, 393 cases were reported to the six regional offices for 2007. In 338 of the 393 cases – 86% of the cases – the persons were treated with psychiatric drugs within one year before their suicide.”
In 304 cases – 77% of the cases – the persons were treated with antidepressant drugs and/or neuroleptics.

In 261 cases – 66% of the cases – the persons were treated with tranquilizers and/or hypnotics; drugs of the class benzodiazepines or similar newer compounds” (pp. 17-19).

![Fig. 1: "Atypical" neuroleptics administered to people who committed suicide in Sweden in 2007 four weeks before they committed suicide](image1)

In addition, many people were treated with other classes of psychiatric drugs, including epileptic drugs used as mood stabilizers (pregabalin, lamotrigine), ADHD drugs (methylphenidate, atomoxetine) and other types of psychiatric drugs like the pain-killer buprenorphine or the sedative clomethiazole.

![Fig. 2: Antidepressants administered to people who committed suicide in Sweden in 2007 four weeks before they committed suicide](image2)
Fig. 3: Tranquilizers/hypnotics administered to people who committed suicide in Sweden in 2007 four weeks before they committed suicide

Larsson summarizes the results of the 2007 report:

“In 86% of the cases of suicide reported to the National Board of Health and Welfare for 2007 (chapter 4) – that is in 338 of 393 cases – the persons were treated with psychiatric drugs. In 0% (!) of these cases was the matter reported as a drug adverse event to the registry for drug adverse events at the Medical Products Agency (...). Instead of Eli Lilly claiming that the drug Zyprexa (olanzapine; neuroleptic) was involved in 0 cases of suicide in Sweden 2007, the fact was that the drug was involved in 52 cases in this subgroup of 338 persons. Instead of Wyeth claiming the same for Effexor (venlafaxine; serotonin/norepinephrine reuptake inhibitor), the fact was that the drug was involved in 41 cases in this group” (ibid., pp. 23-25).

Larsson’s report also includes data about the total number of suicides in Sweden in 2007 and the preceding psychopharmacological treatment in these cases, as well as autopsy data from the Swedish National Board of Forensic Medicine. Larsson’s data discusses the percentage of psychiatric drug classes found in autopsies of people who committed suicide (fig. 4) as well as the classes of psychiatric drugs found in the their blood (fig. 5), and writes:

“The result shows that 1126 definite suicides were committed in Sweden in 2007 (325 women and 801 men). Of these persons, 724 (64%) had filled a prescription for psychiatric drugs within a year of the suicide. Of the 325 women, 250 (77%) had filled a prescription for psychiatric drugs; for the 801 men the figure was 474 (59%). Of the women, 196 (60%) had filled a prescription for antidepressants; for the men the figure was 306 (38%).

In the forensic toxicological analyses, traces of psychiatric drugs were found in 575 persons (52%) of the 1109 analyses done. Traces of antidepressant drugs were found in 132 (41%) of the women investigated.

The conclusion is that a large percentage of the persons who committed suicide in Sweden in 2007 had received extensive treatment with psychiatric drugs within a year of and close to the suicide” (ibid., p. 2).
**Fig. 4:** Percentage of psychiatric drug classes found in autopsies at people who committed suicide in Sweden in 2007

**Fig. 5:** Percentages of traces of psychiatric drugs found in autopsies at men and women who committed suicide in Sweden in 2007
Consequences and Next Steps

Updated product labelling for neuroleptic drugs should include a warning about the increased risk of suicidal thoughts or actions, in order to help patients, supporters and psychosocial staff understand these risks. This must be done even if there is only a fairly low incidence of this risk. Users of psychiatric drugs need to be informed so that they can make carefully considered decisions about whether to take a recommended psychiatric drug. Practices in psychiatric wards could be significantly improved to ensure that patients (and their carers and relatives) have much better information about the prescribed drugs – and their subjectively unwanted effects – and actively consent or deny to taking these. If they so decide, they can then opt for viable alternatives, either outside of pharmacological psychiatry or by using less risky psychopharmacological treatments for their emotional problems.

Reports of (ex-) users and ‘survivors’ of psychiatry who have experienced suicide attempts or suicidality after treatments with psychiatric drugs, electro- and insulin coma shock that have traumatizing effects in many people must no longer be ignored. They must be – and sometimes are – included as keynote speakers, experts and teachers in education programs, conferences and in the public media.

There is, however, a well-established tradition that the segregation of troubled (or troublesome) people is not something that everyone agrees should be prevented. This refers to people who usually have not violated any laws and thus cannot be criminally prosecuted and imprisoned, but whose ideas and actions, values and life styles disrupt (or threaten to disrupt) established relationships. Also psychiatrists can sympathize with population selection by suicide. For example, in 1923, Lenz, one of the most influential German eugenicists and an advocate of racist population selection, praised suicide – with the support of Ernst Bleuler, a leader of mainstream psychiatry (see Lehmann, 1994) – as a measure against “vulgarisation of the race”:

“From this, the selection through suicide lies in the direction of the strengthening of the population’s living will and its cheerful temper.” (p. 23).

As an urgent measure, individually, we must use and improve advance directives to protect ourselves from unwanted treatment (Ziegler, 2007), in which we clearly should mention factors like previous depressive states caused by psychiatric drugs, if we have had such experiences. And, collectively, we could even demand the application of criminal law, not only to penalize professional non-assistance to a person in danger (a breach of standard of care), but also to penalize recklessness – particularly so in the prescription of neuroleptics. In US, German, Swiss and other courts, a wrongdoer who recklessly causes harm can be held as liable as a person who intentionally does so. If psychiatrists continue to administer psychiatric drugs with suicidal effects to people who are known to have underlying risk factors, they should know that laws espousing equality also demand equality in legal responsibility for damages under criminal law. This could be true for the owners and management of drug companies that produce drugs with ‘known’ suicidal effects.

A suicide register with meaningful participation of independent organisations of users and survivors of psychiatry could enhance warnings of suicidal risks of psychiatric treatment methods. It could work, if funds were provided by the authorities, and if it received the

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8 You can find out more information about this principle of law on the internet: http://en.wikipedia.org/wiki/Recklessness_(law)
9 Six decades after the end of World War II the European Union is now funding research about suicides of Jewish people during the Nazi regime ("Freitod," 2006).
authority to gather data, as well as the means to publish and publicize its findings. It could be organized nationally or regionally and be legally authorized; it should then be easily accessible (anonymous upon request) and should operate independently of any medical and psychiatric institutions. As indicated, suicide registers run in this way produce very valuable data, though with perhaps somewhat embarrassing implications.

As a form of user-led or user-controlled research, delegates of independent organisations of users and survivors of psychiatry, as well as competent and independent individuals, should be included in prevention programs and monitoring bodies with adequate remuneration. Again, when this is done, the benefits outweigh the fairly minimal costs.

The rate of suicide in people with emotional problems or those labelled “mentally ill” could be lowered meaningfully with a functioning and independent suicide register. Where the damage has already been done, there may, at least, be a possibility to apply for financial compensation. Enhanced knowledge about the suicidal effects of neuroleptics (as well as other iatrogenic injuries in mental health) could help to protect people with diagnoses like “schizophrenia” from additional burdens and risks from unpleasant effects of the drugs prescribed. Individuals could be more protected from damage caused by toxic elements in drugs. Enhanced knowledge about the suicidal effects of neuroleptics and other iatrogenic injuries would enable mental health professionals, relatives, friends and other carers to support people to live their lives in greater freedom and more peace of mind.

Author


Peter Lehmann does not have any connection to the pharmaceutical industry and to organizations that are dependent on them, nor to Scientology, their subgroups or other sects of whatever colour.

10 It would be counterproductive to include pharmaceutical companies in this research (see, for example, the proposals of the Institute of Medicine, the health arm of the U.S. National Academy of Sciences [see Steinbrook, 2009]) or to include people and organisations which receive(d) funding and other benefits from pharmaceutical companies (Boseley, 2007; Lehmann, 2009, pp. 34-35).
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11 All translations of German citations into English are made by the author or by translators. The explanations in the italic brackets are written by the author. Thanks to Darby Penney, Asmati Augustin, Agita Lüse and Anne-Laure Donskoy for support in translation matters. And thanks to Courtenay Young for support in editorial matters.
ABOUT THE INTRINSIC SUICIDAL EFFECTS OF NEUROLEPTICS


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