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Psychiatric drugs: reconsidering their mode of action and the implications for service user involvement

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Psychiatric drugs: reconsidering their mode of action and the implications for service user involvement

Abstract

Purpose: The purpose of this paper is to examine two competing pharmacological models that have been used to understand how psychiatric drugs work: the disease-centred model and the drug-centred model. In addition, it explores the implications of these two models for mental health service users and the degree to which they are meaningfully involved in decisions about the use of psychiatric drugs.

Design/methodology/approach: The approach is a conceptual review and critical comparison of two pharmacological models used to understand the mode of action of psychiatric drugs. On the basis of this analysis, the paper also provides a critical examination, supported by the available literature, of the implications of these two models for service user involvement in mental health care.

Findings: The disease-centred model is associated with a tendency to view the use of psychiatric drugs as a technical matter that is to be determined by mental health professionals. In contrast, the drug-centred model emphasises the centrality of the individual experience of taking a psychiatric drug and implies a more equitable relationship between practitioners and mental health service users.

Originality/value: Although infrequently articulated, assumptions about how psychiatric drugs work have important consequences for service user involvement in mental health care. Critical consideration of these assumptions is an important aspect of seeking to

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3 maximise service user involvement in decisions about the use of psychiatric drugs as a
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5 response to their experience of mental distress.
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10 **Introduction**

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13 A widespread and enduring notion about psychiatric drugs is that they work by targeting
14 and correcting various biological dysfunctions that supposedly underlie the emergence
15 and maintenance of mental distress. Despite its pervasiveness, this understanding of how
16 psychiatric drugs work is increasingly being contested by those who work within and
17 those who use mental health services (Moncrieff, 2008; Lacasse and Leo, 2015; Whitaker,
18 2015; Roberts, 2018). Such a reconsideration of the mode of action of psychiatric drugs
19 has important implications for the way in which they are used, the manner in which their
20 potential benefits are balanced against their adverse effects and the extent to which those
21 who use psychiatric drugs are meaningfully involved in decisions about their use. In
22 seeking to explore these implications this paper will begin by critically examining the
23 popular notion that psychiatric drugs work by selectively acting upon some form of
24 biological dysfunction or chemical imbalance of which mental distress is a supposed
25 manifestation. However, in contrast to this notion, it will move on to discuss an
26 alternative understanding of psychiatric drugs as powerful psychoactive substances that,
27 by acting upon the central nervous system in a non-specific rather than a targeted way,
28 produce a range of physiological and psychological effects. Finally, this paper will
29 consider critically the implications of these two ways of understanding how psychiatric
30 drugs work for those who use mental health services and for the degree to which service
31 users are meaningfully involved in decisions about the use of those drugs as a response to
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3 the experience of mental distress.
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8 **The disease-centred model of psychiatric drugs**

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10 A popular conception of psychiatric drugs is that they act upon the biological
11 dysfunctions and disease processes that are commonly thought to underlie the emergence
12 and maintenance of mental distress. This explanation of psychiatric drugs draws upon a
13 dominant pharmacological model, what Moncrieff (2015) has referred as ‘the
14 disease-centred model’, that is used to explain how drugs in other fields of medicine are
15 understood as acting upon the biological causes of a disease or the biological processes
16 that produce the symptoms associated with a particular disease. In explaining how
17 psychiatric drugs work by drawing upon this disease-centred model, those drugs are
18 sometimes presented as working in a way that is broadly analogous to how insulin works
19 in diabetes. For example, in a manner somewhat similar to how insulin alleviates the
20 symptoms associated with diabetes by compensating for the body’s inability to produce
21 sufficient quantities of that hormone, psychiatric drugs are presented as alleviating or
22 even eliminating the symptoms associated with mental distress by targeting and
23 correcting the biological dysfunctions that are commonly presented as being implicated in
24 such distress. In highlighting this supposedly selective character of psychiatric drugs,
25 particularly those that emerged during the 1950s and that are said to have revolutionised
26 the treatment of mental distress, Lieberman (2016) has proposed that ‘What made
27 chlorpromazine, imipramine, and lithium so different from the sedatives and tranquilizers
28 that came before was that they directly targeted psychiatric symptoms in a kind of
29 lock-and-key relationship’ (p. 188).
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3 The biological dysfunction that is most commonly presented as being implicated in
4 mental distress, a dysfunction which psychiatric drugs are thought to target and correct,
5 are chemical imbalances in the brain. Indeed, the way in which psychiatric drugs exert an
6 influence on the action of a variety of neurotransmitters has been invoked as evidence for
7 the claim that mental distress is a consequence of some form of chemical imbalance. For
8 example, antipsychotic drugs have been found to block the action of the neurotransmitter
9 dopamine and this has been used to formulate, and as evidence to support, the dopamine
10 hypothesis of schizophrenia. While various versions of this hypothesis have been
11 presented, in its most general sense it maintains that the experiences associated with a
12 diagnosis of schizophrenia arise as a consequence of some form of imbalance, and in
13 particular and over-stimulation, of the neurotransmitter dopamine (Howes and Kapur,
14 2009). Similarly, many antidepressant drugs are understood to inhibit the reuptake of
15 serotonin and noradrenaline, both classed as monoamine neurotransmitters, and this has
16 been used to formulate, and as evidence to support, the monoamine hypothesis of
17 depression. In particular, it has been suggested that the experiences associated with a
18 diagnosis of depression are a manifestation of some form of imbalance, and in particular
19 a deficiency, of either serotonin, noradrenaline or a combination of these two
20 neurotransmitters (Hirschfeld, 2000; Mulinari, 2012).

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Despite its pervasiveness both within and beyond the field of mental health care, the
claim that various chemical imbalances are implicated in mental distress, and that
psychiatric drugs work by acting upon these imbalances, has been a matter of ongoing
dispute. Such claims have been subject to sustained critical consideration from a variety
of perspectives with detailed discussions of, for example, the social, economic and

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3 political factors that have contributed to the emergence of this way of understanding
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5 mental distress and the drugs used to respond to that distress (Double, 2011; Lacasse and
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7 Leo, 2015; Whitaker, 2015). Arguably the most fundamental critique, however, is the
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9 suggestion that there is a lack of reliable, replicable and therefore compelling evidence
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11 that any form of biological dysfunction, including any form of chemical imbalance,
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13 underlies the emergence and maintenance of mental distress. With the exception of
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15 ‘organic mental disorders’ or ‘organic brain syndromes’ such as Huntington’s disease, it
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17 has been suggested that a clear and convincing biological cause has not been
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19 demonstrated for the vast majority of ‘psychiatric disorders’ including schizophrenia,
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21 bipolar disorder and depression (Cromby *et al.*, 2013; Deacon, 2013). Therefore, while it
22
23 is common to encounter claims that mental distress is a consequence of some form of
24
25 chemical imbalance, and that psychiatric drugs work by acting upon these imbalances, the
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27 evidence for such claims is said to be unconvincing, misleading or simply non-existent
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29 (Moncrieff, 2008; Gøtzsche, 2013; Lynch, 2015).
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36 To propose that there is no compelling evidence for the assertion that various forms
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38 of chemical imbalances are implicated in mental distress, imbalances that psychiatric
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40 drugs selectively target and address, may strike some as controversial. It seems intuitively
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42 correct to conclude that because psychiatric drugs can have beneficial effects on the
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44 various symptoms associated with mental distress, and because those drugs act on an
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46 individual’s neurochemistry in certain ways, then those symptoms must have been the
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48 result of some form of dysfunction or imbalance in that neurochemistry. However, while
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50 such an argument appears intuitively plausible, the form of thinking that it involves (what
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52 is referred to as *ex juvantibus* reasoning) is insufficient by itself to establish that the
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3 symptoms associated with mental distress are a consequence of a neurochemical
4 dysfunction. There are many instances in which drugs can produce beneficial effects
5 upon the symptoms associated with a variety of conditions, and do so by modifying an
6 individual's biological systems in particular ways, and yet those systems play no part in
7 the emergence of those conditions (Busfield, 2011). For example, aspirin and paracetamol
8 can alleviate the symptoms associated with a variety of conditions including influenza
9 and migraines; however, the fact that they achieve these effects by modifying biological
10 systems in particular ways does not mean that such conditions are caused by dysfunctions
11 or imbalances in the systems upon which aspirin and paracetamol act (Rose, 2006).
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24 The reasons why psychiatric drugs have come to be understood as precise
25 medications that address various forms of chemical imbalance, despite a lack of
26 compelling evidence for such an understanding, is complex and contested. Indeed, the
27 reasons why any one particular approach to mental distress proliferates within and
28 beyond the mental health field cannot simply be attributed to a supposed theoretical and
29 therapeutic superiority over other approaches to mental distress. In any sphere of human
30 inquiry a range of social, political and historical factors contribute to the establishment,
31 maintenance and dominance of certain ways of understanding and responding to human
32 experience while simultaneously delegitimising and excluding alternative ways of
33 understanding and responding to that experience (Foucault, 1981). In mental health care,
34 for example, it has been suggested that the pharmaceutical industry, driven by a financial
35 imperative to maximise profits, has relentlessly employed various marketing strategies to
36 promote chemical imbalance theories of mental distress and the effectiveness of
37 psychiatric drugs to address those imbalances (Whitaker and Cosgrove, 2015). Moreover,
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3 the proliferation of such claims has been attributed to the way in which they provide a
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5 seductively accessible account of how to understand mental distress which has therefore
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7 been readily perpetuated throughout society by the mass media (Boyle, 2015). In addition,
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9 it has been proposed that such claims are politically expedient in so far as they divert
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11 attention away from a consideration of the wider social and material conditions that can
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13 contribute to mental distress as well as the more financially and politically demanding
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15 task of attempting to address those conditions (Boyle, 2011).
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19 One of the most challenging proposals, however, is that mental health professionals
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21 have been instrumental in perpetuating the notion that mental distress is a manifestation
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23 of a neurochemical dysfunction that psychiatric drugs are able to correct. The reasons for
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25 doing so have variously been attributed to an unfamiliarity with the available research on
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27 psychiatric drugs, to deficiencies in the education and training of mental health
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29 professionals and to a desire on the part of practitioners to be perceived as possessing
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31 treatments that are analogous to those used in other branches of medicine (Moncrieff,
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33 2015). However, one of the most disturbing suggestions is that practitioners have actively
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35 employed chemical imbalance theories of mental distress as ‘productive metaphors’ in
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37 order to achieve a range of clinical objectives (Lacasse and Leo 2015). In doing so, it has
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39 been proposed that such explanations have been used to provide people with an
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41 accessible account of the complex, confusing and sometimes disturbing experiences that
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43 can be associated with mental distress. In addition, claims about the precise and targeted
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45 character of psychiatric drugs are said to have been used to reassure people that
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47 practitioners, when they prescribe and administer those drugs, have a sophisticated
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49 understanding of how they work. Moreover, there are even suggestions that health care
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professionals have perpetuated notions that psychiatric drugs address the chemical imbalances of which mental distress is a supposed manifestation in order to facilitate compliance with psychiatric drugs as a response to that distress (Lacasse and Leo, 2015).

The drug-centred model of psychiatric drugs

In so far as the disease-centred model of psychiatric drugs has been subject to sustained criticism from a variety of perspectives, an alternative pharmacological model has been proposed by which to understand the action of those drugs. This alternative understanding of how psychiatric drugs work, what Joanna Moncrieff (2015) has referred to as ‘the drug-centred model’, maintains that those drugs do not correct some form of chemical imbalance that supposedly underlies mental distress. Rather, this drug-centred model proposes that psychiatric drugs are powerful psychoactive substances that act upon the central nervous system to produce various alterations in a person’s thoughts, feelings and behaviour. While the disease-centred understanding of psychiatric drugs has been illustrated by drawing an analogy with the use of insulin in diabetes, the drug-centred understanding has been illustrated by drawing an analogy with the way in which some people use moderate amounts of alcohol to alleviate certain physiological and psychological experiences associated with social anxiety (Moncrieff, 2008). The use of alcohol in such instances is not understood in terms of the disease-centred pharmacological model as somehow targeting and correcting a chemical imbalance that supposedly underlies social anxiety. Rather, alcohol is understood in a drug-centred way as acting upon the central nervous system to produce an altered state and a range of physiological and psychological effects, effects which some people can find beneficial in

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3 helping them deal with the varied experiences associated with social anxiety.
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6 Rather than being precise medications that selectively target and correct some form
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8 of neurochemical dysfunction, the drug-centred model therefore maintains that
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10 psychiatric drugs are imprecise substances or ‘blunt instruments’. In particular, this way
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12 of understanding psychiatric drugs proposes that they are powerful psychoactive
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14 substances that act upon the central nervous system in a non-specific rather than a precise
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16 manner and that by doing so they change or ‘perturb’ normal neurotransmitter function to
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18 produce a range of altered mental and physical states (Hyman and Nestler, 1996;
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20 Whitaker, 2015). Accordingly, while the disease-centred model suggests that psychiatric
21
22 drugs work by returning supposedly imbalanced, dysfunctional and abnormal brain states
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24 back to normal, the drug-centred model maintains that psychiatric drugs produce
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26 abnormal, artificial or intoxicated brain states by disrupting normal neurotransmitter
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28 function (Breggin, 2006; Moncrieff, 2015). In doing so, each psychiatric drug is
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30 understood as producing its own distinctive drug-induced state that has a variety of
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32 physiological and psychological effects, effects that may be experienced by some people
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34 as being helpful in dealing with the various experiences that can be associated with
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36 mental distress. In illustrating this alternative drug-centred understanding of psychiatric
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38 drugs, and how the altered states that those drugs produce may be experienced by some
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40 people as beneficial, Moncrieff (2013) makes it clear that ‘The drug-centred model
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42 suggests that drugs can sometimes be helpful because the features of the altered
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44 drug-induced state superimpose themselves onto the manifestations of distress’ (p. 161).
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52 In further understanding how the drug-centred model accounts for the way in which
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54 psychiatric drugs can produce effects that some people may find beneficial, it is
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3 productive to consider that model within the context of antipsychotic drugs. As a
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5 response to the experiences associated with a diagnosis of schizophrenia, antipsychotics
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7 are not to be understood as correcting some form of neurochemical imbalance such as an
8
9 overstimulation of dopamine. Rather, by blocking the action of dopamine and affecting a
10
11 range of other brain systems, the drug-centred model suggests that antipsychotics create a
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13 drug-induced state similar to Parkinson's disease in which mental and physical activity is
14
15 suppressed – a condition which has variously been referred to as a state of 'deactivation',
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17 'immobilisation' or 'tranquilisation' (Breggin, 1993; Moncrieff, 2008; Healey, 2016).
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19 However, this tranquilised state is not to be thought of as being comparable to a condition
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21 of relaxation and neither should it be thought of as a 'chemical cosh or straightjacket' -
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23 although antipsychotics can produce a state of immobilised sedation if used in high
24
25 enough doses. Instead, antipsychotics are said to produce a state of indifference or
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27 detachment, a 'who cares feeling' that some may find beneficial for the so-called
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29 'positive symptoms' of schizophrenia by suppressing, or making people less responsive
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31 to, the presence of hallucinations, delusions and the anxiety that can accompany them
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33 (Mizrahi *et al.*, 2005; Kapur *et al.*, 2006).
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40 Rather than being a chemical cure that removes the distressing experiences
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42 associated with schizophrenia, the drug-centred model therefore maintains that
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44 antipsychotics produce an altered state in which people can become less concerned by
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46 those experiences. However, although some people may find antipsychotic drugs
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48 beneficial in this way, it is important to recognise that the general suppression of mental
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50 and physical activity that they produce does not selectively target particular experiences
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52 associated with schizophrenia (Barnes, 2011). That is, while creating a distinctive state of
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3 detachment or indifference, antipsychotics do not simply create a sense of indifference
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5 towards the experiences associated with a diagnosis of schizophrenia but can instead
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7 create a sense of indifference towards all experiences. For example, those who use
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9 antipsychotics report that the full range of human emotions can not only come to be
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11 experienced with less intensity, but there can be an emotional ‘flattening’ or ‘blunting’
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13 that is characterised by a lack or absence of emotion (Moncrieff *et al.*, 2015; Healey,
14
15 2016). Similarly, the generalised suppression of mental and physical activity that
16
17 antipsychotics produce can not only impair a person’s attention, memory and general
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19 thought processes, but it can also reduce their motivation to initiate actions, carry out
20
21 simple tasks and engage with others. Indeed, such effects can present as being similar to
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23 the so-called ‘negative symptoms’ of schizophrenia (which include apathy, social
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25 withdrawal and reduced motivation) and both typical and atypical antipsychotics have
26
27 been found to show limited benefit for the treatment of these symptoms (Erhart *et al.*,
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29 2006; Lewis and Lieberman, 2008).
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35 While the drug-centred model illustrates how psychiatric drugs can produce a range
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37 of physiological and psychological effects that some people may find beneficial, it
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39 therefore also helps to understand how those drugs can produce a range of undesirable,
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41 adverse effects. As powerful psychoactive substances that act upon the central nervous
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43 system to alter normal functioning in a non-specific rather than a targeted way,
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45 psychiatric drugs (in a manner analogous to alcohol) can produce a range of effects that
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47 can not only be unpleasant and unhelpful but can also be experienced as intolerable
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49 (Correll *et al.*, 2015). However, while psychiatric drugs can produce a variety of adverse
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51 effects, it has been suggested that mental health professionals may be unaware of the full
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3 range of those effects, minimise their significance for the person taking the drug or
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5 misinterpret such effects as a deterioration in a person's mental health rather than as
6
7 adverse reactions caused by a psychiatric drug (Breggin, 2006; Read, 2009). Indeed, it
8
9 has been proposed that there can be a tendency in mental health settings to attribute a
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11 deterioration in a person's well-being to a relapse or worsening of their 'underlying
12
13 mental disorder' rather than an adverse effect produced by a psychiatric drug; as a
14
15 consequence, there can be a demand to respond to such a perceived deterioration or
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17 relapse by increasing the dosages of psychiatric drugs which, in turn, can increase the
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19 risks of adverse drug reactions (Healey, 2016).
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24 In order to provide informed, safe and effective mental health care that is responsive
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26 to the needs of people who use mental health services, it has been suggested that
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28 practitioners should develop their ability to recognise the range of adverse effects, both
29
30 short-term and long-term, that psychiatric drugs can produce (Healey, 2016). Doing so,
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32 however, can be a considerable challenge. The briefest of investigations will reveal that
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34 psychiatric drugs can produce an extensive and alarming range of adverse effects. These
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36 will not only include predictable effects that are caused by the drugs normal
37
38 pharmacological activity, such as the extrapyramidal effects associated with
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40 antipsychotics or the risk of toxicity associated with lithium. Rather, psychiatric drugs
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42 can produce a range of idiosyncratic, unpredictable effects that result from processes not
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44 yet fully understood and that may go largely unrecognised, such as the serotonin
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46 syndrome associated with antidepressants and selective serotonin reuptake inhibitors in
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48 particular (Ellahi, 2015). To facilitate the reporting and recognition of the range of
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50 adverse effects that can be produced by psychiatric drugs, it has therefore been suggested
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3 that it is necessary to create a clinical climate in which those taking such drugs feel able
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5 to report potential adverse drug reactions (Healey, 2016). Central to achieving this is a
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7 willingness on the part of practitioners to remain open-minded about the possibility that
8
9 any problems reported, even those that are not generally considered to be established or
10
11 predictable adverse effects, may be a consequence of taking a psychiatric drug.
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17 **Psychiatric drug models and service user involvement**

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20 The enduring influence of the disease-centred understanding of psychiatric drugs has
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22 far-reaching implications for those who use mental health services and for how those
23
24 drugs are used as a response to mental distress. In so far as psychiatric drugs have been
25
26 understood as precise medications that target and correct some form of chemical
27
28 imbalance or biological dysfunction, then decisions about those drugs (such as dose,
29
30 duration and discontinuation) have largely come to be seen as technical matters that are
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32 determined by mental health professionals. As a consequence, the experiences and
33
34 opinions of those who are prescribed and take psychiatric drugs can become subordinated
35
36 to the specialist knowledge and judgements of practitioners. While there is an emerging
37
38 interest in equitable, shared decision-making between mental health professionals and
39
40 those who use mental health services, it has been suggested that the meaningful
41
42 involvement of service users in decisions about psychiatric drugs remains, in practice,
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44 limited (Matthias *et al.*, 2012; Kaminskiy *et al.*, 2013; Morant *et al.*, 2016). Indeed, the
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46 relationship between mental health professionals and those who use psychiatric drugs can
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48 become predominately centred around the issue of 'medication adherence'. By
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50 maintaining assumptions about the selective and corrective character of psychiatric drugs,
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3 practitioners can become almost exclusively focused on seeking to ensure that people
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5 experiencing mental distress adhere to the drug regimens that have been formulated for
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7 them (Deegan and Drake, 2006).
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10 By maintaining assumptions about the precise and targeted character of psychiatric
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12 drugs to address some form of supposed neurochemical dysfunction, there can a tendency
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14 to use a range of strategies to ensure medication compliance (Mitchell and Selmes, 2007;
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16 Chapman and Horne, 2013). While recommendations are often made that compliance
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18 should occur in the context of a therapeutic alliance, it has been claimed that there are
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20 instances in which it is secured through the use of various forms of coercion such as
21
22 ‘subtle persuasion’, ‘strategic dishonesty’ and compulsory enforcement of drug treatment
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24 (Seale *et al.*, 2006; Chaplin, 2007). Moreover, in an attempt to ensure medication
25
26 adherence, the adverse effects of psychiatric drugs can be given minimal consideration by
27
28 mental health professionals. For example, when adverse effects are reported by those
29
30 using psychiatric drugs, it has been suggested that some practitioners contest, disbelieve
31
32 or disregard those reports (Seale *et al.*, 2007; Read, 2009). In so far as psychiatric drugs
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34 are understood as precise medications that selectively correct some form of chemical
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36 imbalance, then consideration of the adverse effects of those drugs can become
37
38 subordinated to a powerful assumption about the short-term and long-term effectiveness
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40 of those drugs. Indeed, such an assumption can be understood as finding its expression in
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42 the description of any adverse consequence of taking a psychiatric drug as a ‘side’ effect
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44 that, while potentially ‘unpleasant’, is seen as a tolerable by-product of that drug’s
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46 supposedly beneficial ‘main’ effect of addressing the dysfunctions that are thought to
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48 underlie mental distress (Moncrieff and Cohen, 2009).
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3 In contrast to the disease-centred model of psychiatric drugs, and the manner in
4 which it can contribute to the marginalisation of the experiences of people who take those
5 drugs, the drug-centred model implies a more equitable or democratic relationship
6 between practitioners and mental health service users. Understanding psychiatric drugs
7 as powerful psychoactive substances that act in a non-specific way to produce a range of
8 physiological and psychological effects, the drug-centred model places fundamental
9 importance on the experiences of the person taking a psychiatric drug. Rather than being
10 subordinated to the knowledge and judgements of practitioners, it is the individual
11 experience of the service user that is given priority when deciding whether the range of
12 effects that can be produced by a psychiatric drug are beneficial for the symptoms that
13 comprise that individual's experience of mental distress. Moreover, against understanding
14 any adverse effect as a tolerable by-product of a drug's supposedly beneficial main effect,
15 the drug-centred model implies that all of the effects produced by a psychiatric drug
16 should be taken into account when deciding upon its effectiveness. In doing so, there is
17 an acknowledgement that while some effects may be experienced by one person as
18 beneficial (such as the manner in which the tranquilising effects of antipsychotics may
19 help some people cope with the positive symptoms of schizophrenia) the same or similar
20 effects might be experienced by another person as undesirable, unhelpful and even
21 disabling.

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47 Against the attempt to ensure compliance with a drug-treatment regimen that has
48 been exclusively formulated by mental health professionals, the drug-centred model
49 suggests that decisions about whether a psychiatric drug may be helpful or not is an open,
50 dynamic and collaborative process. In particular, both the mental health service user and
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3 the practitioner draw upon their respective expertise and experience to investigate which
4 effects induced by a psychiatric drug may, and may not, be beneficial. In doing so, there
5 is a recognition of the actual and potential adverse effects of taking a psychiatric drug,
6 both in the short-term and the long-term, such that the decision about whether a drug is
7 helpful or not becomes an ongoing collaborative attempt to balance the actual and
8 potential benefits of a drug against the adverse effects that it may produce. A variety of
9 potential obstacles can be associated with the attempt to establish such equitable
10 relationships, not least the challenge of how to address the imbalances in power,
11 influence and status that exist between those who use and those who provide mental
12 health services (Beresford, 2013; Faulkner *et al.*, 2015). However, understanding
13 psychiatric drugs as powerful, non-selective psychoactive substances not only places a
14 demand on practitioners to develop a more sophisticated understanding of the range of
15 effects that those drugs can produce. Rather, it also prioritises the need to productively
16 engage with service users as legitimate partners in determining the effectiveness of
17 psychiatric drugs as a response to their experience of mental distress.

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19 By emphasising the centrality of the individual experience of taking a psychiatric
20 drug, and the ongoing need to balance the drug-induced effects that a person may find
21 helpful against those effects which they do not, the drug-centred model provides a
22 rationale for what has variously been referred to as the ‘periodic’, ‘strategic’ or ‘flexible’
23 use of psychiatric drugs (Moncrieff and Cohen 2009; Healey, 2016). In doing so, there is
24 a recognition that the continuous, long-term use of psychiatric drugs can produce effects
25 that may inhibit, rather than facilitate, an individual’s recovery from mental distress
26 (Double, 2011; Wunderink *et al.*, 2013; Götzsche *et al.*, 2015). Therefore, a more
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3 effective way to use those drugs may be to employ them when they are needed, such as in
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5 response to a deterioration in a person's mental health or to help a person cope with
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7 challenging life events. Indeed, there is evidence to suggest that some people already use
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9 psychiatric drugs in this flexible or strategic way by increasing, decreasing or omitting
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11 doses in response to variations in the symptoms that comprise their experience of mental
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13 distress (Deegan and Drake 2006; Britten *et al.*, 2010). In the context of an increased
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15 sensitivity to the potentially harmful effects of the long-term use of psychiatric drugs,
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17 their strategic employment is therefore not only concerned with alleviating the symptoms
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19 of mental distress. Rather, it is also simultaneously concerned with ensuring the need to
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21 maximise a person's long-term physiological, psychological and social functioning.
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26 With its emphasis on the flexible and periodic use of psychiatric drugs, the
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28 drug-centred model therefore not only recognises the role that those drugs can have in
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30 helping people deal with the experiences associated with mental distress but it also
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32 highlights their limitations. Rather than being precise medications that target and correct
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34 a supposed biological dysfunction while leaving the rest of the body undisturbed, the
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36 drug-centred model maintains that psychiatric drugs are imprecise or blunt instruments.
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38 While they can produce effects that some people might find helpful, they also produce
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40 short-term and long-term effects that others may find unhelpful, unpleasant and even
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42 intolerable. As such, there might be some who conclude that the experiences associated
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44 with mental distress are less disruptive to their lives than the adverse effects produced by
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46 the drugs that are used to treat such distress. In the context of the drug-centred
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48 understanding of psychiatric drugs, such a decision should not be understood as evidence
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50 that a person 'lacks insight' or that their mental health is deteriorating for which more
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3 aggressive drug-treatment is required. Moreover, it should not simply be assumed that
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5 people who experience mental distress do not possess, or are unable to develop, the
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7 resilience and strategies to cope without psychiatric drugs (Deegan, 2005). Rather, there
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9 should be an acknowledgement that alternative non-pharmacological means do exist and
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11 practitioners ought to provide the information, support and opportunity for people to
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13 explore such means alongside, or even as an alternative to, the use of psychiatric drugs.
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19 **Conclusion**

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22 Assumptions about the way in which psychiatric drugs are thought to work have
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24 important implications for those who use mental health services. While such assumptions
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26 may be infrequently articulated they can influence the way in which those drugs are used,
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28 the manner in which their potential benefits are balanced against their adverse effects and
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30 the extent to which those who use psychiatric drugs are meaningfully involved in
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32 decisions about their use. In the context of the widespread and enduring disease-centred
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34 model of psychiatric drugs, those drugs are commonly understood as selective agents that
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36 address the various forms of biological dysfunction that supposedly underlie the
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38 emergence and maintenance of mental distress. As such, there can be a tendency to view
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40 psychiatric drugs as predominately being a technical matter in which decisions about their
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42 use are largely determined by the specialist knowledge and judgements of practitioners.
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45 As a consequence of sustained criticism of the disease-centred model of psychiatric drugs,
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47 an alternative pharmacological model has been proposed by which to understand the
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49 mode of action of those drugs. In the context of this alternative drug-centred model,
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52 psychiatric drugs are understood as powerful psychoactive substances that act upon the
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3 central nervous system in a non-specific rather than a targeted way to produce a range of
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5 physiological and psychological effects. As such, this model places increased importance
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7 on investigating which of these effects a person may helpful and which they do not and,
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9 in doing so, implies that decisions about psychiatric drugs should occur within the context
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11 of an open, dynamic and collaborative relationship with those who use mental health
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13 services.
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