

## INVITED REVIEW

# The pharmacology of psilocybin

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### Abstract

*Psilocybin (4-phosphoryloxy-N,N-dimethyltryptamine) is the major psychoactive alkaloid of some species of mushrooms distributed worldwide. These mushrooms represent a growing problem regarding hallucinogenic drug abuse. Despite its experimental medical use in the 1960s, only very few pharmacological data about psilocybin were known until recently. Because of its still growing capacity for abuse and the widely dispersed data this review presents all the available pharmacological data about psilocybin.*

### Introduction

Psilocybin-containing mushrooms are one of the major hallucinogenic drugs of abuse today. These mushroom species are distributed worldwide<sup>1</sup> and their abuse potential produces partially harmful effects in a growing population of psychedelic drug users.<sup>2</sup> No physical damage but many psychiatric complications have been reported worldwide.<sup>3</sup> Recent research has been reported on the treatment of compulsive disorders in humans with psilocybin;<sup>4</sup> therefore, it is important to know the essential pharmacological data about psilocybin.

Despite the fact that pure synthetic psilocybin (Indocybin® Sandoz) was used and marketed for experimental and psychotherapeutic purposes in the 1960s, until recently only limited pharmacological data were available. In recent years some experimental psychophysiological studies were performed in which human pharmacokinetic and

pharmacodynamic data of psilocybin were explored further.<sup>5–10</sup> Because of the widely dispersed material about the pharmacological properties of psilocybin, old and new data are reviewed here. It should be noted that characterization of the complex psychopathological phenomena induced is not in the focus of this review.

### Pharmacology of psilocybin

Psilocybin (4-phosphoryloxy-N,N-dimethyltryptamine) is a substituted indolealkylamine and belongs to the group of hallucinogenic tryptamines. Psilocybin was isolated from Central American mushrooms (*Psilocybe mexicana*) by the renowned Swiss chemist Albert Hofmann in 1957, and in 1958 was produced synthetically for the first time.<sup>11</sup> It has been found in many species of mushrooms worldwide<sup>1</sup> (Fig. 1).

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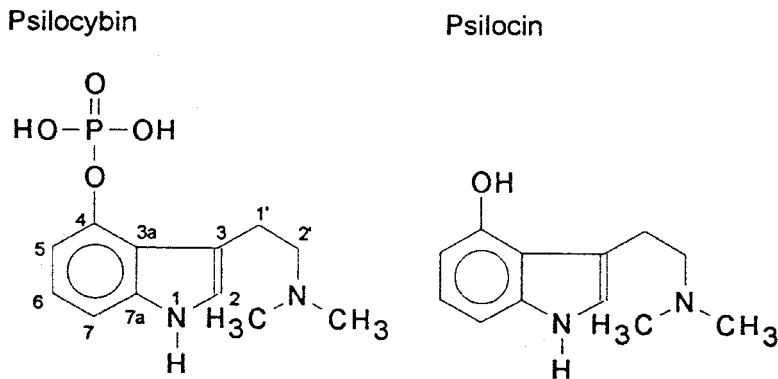


Figure 1. Molecular structures of psilocybin and psilocin.

### Psychic effects

In a medium dosage (12–20 mg p.o.), psilocybin was found to produce a well-controllable altered state of consciousness. This state is marked by stimulation of affect, enhanced ability for introspection and altered psychological functioning in the direction of Freudian primary processes, known otherwise as hypnagogic experience and dreams.<sup>12</sup> Especially noteworthy are perceptual changes such as illusions, synaesthesias, affective activation, and alterations of thought and time sense. The effects last from 3 to 6 hours.

After extensive tests in animals and humans, psilocybin was distributed worldwide under the name Indocybin® (Sandoz) as a short-acting and more compatible substance (than, for example, LSD) to support psychotherapeutic procedures.<sup>13</sup> Experimental and therapeutic use was extensive and without complications.<sup>14</sup>

### Somatic effects

Cerletti<sup>15</sup> reported an LD<sub>50</sub> for mice with intravenous application of 280 mg/kg which may imply an LD<sub>50</sub> of some grams of psilocybin in humans. In some *in vitro* experiments, except for an inhibitory effect on the neurotransmitter serotonin, psilocybin showed no specific effects on isolated organs (intestines, heart) of guinea pigs and rats.<sup>15</sup> Characteristic autonomic effects of the neurovegetative system that were notable for the whole animal (mice, rats, rabbits, cats and dogs) with doses of 10 mg/kg s.c. included: mydriasis, piloerection, irregularities in heart and breathing rate and discrete hyperglycaemic and hypertonic effects.<sup>15</sup> Cerletti interpreted these effects as an

excitatory syndrome caused by central stimulation of the sympathetic system. In contrast to an autonomic excitatory syndrome, motor behaviour was muted.<sup>16–18</sup> Experiments with Rhesus monkeys (2–4 mg/kg i. p.) confirmed the above changes of physiological parameters and a central excitatory syndrome. After 20–40 minutes the EEG showed a disappearance of alpha activity and an increase of beta activity in the neocortex.<sup>19</sup> In two early non-blind studies in healthy volunteers ( $n = 12$ , 0.12–0.15 mg/kg p.o.),<sup>20</sup> ( $n = 22$ , 10 mg p.o.)<sup>21</sup> the EEG showed variations of visual evoked potentials and decrease in alpha and theta frequencies. There were no changes in the electroretinogram.<sup>21</sup>

The somatic effects in humans were investigated first by Quetin<sup>22</sup> in a non-blind study in healthy volunteers ( $n = 29$ , 8–12 mg p.o., i.m.). The physiological changes which were noted regularly are listed in Table 1. These effects were confirmed qualitatively by another early non-blind study ( $n = 16$ , 0.11 mg/kg p.o.).<sup>33</sup> Discrete changes of RR and pulse were also confirmed in a recent double-blind placebo-controlled study ( $n = 8$ , 0.2 mg/kg p.o.), as shown in Table 2.<sup>9</sup> The effects described were barely noticeable and should be interpreted as secondary pharmacological effects, induced mainly by the sympathomimetic excitation syndrome.<sup>24</sup> Hollister *et al.*<sup>25</sup> found no significant aberrations of the aforementioned parameters in one subject after administration of psilocybin for 21 consecutive days with increasing dosages (1.5 mg increased to 25 mg p.o. in three doses per day). Electrolyte levels, liver toxicity tests and blood sugar levels remained unaffected.<sup>23–25</sup> Human leucocytes

**Table 1.** Somatic symptoms

	Percentage of subjects
Midriasis	93%
Heart frequency	
Accelerated	56%
Slowed	13%
Variable	31%
No change	0%
Arterial blood pressure	
Hypotension	34%
Hypertension	28%
Instability	22%
No change	16%
Nausea	44%
Reflexes tendineae	
Increased	80%
Decreased	6%
No change	13%
Dysmetry	16%
Tremor	25%

Modified from ref. 22.  $N = 30$ , 8–12 mg psilocybin i.m., p.o.

were found by Quetin<sup>23</sup> ( $n = 29$ , 8–12 mg p.o., i.m.) and Hollister *et al.*<sup>25</sup> ( $n = 16$ , 0.06–0.2 mg/kg p.o., s.c.) to be reduced in number temporarily between the second and fourth hour after psilocybin. In a recent double-blind placebo-controlled study ( $n = 8$ , 0.2 mg/kg p.o.) endocrine activity (cortisol, prolactin, growth hormone) was found not to be affected significantly by psilocybin.<sup>9</sup>

Experiments in mice (4, 8 and 16 mg/kg) with the micronucleus test, highly sensitive to the chromosome-breaking potential of substances, found no evidence for genetic aberrations through psilocybin.<sup>26</sup> In mutagenicity testing it is not possible at present to prove the mutagenic potential of a compound in a single test system. Results of other tests are required to confirm these negative results.

### Pharmacokinetics

Pharmacokinetic studies showed that 50% of <sup>14</sup>C-labelled psilocybin was absorbed following oral administration. The isotope is distributed almost uniformly throughout the whole body.<sup>27,28</sup> As part of a recent double-blind placebo-controlled psychopathological study ( $n = 13$ , 0.2 mg/kg p.o.), Holzmann<sup>5</sup> assayed psilocybin metabolites in human plasma and urine by HPLC as part of an investigation of the

**Table 2.** Blood pressure and heart rate changes

	Mean/SD
Systolic blood pressure (mmHg)	25.9 ± 11.7
Diastolic blood pressure (mmHg)	10.0 ± 7.6
Heart rate	10.4 ± 12.6

From ref. 9.  $N = 8$ , 0.2 mg/kg psilocybin p.o.

pharmacokinetics of psilocybin and psilocin. In another recent double-blind placebo-controlled study ( $n = 6$ , 0.5–3 mg i.v.;  $n = 6$  0.22 mg/kg p.o.) Hasler *et al.*<sup>6</sup> used HPLC with column-switching coupled with the electrochemical detection procedure for reliable quantitative determination of psilocybin metabolites. Altogether, four metabolites of psilocybin have been identified (Fig. 2):

- 4-hydroxy-N,N-dimethyltrypt-amine (Psilocin);
- 4-hydroxyindole-3-yl-acetaldehyde (4H1A);
- 4-hydroxyindole-3-yl-acetic-acid (41-IIAA); and
- 4-hydroxytryptophol (41-IT).

According to the two above-mentioned pharmacokinetic studies in humans it was found that after oral administration (on an empty stomach), psilocybin is detectable in significant amounts in the plasma within 20–40 minutes. Psychological effects occur with plasma levels of 4–6  $\mu\text{g/ml}$ .<sup>5,6</sup> The threshold dose depends on interindividual differences, but may be in the range of 3–5 mg p.o. for a subjectively detectable sympathomimetic, but not hallucinogenic, effect as found in double-blind placebo-controlled trials.<sup>29</sup> The full effects occur with doses of 8–25 mg p.o. within 70–90 minutes. Psilocin appears in the plasma after 30 minutes. A significant first-pass effect with the vast majority of psilocybin converted into psilocin mainly by hepatic metabolism can be assumed.<sup>29</sup> Another early biochemical study showed psilocin to be the main, if not the solely pharmacologically active substance by decreasing the dephosphorylation of psilocybin to psilocin using a competitive substrate (beta-glycerophosphate) for blocking the alkaline phosphatase.<sup>30</sup> Recent experimentation on rodent tissue presented more evidence for complete conversion of psilocybin to psilocin before entering systemic circulation.<sup>31</sup> This assumption is also supported

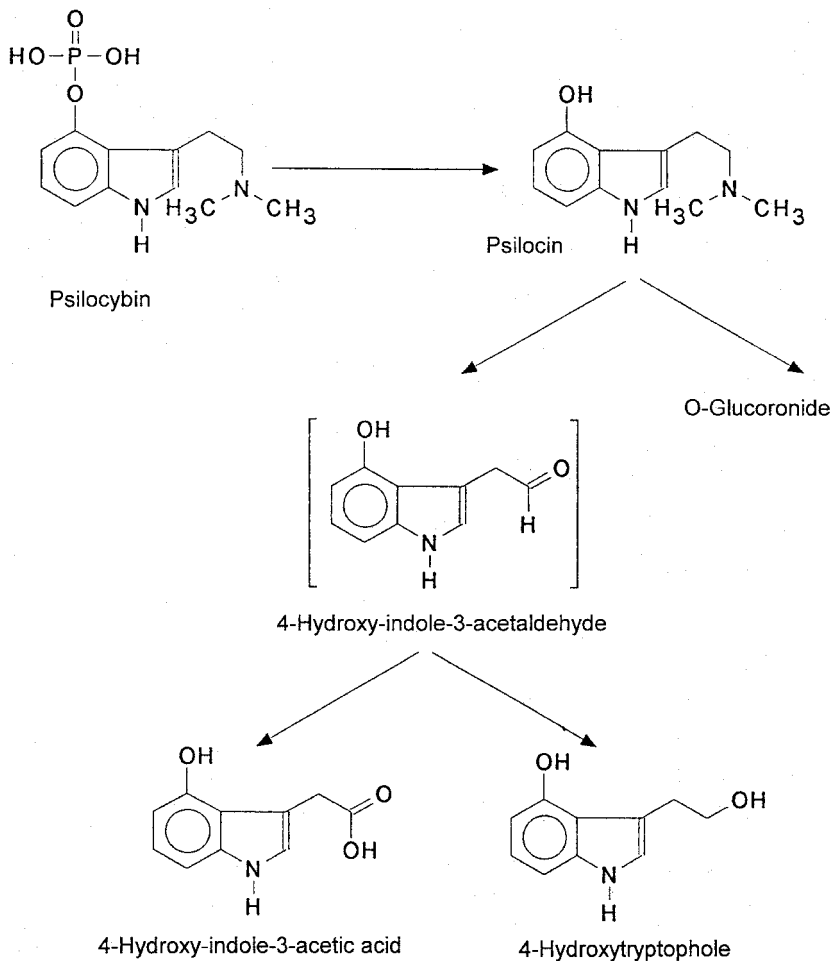


Figure 2. Metabolism of psilocybin.

by the finding that equimolar amounts of psilocybin and psilocin evoke qualitatively and quantitatively similar psychotropic effects in humans.<sup>32</sup> Psilocybin could therefore be referred to as a prodrug. However, because of the lack of reliable analytical methods for the determination of psilocybin in human plasma, it was not possible to prove this assumption by showing the absence of the parent drug in plasma after psilocybin administration. After a rapid increase of psilocin plasma levels a plateau of about 50 minutes follows, after which there is a relatively slow decline of the curve, ending at about 360 minutes. This is confirmed by the subjective impressions of the subjects and Leuner's diagram of the clinical course (Fig. 3).<sup>13</sup> An interesting fact may be the much shorter half-life (mean  $74.1 \pm 19.6$  minutes

i.v. compared to  $163 \pm 64$  minutes p.o.) and duration of action (subjective effects lasting only 15–30 minutes) when psilocybin is given intravenously, as performed in a recent double-blind placebo controlled trial.<sup>29</sup>

Despite weight-specific dosage used in recent human studies, the plasma concentration-time curves indicate highly variable plasma concentrations. However, the timing of the maximum plasma concentration is after approximately 80 minutes (Fig. 4).<sup>6</sup>

The elimination of glucuronidated metabolites as well as unaltered psilocybin (3–10%) was found to occur through the kidneys. Approximately two-thirds of the renal excretion of psilocin is completed after 3 hours, but with great interindividual differences. The mean elimination

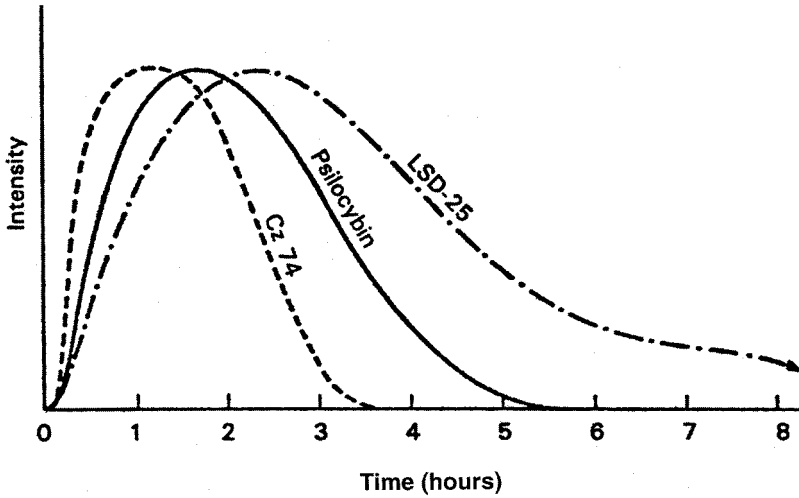


Figure 3. Course of clinical effects of LSD, psilocybin and CZ-74 (a psilocybin-derivative).<sup>13</sup>

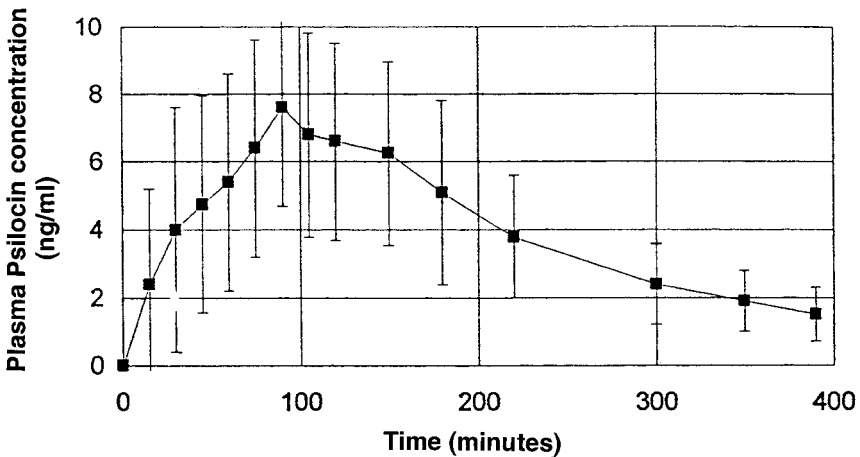


Figure 4. Time-course of plasma levels for psilocin after 0.224 mg/kg body weight psilocybin p.o. ( $n=6$ ).<sup>6</sup>

half-life of psilocin is 50 minutes (Fig. 5, Table 3).<sup>5</sup>

In two early single-blind randomized comparative studies a dose of 100  $\mu$ g psilocybin was reported as equivalent to 1  $\mu$ g LSD and 1000  $\mu$ g mescaline.<sup>33,34</sup> Even though significant tolerance is known to occur with repeated use of psilocybin, the development of physical dependence does not occur.<sup>35,36</sup> Other early single-blind experiments showed cross-tolerance of psilocybin and LSD.<sup>37,38</sup>

#### Pharmacodynamics

Two recent double-blind placebo controlled PET (positron emission tomography) studies using [F-18]-fluorodeoxyglucose showed brain metabolic activation under the influence of psilocybin. Gouzoulis *et al.*<sup>8</sup> ( $n=8$ , 0.20 mg/kg p.o.) found no increase of global brain metabolism, while Vollenweider *et al.*<sup>7</sup> ( $n=15$ , 0.26 mg/kg p.o.) found a general increase of cortical metabolism. Vollenweider *et al.* found increased metabolism bilaterally in the frontomedial and frontolateral

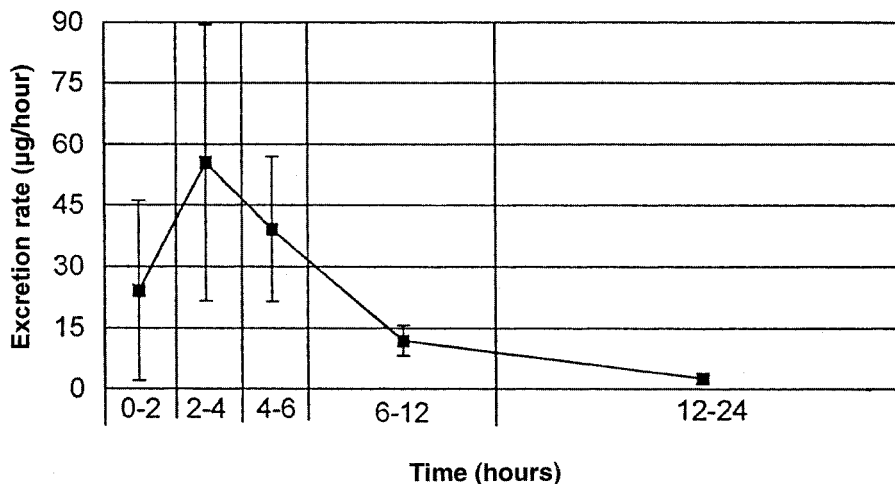


Figure 5. Mean urine excretion rate of psilocin after 0.224 mg/kg psilocybin p.o. ( $n=8$ ).<sup>6</sup>

cortex (24%), as well as in the anterior cingulate gyrus (25%), the temporal-medial cortex (25%) and the basal ganglia (19%). The smallest increases were found in the sensorimotor (15%) and the occipital cortex (14%). Furthermore, an increase of the frontal-occipital metabolic gradient occurs.<sup>7</sup> Regional activation was especially high in the right hemispheric frontotemporal cortical regions and decreased in the thalamus.<sup>8</sup>

Psilocybin interacts mainly with serotonergic neurotransmission (5-HT1A, 5-HT1D, 5-HT2A and 5-HT2C receptor subtypes). It binds with high affinity at 5-HT2A ( $K_i = 6$  nM) and to a lesser extent at 5-HT1A ( $K_i = 190$  nM) receptors.<sup>39</sup> It should be noted that psilocybin and its active metabolite psilocin have—in contrast to the indoleamine LSD—no affinity for dopamine D2 receptors.<sup>40</sup> A recent double-blind placebo-controlled study ( $n=15$ , 0.25 mg/kg p.o.) with ketanserin pre-treatment (20 mg/40 mg p.o.) showed that the psychotomimetic effects of psilocybin can be blocked completely using the preferential 5HT2A receptor antagonist ketanserin.<sup>41</sup> It is probable, therefore, that the effects of psilocybin are mediated mainly via activation

of presynaptic 5HT2A receptors. However, pre-treatment with the D2 receptor antagonist haloperidol also reduces psilocybin-induced psychotomimesis, which raises the possibility that psilocybin-induced psychotomimesis is a secondary response to increased dopaminergic transmission, as demonstrated recently in a double-blind placebo-controlled PET study in humans ( $n=7$ , 0.25 mg/kg p.o.) using the D2-receptor ligand [11C] raclopride.<sup>42</sup> Functional interactions of central dopaminergic and serotonergic systems have been well demonstrated.<sup>43,44</sup>

In experiments with rats, Aghajanian<sup>45</sup> showed psilocybin to interact mainly with serotonin receptors of the dorsal raphe nucleus. Because of its inhibiting influence on neurones of the dorsal raphe nucleus an activation of noradrenergic neurones of the nearby locus coeruleus is induced. The locus coeruleus represents a major center for the integration of sensory input. This may explain some forms of perceptual alterations such as synaesthesias. Another hypothesis generated in the course of recent human studies with psilocybin assumed that alterations of different feedback-loops between cortex and thalamus are

Table 3. Pharmacokinetic parameters of psilocin, the active metabolite ( $N=8$ , 0.224 mg/kg psilocybin p.o.)<sup>6</sup>

	$C_{max}$ [ng/ml plasma]	$t_{max}$ [min]	$AUC_{0-\infty}$ [ng min/ml]	$t_{1/2}$	Fabs [%]
Mean(SD)	8.2 (2.8)	105 (37)	1963 (659)	163.3 (63.5)	52.7 (20.4)

responsible for an "opening of the thalamic filter for sensory input" as the cause of the psilocybin induced frontal hyperfrontality, as shown in PET studies.<sup>7</sup>

The evidence reviewed suggests psilocybin to exhibit low toxicity and may be seen as physiologically well tolerated. However, most studies are old and do not meet contemporary standards for safety studies. In particular, properly conducted safety pharmacology studies are lacking. Complications may result mainly from its psychotomimetic effects in vulnerable individuals, especially under uncontrolled conditions.

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