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Effects of Intraocular Mescaline and LSD on Visual-Evoked Responses in the Rat[1]

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EELLS, J. T. AND D. M. WILKISON. *Effects of intraocular mescaline and LSD on visual-evoked responses in the rat.* PHARMACOL BIOCHEM BEHAV 32(1) 191–196, 1989.—The effects of mescaline and LSD on the flash-evoked cortical potential (FEP) were determined in unrestrained rats with chronically-implanted electrodes. Systemic administration of mescaline or LSD significantly attenuated the primary component of the FEP at three stimulus intensities with the greatest effect observed 60–90 minutes following drug administration. The magnitude and specificity of the effects of these agents on the primary response suggest that they produce deficits in conduction through the retino-geniculato-cortical system. The serotonin receptor antagonists, cyproheptadine and methysergide, antagonized the mescaline-induced depression of the FEP in accordance with neurochemical and behavioral evidence that mescaline acts as a partial agonist on serotonin receptors. Topical or intraocular administration of atropine antagonized the actions of systemically-administered mescaline. In addition, intraocular administration of mescaline or LSD attenuated the FEP indicative of an action of these hallucinogens on visual processing in the retina which is modulated by muscarinic receptor activity.

Mescaline LSD Visual-evoked potential Retina Rats

MESCALINE and lysergic acid diethylamide (LSD) are potent psychoactive agents which produce profound alterations in sensory perception including visual hallucinations (17,26). The clinical effects of the phenethylamine-derived hallucinogen, mescaline, and the indoleamine-derived hallucinogen, LSD, are quite similar, however, differences have been reported in their actions on visual perception and sensory integrative function (6,18). The mechanisms by which these drugs produce visual distortions are not understood, although LSD has been shown to alter electrical activity and the propagation of information at several levels of the visual system including the retina, optic tract, lateral geniculate nucleus, and visual cortex (5, 14, 23). Less information is available on the actions of mescaline on visual processing (20). Behavioral studies have suggested that serotonergic mechanisms are involved in the perceptual modifications induced by mescaline and LSD and recent evidence suggests that these drugs act as partial agonists at postsynaptic serotonin receptors (10, 12, 19). We have investigated the effects of systemic and intraocular administration of mescaline and LSD in unrestrained awake rats using the flash-evoked cortical potential as a measure of net functional neuronal activity in the visual system. We have also investigated the involvement of serotonergic and retinal cholinergic systems in the actions of the phenethylamine hallucinogen, mescaline.

METHOD

Long-Evans hooded rats were anesthetized with pentobarbital (60 mg/kg, IP) and epidural screw electrodes were stereotaxically implanted over the right and left visual cortices at points 7 mm posterior to bregma and 4 mm lateral to the midline. Ground and reference electrodes were placed 2 mm anterior and 2 mm lateral to bregma. All electrodes were attached to an Amphenol connector and the entire assembly was secured to the skull with dental acrylic.

Recording sessions were initiated after a one-week recovery period. Rats were placed into a shielded cage with mirrored panels on the sides and the floor. The animals were dark-adapted for 20 minutes and the averaged flash-evoked potential (FEP) to 50 flashes, 10 μsec in duration presented at 0.4 Hz, was obtained at each of 3 different flash intensities. The flashes were delivered by a Grass photostimulator (PS22) at luminesce intensity settings 1, 4 and 16 which roughly corresponds to 9.4×10^4, 3.8×10^5 and 1.5×10^6 candlepower, respectively. Input/output curves at the three flash intensities were established prior to drug administration and at defined intervals after drug administration up to 3 hours.

Analysis of evoked potentials followed previously described digital methods (24,25). Responses to 50 flashes were averaged to obtain peak amplitudes, their latencies and their

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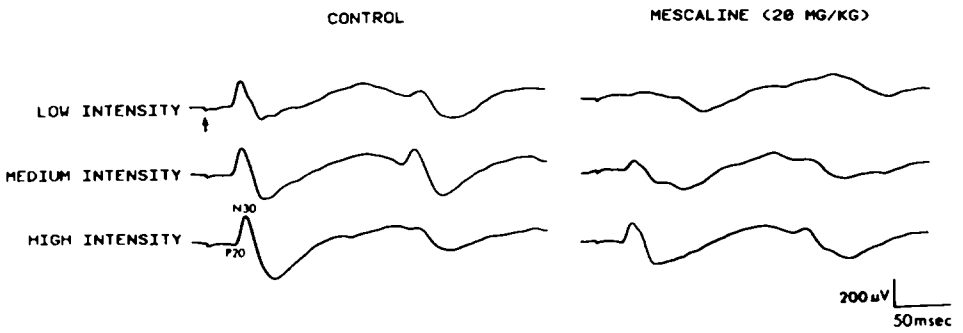

FIG. 1. The effect of mescaline (20 mg/kg) on the flash-evoked potential in the awake rat. The averaged FEP consisted of a primary (P20–N30) component and a more variable secondary (P50–N70–P100) component. Mescaline (20 mg/kg, IP) produced a marked attenuation of the primary (P20–N20) component of the FEP at low and medium stimulus intensities 60 min after administration when compared with baseline measurements.

standard deviations. Amplitude measurements were made in microvolts from the peak of the negative component of the P20–N30 complex of the FEP. Drug effects were calculated as percentage of the baseline amplitude of this component and averaged across animals at each flash intensity.

Mescaline and LSD were obtained from the National Institute on Drug Abuse. Cyproheptadine and methysergide were supplied by Merck Sharp and Dohme (West Point, PA) and Sandoz Pharmaceuticals (Hanover, NJ), respectively. Phenylephrine hydrochloride (2.5% ophthalmic solution) was obtained from Sterling Drug Inc. (New York, NY) and atropine sulfate from Sigma Chemical Company (St. Louis, MO). Mescaline, LSD, cyproheptadine and methysergide were dissolved in saline and administered intraperitoneally in volumes to 0.5 ml. Rats received either cumulative 5–20 mg/kg or single (20 mg/kg) doses of mescaline. In cumulative dose experiments, mescaline was administered in 3 doses given at 45-minute intervals and responses were measured 30 minutes after drug administration. LSD was administered at a single dose of 50 μg/kg. Cyproheptadine (1 mg/kg) or methysergide (1 mg/kg) was administered 30 min prior to mescaline. Atropine sulfate (1%) or phenylephrine (2.5%) was administered optically, 1 drop in each eye. For intraocular administration, LSD (5 ng/μl), mescaline (10 μg/μl) or atropine sulfate (1%) was microinjected (1 μl) into the vitreous humor in both eyes under halothane anesthesia. In these experiments intravitreous saline administration (1 μl) served as a vehicle control.

Statistical determinations used analysis of variance with repeated measures and Student's *t*-test. The null hypothesis was rejected at $p<0.05$.

RESULTS

Effects of Mescaline and LSD on the FEP

Mescaline and LSD were administered systemically (IP) and effects were evaluated for 3 hours after drug administration. Both mescaline and LSD depressed the flash-evoked cortical potential (FEP). The average FEP illustrated in Fig. 1 consisted of a primary (P20–N30) component and a more variable secondary (P50–N70–P100) component. The voltage of the negative deflection of the P20–N30 wave was 50–200 μV with a peak latency of 25–30 msec. The effect of 20 mg/kg mescaline on flash-evoked activity is also shown in Fig. 1.

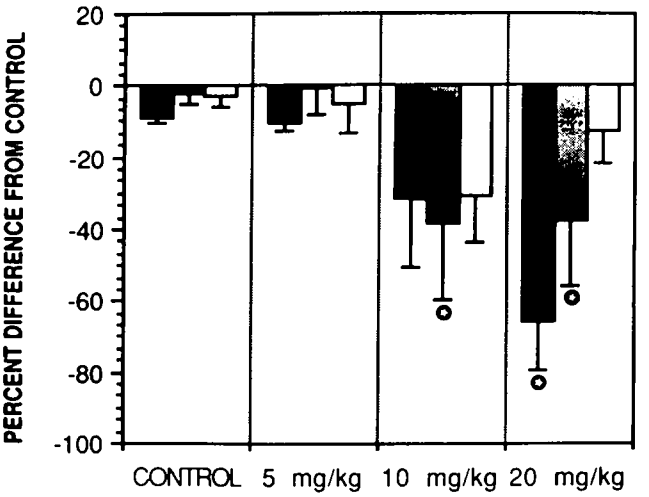

FIG. 2. The effect of mescaline on the FEP. FEP's were measured at 3 flash intensities (1, 4 and 16; Grass PS22 photostimulator) prior to drug administration and 30 minutes after each dose of mescaline. Rats received cumulative doses of mescaline (5–20 mg/kg, IP). Data are expressed as the percent difference from mean baseline (control) values for three levels of flash intensity. Mescaline significantly reduced the amplitude of the P20–N30 primary component of the FEP (☆), $p<0.05$. Solid bars: low; shaded bars: mid; open bars: high.

Mescaline significantly attenuated the amplitude of the primary component of the FEP at all three stimulus intensities with the greatest effect observed at low luminesce intensities. Figure 2 shows the effect of mescaline on the amplitude of the P20–N30 component of the FEP after the administration of cumulative doses of mescaline. In these experiments, mescaline was administered in a cumulative fashion over 3 doses from 5–20 mg/kg (N=5). Responses were measured 30 minutes after drug administration and the dosing interval was 45 minutes. Significant reductions in FEP amplitude at the low and medium flash intensities were observed following the 10 and 20 mg/kg doses. The time course of the effect of a single dose of mescaline (20 mg/kg; N=6) or LSD (50 μg/kg; N=6) on the FEP amplitude is shown in Fig. 3.

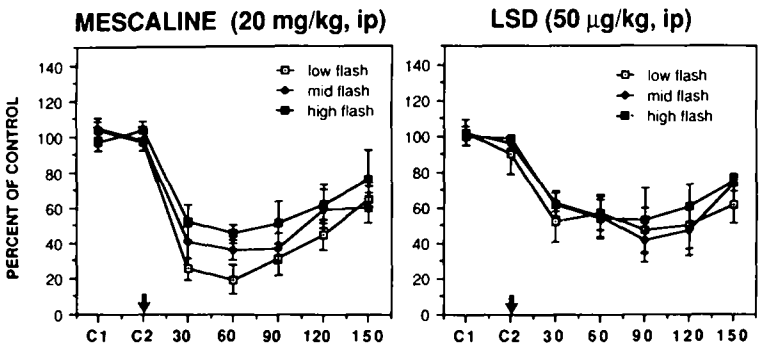

FIG. 3. Time course of a single dose of mescaline or LSD on the FEP. Rats received a single injection of mescaline (20 mg/kg, IP) or LSD (50 μg/kg, IP) and the FEP was determined at 30-minute intervals for 150 minutes after drug administration. Solid arrows indicate the time of drug administration. Data are expressed as percent of the mean baseline (control) value for three levels of flash intensity. Mescaline significantly attenuated the amplitude of the P20–N30 primary component of the FEP at all 3 stimulus intensities [$F(6,36)=25.2$, 17.9, 4.5; low to high intensity respectively: $p<0.001$]. LSD produced similar effects [$F(6,18)=5.9$, 6.8, 4.5; low to high intensity respectively; $p<0.001$].

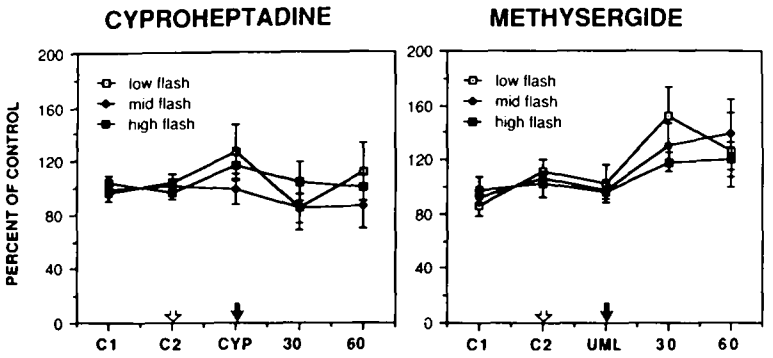

FIG. 4. Effects of Serotonin Antagonists, alone and in combination with mescaline, on the FEP. Cyproheptadine (CYP) or methysergide (UML) were administered (1 mg/kg, IP) as indicated by open arrows and FEPS were measured 30 minutes later (CYP, UML). Rats then received a single injection of mescaline (20 mg/kg) as indicated by solid arrows and the FEP was determined 30 and 60 minutes after mescaline administration. Data are expressed as percent of mean baseline (control) values for three levels of flash intensity.

Both mescaline and LSD significantly attenuated the amplitude of the primary component of the FEP at all three stimulus intensities [Mescaline: $F(6,36)=25.2$, 17.9, 7.6, respectively, $p<0.001$; LSD: $F(6,18)=5.9$, 6.8, 4.5, respectively, $p<0.01$]. Maximal reduction of the FEP amplitude occurred approximately 60 minutes after the administration of mescaline ($65\pm5\%$ reduction at medium flash intensity) or LSD ($50\pm11\%$ reduction at medium flash intensity) and returned toward baseline values by two hours. The P20–N30 component of the FEP recovered to baseline values by 3 hours after the administration of mescaline or LSD.

Effects of Serotonin Antagonists on the FEP

The effect of the serotonin antagonists, cyproheptadine and methysergide, alone and in combination with mescaline on the FEP in the rat were determined. Rats were administered cyproheptadine (1 mg/kg, IP; N=6) or methysergide (1 mg/kg, IP; N=6) and the FEP was recorded 30 min later. Animals then received a single injection of mescaline (20 mg/kg,) and the FEP was again measured 30 and 60 minutes after administration of mescaline. The data in Fig. 4 show that cyproheptadine and methysergide had no effect alone on the P20–N30 component of the FEP and that both of these agents antagonized the mescaline-induced reduction of the P20–N30 component of the FEP. FEP amplitudes at all three flash intensities were significantly different in cyproheptadine-pretreated rats ($p<0.01$) and methysergide-pretreated rats ($p<0.05$) compared with animals receiving only mescaline.

Effects of Atropine and Phenylephrine on the FEP

Doses of mescaline and LSD which produce visual perceptual distortions in humans also produce mydriasis (26).

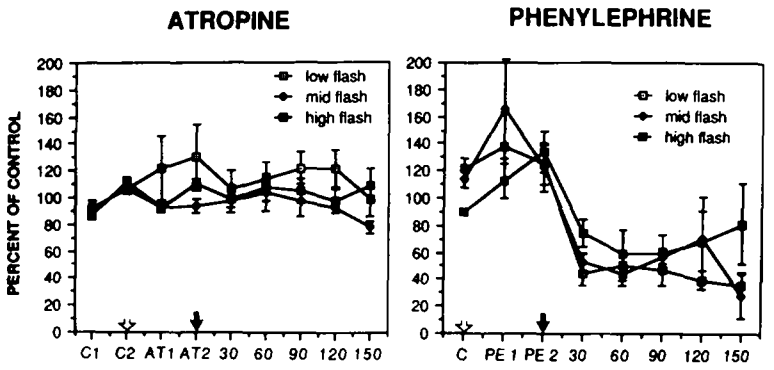

FIG. 5. Effects of topical atropine or phenylephrine alone and in combination with mescaline on the FEP. A solution of 1% atropine sulfate (AT) or 2.5% phenylephrine HCl (PE) was topically applied as indicated by open arrows to induce mydriasis. FEPS were measured 20 (AT1 and PE1) and 40 minutes (AT2 and PE2) following atropine or phenylephrine administration. Rats then received a single injection of mescaline (20 mg/kg) as indicated by solid arrows and the FEP was determined at 30, 60, 90, 120 and 150 minutes after mescaline administration. Data are expressed as percent of mean baseline (control) values for three levels of flash intensity.

Mydriasis was observed following the administration of mescaline (20 mg/kg) or LSD (50 μg/kg) in the present study to determine if the mydriatic actions of mescaline contributed to the observed reduction in FEP; the effects of two mydriatic agents, atropine and phenylephrine, alone and in combination with mescaline on the FEP were examined. Atropine (1% ophthalmic solution; N=6) and phenylephrine (2.5% ophthalmic solution; N=6) were topically applied to induce mydriasis and the FEP was measured at 20 minutes and again at 40 minutes following either drug. Pupillary dilation was observed in all animals within 5 minutes of drug administration. Animals then received a single injection of mescaline (20 mg/kg, IP) and the FEP was recorded at 30-minute intervals to 150 minutes.

The data in Fig. 5 show that atropine and phenylephrine did not alter the amplitude of the P20–N30 component of the FEP at any level of flash intensity. A slight, but nonsignificant increase in FEP amplitude was observed at low luminescence intensities with both agents. The mescaline-induced reduction in the FEP was not affected by phenylephrine. However, atropine antagonized the action of mescaline abolishing the reduction in FEP amplitude produced by the hallucinogen. To determine if atropine was acting on the retina to antagonize the effects of mescaline, studies were conducted in which atropine was administered intraocularly followed by systemic administration of mescaline. The data in Fig. 6 show that intraocular administration of atropine (1 μl of 1%) also antagonized the actions of systemically-administered mescaline (20 mg/kg, IP). FEP's in these experiments were measured at medium flash intensity.

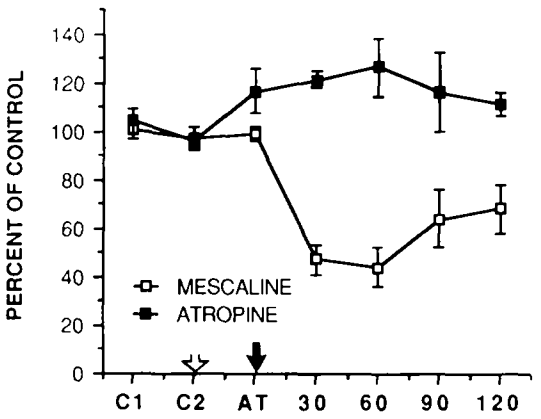

FIG. 6. Effects of intraocular administration of atropine alone and in combination with mescaline on the FEP. Atropine sulfate (1%) (ATROPINE) or saline (MESCALINE) was administered by injection (1 μl) as indicated by the open arrow into the vitreous humor of both eyes of rats anesthetized with halothane. Animals were allowed to recover and the FEP was determined 30 minutes later (AT). Rats then received a single injection of mescaline (20 mg/kg, IP) as indicated by the solid arrow and the FEP was measured at 30, 60, 90, and 120 min after mescaline administration. Data are expressed as percent of mean baseline (control) values at medium flash intensity. Mescaline significantly reduced the amplitude of the P20–N30 component of the FEP in saline-pretreated rats as compared with atropine-pretreated rats, $F(1,6)=15.5$, $p<0.01$.

Effects of Intraocular Injection of Mescaline and LSD on the FEP

Mescaline and LSD were administered bilaterally into the vitreous humor of the eye to assess their actions on retinal components of the photically-evoked cortical potential. As shown in Fig. 7, both drugs attenuated the primary component of the FEP. Intraocular injection of mescaline (10 μg; N=6) or LSD (5 ng; N=6) significantly reduced the amplitude of the FEP at medium flash intensity as compared with saline control [$F(1,6)=16.3$ for mescaline ($p<0.01$) and $F(1,6)=11.5$ for LSD ($p<0.01$)]. The FEP was recorded only at medium flash intensity in these experiments to enable data collection at 10-minute intervals. Maximal reduction in the FEP amplitude occurred between 10–30 min after intraocular administration of mescaline or LSD shifting towards baseline values by 90 min.

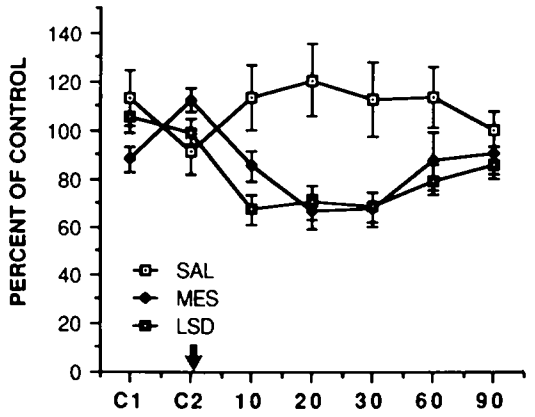

FIG. 7. Effects of intraocular administration of mescaline and LSD on the FEP. Saline, mescaline (10 μg) or LSD (5 ng) were administered by injection (1 μl) as indicated by the solid arrow into the vitreous humor of both eyes of rats anesthetized with halothane. Animals were allowed to recover and the FEP was determined at 10, 20, 30, 60 and 90 minutes after drug administration. Both mescaline and LSD significantly reduced the amplitude of the P20–N30 component of the FEP at medium flash intensity as compared with saline-injected controls [$F(1,6)=16.3$, $p<0.01$ for mescaline; $F(1,6)=11.5$, $p<0.01$ for LSD].

DISCUSSION

The locus of action of mescaline and LSD in the visual system of the rat was investigated. Systemic administration of mescaline and LSD depressed the primary component of the photically-evoked cortical response indicative of a drug-induced deficit in conduction through the retino-geniculato-cortical system. Studies in which LSD and mescaline were administered intraocularly suggest that the action of these drugs are mediated, in part, at the level of the retina and are modulated by cholinergic systems in the retina.

Experiments were conducted in freely-moving awake animals, thus allowing for behavioral observations and avoiding the complications in interpretation of drug effects on sensory processing common to experiments conducted in anesthetized animals (21, 24, 25). At the doses of mescaline and LSD used in these studies only minor behavioral effects were observed. Following the injection of mescaline or LSD, rats exhibited a decrease in spontaneous activity and appeared to be fixated on the strobe light.

The distortion of visual perception produced by mescaline and LSD most likely results from a multiplicity of actions at various sites within the central nervous system. These drugs have been reported to facilitate and to inhibit photically-evoked activity of neurons in the primary visual pathway (5, 14, 20, 22). Our findings confirm reports that LSD depresses the photically-evoked cortical response and extends this work by demonstrating that the phenethylamine hallucinogen, mescaline also depresses the FEP. Furthermore, our data suggest that mescaline increases the threshold and slope of the visual response to flash. The primary components of the FEP are purported to be the result of retinogenulostriate activity, whereas the secondary components are the result of a multitude of modulatory systems including geniculostriate connections and thalamic, limbic and reticular nuclei (4). The magnitude and specificity of the effects of these agents on the primary component of the FEP suggest that these drugs attenuate conduction through the retino-geniculato-cortical system. LSD has been reported to block the optic afferent synapse at the level of the dorsal lateral geniculate nucleus (dLGN) and to alter the evoked activity of visual cortical neurons in addition to subcortical effects (6).

Intraocular administration of mescaline and LSD also depressed the FEP indicating that these agents affect visual processing in the retina in addition to their actions on the dLGN and the visual cortex. Studies by other investigators also support a retinal site of action for LSD and mescaline (1, 15, 18, 26). LSD has been shown to improve discrimination of flicker in rats, whereas mescaline degrades it (18). The mechanisms determining flicker sensitivity are believed to be governed by receptor cell and bipolar cell layers in the retina (26). In other studies, LSD has been reported to increase spontaneous activity and decrease evoked activity of retinal ganglion cells in vitro (1) and in vivo (15). Changes in the response patterns of ganglion cell receptive fields may be sufficient to explain much of the visual distortion produced by LSD and mescaline at a purely retinal level.

The serotonin antagonists, cyproheptadine and methysergide antagonized the mescaline-induced depression of the FEP in accordance with neurochemical and behavioral evidence that mescaline, like LSD, acts as a partial agonist in serotonergic systems to alter perception (3, 4, 8, 10, 12). However, these findings differ from those obtained in our laboratory in α-chlorolose-anesthetized cats in which cyproheptadine antagonized and methysergide potentiated the LSD-induced depression of electrically-evoked activity in the visual system (23). These discrepancies may reflect species differences, anesthetic effects or differences in the actions of mescaline and LSD on serotonergic systems.

Atropine, administered topically or by intraocular injection, antagonized the actions of systemically-administered mescaline. These findings suggest that the effects of mescaline are modulated by cholinergic systems in the retina. Cholinergic amacrine cells have been well characterized in the retinae of several species including the rat. Acetylcholine is well established as an excitatory neurotransmitter in the retina (9, 13, 16). Light-evoked release of acetylcholine from rabbit retina has been shown to be enhanced by atropine and inhibited by muscarine indicative of presynaptic muscarinic receptors on these cholinergic amacrine cells (16). This provides the substrate for a muscarinic link in the actions of hallucinogens in the retina. Although the role of serotonin as a neurotransmitter in the mammalian retina is controversial, there are clear indications of indoleamine actions on retinal neurotransmission. Serotonin receptors have been characterized in the retinae of rats and other mammals (3, 9, 13). Furthermore, electrophysiological studies in vitro and in vivo have shown that serotonin agonists and antagonists modulate spontaneous and light-evoked activity of retinal ganglion cells (1, 15, 23). LSD and mescaline may alter retinal ganglion cell activity by reducing the activity of cholinergic amacrine cells. Mescaline and LSD have been shown to attenuate the evoked release of acetylcholine from cholinergic interneurons in the rat striatum by interacting with serotonin receptors on these interneurons (7). If cholinergic amacrine cells possess similar serotonin receptors then LSD and mescaline would reduce the light-evoked release of acetylcholine from these cells, whereas the muscarinic antagonist atropine would enhance evoked release resulting in a physiological antagonism (11,16). Such a mechanism would be consistent with the observed actions of mescaline and atropine on the flash-evoked cortical potential.

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