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INHIBITION OF LECTIN STIMULATION OF MURINE LYMPHOCYTES BY MESCALINE*

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Abstract—Evidence is presented that the T-cell specific stimulation of mouse lymphocytes by concanavalin A is inhibitable by mescaline. The degree of lectin inhibition is directly proportional to the amount of mescaline added to the *in vitro* cell incubation. Results also suggest that, during *in vitro* incubation, mescaline becomes covalently conjugated to a cellular protein which has a minimum nonreduced molecular weight of approx 55,000 daltons. The dynamics of mescaline incorporation is relatively rapid and the kinetics do not correlate directly with active *de novo* protein synthesis. Selected inhibitors of oxidation, and metabolic antagonists show no effect on the incorporation of mescaline into protein.

Suppression of the immune response can be achieved either partially or totally through the use of chemical and/or biological reagents [1]. Due to the complexity of the immune response, the mechanisms of action of immunosuppressive agents are frequently diverse [2]. For example, multiplicity of sites of immunosuppressive action can be exemplified by the following examples. Δ^9-THC†, the active principal in marijuana, decreases the responsiveness of lymphocytes from Rhesus monkeys to the lectin concanavalin A (Con A) and results in the lowering of immunoglobulin levels in serum [3]. Rabbit antibody-producing lymphoid cells have been shown to secrete low molecular weight peptides when incubated *in vitro* in the presence of LSD [4, 5]. The lysergyl moiety is secreted attached to peptides which are produced during *de novo* biosynthesis [6]. In addition, LSD incorporation competes with *de novo* incorporation of L-tryptophan [4]. Evidence that the lysergyl moiety is covalently attached to the peptides has been varied and extensive [6, 7].

This report will present evidence that mescaline interferes with the ability of Con A to stimulate mouse lymphoid cells *in vitro*. Mescaline is apparently incoporated into macromolecules during *in vitro* incubations of hyperimmune mouse lymphoid cells and Con A stimulated cells.

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†Abbreviations: Δ^9-THC, delta 9-tetrahydrocannabinol; Con A, concanavalin A; LSD, *d*-lysergic acid diethylamide; L-trp, L-tryptophan; $DNP_{52}BGG$, immunogen of bovine gamma globulin substituted with an average of 52 2,4-dinitrophenyl groups/molecule; MEM, minimal essential media; BSS, balanced salt solution; TCA, trichloroacetic acid; TMPA, trimethoxyphenylacetic acid; Gu–HCl, guanidine–HCl; PBS, phosphate buffered saline; V_0, void volume; V_t, total excluded volume; and PPO. 2,5-diphenylopazole.

MATERIALS AND METHODS

Immune cell incubations. Balb/c mice were boosted 3 weeks or longer after a primary immunization with $DNP_{52}BGG$ in complete Freunds adjuvant. Five days later the mice were killed by cervical dislocation and the spleens were removed into Eagle's MEM with Earle's BSS, penicillin, glutamine, $NaHCO_3$, non-essential amino acids and 0.5% carbowax (to increase osmolarity). All immune cell incubations were performed in this medium.

Cells were teased from the spleens with sterile forceps and scalpel, and the resulting suspension was allowed to stand for approx 5 min in a 10 ml tissue culture tube. The supernatant fraction, containing single cells, was separated from the clumps of cells and debris that had settled, by gentle aspiration. The single cell suspension was centrifuged for 5 min at ~2500 rev/min and the cell pellet suspended in fresh media. White cell concentration was determined in a model ZBI Coulter Counter after which the suspension was diluted to give about 2×10^7 cells/ml in the final incubation mixture.

The total volume for animal cell incubations was adjusted to 1 ml/10 × 35 mm sterile plastic tissue culture plate (Falcon). Each incubation contained 0.2 μmole [^{14}C]mescaline (New England Nuclear, Boston, MA) with a specific activity of 21.54 mCi/mmole. Thus, each incubation possessed the same molar concentration of mescaline as tyrosine and phenylalanine in MEM. Tissue culture plates were transferred from a 37°, 5% CO_2 humid atmosphere to a 4° cold room after an appropriate incubation time (usually 8 hr). The contents of the plates were pipetted into test tubes and the cells separated from the supernatant fractions by 15 min of centrifugation at ~2000 rev/min. All experiments were run in triplicate. Incorporation of radioactive label into macromolecules was determined by precipitation of cell lysates and supernatant fractions with a final concentration of 10% cold TCA. Precipitates were collected on Whatman GF/C 24 mm diameter glass fibre filters, washed three

times with 10% cold TCA and one time with methanol. Filters were dried in glass mini-scintillation vials and counted in a Beckman LS150 scintillation counter after the addition of 5 ml Toluene–0.4% PPO scintillation fluid. Samples from some experiments were hydrolyzed prior to counting by the incubation of the GF/C filters in capped scintillation vials with 0.5 ml of 0.1 N HCl at 80° for 8 hr. One milliliter ethyl acetate and 8 ml Aquasol (New England Nuclear) were added for scintillation counting [8].

An experiment designed to inhibit incorporation of [^{14}C]mescaline into macromolecules compared incubations with mescaline (as above) to incubations containing mescaline and a final concentration of 1 mM ascorbate, TMPA, cysteine or 20 μg/ml of cycloheximide.

Gel chromatography. Gel chromatography in 6 M Gu–HCl was performed using Sephadex G-75 equilibrated in 6 M Gu–HCl in a Pharmacia K16/40 jacketed column with adjustable plungers using ascending flow (39 × 1.6 cm). Approximately 1-ml fractions were collected using a drop counter (Gilson 100 sample microfraction). All samples were chromatographed with either blue dextran 2000 (average mol. wt. 2×10^6) or porcine gamma globulin (155,000 daltons) as an internal marker for void volume (V_0) and with L-tryptophan as an internal end point marker (V_t). The position of each marker was determined by u.v. absorbance at 278 nm. Radioactive samples were determined by counting 0.5 ml aliquots in 5 ml Biofluor with 75 μl Protosol (both New England Nuclear) and 5 μl of 2 N acetic acid to adjust the pH. A standard curve was constructed by determining the distribution coefficient $K_D(K_0 = [V_e - V_0/V_t - V_0]$, where V_e is the elution volume) of known molecular weight proteins [9] and plotting by the method of Porath [10]. This consisted of plotting $K_D^{1/3}$ vs mol. wt.$^{1/2}$ for seven proteins of known molecular weight, each run separately on the Sephadex G-75 column and including internal markers for V_0 and V_t. The nanopeptide was obtained from Beckman (Fullerton, CA); insulin, ribonuclease and pepsin were obtained from Sigma (St. Louis, MO); glucagon was obtained from Cal-Biochem (La Jolla, CA); and bovine gamma globulin was obtained from Miles Laboratories (Elkhart, IN).

Enzyme digestions. Pools of immune cell incubation supernatant fractions and lysates were made. Digestions were performed in triplicate on either a 100 μl supernatant pool or a 100 μl lysate pool. These were incubated with 25 μl deoxyribonuclease (1–2 mg/ml in H_2O, Worthington, Freehold, NJ), 25 μl ribonuclease A (1 mg/ml), pronase (1–2 mg/ml in 0.01 N HCl, both Sigma) or 2 mg/ml bovine serum albumin (BSA) as a control. After 16 hr of incubation at 37°, all samples were precipitated in the presence of 4 mg BGG carrier by a 10% final concentration of TCA. After the precipitate had developed in the cold, it was collected by centrifugation at 1500 rev/min for 15 min in a refrigerated International centrifuge. The supernatant fraction was discarded and the pellet dissolved in 1.0 N NaOH. This was reprecipitated by the addition of 1.0 ml of 20% TCA and centrifuged as before. The pellet was dissolved in 200 μl of 1 N NaOH and added to a glass minivial. Test tubes were rinsed with 0.5 ml methanol which was added to the minivial. Six milliliters Biofluor and 100 μl of 2 N acetic acid (to adjust pH) were then added for scintillation counting.

Mitogen stimulation. Spleens were removed from normal Balb/c mice and cell suspensions prepared as described for immune cell incubations, except for the following differences. RPMI 1640 media, supplemented with 10% fetal calf serum, penicillin, streptomycin, glutamine and $NaHCO_3$, were used for all stimulation and transport experiments. The cell concentration was adjusted to 5×10^6 cells/ml in the final incubation. Con A was added to the incubations at a concentration of 5 μg/ml. [^{14}C]mescaline or unlabeled mescaline was added in concentrations up to 200 μg/ml. The final volume of each incubation was adjusted to 250 μl in a 10 × 80 mm sterile polystyrene tissue culture tube. Cell viability, tested by Trypan Blue dye exclusion, remained greater than 80 per cent throughout all experiments. After 48 hr of incubation at 37° in a humid 5% CO_2 atmosphere, 2.5 μCi [^{3}H]thymidine (thymidine[methyl-^{3}H], 6.7 Ci/mole, New England Nuclear) was added to each test tube and incubations were allowed to continue an additional 6 hr. Incubations were terminated at that time by the addition of 2 ml of cold 10% TCA. Precipitates were collected on Whatman GF/C glass fibre filters and treated as described for immune cell incubations.

Transport of thymidine in the presence and absence of mescaline was determined on the same day using the same cells as in the stimulation experiments. Mescaline was tested at concentrations of 20 and 200 μg/ml. Ten micro-Curies [^{3}C]thymidine was added per ml of pooled cells (5×10^6 cells/ml) at time zero. The media were the same as above, except that they did not contain serum or Con A. Controls without mescaline were run at the same time. A 1.0-ml sample was removed at intervals over a 30-min time period. Immediately upon removal, cells were diluted with 4 ml of cold PBS, collected by vacuum filtration on Whatman GF/C glass fibre filter paper and washed two times with cold PBS. Filters were dried in glass minivials and counted in Toluene–0.4% PPO scintillation fluid.

Calculations. All statistical calculations and corrections for quenching and spillage in double labeled radioactive experiments were done with the aid of a Hewlett Packard 9821A programmable calculator.

Con A binding. Con A (Sigma) was radioiodinated with ^{125}I Na (Amersham, Arlington Hts, IL) by a solid phase lactoperoxidase catalyzed reaction (Worthington lactoperoxidase bound to Sepharose). To 1.0 ml of lectin (2.0 mg/ml in PBS) was added 100 μl of 10^{-4} M KI, 0.1 ml lactoperoxidase-Sepharose suspension, 20 μl ^{125}I Na (2 μCi) and 50 μl of 0.003% H_2O_2. The reaction was incubated for 20 min at 25°. The lactoperoxidase-Sepharose was collected by centrifugation and the radiolabeled Con A harvested by passage over a 50-ml (packed volume) Biogel P-6 (equilibrated in PBS). Specific activity of the labeled Con A was calculated to be 1.53×10^6 cpm/mg.

Mouse spleen cells were prepared as for nitrogen stimulation experiments. Cells and all additions were in PBS and final volumes were adjusted to 1.0 ml. Triplicate incubations were set up with 2×10^7 cells/ml and 50 μg Con A present in each. Inhibition of Con A binding to cells was attempted with 0.1 M α-methyl-D-mannoside (Sigma), 10^{-3} M and 2.5 ×

10^{-4} M mescaline and 10^{-4} M and 10^{-6} M colchicine. After a 1-hr incubation, cells were diluted with 4 ml PBS and harvested by filtration over GF/C filters. The filters were washed five times with 4 ml PBS and counted in a Nuclear Chicago gamma counter.

RESULTS

Incorporation of mescaline. The *in vitro* incubation of [^{14}C]mescaline with hyperimmune murine lymphocytes resulted in the incorporation of the drug into TCA precipitable material. Time studies indicated that the incorporation of [^{14}C]mescaline (i.e. into TCA precipitable material) was most significant quantitatively during the first 60 min period. Figure 1 shows that the increase in mescaline incorporation between 1 and 8 hr was minimal.

Table 1. [^{14}C]mescaline incorporation in *in vitro* incubations of hyperimmune mouse lymphoid cells*

Treatment	[^{14}C] cpm ± S. E.
Mescaline (control)	1776 ± 112
Inhibitor	
Ascorbate	1784 ± 79
TMPA	1786 ± 60
Cysteine	1800 ± 21
Cycloheximide	1734 (one sample only)

*The concentration of all inhibitors except cycloheximide was 1 mM in the final incubation mixture. Cycloheximide was at a final concentration of 20 μg/ml. All incubations were set up and determinations made in triplicate except only one cycloheximide determination was made due to loss of the replicates. Incubation was for 8 hr.

Table 1 shows that various oxidation inhibitors were tested in the *in vitro* incubations with [^{14}C]-mescaline. Ascorbate, TMPA and cysteine tested at 1 mM did not inhibit *in vitro* incorporation of mescaline. Similarly, cycloheximide at 20 μg/ml did not inhibit the incorporation of mescaline.

Molecular sieve chromatography of mescaline derivatized material. When the [^{14}C]mescaline derivatized material, which was produced during the *in vitro* cellular incubations, was chromatographed on Sephadex G-75 in 6 M Gu–HCl, it showed a minimum molecular weight of 55,000 daltons. The column (1.6 × 40 cm) was calibrated with molecular weight markers; Fig. 2 shows a plot of $K_D^{1/3}$ vs mol. wt.$^{1/2}$, obtained with the Sephadex G-75 column (in 6 M Gu–HCl) used in the calibration studies. Figure 3 shows the elution pattern of the mescaline associated material from the Sephadex G-75 column; the material eluted near the void volume of the column. Since the column was equilibrated in an efficient denaturing agent (6 M Gu–HCl), the data are further evidence that the [^{14}C]mescaline was strongly associated with the cell-derived material.

Enzyme digestions of mescaline-associated material. The [^{14}C]mescaline associated material, derived from the *in vitro* incubations, was subjected to digestion with deoxyribonuclease, pronase and ribonuclease. Table 2 shows that only pronase digestion resulted in a reduction in the amount of TCA precipitable ^{14}C-radiolabeled material. Both lysate and supernatant labeled materials (i.e. from the *in vitro* incubations) were subjected to enzyme digestions.

Effect of mescaline on Con A stimulated lymphocytes. To define the effect of mescaline on lymphocytes, the drug was incubated at various concentrations with Con A stimulated murine lymphocytes. Lectin stimulation of cells was determined by cellular uptake and incorporation of [^{3}H]thymidine. Figure 4 shows the results of increasing mescaline concentrations on [^{3}H]thymidine incorporation. Correspondingly, [^{14}C]mescaline incorporation was studied in respect to increasing concentrations of mescaline. In Fig. 4, it is evident that thymidine incorporation is inhibited 90 per cent or more at relatively high concentrations of mescaline. The effect was similar with either ^{14}C-labeled mescaline or nonradioactive mescaline. When [^{14}C]mescaline incorporation was studied as a function of increasing amounts of mescaline, there was a significant increase in drug incorporation. This apparent synergistic effect suggests that the amount of drug incorporated is a function of the amount of drug used in a given time period.

Figure 5 shows that inhibition of thymidine uptake (Fig. 4) by mescaline is probably not due to an effect upon nucleotide transport. Thymidine transport was not affected significantly at low concentrations of mescaline (20 μg/5 × 10^6 cells). However, a modest effect was noted at high concentrations of mescaline (200 μg/5 × 10^6 cells). Both experiments were monitored over a 30-min period.

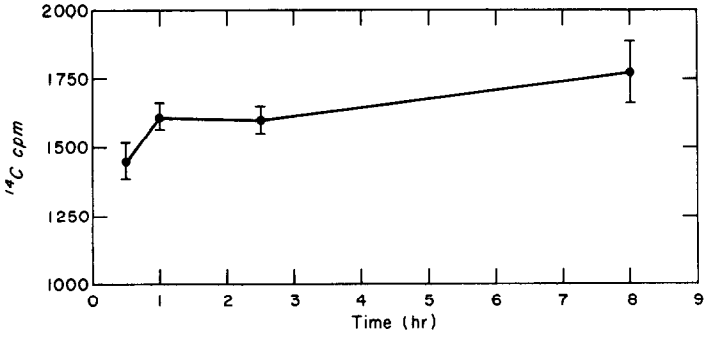

Fig. 1. Total [^{14}C]mescaline radioactivity incorporated into TCA precipitable material after incubation of 0.2 μmole [^{14}C]mescaline with 2 × 10^7 immune lymphoid cells. Counts/min. ± S.E. are plotted (n = 3).

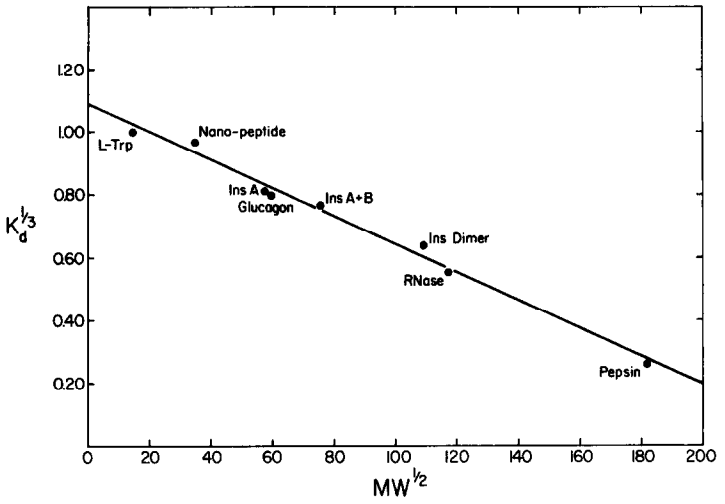

Fig. 2. Calibration markers and least squares best fit line for Sephadex G-75 column in 6 M guanidine–HCl. All samples were run with L-tryptophan (V_t, 204 daltons) and either bovine gamma globulin (V_0, 155,000 daltons) or blue dextran 2000 (V_0, 2×10^6 daltons). Molecular weights of markers were (in daltons): nanopeptide, 1212; insulin A chain, 3330; glucagon, 3483; insulin A-SS-B, 5733; insulin dimer (A + B) times two 11,466; ribonuclease, 13,640; and pepsin, 33,000.

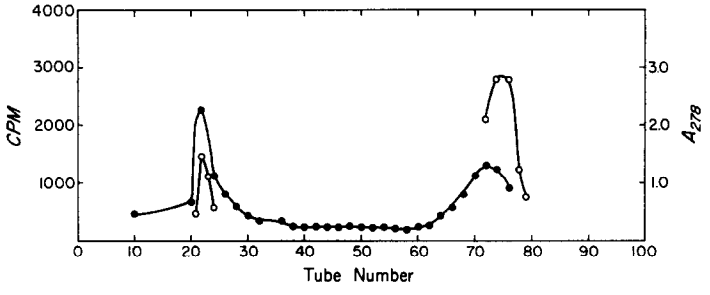

Fig. 3. Sephadex G-75 elution profile of [^{14}C]mescaline cpm material from immune cell incubations (cpm, ●——●), and internal markers for V_0 and V_t (L-tryptophan and BGG were monitored by absorbance at 278 nm, ○——○).

The effect of the length (i.e. time) of mescaline incubation with stimulated lymphocytes on [^{3}H]-thymidine incorporation was investigated. To test the effect of time, cells were compared which received either no mescaline, mescaline at the beginning of the incubation, or mescaline immediately prior to the addition of [^{3}H]thymidine (i.e. after approx 48 hr). A separate experiment compared cell incubations with or without mescaline (added initially). However, in the former, mescaline was removed prior to the addition of [^{3}H]thymidine and replaced with fresh media without mescaline, or with fresh media and mescaline. Both experiments included nonstimulated control incubations. Results shown in Fig. 6A demonstrate more effect on the inhibition of thymidine uptake by cells exposed for 48 hr to mescaline than by those exposed only during the 6-hr thymidine incubation. Figure 6B shows that removal of mescaline prior to [^{3}H]thymidine incubation results in only a modest reversal of the inhibition of [^{3}H]thymidine uptake. Adding fresh mescaline causes a further decrease in thymidine incorporation.

Table 2. Trichloroacetic acid precipitable cpm of immune cell incubations after enzyme digestions

Digested material	Mean [^{14}C] cpm ± S.E.
Lysate	
Control	540 ± 20
Deoxyribonuclease	622 ± 55
Pronase	279 ± 22
Ribonuclease	552 ± 22
Supernatant fraction	
Control	6723 ± 350
Deoxyribonuclease	8079 ± 693
Pronase	3083 ± 73
Ribonuclease	7982 ± 607

Effects of mescaline on Con A binding to cells. Table 3 shows that mescaline does not alter Con A binding to cells. Equivalent amounts of [^{125}I]Con A were bound by cells in the presence of mescaline as in buffer controls or cells in the presence of colchicine. Only α-methyl-D-mannoside, for which Con A has known binding specificity, inhibited Con A binding to cells.

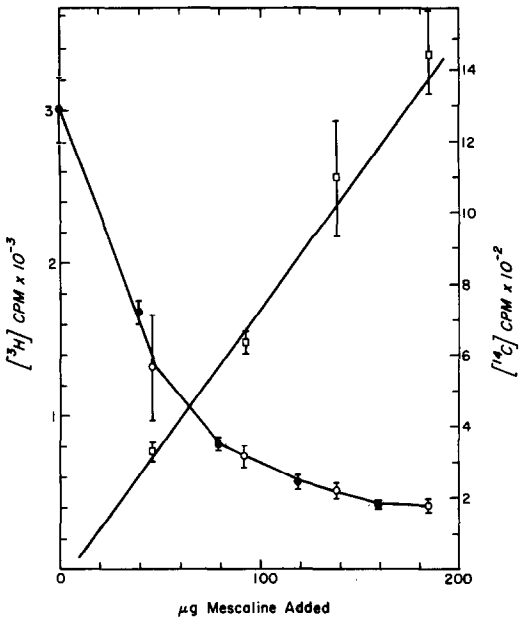

Fig. 4. *In vitro* [^{3}H]thymidine incorporation into DNA of Con A stimulated murine lymphocytes in presence of mescaline. Incubations are designated as: [^{3}H]thymidine uptake in presence of [^{14}C]mescaline (○——○); [^{3}H]thymidine uptake in presence of unlabeled mescaline (●——●); and [^{14}C]mescaline in TCA precipitable material (□——□). All values are shown as mean ± S. E. (n = 3).

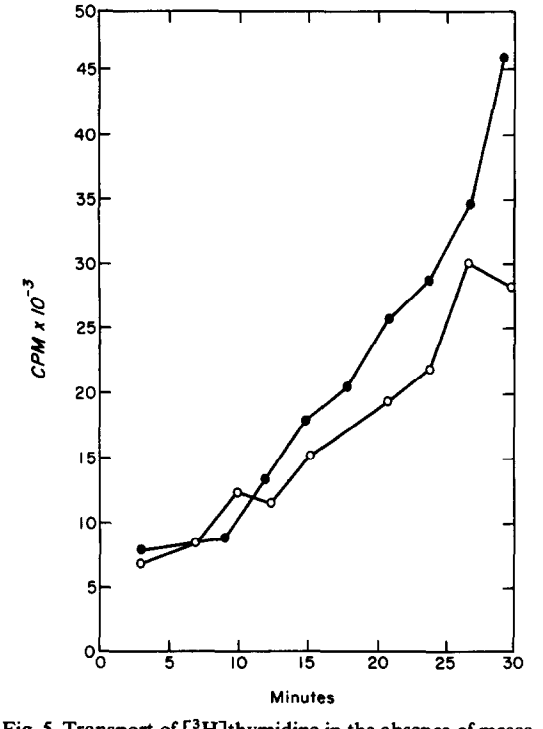

Fig. 5. Transport of [^{3}H]thymidine in the absence of mescaline (●——●) and in the presence of 400 μg/ml of mescaline (○——○).

Table 3. [^{125}I]Con A binding to mouse spleen cells in the presence of mescaline and other inhibitors

Inhibitor	Inhibitor concn (M)	Mean cpm [^{125}I] Con A bound ± S.E.
Buffer control		31,684 ± 72
α-Methyl-D-mannose	1.0×10^{-1}	2626 ± 69
Mescaline	1.0×10^{-3}	33,172 ± 447
Mescaline	2.5×10^{-4}	33,828 ± 496
Colchicine	1.0×10^{-4}	32,513 ± 1831
Colchicine	1.0×10^{-6}	35,564 ± 810

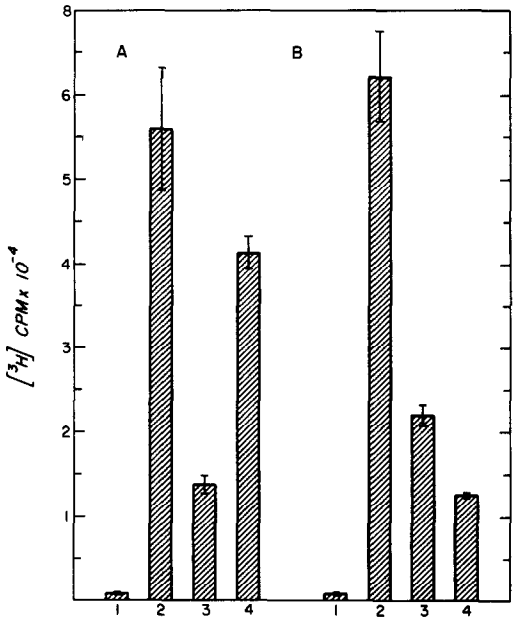

Fig. 6. Effect of length of exposure to mescaline on [^{3}H]-thymidine incorporation into Con A stimulated murine lymphoid cells. Non-stimulated controls are represented by 1A and 1B, while the lectine stimulated standard (i.e. no mescaline) is designated 2A and 2B. Mescaline was added to the cell incubation (final concentration of 200 μg/ml) at the beginning of the experiment (3A) and after 48 hr (4A). 3B and 4B reflect an initial incubation with mescaline. In both cases the cells were harvested, washed and resuspended in fresh media. In 4B the fresh media contained mesclaine, while in 3B mescaline was depleted. All cpm are listed as mean ± S.E. (n = 3).

DISCUSSION

These studies indicate that upon *in vitro* incubation with cells mescaline is found associated with macromolecular material. This is primarily based on the measurement of ^{14}C-label in trichloroacetic acid precipitable material (Fig. 1). The substrate for mescaline attachment is probably protein, since only digestion with the proteolytic enzyme pronase resulted in reduction in the size of the carrier molecule (Table 2). The carrier or substrate is resistent to nuclease digestion, suggesting that it is not a nucleic acid. It is conceivable that the material is a glyco- or lipoprotein,

since studies were not conducted to determine its chemical composition.

The suggestion that the mescalyl moiety is apparently covalently attached to the protein carrier was based on the observation that the ^{14}C-label was firmly associated with the carrier. It is unlikely that, during the incubation time periods employed, recycling of the ^{14}C-label could have occurred. The specificity of the competitive *in vitro* incubation experiments also suggested that mescaline is being incorporated (Fig. 4). Covalent bonding was indicated in several ways. Non-dissociability of the ^{14}C-label under denaturing conditions of 10% trichloroacetic acid and 6 M Gu–HCl was evidence for a covalent bond (Fig. 3). In addition, the dilution effect of the molecular sieve column (Fig. 3) further suggested it was not just avid binding as in some antigen–antibody complexes or in the biotin–avidin complex [11].

If one assumes covalent bonding, then the mode of incorporation remains unclear. The kinetic studies (Fig. 1), and the lack of inhibition by cycloheximide, indicate that incorporation of [^{14}C]mescaline does not conform to a requirement of *de novo* protein biosynthesis. If the latter were required, then kinetics such as those observed with the incorporation of LSD should have been observed [5]. In addition, LSD incorporation was puromycin and cycloheximide sensitive. The experimental results show that [^{14}C]-mescaline incorporation is enhanced in direct proportion to increasing amounts of mescaline. This may indicate that, although the incorporation process is not linked to *de novo* protein biosynthesis, it is concentration dependent. The latter may imply existence of specific cell receptors, permeases or metabolic enzymes for the transport and/or processing of the drug.

The β-phenethylamines (α-methyl dopa, L-dopa and dopamine) are structurally related to mescaline, and have been shown by others to be covalently bound to protein under certain conditions [12, 13]. Suggested mechanisms for covalent incorporation of these β-phenethylamines have included auto-oxidation or enzymatic oxidation to a reactive intermediate metabolite which might react with proteins (e.g. with sulfhydryl groups) [12–14]. Table 1 shows that ascorbate and TMPA should have inhibited mescaline incorporation if an oxidized derivative of mescaline were being incorporated. Ascorbate is an antioxidant, while TMPA is a known oxidized metabolite of mescaline [15]. Similarly, cysteine would have inhibited mescaline incorporation into macromolecules (i.e. proteins) if the attachment were by means of the free sulfhydryl group. Collectively, these results suggest that oxidation of mescaline is not an important prerequisite to incorporation. The results presented in this study do not elucidate what protein(s) serves as the substrate for mescalyl conjugation, only that the minimum molecular weight is 55,000 daltons.

Another effect of mescaline described in these studies is the inhibition of lymphocyte stimulation by a lectin. Normal mouse lymphoid cells, stimulated by Con A show a decrease in [^{3}H]thymidine uptake in the presence of mescaline (*in vitro*). [^{14}C]mescaline was incorporated into Con A stimulated cells in direct proportion to the amount of mescaline added (Fig. 4). However, there was an inverse relationship between the degree of stimulation and the amount of [^{14}C]-mescaline added and incorporated. Table 3 shows that Con A binding is not altered by the presence of mescaline.

Since stimulation was measured by [^{3}H]thymidine incorporation, the effect of mescaline on the transport of thymidine was investigated. Figure 5 shows a definite, though moderate effect, on the rate of thymidine transport in the presence of a high concentration of mescaline. A similar experiment, using only one-tenth the concentration of mescaline, showed no effect on thymidine transport.

Finally, experiments were designed to determine if the results from Con A stimulation experiments with mescaline present could be explained solely by transport, or if there were additional effects of mescaline on the cell. Figure 6A shows results of *in vitro* cell incubations, all having [^{3}H]thymidine at 48 hr, and compares the effects of adding mescaline at zero time and at 5 min prior to the addition to thymidine. If mescaline only acted with a transport component of the cell in a reversible manner, such as the majority of transport inhibitors act, there should be no difference in the decrease of thymidine incorporation compared to the control. Figure 6A shows there is a large difference. Incubations receiving mescaline early have a large decrease in thymidine incorporation, indicating transport alone is not sufficient to explain the decrease.

An alternative approach examined the effect of removal of mescaline prior to the addition of [^{3}H]-thymidine. Most inhibitors of transport inhibit reversibility, normal transport being restored by removal of the inhibitor. Figure 6B shows that removal of mescaline did not return the thymidine incorporation level to normal. Adding fresh mescaline prior to thymidine did cause a further reduction in thymidine incorporation. This difference may represent the effect on transport by mescaline.

Data presented in these studies are insufficient to draw conclusions as to the mechanisms of mescaline incorporation into protein of immune and mitogen stimulated lymphoid cell cultures and of interference with Con A stimulation of normal lymphoid cells. However, the data clearly indicate that the mescaline becomes firmly attached to a cellular component and inhibits lectin stimulation of lymphocytes. It is not evident that these two observations are interrelated, but investigations are currently in progress to further resolve the effect of mescaline on lymphocytes and to characterize the component in the mescalyl–protein conjugate.

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