dinucleotide (NADH) (data not presented). Several other workers have also reported the oxidation of NADPH and NADH by peroxidase[21-25]. In this context the low levels of peroxidase become significant in the nodules, as peroxidase if present in high amounts may oxidize NADPH and NADH, which are required for the removal of an excess of ammonia by the ammonia assimilatory enzymes like glutamate dehydrogenase, glutamine synthetase/glutamate synthase[9,26] and as the reductant for nitrogenase.

1 Acknowledgment. This investigation was supported by grants from the Indian Council of Agricultural Research, New Delhi, India.
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0014-4754/83/020155-02$1.50+0.20/0

Treatment of mice with monosodium glutamate does not inhibit a subsequent response to gold thioglucose

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Summary. Neonatal mice were injected with monosodium glutamate (MSG) followed 10 days later by treatment with gold thioglucose (GTG). Weight gain, food consumption and body lipid determinations were made. Results indicated that pretreatment with MSG does not block subsequent response to GTG and the resulting changes are similar to those induced by GTG alone.

Gold thioglucose (GTG) and monosodium glutamate (MSG) are frequently used to induce metabolic alterations in mice. Damage in the ventromedial area of the hypothalamus (VMN) after GTG administration results from pericapillary changes[1]. Physiological alterations induced by VMN damage include excessive weight gain, lipid accumulation, and changes in energy balance among others. For example, GTG-injected mice reached a body weight of 56.3 ± 0.9 g 4 months post-injection vs 30.7 ± 3.6 g for the controls. Mean body lipid content was 51.3% for the injected animals and 14.9% for the controls[2].

In 1969 Olney[3] reported that neonatal mice treated with MSG had increased body weight, substantially elevated amounts of depot lipid and a slight suppression in food intake compared to control animals. Examination of the hypothalamic region of the MSG-treated mice revealed extensive damage to the arcuate-median eminence area with no observable change in the VMN. Djazayery and Miller[2] reported little change in body weight or food comsumption of MSG-treated neonatal mice; however, at 4 months of age the treated animals had a body lipid content of 26.3% compared to 14.9% for the non-injected animals.

When GTG or MSG treatments are utilized one can expect to see changes in body lipid content; however, food intake and total body weight alterations depend on the compound administered and the protocol used. Since these 2 compounds seem to affect different hypothalamic regions and somewhat different metabolic parameters we sought to determine if treatment with MSG might eliminate a subsequent response to GTG. The results indicated that the MSG-treated mice are still susceptible to damage from GTG. Mice used in this study were a CF1 strain derived, from Sprague-Dawley stock. The animals were housed in plastic box cages in a temperature controlled room (22 ± 1 °C) with a 12:12 light cycle. Feed (Purina Chow) and water were available ad libitum. Upon delivery of the young, litter size was adjusted to 8-10 per female. Beginning at 5 days of age, one half of the pups in each litter were s.c. injected for 10 days with MSG in sterile saline

The effect of MSG and/or GTG treatment on selected parameters in male CFI mice

Parameter measured	Experimental group Control	MSG	GTG	MSG + GTG
Food consumption (g/week at 100–125 days of age)	41.5 ± 1.2 (6)	37.2 ± 1.2 (6)*	45.3 ± 1.0 (6)	43.4 ± 1.5 (6)
Body weight (g at 125 days of age)	35.5 ± 0.6 (21)	36.8 ± 1.1 (21)	46.8 ± 2.3 (7)	45.7 ± 2.4 (13)*
% Body lipid (% of wet wt)	10.7 ± 3.1 (6)	18.7 ± 2.7 (6)*	27.2 ± 1.3 (6)*	30.2 ± 2.8 (6)**
% Liver lipid (% of wet wt)	2.9 ± 0.9 (6)	8.6 ± 2.3 (6)*	3.2 ± 0.5 (6)	3.6 ± 0.1 (6)
Average total liver lipid (mg)	69 ± 8 (6)	189 ± 18 (6)*	87 ± 10 (6)	101 ± 6 (6)

Mean ± SEM (n). *Significantly different from controls ($p < 0.05$). **Significantly different from control and MSG groups ($p < 0.05$) but not from GTG group.

(10% w/v) at 4.0 mg/g b.wt. The remaining pups were injected with a comparable volume of saline. Animals were weaned at 25 days of age, separated by sex, and one half of the controls and one half of the MSG-treated animals were injected with GTG. The GTG was dissolved in saline (100 mg/ml) and s.c. administered at 1 mg/g of b.wt. Body weights were recorded each week. At 100 days of age 6 male animals were randomly selected from each group (controls, MSG, GTG and MSG + GTG) and placed in suspended wire-bottomed cages for a 3-week food consumption study. All animals were sacrificed at 125 days of age. 6 male animals were randomly selected from each group for total lipid analysis. Wet carcass weight was recorded, the liver removed and after drying to a constant weight the total amount of lipid in the body and liver was determined by extracting with petroleum ether. All data were subjected to statistical analysis including T-tests and ANOV.

Data for male animals are presented in the table. Food consumption data indicated a significant inhibition in the MSG treated animals with no difference between the other groups. Body weight was elevated in the GTG and MSG + GTG-treated groups, indicating that MSG-treated animals are capable of responding to GTG. The data for total body lipid in the control, MSG and GTG treated groups are similar to other published information[2,3]; however, the 30.2% value for the MSG + GTG-treated animals is not significantly different from GTG treatment alone. In the MSG-treated mice both percent and total liver lipid are elevated. These results indicate that 2 relatively separate metabolic systems leading to body lipid accumulation may be affected by the treatments. The MSG lesions favor liver lipid deposition while GTG-treatment results in greater depot lipid accumulation. In the combined MSG + GTG treatment group, the MSG does not block a subsequent response to GTG and the resulting changes appear to be the effect of the GTG rather than the MSG.

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0014-4754/83/020156-02$1.50 + 0.20/0

Binding of levonorgestrel to monkey plasma proteins[1,2]

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Summary. ^{3}H-levonorgestrel, a protein progestational steroid, showed a high affinity saturable binding to monkey plasma. Competitive protein binding experiments suggested that the levonorgestrel binds to a protein which resembles sex hormone-binding globulin.

The initial step in the mechanism of steroid hormone action is the binding of the hormone to a specific receptor protein in the cytosol of the target cell[3]; binding to plasma proteins, however, also occurs. The binding of steroid hormones to plasma proteins is of great physiological significance because steroids are biologically inactive when associated with protein; they can be activated by dissociation to form the unbound hormone. In fact, steroid hormone binding to specific plasma proteins provides a pool of the steroid in equilibrium with unbound fraction and thus limits the availability of steroid hormone to the target tissue. Specific high affinity binding proteins in plasma have been identified for cortisol and sex steroids and the physiological effect of steroid protein interactions have been well established[4,5]. Levonorgestrel is a potent progestin widely used as a contraceptive agent in women. In the present study, we report the ^{3}H-levonorgestrel binding to plasma proteins of rhesus monkey *(Macaca mulatta).*

Material and methods. ^{3}H-levonorgestrel (D-15,16-(^{3}H) 13β ethyl-17β-hydroxy-18,19-dinor-17-pregn-4-en-20-yn-3-one; sp.act. 39 Ci/mmole) was a generous gift from Schering AG, Berlin. The unlabeled levonorgestrel was obtained from Wyeth Chemicals, USA. Other radioinert steroids were authentic standards provided by Prof. D.N. Kirk, Steroid Reference Collection, England. Plasma samples were collected from healthy adult female monkeys (b.wt