Table 1. Labellar taste hair resistivity (MΩ) of males and females of *Phormia* when tested with NaCl, KCl and LiCl equiconductive solutions

	Test solution NaCl	KCl	LiCl
Males	39.23±0.71 (56)	38.58±0.73 (56)	38.95±0.67 (56)
Females	35.21±0.91 (40)	33.91±0.94 (40)	33.98±0.99 (40)

Mean values±SE. Males differ significantly from the corresponding females (Student's t-test, $p < 0.001$). The number of experiments in brackets.

Table 2. Labellar taste hair resistivity (MΩ) of males and females of *Phormia* when tested with $MgCl_2$, $CaCl_2$ and $BaCl_2$ equiconductive solutions

	Test solution $MgCl_2$	$CaCl_2$	$BaCl_2$
Males	50.35±0.69 (55)	50.27±0.96 (56)	51.97±1.17 (56)
Females	46.75±1.23 (38)	46.27±1.20 (38)	45.48±1.02 (39)

Mean values±SE. Males differ significantly from the corresponding females (Student's t-test, $p < 0.001$). The number of experiments in brackets.

$MgCl_2$, $CaCl_2$ or $BaCl_2$ equiconductive solutions. Also in this case, the differences were statistically significant (table 2). Since the resistivity of labellar hairs is always lower in female *Phormia*, irrespective of the kind of bathing solution, one can assume that the concentrations of the stimulating ions we used in our experiments is higher, at the chemosensory dendrites, in females than in males. In fact, the lower resistivity of labellar taste hairs of females indicates a higher ion diffusion from the ambient solution to the dendrite. Consequently, females can be affected to a greater extent by the stimulating action of ions.

In conclusion, it seems conceivable that our results can contribute to an explanation, on the basis of a different response to the environmental stimulating agents, of the unequal food intake of males as compared to females.

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Enhancement of ethanol-induced sleep by whole oil of nutmeg

C.J. Sherry and R.E. Burnett[1]

Department of Biology, College of Science, Texas A & M University, College Station (Texas 77843, USA), 1 September 1977

Summary. In young chickens, the whole oil of nutmeg (200 mg/kg) increased the duration of sleep induced by ethanol (1-4 g/kg), particularly deep sleep. Iproniazid (50-400 mg/kg), a monoamine oxidase inhibitor, did not mimic this effect.

Due to its easy availability and potential for abuse as a psychotropic agent (i.e. a hallucinogen)[2,3], the basic pharmacology of the whole nutmeg (N), which is the dried seed of the nutmeg tree (*Myristica fragrans* Houtt.), its essential oil (NO), and the constituents of the aromatic fraction of the essential oil (e.g. myristicin, elemicin, etc.) have received some attention in the literature[3-8]. The mechanism of action of N is still rather poorly understood, although it has been suggested that N, NO, or myristicin may act as a weak monoamine oxidase inhibitor (MAOI)[9,10]; that amphetamine derivatives might be formed from myristicin and/or elemicin[8,11]; or that they might be transformed into the amino derivatives, either by direct transamination or oxidation and transamination[4]. While myristicin, one of the key components of the aromatic fraction of the oil of nutmeg, has some psychotropic activity, it does not account for the activity of the whole oil in humans[3,6]. It should be noted that elemicin, which is also a component of the aromatic fraction of the oil, but usually present in smaller amounts than myristicin, is approximately twice as potent as myristicin in rodents[12]. The interactions of N, NO, and the potentially active components, myristicin and elemicin, with other pharmacological and particularly psychopharmacological agents have received relatively little attention[7]. Since it has been reported that alcohol and MAOIs synergize each other[13], and since it is likely that alcohol and nutmeg might be consumed together by drug experimenters, and since myristicin and elemicin have been reported to alter the duration of ethanol-induced sleep in rodents[7], we felt that it was important to determine the interactions of the whole oil of nutmeg, which is likely to be more potent than its fractions, with ethanol. Since we found that young chickens were relatively susceptible to the effects of the whole oil of nutmeg[14], we decided to determine the interactions of ethanol and the whole oil of nutmeg in the young chicken.

Methods. Male White Leghorn chickens were obtained at 1 day of age from the Kazmeier Hatchery (Bryan, Texas) and housed in temperature controlled brooders, with food and water available ad libitum. The whole oil of nutmeg (F.C.C. East Indian Extra) was obtained from Fritsche D&O (New York, N.Y.). Weighted samples (i.e. 200 mg) of the whole oil were dissolved in 10 ml of distilled water which contained 0.05 ml Triton X-100 (Sigma Chemical Co., St. Louis, Mo.). This allowed a dose level of 0.1 ml/g of b.wt. Iproniazid phosphate (Sigma) was dissolved in similar volumes of distilled water and administered at dose levels of 50, 100, 200, and 400 mg/kg. Ethanol 95% (IMC Chemicals, Harvey, La.) was appropriately diluted with distilled water to allow the same relative volumes of alcohol solution to be injected. The dose levels of alcohol were 1.0, 1.5, 2.0, 2.5, 3.0 and 4.0 g/kg. All drugs were

administered i.p. Immediately after injection, each animal was placed in a standard galvanized steel mouse cage (i.e. 17 × 24 × 17 cm), 1 animal per cage, and closely observed for 2 h (i.e. 7200 sec). After the initial 2 h, the animals were observed periodically, at approximately half-hour intervals, until the animal recovered from the drug effect. The average latency and duration of the 2 classes of behavioral sleep during the 2 h observation period were noted. The 2 classes of behavioral sleep were: 1. light sleep (i.e. the animal sat or stood quietly, without moving or peeping, with eyes closed, and head up); 2. deep sleep (i.e. the animal either sat down without moving or peeping, with eyes closed, and head down or lost posture and lay on his side). The total duration of drug effect (i.e. the total amount of time that the animals remained asleep), as noted above, is accurate to approximately 30 min (i.e. total sleep time in min ± 30 min).

Results. When examining table 1, it should be noted that when 200 mg/kg of oil of nutmeg was combined with any dose level of ethanol higher than 1.0 g/kg, there was a marked increase in average sleep time (i.e. average light sleep + average deep sleep). With increasing-dose levels of ethanol, there was an increase in the amount of time spent in deep sleep and an increase in the total sleep time. When these results are compared to those in table 2, where increasing dose levels of the MAOI, iproniazid phosphate, are combined with 3 g/kg ethanol, it should be noted that dose levels up to 400 mg/kg did not cause as large an increase in deep sleep as 200 mg/kg oil of nutmeg and 1.0 g/kg ethanol.

Discussion. It has been suggested that ethanol and/or its key metabolite, acetaldehyde, may alter the metabolism of the biogenic amines and that this alteration is probably due to a competitive inhibition of aldehyde dehydrogenase by acetaldehyde, which tends to cause the amine derived aldehyde to be reduced to the alcohol, rather than oxidized to the acid[15,16]. In addition, it has been suggested that MAOIs and ethanol synergize each other[13,17] and that MAOI may increase acetaldehyde levels in ethanol-treated mice[18]. By examining table 1, it can be clearly seen that NO causes a marked increase in duration of ethanol-induced sleep, particularly in time spent in deep sleep (i.e. a 17–20fold increase), while similar to higher dose levels of iproniazid, a MAOI, did not cause a similar increase in time spent in deep sleep. This is especially interesting in light of the fact that myristicin (100 mg/kg) slightly decreased and elemicin (100 mg/kg) slightly increased ethanol sleeping time in rodents[7]. It is therefore likely that the oil of nutmeg is a more potent MAOI than is suggested in the literature[9,10] and/or the oil has other pharmacological activity. We are currently investigating these possibilities, but we already have data that suggest that the oil of nutmeg may not synergize amphetamine[19], which would tend to indicate the pharmacology of the oil of nutmeg may be more complex than anticipated.

Table 1. The effect of 200 mg/kg oil of nutmeg (East Indian Extra) on duration of ethanol-induced sleep in the young chicken

Drug	Average sleep time		
	Light sleep	Deep sleep	Total sleep
200 mg/kg Nutmeg (N)	3,167 (1,629)	1,307 (721)	*
3 g/kg Alcohol	4,128 (1,632)	352 (421)	*
N + 1.0 g/kg Alcohol	3,552 (1,497)	1,163 (1,186)	11,472 (4,832)
N + 1.5 g/kg Alcohol	4,270 (1,578)	2,052 (942)	12,050 (4,435)
N + 2.0 g/kg Alcohol	4,335 (1,452)	2,064 (1,191)	18,150 (3,290)
N + 2.5 g/kg Alcohol	4,142 (1,488)	2,753 (1,595)	16,512 (4,083)
N + 3.0 g/kg Alcohol	769 (753)	6,297 (945)	21,720 (8,883)
N + 4.0 g/kg	20 (49)	7,035 (54)	

All times are expressed in sec. Light, deep and total sleep defined in text. The numbers in parentheses are the SD. 6 chicks were used at each dose level.
*Awake before 7,200 sec.

Table 2. The effect of varying dose levels of iproniazid phosphate (a monoamine oxidase inhibitor) on duration of sleep induced by 3.0 g/kg ethanol

Drug	Average sleep time	
	Light sleep	Deep sleep
50 mg/kg	1,214 (1,660)	544 (556)
100 mg/kg	1,518 (1,077)	381 (378)
200 mg/kg	4,219 (2,176)	400 (312)
400 mg/kg	4,912 (489)	1,150 (584)

All times are expressed in sec. The numbers in parentheses are the SD. 6 chicks were used at each dose level.

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