states, especially since the identification of a peripheral dopamine receptor-blocking agent, domperidone, which eliminates emetic and other peripheral side effects of dopamine agonists (*17*). Schizophrenia has recently been found to be dramatically improved by the administration of low, sedative doses of apomorphine (*18*). Since the underlying neurochemical defect of schizophrenia has been viewed as overactivity of dopamine neurons, apomorphine may exert its therapeutic action through presynaptic inhibition of dopaminergic neurotransmission. Therefore, as a selective presynaptic dopamine receptor agonist, TL-99 provides a pharmacologically unique alternative to classical neuroleptic therapy not only of schizophrenia, but also of other disease states, such as Huntington's chorea (*5*), hyperkinetic disorders (*4*), and tardive dyskinesias (*19*) where dopaminergic neuronal hyperactivity is the pathophysiological alteration.

DAVID B. GOODALE
DAVID B. RUSTERHOLZ, JOHN P. LONG
JAN R. FLYNN, BRIAN WALSH
JOSEPH G. CANNON, TERESA LEE
Departments of Pharmacology and *Toxicology, College of Medicine* and *Division of Medicinal Chemistry, College of Pharmacy, University of Iowa, Iowa City 52242*

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13. Test substances were administered 5 minutes before γ-hydroxybutyrate (750 mg/kg) was administered intraperitoneally; 5 minutes later a central decarboxylase inhibitor, *m*-hydroxybenzylhydrazine dihydrochloride (NSD 1015, 100 mg/kg) was administered intraperitoneally. The rats were decapitated 30 minutes later, and the brains were frozen in liquid nitrogen. A coronal section, 3 mm wide, was taken; the anterior and posterior limits of the olfactory tubercle being the landmarks for slicing on the ventral surface of the brain. Tissue punches, 4.5 mm in diameter, of the left and right corpus striatum were combined and placed in a 1.5-ml Sarstedt tube and weighed on a Mettler H15 balance. The combined striatal wet weights averaged 50.5±1.5 mg. Samples were then homogenized in 200 μl of 0.1*N* $HClO_4$ with 0.03 percent ascorbic acid and placed on ice for 30 minutes after manual homogenization. Thirty microliters of the sample were then injected by means of an on-line septum injector into a strong cationic exchange resin (Vydac) in a 750 by 2 mm microbore glass column. The mobile phase was a buffer solution of sodium acetate (5 m*M*) and sodium citrate (10 m*M*) with *p*H = 3.75. The mobile phase was pumped at 0.55 ml/min and the working electrode was held at a potential of +0.62 V compared to a standard calomel electrode. The dopa retention time was 8 minutes and dopa was clearly separated from the solvent front. Variability with repeated injections of the same sample was less than 1 percent, and the recovery of a known amount of the dopa standard homogenized with a preextracted tissue sample was 93.3 ± 0.5 percent (N = 4).
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Electrophysiological Correlates of Ethanol-Induced Sedation in Differentially Sensitive Lines of Mice

Abstract. *Acute electrophysiological effects of ethanol were studied in two lines of mice that differ markedly in their response to the soporific effects of systemic alcohol administration. Cerebellar Purkinje neurons from the genetic line that had long sleep times were one to two orders of magnitude more sensitive to the depressant effects of locally administered ethanol than those from the line that had short sleep times. The data suggest that there are genetically determined specificities in the acute effects of ethanol on central neurons and that such specificities might be used to determine which regions of the cerebellum participate in differences in behavioral responses to this substance.*

Among promising developments in analyzing acute effects of ethanol has been the selective breeding of mouse lines that differ markedly in sensitivity to the soporific effects of this drug. A given dose of ethanol produces a 25-fold longer sleep time in mice that have long sleep times (LS mice) than in those with short sleep times (SS mice) (*1*, *2*), as measured by the disappearance and return of the righting reflex. The rate of disappearance of ethanol from blood is identical in the two lines, and the alcohol content of the blood is much higher for SS mice than for LS mice at their respective times of awakening; this suggests a differential central nervous system sensitivity to the depressant effects of alcohol.

The cerebellum has been implicated in acute central nervous effects of ethanol both electrophysiologically (*3*, *4*) and behaviorally (*4*-*6*). We report here that cerebellar Purkinje neurons in LS mice show a much greater sensitivity to the depressant effects of ethanol than those in SS mice (*7*).

For these in situ experiments, mice were anesthetized with urethan and prepared for recording as previously described (*8*). Spontaneous discharge of Purkinje neurons, identified by their characteristic pattern of single and complex spikes, were recorded by use of multibarreled micropipettes (*9*). Ethanol was administered locally by pressure ejection from an adjacent barrel of the micropipette; release of substances from such pipettes is linearly related to ejection time and pressure (*10*). Effects of alcohol were considered reliable only if they could be obtained at least twice, with recovery of control values in each case. Rate-meter records of electrophysiological changes were quantitated with a digitizing tablet and computer (*11*).

The most striking finding in this study was the marked difference in sensitivity of cerebellar Purkinje cells to ethanol in the different lines of mice (Fig. 1). For each of the 87 neurons suitable for analysis and shown in the bar graph, we se-

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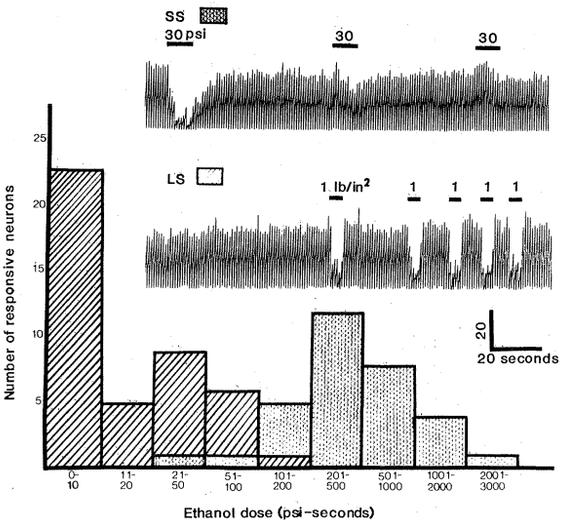

Fig. 1. Effects of acute ethanol administration by micropressure ejection on spontaneous activity of Purkinje neurons in situ. The number of neurons is plotted against the minimum ethanol dose in pounds per square inch × seconds required to elicit significant depression (30 to 70 percent inhibition). The difference between the mean ethanol dose needed to produce roughly half-maximal depressions of spontaneous Purkinje cell firing in SS mice (888 ± 147 psi-seconds; average depression, 52 percent) and LS mice (29 ± 5.5 psi-seconds; average depression 54 percent) was significant at $P < .001$. Nonlinearity of abscissa permits display of LS and SS data on the same graph. (Inset) Representative rate-meter records of spontaneous firing from single Purkinje cells. There is a reproducible depression with repeated ethanol application at 5 psi-seconds for the LS neuron. Acute tolerance of response to ethanol at 300 psi-seconds is shown for the SS neuron; the first depressant response is half-maximal, the second response is just threshold, and subsequent ethanol applications elicited no depression.

lected an ethanol dose sufficient to elicit a 30 to 70 percent inhibition of spontaneous Purkinje cell firing. Threshold or supramaximal responses were not used for these calculations to avoid problems in interpretation (*12*). The doses needed to produce similar effects in the two lines differed by at least 1.5 orders of magnitude, with SS neurons requiring a mean of 888 psi-seconds (pounds per square inch × seconds) compared to 29 psi-seconds for LS neurons. This difference was highly significant ($P < .001$). Moreover, often only the initial response of cells from the SS mice could be used, since many cells showed rapid onset of tolerance (*13*) to the effects of ethanol after several applications (Fig. 1, rate-meter inset). No tolerance was seen in any Purkinje neuron from LS mice (Fig. 1, rate-meter inset). Pressure ejection of saline or of 670 m*M* sucrose in saline from another barrel of the pipette, at similar times and pressures, did not alter Purkinje cell discharge in either LS (Fig. 2) or SS mice. Moreover, in a number of cases, cells from LS and SS animals were alternately recorded, with ethanol ejected from the same multibarreled micropipette, to ensure that differences in ethanol release were not involved in the differential sensitivity to this substance.

The correlation of behavioral differences between the two mouse lines to cerebellar electrophysiological differences suggests that the behavioral depressant effects of ethanol are due to direct effects in the central nervous system. The amount of ethanol administered by micropressure ejection would be insufficient (*10*) to influence peripheral metabolism. Moreover, since the two mouse lines had similar time courses of changes in the alcohol content of the blood after parenteral injection, simple pharmacokinetic differences are unlikely (*2*).

The previously reported differences in ethanol-induced behavioral depression in LS and SS mice are reflected in profound differences in electrophysiological activity of Purkinje neurons. If ethanol has a direct action on the molecular structure of protein or lipid constituents in excitable membranes (*13*), then a differential distribution of such ethanol-sensitive elements must exist in LS and SS mice. Biochemical studies of the cerebellum in these lines of mice may provide definitive answers as to the locus of ethanol action at the macromolecular level.

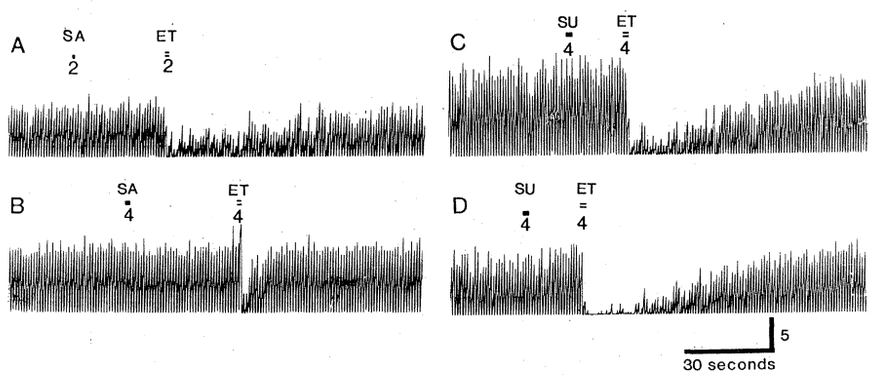

Fig. 2. Rate-meter records showing a comparison of the effects of pressure ejection of ethanol (*ET*), saline (*SA*), and sucrose (*SU*) on Purkinje cell discharge. Traces A to D are from four different LS mice; the duration of ejection is indicated by the single or double bar over each trace, and ejection pressure (pounds per square inch) is given by the underlying number. Note that, in each case, ethanol elicits a potent depression of spontaneous activity whereas normal saline or a sucrose solution isotonic to the ethanol (*9*), administered from an adjacent barrel, has no effect.

STEVE SORENSEN
MICHAEL PALMER
THOMAS DUNWIDDIE
BARRY HOFFER

Department of Pharmacology, Alcohol Research Center, University of Colorado Health Sciences Center, Denver 80262

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7. Mice from the LS and SS lines were obtained from the Institute of Behavioral Genetics [G. E. McClearn, G. Lindzey, G. G. Lindzey, D. D. Thiessen, Eds., *Contributions to Behavior—Genetic Analysis: The Mouse as a Prototype* (Appleton-Century-Crofts, New York, 1970), p. 3]. The animals used in this study were from the 25th generation. Behavioral studies on other animals from this generation showed that the differences in sleep times were consistent with those of earlier generations.
8. Our electrophysiological techniques for recording from cerebellum have been reported [B. J. Hoffer, G. R. Siggins, A. P. Oliver, F. E. Bloom, *J. Pharmacol. Exp. Ther.* **184**, 553 (1973); R. Freedman, B. J. Hoffer, D. J. Woodward, *Br. J. Pharmacol.* **54**, 529 (1975); D. Taylor, J. Nathanson, B. Hoffer, L. Olson, A. Seiger, *J. Pharmacol. Exp. Ther.* **206**, 371 (1978)]. Mice were anesthetized with urethan administered intraperitoneally. The animals were intubated, allowed to breathe spontaneously, and placed in a stereotaxic instrument. The bone over the vermis was removed and, after cisternal drainage, the dura was incised. The surface of the cerebellum was then covered with warm 2 percent agar in saline. Spontaneous discharge of Purkinje neurons, identified by their characteristic pattern of complex and simple spikes, was recorded with multibarreled micropipettes.
9. For multibarreled micropipettes, five glass fiber-filled capillary tubes (Corning 7740; 1.5 mm outer diameter, 1.1 mm inner diameter) were ce-

mented together with ends staggered and pulled to a total tip diameter of 5 to 6 μm. The recording barrel was filled with 5*M* NaCl. The barrels to be used for pressure ejection were filled with 750 m*M* ethanol in normal saline, with normal saline alone, or with 670 m*M* sucrose in normal saline, which is isosmotic with the ethanol solution.

10. For pressure ejection of ethanol, nitrogen gas at 100 psi was connected to a pressure ejection control unit (Medical Systems), permitting precise control of the amplitude and time of pressure applied to the ethanol-containing pipette barrels. All micropressure ejection doses are given as pounds per square inch times seconds. Recent investigations with micropressure ejection have shown that release of drug volume is reproducible and is linearly related to both time and pressure [R. F. McCaman, D. G. McKenna, J. K. Ono, *Brain Res.* **136**, 141 (1977); M. Sakai, B. Swartz, C. Woody, *Neuropharmacology* **18**, 209 (1979); M. Palmer, S. Wuerthele, B. Hoffer, in preparation]. Furthermore, these studies suggest that the pressure and time parameters can be interchanged (that is, 5 psi × 10 seconds equals 10 psi × 5 seconds) without significantly altering the volume of drug released, and also suggest that there is little drug leakage between ejection trials. In addition, these studies show that the volume ejected in a 50 psi-second pulse is on the order of 10^{-5} μl, so that even though there is an ethanol concentration of 750 m*M* in the micropipette barrel, the concentration at the cell membrane is far less.
11. The rate of spontaneous discharge of Purkinje cells was integrated over 1-second intervals and displayed as spikes per second on an inkwriter. These ratemeter records were subsequently digitized on a digitizing tablet interfaced with a Data General NOVA 3/12 computer. With appropriate software, it was possible to determine the average discharge rate before, during, and after ethanol administration and to compute the percent changes. To be used for analysis, all single-unit recordings had to show stable patterns during the control period, consistent changes after ethanol administration, and subsequent recoveries of stable patterns. This usually required continuous recording for 1 to 3 hours.
12. If a response is supramaximal, the question of whether a smaller dose of ethanol would give the same response arises. If a response is just threshold or subthreshold, it is conceivable that a larger dose of ethanol could elicit an effect of similar magnitude. Hence, after determinations of dose-response relationships for each Purkinje cell, the dose giving a 30 to 70 percent response was selected because it was certain that larger and smaller doses would elicit greater and lesser effects, respectively. Overall, however, the mean depression in the firing rate of the population of LS Purkinje cells produced by an average dose of 29 psi-seconds was 54 percent, and the mean depression in firing rate for the population of SS Purkinje cells produced by an average dose of 888 psi-seconds was 52 percent. There was no significant difference in these mean population depressions.
13. The question of long-term tolerance has been extensively reviewed [D. B. Goldstein, *Fed. Proc. Fed. Am. Soc. Exp. Biol.* **39**, 1943 (1975); E. Majchrowicz, *Psychopharmacologia* **43**, 245 (1975)] and has been seen electrophysiologically in the hippocampal slice in vitro [W. A. Corrigal and P. L. Carlen, *Proc. Annu. Meet. Soc. Neurosci 8th*. (1978), Abstr. 1329]. It has been shown that SS mice develop tolerance to the hypnotic effects of alcohol much more rapidly than LS mice do, with a regimen of multiple injections of ethanol [B. Tabakoff, R. Ritzman, T. Raju, R. Deitrich, *Alcoholism Clin. Exp. Res.* **4**, 70 (1980)]. Short-term tolerance to ethanol has also been described with respect to motor behavior [A. E. LeBlanc, H. Kalant, R. Gibbons, *Psycholpharmacologia* **41**, 43 (1975)], nystagmus [I. A. Mirsky, P. Piker, M. Rosenblum, M. Lederer, *Q. J. Stud. Alcohol* **2**, 35 (1941)], and EEG phenomena [L. V. Story, E. Eidelberg, J. French, *Arch. Neurol.* **5**, 565 (1961)].
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Osmolality and Potassium Ion: Their Roles in Initiation of Sperm Motility in Teleosts

Abstract. *Spermatozoa that are quiescent in electrolyte and nonelectrolyte solutions isotonic to seminal plasma show motility when the semen is diluted with hypotonic solution in freshwater teleosts (four species tested) and with hypertonic solution in marine teleosts (five species tested). Decrease or increase, respectively, in osmolality of the environment may be the factor initiating sperm motility in these species. The motility of chum salmon spermatozoa in a sodium chloride solution isotonic to seminal plasma is completely suppressed by approximately 10 millimoles of potassium per kilogram; topminnow spermatozoa, however, were immotile in a nonelectrolyte solution, and motility was induced by electrolytes, especially potassium. Thus ions, rather than osmolality, may be an essential determinant of sperm motility in salmonid and viviparous teleosts.*

In many animals, hypotonicity is harmful to sperm structure and sperm motility. Sperm heads of marine invertebrates swell immediately after immersion in media hypotonic to seawater. Diluted Ringer solution causes the sperm tails of mammals to spiral and cease motility (*1*). In salmonids, the life span of spermatozoa is longer in 20 percent seawater than in freshwater (*2*). In teleosts, spermatozoa that are immotile when normally suspended in a seminal plasma having an osmolality of about 300 to 350 mosmole/kg in freshwater or marine species (*3*) or containing high concentrations of potassium ion in Salmonidae (*4*) become exposed at spawning to environments that are hypotonic, hypertonic, or low in K^+, respectively.

In a freshwater teleost, the goldfish *Carassius auratus*, and in a marine teleost, the puffer *Fugu niphobles*, the sperm were immotile when the semen (*5*) was mixed with 100 to 200 volumes of either an electrolyte (150 mmole/kg NaCl or KCl) or a nonelectrolyte (300 mmole/kg mannitol) solution isotonic to the seminal plasma (300 mosmole/kg) (Fig. 1, a and b). Motility occurred only when the semen was diluted with a solution (electrolyte or nonelectrolyte) of lower osmolality for the freshwater species or higher osmolality for the marine species. Thus, the experimental conditions parallel the natural conditions in which sperm motility is suppressed by the osmolality isotonic to the seminal plasma in the testis and initiated by a decrease or an increase in the osmolality of the environment surrounding the spermatozoa at spawning. Duration of sperm motility in the goldfish increased with a decrease in osmolality in both NaCl and mannitol solutions, reaching a maximum motility at 100 mosmole/kg before decreasing. This decrease in amount and duration of sperm motility is probably the result of disruption of sperm structure by hypoosmotic shock, since swollen sperm heads and fragmented tails were noted. Because spermatozoa have a longer life span at an osmolality between 100 and 200 mosmole/kg, they must reach spawned oocytes within a short period in an environment constituted by the mixture of seminal plasma and freshwater. In fact, the duration of sperm motility of teleosts—including goldfish—is relatively short, and generally the mature male of many fishes approaches the female and releases spermatozoa immediately after oviposition. In the puffer, the duration of sperm motility reached a maximum at 400 mosmole/kg and then decreased with further increase in osmolality. The spermatozoa ceased to move at osmolalities (1200 mosmole/kg) above that of seawater (1000 mosmole/kg).

Sperm motility was induced in other freshwater teleosts—*Cyprinus carpio, Carassius carassius*, and *Tribolodon hakonensis*—by reducing the osmolality, and in other marine teleosts—the cod *Gadus morrhua macrocephalus* and the flounders *Limanda yokohamae, Microstomus achne*, and *Kareius bicoloratus*—by increasing the osmolality. In the seawater teleosts, sperm motility ceased at an osmolality greater than 1200 mosmole/kg.

Spermatozoa of the chum salmon *Oncorhynchus keta*, which belongs to a primitive group of Teleostei, the Salmonidae, were motile in NaCl solutions between 0 and 500 mosmole/kg (Fig. 1c); osmolalities of 300 to 400 mosmole/kg, which are similar to that of the seminal plasma of chum salmon (*4*), did not inhibit motility. The seminal plasma of the rainbow trout *Salmo gairdnerii* contains a high concentration of K^+, which inhibits sperm motility, and K^+ is also a major constituent of the seminal plasma of chum salmon (*4*). In our study, sperm motility in chum salmon and rainbow trout (*6*) did not occur at any KCl concentration examined. When ion concentration was increased by adding various concentrations of KCl to 100 mmole/kg NaCl, sperm motility ceased, although

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