the seeded cloud passed over the radar.

The seeding of the two tracks described above produced significant changes in the structures of the clouds that enhanced their radar reflectivities. However, it appeared that the seeding was being carried out too close to the radar to allow sufficient time for the large particles produced by the seeding to reach the ground at the radar site. Consequently, the third seeded track was located 18 km upwind of the radar (twice the spacing used in the second track). In addition, the rate of Dry Ice seeding was doubled (to 0.1 kg km^{-1}).

The third seeded cloud passed over the radar between ~ 1326 and 1328 PST (~ 40 minutes after seeding). Precipitation trails from this cloud reached the ground between ~ 1331 and 1334 PST (Fig. 1). (An expanded view of the radar display of this seeded track is shown in Fig. 3, where various radar echo intensities are depicted in different colors.) The amount of precipitation that reached the ground at the radar site was quite small (observers on the ground reported a trace). The fact that the radar detected a strong signal all the way to the ground illustrates its sensitivity. The particle size spectra measured in this third seeded track are shown as curves A and B in Fig. 2c; curve C shows the measured spectrum in an adjacent cloud that was not seeded. The spectrum shown in curve B was measured at an altitude of 1.6 km above MSL (−4.2°C) just 1 minute after the seeded cloud passed over the radar. Aggregates of ice particles 2 mm in size in concentrations of ~ 30 per liter were measured on this pass.

Each one of the three seeded tracks passed over the radar within a few minutes of the times predicted by the scientist aboard the B-23 aircraft. These predictions were based on the location of the seeding with respect to the radar and the velocity of the winds.

These observations illustrate that a short-wavelength radar can provide a unique and powerful tool in evaluating the effects of cloud seeding. It permits continuous remote sensing of precipitable particles from cloud to ground, a capability that thus far has not been used in cloud-seeding research.

PETER V. HOBBS
JAMIE H. LYONS
JOHN D. LOCATELLI
KUMUD R. BISWAS
LAWRENCE F. RADKE
RICHARD R. WEISS, SR.
ARTHUR L. RANGNO
Atmospheric Sciences Department, University of Washington, Seattle 98195

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2. Our 8.6-mm-wavelength radar is an adaptation based on the use of modern technology of the AN/TPQ-11 radar developed by the Air Force [W. H. Paulsen, P. J. Petrocchi, G. McLean, *Operational Utilization of the AN/TPQ-11 Cloud-Detection Radar* (Instrumentation Papers, No. 166, Air Force Cambridge Research Laboratories, L. G. Hanscom Field, Bedford, Mass., 1970)].
3. There have been a few reports describing the use of radars with wavelengths of several centimeters for detecting precipitation produced by artificial seeding. For example, D. Atlas [*Bull. Am. Meteorol. Soc.* **46**, 696 (1965)] mentioned some demonstrations of this type in the Soviet Union. P. V. Hobbs [*J. Appl. Meteorol.* **14**, 805 (1975)] described the use of a 3.2-cm Doppler radar for detecting changes in particle fallspeeds produced by seeding. However, to our knowledge, the radar measurements reported here are unique in documenting the effects of cloud seeding from cloud to ground.
4. The University of Washington's B-23 research aircraft was used in these experiments. The extensive instrumentation aboard this aircraft for measuring the properties of clouds and precipitation has been described by P. V. Hobbs, T. J. Matejka, P. H. Herzegh, J. D. Locatelli, and R. A. Houze, Jr. [*J. Atmos. Sci.* **37**, 568 (1980)].
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Sodium Pump in Skeletal Muscle: Central Nervous System–Induced Suppression by α-Adrenoreceptors

Abstract. *The central nervous system regulates active sodium and potassium transport in rat skeletal muscle during hypokalemia. This regulation is achieved by the apparent release of catecholamines onto muscle following nerve activity. This effect can be prevented by treatment with agents that block the α-adrenoreceptor.*

Potassium-deficient diets cause a decrease in K^+ concentration in blood plasma and skeletal muscles but not in cerebrospinal fluid or brain tissue. During the first 4 to 5 weeks of potassium deficiency, decreases in the rat muscle K^+ are not compensated by equivalent increases in muscle Na^+. However, during further hypokalemia, the ratio of the number of Na ions entering the muscle to that of K ions coming out approaches one (*1*). The plasma K^+ concentration in hypokalemic rats decreases from a normal level of about 4.6 m*M* to about 1.6 m*M*, a value that is sufficiently high to maintain activity of the Na-K pump in muscle. Furthermore, when muscles from hypokalemic rats are excised and placed in a Krebs solution at 37°C, the intracellular Na^+ concentration, $[Na]_i$, is promptly reduced, whereas the intracellular K^+, $[K]_i$, is increased even when the external K^+ concentration is as low as 1 m*M* (*2*). Therefore, in hypokalemic rats the high $[Na]_i$ and low $[K]_i$ of muscles in situ cannot be attributed to pump inhibition by plasma hypokalemia; there must be an additional mechanism by which activity of the Na-K pump in muscle of hypokalemic rats is suppressed in vivo.

We report here that sectioning of a peripheral nerve, decerebration, or spinal transection can remove suppression of the active Na-K transport in rat soleus muscle during hypokalemia and that the neurally mediated inhibition of the muscle Na-K pump is also abolished by α-adrenoreceptor blocking agents.

The experiments were performed on soleus muscles of male Wistar rats that had been given free access to a potassium-deficient diet and deionized water for 5 to 7 weeks. Chemical analysis of Na^+ and K^+ contents in muscles and plasma samples were carried out with a flame spectrophotometer. The $[Na]_i$ and $[K]_i$ of tissues were calculated from the tissue cation contents, plasma cation contents, extracellular space, and the ratio of dry weight to wet weight according to the conventional method described by Desmedt (*3*).

The most striking finding was a dramatic reversal of the effects of hypokalemia on muscle $[Na]_i$ and $[K]_i$ by the sectioning of the tibial nerve branch or the sciatic nerve (Fig. 1 and Table 1) (*4*). After denervation, the soleus muscles promptly restored their $[Na]_i$ and $[K]_i$ to values close to those of "fresh" soleus muscles of normal rats (*5*). The half-recovery time of these internal cations was about 20 minutes. The recovery values leveled off within a few hours after denervation and the recovered $[Na]_i$ and $[K]_i$ remained unchanged for several days. The results under different experimental conditions are summarized in Table 1. When ouabain, a specific inhibitor of the Na-K pump, was administered prior to denervation, there was no recovery of the effects of hypokalemia on $[Na]_i$ or $[K]_i$ after denervation (see Table 1). Thus, the ouabain-sensitive Na-K pump must be involved in the denervation-induced recovery of the sodium and potassium contents of the muscle of hypokalemic rats.

Further characterization of the neural

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control of the active Na-K transport was achieved by transecting the spinal cord at the level of C_3 to C_5, during which time the animals were maintained on an artificial respirator. The effects of both brainstem transection and decerebration were also examined. As shown in Table 1, these treatments had the same effect on muscle Na^+ and K^+ contents as did peripheral denervation. Furthermore, there were no differences among the time courses in the dramatic shifts of $[Na]_i$ and $[K]_i$ after peripheral sections, central nervous system (CNS) transection, and decerebration. These results suggest that the CNS of hypokalemic rats may inhibit the Na-K pump mechanism. It becomes important to know whether the activation of the muscle Na-K pump may result from the deprivation of neural activity or whether the interruption of axonal flow may be responsible for it. It is well known that tetrodotoxin (TTX) blocks nerve conduction but not axonal flow. Therefore, the sciatic nerve on one side of hypokalemic rats was treated with TTX by means of the slow-release technique (*6*). After 3 hours, both innervated soleus muscles were dissected out and analyzed for their Na^+ and K^+ content. In the innervated muscles on the TTX-treated side, Na-K transport was considerably activated. This result suggests that some factors associated with peripheral neural activity suppresses Na-K transport in the soleus muscle of hypokalemic rats.

We then examined the possibility that a neurotransmitter receptor was involved in the suppression of the Na-K pump by the CNS. The block of neuromuscular transmission by curare did not activate the Na-K transport in soleus muscles of hypokalemic rats. In addition, the activation of Na-K transport after denervation of curarized hypokalemic rats was completely removed by electrical stimulation of the distal stump of the cut sciatic nerve (*7*). Thus, it seems clear that the muscle Na-K pump during hypokalemia is regulated by neural activity. This complements the interpretation of the TTX data described above.

According to Dury (*8*) and Leitch *et al.* (*9*), catecholamines produce hypokalemia in the intact organism. Moreover, catecholamines can augment the resting membrane potential of skeletal muscle isolated from normal rats by activation of the β-adrenoreceptors (*10*). Therefore, I used agents that block the α- and β-adrenoreceptors (*11*) to determine whether catecholamines might mediate the CNS-induced inhibition of the muscle Na-K pump during hypokalemia. At the dosages tested, three α-adrenoreceptor antagonists (dibenamine, phentolamine, and phenoxybenzamine) markedly reduced the CNS-induced inhibition of the active transport. Propranolol, a β-adrenoreceptor antagonist, did not produce any detectable changes in internal Na^+ and K^+ contents. These results suggest that an α-adrenoreceptor on the muscle membrane of hypokalemic rats plays an important role in mediating the CNS-induced inhibition of active Na-K transport and that the endogenous transmitter may be norepinephrine (*12*).

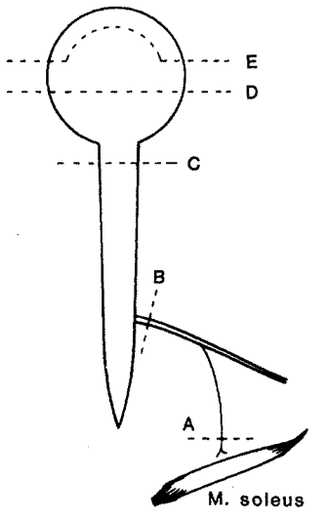

Fig. 1. Schematic illustration of the experimental arrangement of denervation of rat soleus muscle during hypokalemia. Dashed lines connected with *A*, *B*, *C*, *D*, and *E* indicate the transection levels: *A*, tibial nerve branch; *B*, sciatic nerve; *C*, spinal cord at C_3–C_5; *D*, brain stem; and *E*, cerebrum.

These experiments show that the CNS of hypokalemic rats suppresses the muscle Na-K pump. It appears that CNS-induced inhibition of the pump is not mediated by acetylcholine at the neuromuscular junction nor by blood-borne hormones but probably by catecholamines released from the peripheral nerve. According to Tashiro (*13*) and Clausen and Flatman (*14*), epinephrine and isoproterenol stimulate Na^+ efflux and K^+ uptake in fresh soleus and extensor digitorum longus muscles isolated from normal rat or guinea pig, and the effects are mediated by way of β-adrenoreceptors. It is likely that both adrenergic α- and β-receptors may exist on the mammalian skeletal muscle membrane and that the catecholamine-induced stimulation of the Na-K pump may result from activation of the β-adrenoreceptor or from inhibition of the α-adrenoreceptor. Which of these effects predominates may depend on the condition of the animal.

Potassium-deficient diets cause hypokalemia and a considerable decrease in cellular K^+ in skeletal and smooth mus-

Table 1. Electrolyte contents in rat soleus muscle during hypokalemia. Muscles were maintained in vivo for 3 hours after denervation or drug treatments, and then prepared for analysis of electrolytes. In curare- or atropine-treated animals, however, the muscles were dissected out after 1 hour of the treatment. The drugs were used in the following dosages (per kilogram of body weight): ouabain, 1 to 3 mg intravenously; atropine, 1.5 mg intraperitoneally; phenoxybenzamine and dibenamine, 1 mg intraperitoneally; phentolamine, 1 mg intraperitoneally for each hour; propranolol, 0.5 mg intraperitoneally; and tetrodotoxin, 10^{-5} g/ml. Plus signs indicate the activation of muscle Na-K transport; minus signs indicate no significant change of internal cation contents by the treatments. Internal cation concentrations are given as means ± standard deviation. The number of animals is shown in parentheses.

Experimental conditions	Activation of sodium pump	Intracellular electrolyte concentration (mmole/liter fiber water)	
		Sodium	Potassium
Control		73.3 ± 1.5 (25)	75.4 ± 1.9
Tibial nerve cut	+	36.8 ± 2.2* (11)	113.1 ± 2.8*
Sciatic nerve cut	+	38.6 ± 1.4* (15)	116.0 ± 3.5*
Spinal cord transection	+	39.2 ± 0.9* (12)	108.9 ± 1.3*
Brainstem transection	+	42.7 ± 1.2* (7)	106.1 ± 3.6*
Decerebration	+	40.7 ± 2.0* (8)	108.0 ± 3.2*
Ouabain plus denervation	−	71.8 ± 3.0 (10)	80.5 ± 3.2
Tetrodotoxin	+	43.3 ± 2.1* (6)	102.5 ± 2.8*
Curare	−	72.2 ± 3.1 (6)	74.5 ± 2.3
Curare plus denervation	+	44.4 ± 2.7* (8)	108.5 ± 2.2*
Curare plus denervation plus electrical stimulation	−	70.1 ± 3.0 (8)	77.1 ± 3.1
Atropine	−	73.4 ± 2.7 (6)	78.1 ± 2.3
Atropine plus denervation	+	42.0 ± 3.3* (6)	105.2 ± 3.1*
Dibenamine	+	48.7 ± 2.2* (7)	97.2 ± 3.4*
Phentolamine	+	50.7 ± 2.7* (7)	91.2 ± 2.9*
Phenoxybenzamine	+	49.0 ± 1.8* (8)	93.7 ± 3.0*
Propranolol	−	71.1 ± 3.1 (8)	78.3 ± 3.3

*Significant difference at $P < .001$ between control and treated muscles.

cles (*2*, *13*, *14*). However, the CNS, including the cerebrum, cerebellum, medulla oblongata, and spinal cord, is spared this K^+ decrease, presumably as a result of the maintenance of normal K^+ levels in the cerebrospinal fluid by the choroid plexus (*15*). Also, heart and liver can maintain their normal $[Na]_i$ and $[K]_i$ values even though these organs are equally exposed to plasma hypokalemia (*16*). Perhaps the CNS can influence the distribution of Na^+ and K^+ in the body by selectively inhibiting the pump in certain skeletal and smooth muscles during hypokalemia. At least in the initial stages of hypokalemia, such peripheral organs may serve as a reservoir for K^+ without seriously compromising their function. Consequently, a slow net release of K^+ from these organs may serve as a buffer between the animal's intake of K^+ and its plasma K^+ concentration, thus maintaining homeostasis.

NORIO AKAIKE

Department of Pharmacology, Kumamoto University Medical School, Kumamoto 860, Japan

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5. The values for $[Na]_i$ and $[K]_i$ in 30 fresh muscles in normal rats are (mean ± 1 standard deviation) 24.2 ± 3.2 and 134.1 ± 7.4 mmole per liter of fiber water, respectively [see Akaike (*2*)].
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Release of Immunoreactive Serotonin into the Lumen of the Feline Gut in Response to Vagal Nerve Stimulation

Abstract. *Immunoreactive serotonin was detected in the lumen of the proximal jejunum of food-deprived cats. During perfusion of this intestinal segment in vivo, there was a constant basal rate of intraluminal secretion of this amine. The rate of secretion was significantly increased during efferent electrical stimulation of the cut cervical vagal nerves. This stimulatory effect was not altered after bilateral adrenalectomy was performed in the same animals. A synchronous release of substance P into the gut lumen was also demonstrated during vagal stimulation. During the period of increased intraluminal secretion of immunoreactive serotonin, there was no demonstrable change in the portal or systemic blood levels of this amine.*

In the mammalian gastrointestinal tract, serotonin is largely stored within the enterochromaffin cells (EC) of the intestinal mucosa (*1*). Efferent electrical stimulation of the feline cervical vagal nerves in vivo decreases the amount of serotonin in individual EC cells prepared for fluorescence microscopy by the Hillarp-Falck technique for visualizing intracellular monoamines (*2*). This observation supports the concept of a vagal release of the amine from these cells (*2*). Furthermore, a direct innervation of EC cells has been shown at the ultrastructural level (*3*). Hypothetically, the released amine may be extruded through the basolateral membrane of these cells and diffuse into subepithelial capillaries or may be secreted into the gut lumen through the apical cytoplasmic processes, or perhaps both. Ultrastructural studies of vagally stimulated gut mucosa obtained from biopsies demonstrated signs of active exocytosis of the basolateral membrane but also indicated accumulation of electron-dense material in the most apical portions of the cells (*4*). Combined electron microscopy and autoradiography after injection of 5-[^{3}H]hydroxytryptophan (the serotonin precursor) demonstrated release of the radioactive tracer into the gut lumen in response to electrical vagal nerve stimulation (*5*). We have now established the presence of intraluminal serotonin in the feline proximal jejunum and determined the effect of nerve stimulation in vivo on the levels of this amine in the lumen and in the portal and femoral veins.

Six cats weighing 2.6 kg were deprived of food for 18 hours. The cats were anesthetized with chloralose (100 mg/kg, intravenously), and a 15-cm segment of the proximal jejunum was isolated by dividing the bowel but not the mesentery; the lumen was constantly perfused with saline (37°C) at a rate of 1.0 ml/min. Venous blood samples were drawn from heparinized Teflon catheters in the portal and left femoral veins, and the perfusates were collected every 5 minutes. The concentrations of serotonin were measured by a sensitive radioimmunoassay technique developed in our laboratory (*6*); extractions of serotonin from the blood samples and perfusates were carried out with the same technique (*6*). Recovery of both ^{3}H-labeled and unlabeled serotonin added to portions of the blood samples averaged 64.5 ± 1.9 percent and to portions of the perfusates averaged 91.2 ± 2.4 percent. High intraluminal levels of immunoreactive serotonin were detected (80 to 720 ng per 5 minutes) during a 15-minute washout period. After the washout, the basal rate of intraluminal serotonin secretion averaged 64 ± 12 ng per 5 minutes ($N = 6$). The presence of serotonin in the intestinal lumen was confirmed by high-performance liquid chromatography (*7*), which yielded comparable data.

After two such basal periods, bilateral efferent electrical stimulation (10 V, 5 msec, 8 to 10 Hz) of the cut cervical vagal nerves inserted into silver ring electrodes caused a rapid increase in the rate of secretion of the amine in all six cats (Table 1). The *t*-test for paired data was used to confirm that the 1.93 ± 0.35-fold increased rate of luminal secretion of serotonin during vagal stimulation was significantly ($P < .01$) higher than the basal rate. When stimulation was stopped, the rate of intraluminal secretion of immunoreactive serotonin abruptly returned to the basal value.

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