though the normal patterns remain intact. This has been observed in hypocalcemic hypoparathyroid children who daily are treated with two suboptimal doses of 1,25-dihydroxyvitamin D_3 (*13*).

Our data establish the presence of circadian patterns of Ca^{2+} and P_i in healthy, young adult males. Models of these patterns provide a physiological basis for future studies of blood minerals and calcium-regulating hormones in other healthy populations and at specific stages of metabolic bone disease.

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Brain Acetylcholine Synthesis Declines with Senescence

Abstract. *The synthesis of whole brain acetylcholine is reduced in two strains (C57BL and BALB/c) of senescent mice. The incorporation of [U-^{14}C]glucose into acetylcholine decreased in both strains by 40 ± 4 percent in 10-month-old mice and by 58 ± 9 percent in 30-month-old mice compared with mice 3 months old. The incorporation of [$^2H_4$]choline into acetylcholine declined 60 and 73 percent in 10- and 30-month-old mice, respectively. Deficits in the cholinergic system may contribute to brain dysfunctions that complicate senescence.*

Senescence is a complicated process characterized by many morphological and chemical alterations in the brain. Cognitive function declines with aging in humans and other animals (*1*). The biochemical mechanism of this decrease is unknown, however. Several studies suggest that impaired cholinergic function may be an important factor in the production of geriatric memory deficits. Pharmacological manipulation of the cholinergic system in aged animals or in humans (*1–3*) implies an age-related decrement in cholinergic function. Neurophysiological studies also demonstrate these reductions (*4*, *5*). Cholinergic receptors, choline acetyltransferase, and acetylcholinesterase activities in the brain are reduced with aging and further depressed with Alzheimer-type dementia (*6*, *6a*, *7*, *8*). Some of these changes are controversial. For example, Davies and Verth (*7*) found no decrease in muscarinic receptors with aging in humans. Furthermore, the decline may be specific to discrete brain regions, since Morin and Wasterlain (*8*) observed a decrease in receptors in the striatum and cerebellum but not in the hippocampus, hypothalamus, or amygdala. Similar disputes exist over the decline in choline acetyltransferase and acetylcholinesterase.

Brain oxidative metabolism decreases with senescence in brain slices, homogenates, mitochondria, and in vivo (*9*, *9a*). In aged patients, decrements in cerebral oxygen consumption correlate with the degree of dementia, but in normal elderly subjects, inconsistent changes are reported (*10*). In vitro, reduced oxygen utilization produces a proportional inhibition in acetylcholine synthesis even though less than 1 percent of the oxidized precursor becomes acetylcholine

Table 1. Lactate, acetylcholine, and choline brain concentrations (in nanomoles per milligram of protein) in senescent mice. Values are means ± standard errors of the means. Lactate was determined fluorometrically. Acetylcholine and choline were separated and their concentrations measured by gas chromatography–mass spectrometry. Protein was determined by the biuret reaction with bovine serum albumin as the standard (*14*).

Strain	Age (months)		
	3 (N = 11)	10 (N = 11)	30 (N = 11)
	Lactate		
BALB/c	9.47 ± 0.62	11.59 ± 0.99	12.92 ± 1.18*
C57BL	10.80 ± 0.73	11.61 ± 0.80	27.08 ± 4.60†
	Acetylcholine		
BALB/c	0.26 ± 0.02	0.26 ± 0.01	0.22 ± 0.01
C57BL	0.33 ± 0.02	0.32 ± 0.02	0.29 ± 0.02
	Choline		
BALB/c	0.45 ± 0.04	0.46 ± 0.07	0.45 ± 0.07
C57BL	0.42 ± 0.06	0.42 ± 0.07	0.42 ± 0.08

*Significantly different ($P < .05$) from 3-month-old mice (analysis of variance with least significant difference multiple comparison test) (*15*). †Significantly different ($P < .05$) from both 3-month-old and 10-month-old mice.

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(*11*). In vivo, histotoxic hypoxia (injection of KCN), anemic hypoxia (injection of $NaNO_2$), and hypoxic hypoxia (reduced levels of O_2) decrease acetylcholine synthesis. When the O_2 content of the inspired air is reduced from 30 percent to 15 or 10 percent, whole-brain acetylcholine synthesis declines 43 or 52 percent, respectively (*12*). These degrees of hypoxia impair short-term memory and the ability to perform complex tasks, but do not decrease the level of energy metabolites in the brain (*13*).

We examined the effects of aging on whole brain acetylcholine synthesis. The incorporation of isotopic precursors into acetylcholine provides a direct method for assessing the dynamics of the cholinergic system. The measurements of the concentrations of acetylcholine or the cholinergic enzymes or receptors are more indirect methods for determining cholinergic function. Male mice of two strains (C57BL and BALB/c) were studied at 3, 10, and 30 months of age. An intravenous pulse injection of [U-^{14}C]glucose (3 μCi/g) or [$^2H_4$]choline (20 nmole/g) labeled the acetyl or choline moieties of acetylcholine, respectively. Mice were killed by focused microwave irradiation (2.2 kW for 0.3 second) either 2 minutes ([U-^{14}C]glucose) or 30 seconds ([$^2H_4$]choline) after the isotopic injection. Acetylcholine and choline were extracted as ion pairs with dipicrylamine, and their concentrations were measured by gas chromatography–mass spectrometry. The incorporation of [U-^{14}C]glucose was determined by liquid scintillation. Labeling of either the acetyl or choline moieties of acetylcholine gave similar results; thus substrate compartmentation probably does not complicate the interpretation of the results. The use of [U-^{14}C]glucose permitted the simultaneous estimate of whole brain glucose utilization. Lactate concentrations were determined fluorometrically (*14*).

Carbohydrate metabolism changes with senescence. The concentration of lactate, an indirect measure of carbohydrate metabolism, increased significantly in the 30-month-old mice (Table 1), but whole brain glucose utilization remained the same in all age groups (Table 2). Brain glucose utilization and cerebral blood flow decrease in aged rats and perhaps in aged humans as well (*9*, *9a*). These changes are more striking on a regional basis. In these studies we did not observe such a decrease. Contributing factors may have been our use of mice, the use of whole brain rather than regional studies, or our method of determining glucose utilization (*14*).

The synthesis of acetylcholine from either precursor decreased by 10 months and declined further by 30 months (Table 2), whereas the concentration of acetylcholine and choline remained unchanged (Table 1). In other studies, the age-related decreases in oxidative metabolism are apparent by 10 months and do not decline further by 30 months (*9*). In the C57BL strain, the rate of acetylcholine synthesis from [U-^{14}C]glucose declined 49.3 and 68.6 percent in 10- and 30-month-old animals compared with 3-month-old mice (Table 2). In the BALB/c strain, the estimated rate decreased 42.3 and 65.1 percent at 10 and 30

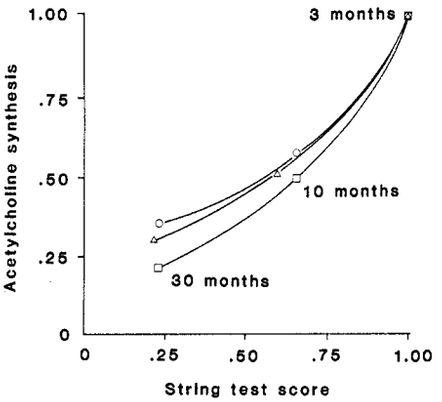

Fig. 1. Relation of string-test scores to the synthesis of acetylcholine. Scores are shown as proportions of 3-month scores. Group sizes of 3-, 10-, and 30-month-old mice, respectively, were (i) BALB/c: 37, 45, and 36; and (ii) C57BL: 34, 34, and 32. In each strain, the scores at the three ages were significantly different (least significant difference test; F statistic for BALB/c = 62.93, d.f. = 122, $P < .05$; F statistic for C57BL = 54.54, d.f. = 103, $P < .05$). The decline in score was highly correlated (*15*) with the decrease in acetylcholine synthesis (synthesis divided by 3-month synthesis from [U-^{14}C]glucose in C57BL mice (△) ($r = .98$, $y = 0.216e^{1.52x}$), in the BALB/c mice (□) ($r = .98$, $y = 0.255e^{1.34x}$), and from [$^2H_4$]choline in BALB/c mice (○) ($r = .98$, $y = 0.150e^{1.88x}$).

Table 2. Alterations in glucose and acetylcholine metabolism with age. Values are means ± standard errors of the mean. The synthesis of acetylcholine from [U-^{14}C]glucose was calculated according to the following equation:

$$\text{Rate of synthesis} = 3 \times \frac{1}{2 \text{ min}} \times \frac{\text{dpm in acetylcholine}}{100 \text{ mg protein}} \times \frac{\text{nmole of glucose}}{\text{dpm in glucose}}$$

A factor of 3 was used because there are three times as many radioactive carbons in glucose as in the acetyl group of acetylcholine. The synthesis of acetylcholine from [$^2H_4$]choline was estimated by the following equation:

$$\text{Rate of synthesis} = \frac{1}{0.5 \text{ min}} \times \frac{\text{nmole of [}^2H_4\text{]acetycholine}}{100 \text{ mg protein}} \times \frac{\text{nmole of choline}}{\text{nmole of [}^2H_4\text{]choline}}$$

Acetylcholine synthesis from [$^2H_4$]choline in the C57BL strain was not determined because [$^2H_4$]choline was not detectable 30 seconds after the injection of [$^2H_4$]choline.

Strain	Age (months)		
	3 (N = 11)	10 (N = 11)	30 (N = 11)
	Glucose utilization (nmole per milligram of protein per minute)		
BALB/c	7.35 ± 0.52	8.26 ± 0.57	8.27 ± 1.04
C57BL	7.25 ± 0.90	6.86 ± 0.69	8.19 ± 0.05
	[^{14}C]Acetylcholine (dpm/nmole)		
BALB/c	409.6 ± 25.9	228.3 ± 15.0*	144.6 ± 21.4†
C57BL	751.1 ± 29.9	496.6 ± 58.6*	358.5 ± 21.4†
	[^{14}C]Glucose (dpm/nmole)		
BABL/c	2663 ± 128	2634 ± 229	2554 ± 195
C57BL	5290 ± 350	6110 ± 711	5623 ± 669
	Rate of [^{14}C]acetylcholine synthesis (nmole per 100 mg of protein)		
BALB/c	5.28 ± 0.33	3.04 ± 0.30*	1.84 ± 0.30†
C57BL	6.87 ± 0.45	3.48 ± 0.30*	2.16 ± 0.30†
	[$^2H_4$]Acetylcholine (nmole per 100 mg of protein)		
BALB/c	0.37 ± 0.04	0.15 ± 0.01*	0.10 ± 0.04†
	[$^2H_4$]Choline/total choline		
BALB/c	0.10 ± 0.01	0.09 ± 0.01	0.10 ± 0.01
	Rate of [$^2H_4$]acetylcholine synthesis (nmole per 100 mg of protein)		
BALB/c	6.65 ± 0.90	3.30 ± 0.50*	1.60 ± 0.40†

*Significantly different ($P < .05$) from 3-month-old mice (analysis of variance with least significant difference test). †Significantly different ($P < .05$) from both 3-month-old and 10-month-old mice.

months. The decreased incorporation of glucose was not due to a dilution of the specific activity of the precursor pool [glucose disintegrations per minute (dpm) per nanomole]. The incorporation of [$^2H_4$]choline into acetylcholine also declined with senescence. In the BALB/c strain, the estimated rate of synthesis from [$^2H_4$]choline decreased by 50.4 and 75.9 percent in 10- and 30-month-old mice. Neither the specific activity of the precursor pool nor the uptake of [$^2H_4$]-choline into the brain accounted for the decreased incorporation.

The depressed acetylcholine synthesis in senescent mice was correlated (*15*) with behavioral deficits as measured with a string test (r = .98; Fig. 1), which quantitates the ability of a mouse to traverse an elevated taut string (*16*). Scores for 10- and 30-month-old mice of both strains were 35 to 42 and 77 to 78 percent lower than those of the 3-month-old animals. In thiamine deficiency, a decrease in the string-test score seems attributable to a central cholinergic muscarinic lesion (*15*). Whether this is also a causative factor in aged animals remains to be determined. Decreased acetylcholine synthesis correlates well with a decrement in geriatric memory deficits previously reported (*2*). The latencies in passive avoidance tasks declined 31 to 40 percent in 12-month-old mice (acetylcholine declined 32 percent) and by 58 to 64 percent in 30-month-old mice (acetylcholine declined 59 percent). Other studies in rats by Lippa *et al.* (*4*) suggest that memory impairment may not be detectable until 20 months.

Our studies directly demonstrate reduced acetylcholine synthesis in senescent mice. This decrease is correlated with the development of progressive behavioral deficits and may underlie some of the brain dysfunctions which complicate senescence. The mechanism linking these two findings requires further investigation.

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17. The aged animals were from Charles Rivers Breeding Laboratories, which is under contract with the National Institute on Aging. They are kept in our temperature- and humidity-controlled animal facilities for no longer than 3 days. This work was supported in part by NIH grants NS16997, MS15649, and MH17691; the Winifred Masterson Burke Relief Foundation; and the Will Rogers Institute.

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Short-Term Variations in Diet Composition Change the Pattern of Spontaneous Motor Activity in Rats

Abstract. *The nocturnal activity patterns of rats changed significantly within 3 days after they were given unrestricted access to isocaloric diets in which the ratio of carbohydrate to protein was systematically varied. As the ratio increased, the rats were more continuously active. The subjects showed similar responses to variations in this ratio whether the diet contained 15 or 45 percent fat. No correlation was found between the number of calories an animal ate and its activity pattern.*

Does diet composition affect the behavior of omnivores like rats and humans? Learning, sleep, and spontaneous motor activity can be altered experimentally by starvation (*1*), malnutrition (*2*), or excesses or deficiencies of various dietary components (*3*). Animals or humans may occasionally be exposed to severe and protracted dietary changes; however, they usually are able to search for and choose among a variety of foods (*4*). The behavioral effects of normal short-term variations in diet composition have not, to our knowledge, been studied.

The proportions of protein and carbohydrate in each meal can affect the amounts of tryptophan and tyrosine taken up into the brain (*5*) and, consequently, synthesis of serotonin and the catecholamine neurotransmitters. Similarly, dietary lecithin or choline content can affect neuronal acetylcholine synthesis (*6*). There is evidence that these neurotransmitters participate in brain mechanisms underlying behavior such as spontaneous motor activity (*7*). We now report that short-term changes in diet composition, similar to those that may occur naturally, can modify patterns of spontaneous motor activity in rats.

Male Sprague-Dawley rats (Charles River) were housed singly for several weeks in specially constructed plexiglass cages (14 by 14 by 10 inches) that allowed them unrestricted access to food and water. The cages were kept in an isolated room and could be cleaned without disturbing the animals (*8*). Between 4 a.m. and 4:40 p.m. daily, the cages were lighted by fluorescent bulbs (Vita-Lite, Duro Test Corp.) emitting a spectrum

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