explanation on a chemical-physical basis, its significance may be of interest also on the biological level, with regard to both fat transport and fat metabolism.

All that the above data appear to suggest at this point is that (1) liver suspensions of normal and of depancreatized dogs do not behave in the same manner as far as their lipid content is concerned when incubated for varying periods of time, (2) the addition of pancreatic extracts to the liver suspensions of depancreatized dogs seems to nullify this difference in behavior, and (3) the pancreatic extract called "lipocaic" appears to act, though less efficiently, in the same manner as the more easily obtainable extract first used.

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Aspects of the Biologic Decay Periods of Sodium in Normal and Diseased Man[1]

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A knowledge of the duration of time for which a radioactive element is retained in the body has important significanee physiologically, in safety considerations, and in dosage calculations in tracer studies and isotope radiation therapy. The biologic decay-life of sodium in man can be determined satisfactorily with the long half-life Na^{22} ($T_{\frac{1}{2}} = 3$ yrs) but not with Na^{24} ($T_{\frac{1}{2}} = 14.8$ hrs). During the course of experiments using Na^{22} in man and designed for other purposes, data were obtained which indicated the rates of elimination of sodium. Na^{22} with an activity such that 10,000,000–17,725,000 disintegrations occurred per minute was injected into each subject. A daily blood serum concentration of Na^{22} was determined for from 30 to 60 consecutive days. All samples of urine were collected separately and the Na^{22} excretion determined for each. The subjects consisted of 4 control subjects free from cardiovascular and renal disease, 6 subjects with chronic congestive heart failure (2 were slowly improving, 2 rapidly improving, and 2 becoming worse), and 2 subjects with chronic glomerular nephritis of the nephrotic type.

Since only the serum concentration of Na^{22} and the rate of urine excretion of the radioelement were measured, it was not possible to know the time required to excrete one-half of the injected Na^{22}, the biologic half-life period ($B_{\frac{1}{2}}$), *per se*. Therefore, it was necessary to introduce the following terms:

(1) $C_{\frac{1}{2}}$ = time required to reduce the serum concentration of Na^{22} to one-half the value obtained at any time after equilibrium of distribution has been reached.

(2) $U_{\frac{1}{2}}$ = time that would be required to eliminate one-half of the administered Na^{22} if it were being excreted only in the urine at the rate observed.

The results are briefly summarized in Table 1. It can be seen that with the exception of Subject No. 2 the $C_{\frac{1}{2}}$ periods were less than the $U_{\frac{1}{2}}$. This is to be expected because of the influence of other factors, such as other avenues of sodium excretion, shifts of sodium within the sodium compartments of the body, and variations in the volume of these compartments. The subjects with chronic congestive heart failure and those with chronic glomerular nephritis of the nephrotic type had $C_{\frac{1}{2}}$ and $U_{\frac{1}{2}}$ periods which were much longer than those of the controls (Table 1). Sodium and water diuresis in the subjects whose congestive heart failure rapidly improved resulted in a definite shortening of the $C_{\frac{1}{2}}$ and $U_{\frac{1}{2}}$ periods, the times be-

TABLE 1

Subject No.	$C_{\frac{1}{2}}$*	$U_{\frac{1}{2}}$†	Days of continuous observation	Weight change (lb)
		Control		
1	14	30	62	− 3.5
2	13	9	22	−14
3	12	42	45	−11
4	14	34	65	+ 2.3
Mean	13.3	28.8	48.5	− 6.6
		Congestive heart failure (Slowly improving)		
5	40	60	35	−18
6	42	72	46	− 7
Mean	41	66	40.5	−12.5
		Congestive heart failure (Rapidly improving)		
7	13	26	62	−29
8	28	33	58	−17
Mean	20.5	29.5	60	−23
		Congestive heart failure (Slowly becoming worse)		
9	24	72	68	+17
10	30	48	58	− 5.5
Mean	27	60	63	+ 5.8
		Chronic glomerular nephritis (Nephrotic type)		
11	58	660	45	+15
12	54	366	71	−86
Mean	56	513	58	−35.5

* Time in days required for the serum Na^{22} concentration to reach one-half at any time after equilibrium of distribution.

† Time in days required for one-half of the total Na^{22} injected to be eliminated by the urine.

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coming even less than that for the normal subjects on high sodium intake. The $C_{\frac{1}{2}}$ periods in the control subjects were essentially the $B_{\frac{1}{2}}$ periods. This is less likely to be true for the abnormal subjects.

The $C_{\frac{1}{2}}$ and $U_{\frac{1}{2}}$ periods were influenced by measures which influenced sodium metabolism and excretion, such as sodium intake, desoxycorticosterone acetate, mercurial diuretics, water intake, and pitressin. Fig. 1 shows the influence of sodium intake on the $C_{\frac{1}{2}}$ and $U_{\frac{1}{2}}$ periods.

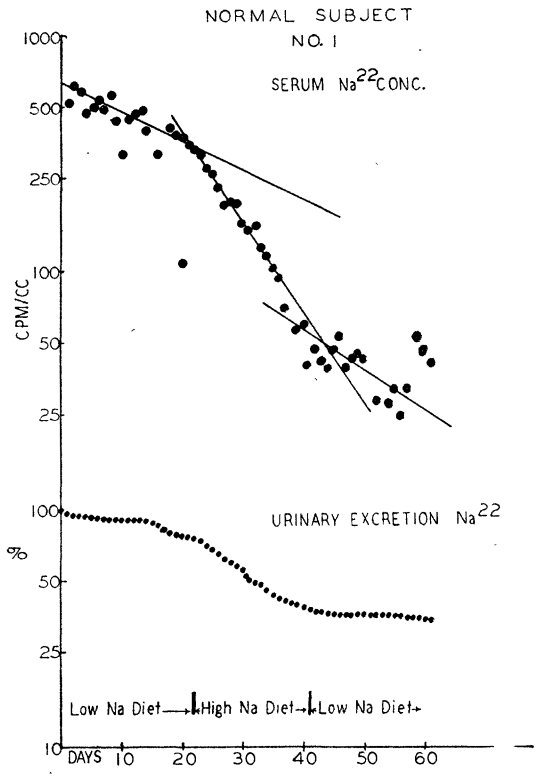

FIG. 1. Semilogarithmic graphs of changes in serum Na^{22} concentration (cpm/cc) and the rate of urinary excretion of Na^{22} (per cent of injected Na^{22} not eliminated in the urine) in Normal Subject No. 1.

The relationships of the changes in the rate of decrease in serum concentration and rate of urinary excretion to the changes in dietary sodium are shown. During the first 22 days while the subject was on a low Na diet (< 1.7 gm of NaCl/day) the rates were such that $C_{\frac{1}{2}}$ was 25 days and $U_{\frac{1}{2}}$ 100. During the next 19 days while the subject was on a high Na diet (13.7 gm of NaCl/day) $C_{\frac{1}{2}}$ was reduced to 8 days and $U_{\frac{1}{2}}$ to 19. During the last 19 days the subject was again on a low Na diet and antidiuretics. $C_{\frac{1}{2}}$ increased to 18 days and $U_{\frac{1}{2}}$ to 250.

These experiments have shown that the $B_{\frac{1}{2}}$ as well as the $C_{\frac{1}{2}}$ and $U_{\frac{1}{2}}$ for sodium are quite variable, being influenced not only by normal physiologic phenomena but particularly by disease and drugs. These variations must be taken into consideration when calculating the safety doses for radiosodium. $C_{\frac{1}{2}}$ and $U_{\frac{1}{2}}$ periods found for Na^{22} indicate the length of time required to turn over Na^{23} in man. The experiments will be published in detail elsewhere.

Isolation From Wild Bird Mites (*Liponyssus sylviarum*) of a Virus or Mixture of Viruses From Which St. Louis and Western Equine Encephalitis Viruses Have Been Obtained[1]

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In a previous communication by Reeves, Hammon, Furman, McClure, and Brookman (*1*) it was reported that in addition to three strains of Western equine encephalomyelitis virus isolated from mites, *Liponyssus sylviarum* (Canestrini and Fanzago),[2] found in the nest of a yellow-headed blackbird, another virus was isolated which was not as yet identified. Several months of laboratory work, including serial passages in several species of animals and extensive immunological tests, have led to the results summarized in this preliminary paper. When the studies are complete, they will be reported elsewhere in detail.

This agent, following isolation in mice and after several serial mouse passages, killed mice, guinea pigs, and chick embryos, but failed to kill guinea pigs which previously had been vaccinated with Western equine virus. It was not neutralized by hyperimmune Western equine serum or by St. Louis or Japanese B serum alone, yet a mixture of the three was effective in neutralizing the virus. A complement-fixing antigen prepared from the brains of mice infected with this virus reacted with specific antisera against Western equine, St. Louis, and Japanese B viruses, and antigens prepared from each of these three viruses in turn reacted with the sera from animals immunized against the mite virus. In cross-vaccination tests, however, there was no immunity in either direction in so far as Japanese B virus was concerned.

After 8 serial passages in mice the virus had only the immunological characteristics of St. Louis virus, and it would not kill guinea pigs. After 10 passages in chick embryos it had only the characteristics of a Western equine virus.

Two possibilities presented themselves: (1) that this was a simple mixture of two viruses or (2) that it was a stem virus maintained by mite-to-bird passage which could develop as either virus after passage in more selective hosts. The first possibility appeared more likely; but the second was challenging and not incredible, since these

[1] This investigation was carried out in collaboration with the Commission on Virus and Rickettsial Diseases, Army Epidemiological Board, Preventive Medicine Division, Office of the Surgeon General, U. S. Army, aided by a grant from the National Foundation for Infantile Paralysis, and under a contract with the California State Department of Public Health.

[2] These mites were collected and identified by entomologists and the ornithologist of the Neurotropic Virus Research Unit, including W. C. Reeves, D. P. Furman, B. Brookman, and H. E. McClure.