activity) and from Oahu (high organic matter, low activity). The Oahu soil was the red, porous, granular volcanic soil characteristic of that island. The fact that the Riverside soil was under cultivation may have been a factor in its higher activity. With the exception of the Riverside soil, however, cultivated soils were not appreciably active. As a general rule, the more acidic soils were the most active. The Riverside orchard soil (high *p*H, high activity) and the Oahu soil (low *p*H, low activity) were also exceptions to this rule. The soil from the Yosemite wall taken under white fir also defied this classification. No relation was apparent in this listing between soil activity and the prevalence of air pollution conditions.

On the basis of measurements listed in Table 2, an estimate of the total capacity of soil to remove CO from the atmosphere can be attempted. The average activity of the soils tested was 8.44 mg of CO per hour per square meter of soil, equivalent to 191.1 metric tons per year per square mile. If we assume that this value is representative of the average capacity of soils in the temperate zone, the capacity of the total soil surface of the continental United States [2,977,128 square miles (7,792,533 km^2)] is estimated to be 569 million metric tons per year, which is 6.5 times the annual estimated production of CO attributed to the United States and almost three times the estimated worldwide production due to man's activities. The soil, therefore, must now be considered as a major natural sink for CO that is released into the atmosphere by natural emitters or by the burning of fossil fuels.

ROBERT E. INMAN
ROYAL B. INGERSOLL
ELAINE A. LEVY
Stanford Research Institute-Irvine, Irvine, California 92664

References and Notes

1. R. C. Robbins, K. M. Borg, E. Robinson, *J. Air Pollut. Contr. Ass.* **18**, 106 (1968).
2. L. S. Jaffe, *ibid.*, p. 534.
3. J. W. Swinnerton, V. J. Linnenbom, R. A. Lamontagne, *Science* **167**, 984 (1970); D. F. Wilson, J. W. Swinnerton, R. A. Lamontagne, *ibid.* **168**, 1577 (1970).
4. D. R. Bates and A. E. Witherspoon, *Month. Notic. Roy. Astron. Soc.* **112**, 101 (1952).
5. B. Weinstock, *Science* **166**, 224 (1969).
6. A. J. Kluyver and Ch. G. T. P. Schnellen, *Arch. Biochem.* **14**, 57 (1947).
7. S. A. Waksman, *Principles of Soil Microbiology* (Williams and Wilkens, Baltimore, 1929), p. 98.
8. M. Stephenson, *Bacterial Metabolism* (Longmans, Green, London, ed. 3, 1949), p. 95.
9. T. Yagi, *Biochim. Biophys. Acta* **30**, 194 (1958).
10. G. W. Jones and G. S. Scott, *Ind. Eng. Chem.* **31**, 775 (1939).
11. K. Porter and D. H. Volman, *Anal. Chem.* **34**, 748 (1962).
12. Work supported by the Environmental Protection Agency and the Coordinating Research Council through CRC-APRAC contracts CAPA-4-68 (1-69) and CAPA-4-68 (2-68) and APCO contract CPA 22-69-43.

29 March 1971

Net Kinetic Energy in Littoral Transport

Abstract. *Studies of coastal erosion and coastal management can be put on a firm physical basis only after methods have been developed for closely estimating the energy expended in actual unidirectional net littoral transport of sediment. Such measures have now been obtained for six coastal drift cells. The results, for a 68-year period, vary from a minimum of 0.6 × 10^4 ergs (very low energy) to a maximum of 340 × 10^4 ergs (moderately high energy) per cell.*

There have been two ways of approximating the energy level of the surf zone: (i) by making a precise computation for actual total energy delivered to the coast, regardless of whether it produces any net changes or not (and most of it typically does not); and (ii) by estimating the energy from some convenient observation such as ramp slope (*1*) or breaker height (*2*). Neither method has been completely satisfactory (from the standpoint of the student of coastal processes); yet, if we are to deal effectively with problems of coastal erosion, it is important that we know the energy involved. A third method is now available.

Stapor (*3*) showed that the coast of the Gulf of Mexico southwest of Tallahassee, Florida (low to moderate energy), can be divided into essentially independent cells, separated by inlets or by large shoals which act as transverse bars (*4*). Stapor isolated six such cells in a distance of about 125 km and studied them, using a technique which involved a numerical comparison of measured water depths from selected nautical charts, which he checked carefully for accuracy; a seventh and an eighth cell, in the same array, could not be treated on the charts available.

Stapor's data, for each cell, included the mass of sand moved (volume measure), the distance moved, and the time interval covered. The distance and time terms can be set in a ratio to provide a velocity; the volume term can be converted into a mass (inasmuch as the sand is essentially pure quartz). Kinetic energy, as a measure of work, can be expressed as:

$$\text{K.E.} = mv^2/2$$

where m is the mass and v is the velocity. This formula gives a minimum estimate of the work done, inasmuch as the shuffling of individual grains back and forth, important in absorbing wave energy, is not included; that is, this formula provides an estimate of net unidirectional work.

The results of the calculation, for six cells from east to west, are as follows (for a 68-year period ending about 1940): cell 1, northeastern Dog Island: 0.6×10^4 ergs; 0.3×10^{-5} erg/sec; cell 2, southwestern Dog Island: 7.1×10^4 ergs, 3.3×10^{-5} erg/sec; cell 3, northeastern St. George Island: 5.4×10^4 ergs, 2.5×10^{-5} erg/sec; cell 4, Cape St. George (St. George Island): 9.4×10^4 ergs, 4.4×10^{-5} erg/sec; cell 5, Cape San Blas: 9.9×10^4 ergs, 4.6×10^{-5} erg/sec; and cell 6, St. Joseph peninsula: 340×10^4 ergs, 170×10^{-5} erg/sec. Individual cells are between 5 and 25 km in length.

In general, the energy level increases toward the west (that is, from cell 1 to cell 6). Furthermore, westward-moving and northwestward-moving drift systems (cells 2, 4, and 6) represent more energetic conditions than eastward-moving and northeastward-moving drift systems (cells 1, 3, and 5). Both of these results are consonant with the easterly prevailing winds in the region and the resulting westward overall drift.

I would estimate that this kinetic energy is perhaps roughly 10 percent of the available bidirectional littoral energy, and a much smaller fraction of the total wave energy delivered to points along this coast. For purposes of comparison, measured wave heights within cell 5 (*5*) are about 13 cm, on the average; and estimated wave heights, based on repeated field observations, are markedly larger to the northwest (cell 6) and somewhat smaller toward the northeast (cells 1, 2, 3, and 4) toward the "zero" energy coast southeast of Tallahassee.

WILLIAM F. TANNER
Geology Department, Florida State University, Tallahassee 32306

References

1. W. A. Price, *Texas A & M. Dep. Oceanogr. Proj. 63* (1955).
2. W. F. Tanner, *J. Sediment. Petrol.* **29**, 564 (1959).
3. F. W. Stapor, *Mar. Geol.* **10**, M1 (1971).
4. W. F. Tanner, *Science* **132**, 1012 (1960); A. W. Niedoroda and W. F. Tanner, *Mar. Geol.* **9**, 41 (1970).
5. J. R. Helle, *Beach Erosion Board Tech. Mem. 108* (1958).

18 February 1971; revised 14 April 1971

Vitamin D Metabolism: The Role of Kidney Tissue

Abstract. *The appearance of a polar metabolite of 25-hydroxycholecalciferol has been studied in the intestinal mucosas of nephrectomized rats and rats which have been made uremic by ureter ligation. In confirmation of previous work by Fraser and Kodicek, it was found that nephrectomy prevents the appearance of this metabolite in the intestine. On the other hand, equivalent amounts of the metabolite were found in uremic rats and in sham-operated controls, showing that the production of this metabolite is independent of the uremic state of the animals. In addition, it was shown that the metabolite produced in vitro by kidney homogenates is identical to that found in vivo in the intestine.*

Impaired renal function has for several years been known to be associated with alterations in vitamin D and calcium metabolism. Kessner and Epstein (*1*) showed the active transport of calcium by everted duodenal sacs to be reduced in the chronically uremic rat, and Avioli *et al.* (*2*) have in addition demonstrated decreased intestinal calcium binding protein activity in such animals. These situations are not reversed by physiological amounts of vitamin D, though they may be partially reversed by 25-hydroxycholecalciferol (25-HCC), the "circulating" or "hormonal" form of vitamin D (*3*, *4*). There is, however, no evidence that the hepatic synthesis of 25-HCC from vitamin D is impaired in uremia.

Recently, Fraser and Kodicek (*5*) have shifted attention from the role of renal function to the role of renal tissue in vitamin D metabolism by their report that the conversion of 25-HCC to a more polar metabolite takes place exclusively in the kidney. This metabolite (designated "peak V" in this laboratory) has been shown to act even more rapidly than 25-HCC in the stimulation of calcium absorption and may very well be the active form of the vitamin in the intestine (*6*). The evidence for the role of kidney tissue in the production of this metabolite is based principally on the observation that the normal appearance of this metabolite in the intestinal mucosa after administration of vitamin D is not seen in nephrectomized rats (*5*). Furthermore, Fraser and Kodicek reported kidney to be the only tissue capable of producing this metabolite in vitro (*5*).

The present study was undertaken with two objectives in mind. The first was to determine whether the absence of the peak V metabolite in the intestine of nephrectomized rats is due to toxic effects of the uremia produced by nephrectomy or whether kidney tissue is required for the further metabolism of 25-HCC. The second objective was to determine whether the peak V produced in vitro by kidney homogenates is identical to that observed in the intestine after in vivo administration of 25-HCC or vitamin D_3.

Male weanling Holtzman rats were fed a vitamin D–deficient diet for 5 to 6 weeks prior to being used for experiments (*7*). The [26,27-^{3}H]-25-HCC used in these experiments was prepared in this laboratory and had a specific activity of 1.3 c/mmole (*8*). Groups of three rats were either bilaterally nephrectomized or sham-operated and were injected intrajugularly with 125 ng of ^{3}H-25-HCC in 95 percent ethanol immediately after surgery. A third group of rats was ureter-ligated 6 hours before being injected with 125 ng of ^{3}H-25-HCC, the 6-hour delay being to ensure a degree of uremia similar to that of the nephrectomized animals at the time of killing. The rats in all groups were killed 12 hours after the injection of ^{3}H-25-HCC, having also been fasted 16 hours prior to killing. The intestinal mucosas from the rats in each group were pooled, homogenized in 0.9 percent saline, and extracted with $CHCl_3$-methanol as previously described (*4*). Identification of ^{3}H metabolites was

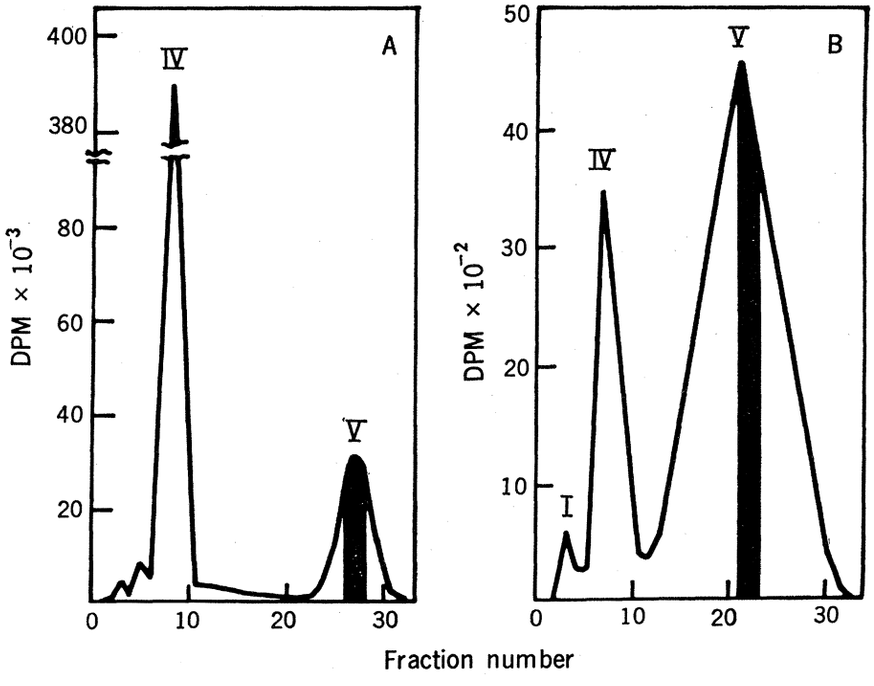

Fig. 1. (A) Sephadex LH-20 chromatography of the combined lipid extracts from six in vitro incubation mixtures. Incubation was for 2 hours at 37°C. A column containing 12 g of Sephadex LH-20 was used and 5.3-ml fractions were collected. The fractions in the shaded area were pooled and taken up in a known volume of solvent. (B) Sephadex LH-20 chromatography of the lipid extract of the mucosal scrapings from six vitamin D–deficient chicks each given 125 ng of [26,27-^{3}H]-25-HCC 12 hours prior to killing. A column containing 10 g of Sephadex LH-20 was used and 5.5-ml fractions were collected. The fractions in the shaded area were pooled and taken up in a known volume of solvent.