1½ to 2 hours after eating. The complete absorption of a small, orally ingested dose of methanol requires about 2 hours in man (*12*) and 4 to 6 hours in rabbits (*13*). In view of this absorption pattern a dietary source of the concentrations in breath is possible.

According to the available biologic half-life data, which is 10 to 13 hours in rabbits and 2 hours in man, the peak observed after eating would drop to one half its value in about 2 hours for a living person. Thus a peak of about 6×10^3 μg/liter of expired air would be required to produce 0.1 μg per liter in the breath after 16 hours; this is quite unreasonable in view of the data presented.

That intestinal bacteria may be the source for the observed methanol is not ruled out from the human data presented. We did not attempt to sterilize the gut to study this point as has been done in other studies (*14*). The wide racial, age, and dietary differences presented by the subjects, who were Indian, Iranian, Iraqi, Egyptian, American, and German, and the differences observed (less than one order of magnitude) suggest strongly, however, that diet is only a minor contributor of variations, not a source.

Although the presence of methanol might be explained from a bacteriologic standpoint, it is more likely that it is the result of some metabolic process. There seems to be, however, no currently available explanation how any carbon metabolic degradation process would produce methanol (*15*) so that its rather universal presence seems to be somewhat of a mystery (*16*).

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Iodine-131 in Utah during July and August 1962

Abstract. *Nuclear explosions in Nevada in July 1962 caused an average intake of about 58,000 picocuries of* I^{131} *and a peak intake of 800,000 picocuries of* I^{131} *by Utah residents consuming one liter of milk per day. Corresponding infant thyroid doses were about 1 rad (average) and 14 rad (peak).*

Fallout from nuclear tests in Nevada during July 1962, deposited iodine-131 in Utah. Published meteorological trajectories suggest that fallout from the tests on 7 July, 11 July, and 17 July went west of the main Utah milkshed (*1*), while peak gross beta activities in the air at Salt Lake City of 900 picocuries per cubic meter on 7 to 8 July and 450 pc per m^3 on 15 to 16 July implicate the "Sedan" Plowshare test of 6 July and the "Small Boy" weapons test of 14 July (*2*). Much of the high Sedan activity was due to neutron-activated tungsten (*3*). Our measurements of I^{131} in milk collected between Sedan and Small Boy indicate that Sedan contributed 10 to 30 percent of the total I^{131} intake in Utah resulting from these tests.

On 7 July 1962, the day after the 100-kiloton Sedan nuclear test in Nevada, one of us (R.C.P.) was measuring background radiation 20 miles southeast of Salt Lake City. I observed the approach of a large dusty cloud which increased the γ-ray intensity to about 100 times that of normal background. Because of this, we counted the milk scheduled for collection from several farms near Salt Lake City on 12 and 13 July. The radioiodine in this milk ranged from 10 to 2600 pc of I^{131} per liter. At the request of the Utah State Department of Health, we then collected milk from each of our 39 stations located throughout the state in order to measure the extent and degree of the I^{131} contamination. Each station was an individual farm already participating in our study of the ecological factors affecting Cs^{137} uptake in milk and man.

Iodine-131 was evaluated by means of its 364-kev γ-ray detected by a sodium iodide crystal (20 × 10 cm) and recorded on a 400-channel pulse-height analyzer. The accuracy (S.D.) of a measurement was about ± 10 percent or ± 20 pc of I^{131} per liter, whichever was larger. All measured activities of I^{131} in milk were adjusted to the day of sampling.

The buildup and subsequent decrease of I^{131} in milk taken from several individual farms is shown in Fig. 1. Because of the limited data during the period of buildup, we assumed that the concentrations of I^{131} in milk increased in linear fashion from negligible values on 11 July to peak values on 20 July. In equation form:

$$C_1 = C_p\, 0.111\ \text{day}^{-1}\, t_1 \qquad (1)$$

where C_1 = I^{131} concentration during buildup phase, C_p = peak I^{131} concentration on 20 July 1962, and t_1 = time after 11 July 1962. It will be shown later that a moderate error in Eq. 1 will cause only a small error in computing the total I^{131} intake.

After 20 July 1962, the concentration of I^{131} in milk was evaluated serially for seven separate farms. The concentrations (from the dates of collection) decreased exponentially with effective half-periods ranging from 3.8 to 9.8 days and averaging 5.8 days. Thus, after 20 July, the concentration of I^{131} in milk could be expressed as:

$$C_2 = C_p \exp\left(-0.12\ \text{day}^{-1}\, t_2\right) \qquad (2)$$

where C_2 = I^{131} concentration during decreasing phase, C_p = peak I^{131} concentration on 20 July 1962, and t_2 = time after 20 July 1962.

The daily intake of I^{131} was obtained by multiplying Eqs. 1 and 2 by the volume of milk consumed per day. Then the total I^{131} intake was calculated by integrating the daily intake over the buildup and decreasing phases and is:

$$\text{total } I^{131} \text{ intake} = 12.8\ VC_p \qquad (3)$$

where V = volume of milk consumed per day, and C_p = peak I^{131} concentration in milk.

About one-third of the calculated total intake occurred during the buildup

phase and two-thirds during the decreasing phase. Thus, a 30-percent error in the assumed buildup (Eq. 1) would produce an error of only 10 percent in the total intake.

The total I^{131} intake resulting from the continuous consumption of one liter of milk per day was computed for each station as follows: (i) for each measured milk sample, the corresponding peak concentration on 20 July was computed by rearranging Eq. 1 or 2, (ii) the computed peak concentrations were averaged for each station, and (iii) the total I^{131} intake was calculated from Eq. 3.

Total intake (for 1 liter of milk per day) ranged up to 800,000 pc of I^{131}. Seven stations exceeded 100,000 pc of I^{131}. The highest exposures were near Duchesne, Utah, and additional high exposures occurred near Heber and Price. An extremely important finding was that very little I^{131} appeared in the milk of cattle eating exclusively feed which had been stored prior to the Sedan explosion on 6 July 1962. Every case of high I^{131} in milk could be traced to grazing on contaminated pasture or to using contaminated feed.

Our 39 stations had a median total intake of 6100 pc of I^{131} and an average total intake of 58,000 pc of I^{131}, indicating a highly skewed distribution. The mean population intake for the state may differ somewhat from our average since our stations comprise only a small fraction of Utah's milk producers. An average total intake of 37,000 pc of I^{131} from 1 September 1961 to 31 August 1962 has been reported for the Salt Lake City "milk pool" (*2*). However, milk-pool values can be misleading indexes of group exposure. Our measurements of milk from one major Salt Lake dairy indicated a total intake of 117,000 pc of I^{131}, for individuals consuming 1 liter of milk per day; whereas for a second major Salt Lake dairy the total intake was only 3400 pc of I^{131}. For comparison, the current yearly radiation protection guide of 0.5 rad to the thyroid gland of members of the general population is considered to correspond to the intake of about 29,000 pc of I^{131} by the young child (*4*).

The internal I^{131} of 24 farmers from our milk stations was evaluated by γ-ray counting of the thyroid or the total body. Accuracy for thyroid counting was about $\pm$ 20 percent or $\pm$ 150 pc of I^{131}, whichever was larger. Accuracy for total-body counting was about $\pm$ 30 percent or $\pm$ 400 pc of I^{131}, whichever was larger.

The results are listed in Table 1. The percentage uptake of I^{131} was calculated as the ratio of that measured in the body to that computed for 100-percent uptake from milk. Each day's I^{131} intake was corrected from the day of consumption to the day of human counting for radioactive decay and biological elimination, assuming that 7.6 days is the effective half-time for I^{131} in man (*5*). Our average uptake of 17 percent agrees well with the value of 20 percent obtained by integrating (over constant continuous exposure) Lushbaugh's single-intake retention equation based on 26 normal subjects, most of whom were adults (*6*).

The cumulative radiation dose to M grams of thyroid tissue resulting from the intake of A microcuries of I^{131} of which a fraction U is taken up in the thyroid gland and retained with an effective half-time of T days had been given (*7*) as:

$$\text{thyroid dose} = 15\frac{A\,U\,T}{M}\ \text{rad} \qquad (4)$$

Young children 0 to 2 years old are regarded as most susceptible to I^{131} damage because of (i) the small size of the child's thyroid, (ii) its presumed greater sensitivity to irradiation at this stage, and (iii) the long post-irradiation life span during which delayed effects

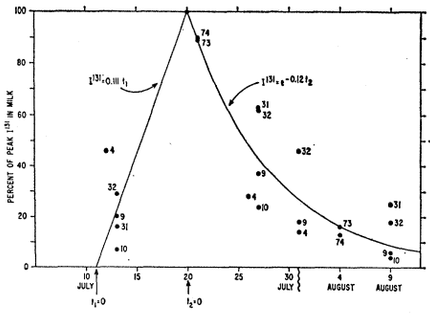

Fig. 1. Buildup and subsequent decrease of I^{131} in milk. Concentrations are relative to peak values observed on 20 July 1962. The numbers designate the farms from which the samples were taken.

could appear. W. S. Snyder (Oak Ridge National Laboratory) has pointed out to us the urgent need for good data on iodine uptake and retention in the infant thyroid, because values for the infant may differ from those established for adults and older children. Recent work by Morrison *et al.* (*8*) indicates an I^{131} thyroid uptake of 50 percent and effective half-time of 6 days in the newborn infant. In the absence of precise information, a young child is frequently assumed to have 2 g of thyroid tissue, a 30-percent uptake of I^{131}, and an effective retention half-time of 7.6 days (*4*).

With these assumptions, Eq. 4 indicates a thyroid dose of 1.0 rad for infants consuming our average intake of

Table 1. I^{131} in Utah dairy families measured by thyroid counting and total-body counting.*

Person	Age (yrs)	Milk (liters/day)	Date counted (Aug 62)	I^{131} (10^3 pc)	Uptake (%)
9-a, M	19	3.0	1	11.0	8
9-a			22	2.9	8*
9-b, F	11	0.7	1	6.3	19
10-a, F	7	0.7	2	4.1	26
10-b, M	5	1.1	2	4.2	17
10-c, M	30	2.1	2	7.6	16
10-d, F	26	0.5	24	0.8	28*
31-a, F	11	0.8	1	7.8	19
31-a			24	2.2	23*
31-b, F	8	0.8	1	10.2	24
31-b			24	1.8	19*
31-c, F	4	0.5	1	4.0	15
31-d, M	35	1.0	1	3.6	7
32-a, M	10	1.5	6	4.9	9
32-b, F	14	4.0	6	7.3	5
32-b			22	3.6	7*
32-c, F	13	1.7	6	4.2	7
32-d, M	41	1.7	6	3.7	6
32-e, M	9	1.7	6	4.1	6
32-f, F	45	1.0	22	0.7	5*
57-a, M	61	1.0	25	2.8	9*
57-b, F	49	1.0	25	3.0	10*
59-a, M	48	0.5	25	4.2	46*
59-b, F	13	0.7	25	2.2	18*
60-a, M	27	2.0	31	10.9	14*
60-b, F	16	1.0	31	8.7	23*
63-a, F	41	1.0	29	2.4	34*
63-b, M	42	1.0	29	3.4	47*
Average					17

* Starred numbers represent total-body counts.

58,000 pc of I^{131} (0.058 μc), whereas an intake of 800,000 pc of I^{131} (1 liter of milk per day from our highest station) would deliver 14 rad. About 53,000 Utah children between 0 and 2 years of age were subject to I^{131} exposure in 1962 (from extrapolation of the 1950 and 1960 U.S. Census) (*9*).

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Lactate Dehydrogenase Variant from Human Blood: Evidence for Molecular Subunits

Abstract. *A variant of human lactate dehydrogenase is described. The occurrence of lactate dehydrogenase-1, -2, -3, and -4 as five, four, three, and two components, respectively, is interpreted as supporting the hypothesis that LDH isozymes are tetramers formed from various combinations of two types of subunits.*

The lactate dehydrogenase (LDH) of an organism may occur in multiple molecular forms called isozymes (*1*). LDH isozymes of approximately 135,000 molecular weight can be dissociated into subunits of 34,000 molecular weight (*2*). These subunits occur in two electrophoretically distinct forms (*2*) designated A and B. On the basis of this evidence several groups of investigators (*2, 3*) have suggested that distinction between individual isozymes arises, as shown in the second column of Table 1, from various tetrameric associations of A and B subunits. We have observed a variant of human LDH which supports this thesis.

The LDH variant appeared in a healthy 25-year-old Nigerian male of the Yoruba tribe who was initially examined during a survey for hemoglobin heterogeneity. Two separate samples of whole blood were obtained during a 2-month interval. Each sample was aseptically collected in acid-citrate-dextrose solution, sent to Baltimore, and received there 4 to 5 days later. Erythrocytes were washed in 0.9-percent NaCl, hemolyzed in water, extracted with toluene, and centrifuged at 40,000*g* for 20 minutes at 0°C. Hemolyzates containing approximately equal amounts of LDH were subjected to vertical starch-gel electrophoresis (*4*) (4°C, 4.5 volts/cm) for approximately 16 hours. After electrophoresis, LDH activity was localized by methods (*5*) modified from those of Dewey and Conklin (*6*).

As shown in Fig. 1 the variant specimen contains five components in the LDH-1 position, four in the LDH-2 position, three in LDH-3 position, and two in the LDH-4 position. At each isozyme position the most rapidly migrating variant component has the mobility of the corresponding isozyme from normal subjects. The LDH-5 component is absent in this and other hemolyzates (*7*). A similar, less intensely staining pattern is observed in plasma. The variant pattern is identical in separate blood specimens. Dilution of the sample had no effect on electrophoretic pattern. Other proteins present in the hemolyzate from the variant were indistinguishable from those of normal Negro subjects. The variant is demonstrable in gels prepared with many buffers including ethylenediaminetetraacetic acid-boric acid-tris (EBT) (*8*), borate (*4*), and the discontinuous system of Poulik (*9*). The mobilities and patterns of variant LDH-1 and LDH-2 are preserved after successive elution from starch gel, electrophoresis in starch granules (*10*), and repeated analysis in starch gel. Normal and variant LDH-1, isolated by successive starch gel-starch granule electrophoresis, possess similar Michaelis-Menten constants (K_m) for lactate (*10*) and nearly identical reaction rates with several nicotinamide adenine dinucleotide (NAD) analogs (*11*). For example, the reaction-rate ratio of the 3-acetylpyridine analog to the thionicotinamide analog of NAD was 0.15 for normal LDH-1 and 0.14 for variant LDH-1. In contrast, this ratio was 0.73 for normal LDH-5 from human skeletal muscle. The variant pattern is also detectable in LDH from erythrocytes which has

Table 1. Explanation for multiple components appearing within five major LDH zones.

Isozyme	Normal	Variant
LDH-1	B_4	B_4
		$B_3\beta_1$
		$B_2\beta_2$
		$B_1\beta_3$
		β_4
LDH-2	B_3A_1	B_3A_1
		$B_2\beta_1A_1$
		$B_1\beta_2A_1$
		β_3A_1
LDH-3	B_2A_2	B_2A_2
		$B_1\beta_1A_2$
		β_2A_2
LDH-4	B_1A_3	B_1A_3
		β_1A_3
LDH-5	A_4	A_4

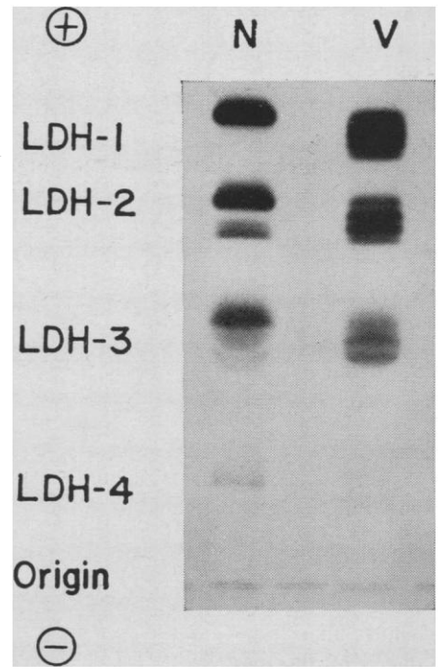

Fig. 1. Starch-gel electrophoretic pattern of normal (N) and human variant (V) LDH from erythrocyte LDH in EBT (*8*) buffer. The nature of the two bands lying just cathodic to normal LDH-2 is unknown (*5*). These components are uniformly present in normal hemolyzates but lacking in LDH of skeletal muscle. We observe such components in both horizontal and vertical starch-gel systems. A similar but very faint set of two bands lies midway between LDH-2 and LDH-3 in the variant hemolyzate. The LDH-3 components are slightly distorted by the coincident migration of hemoglobin A.