Reports

Caries Experience in Twins

Despite widespread and persistent speculation that susceptibility to dental caries in man is a heritable trait, limited data support this belief. Studies of inherited variation in human dental caries experience have produced conflicting findings. Studies of young subjects (*1*) have shown only slight differences in the average intrapair variation in caries experience between monozygotic and dizygotic twins. In data obtained with adults (*2*), however, the mean intrapair difference observed for dizygotic twins was four times as great as that observed for monozygotic twins.

Investigations of susceptibility, or resistance, to dental caries in parents and offspring (*3*) and in siblings (*4*) have generally substantiated the concept of a familial pattern in caries incidence. A critical review of the literature on the relationship of heredity and caries incidence has been presented by Böök and Grahnén (*5*), whose original data on caries-free propositi indicated that genetic factors played an appreciable part in determining individual resistance to dental decay.

Presumably, both environmental and genetic factors are responsible for the initiation of the caries lesion. For genetic problems of this nature, the study of twins offers a suitable investigative procedure and has been utilized in the present investigation.

The study sample consisted of 49 like-sexed pairs of Caucasian twins in good general health, drawn mainly from middle-income residents in New York City. The ages ranged from 18 to 55 years at the time of the study, with the median age 24 years. The diagnosis of zygosity was based on serological and morphological criteria (*6*).

The single tooth surface is considered the unit of measurement most appropriate for caries studies of a limited number of subjects (*7*). Using the method of Klein, Palmer, and Knutson (*8*), we assigned each decayed or filled surface a score of 1 and each tooth missing as the result of caries a score of 5. It is necessary in small-sample studies to account for teeth which through failure to erupt or congenital absence were never available for decay. Teeth lost through trauma or periodontal disease also bias the results if it is assumed that they were extracted because of caries. For these reasons a caries experience ratio (CER) was determined for each subject in the following manner:

$$\text{CER} = \frac{\text{Observed No. of decayed, missing, or filled surfaces}}{\text{Total No. of surfaces originally available for decay (based on 28 teeth, 128 surfaces)}}$$

The use of this ratio makes it possible to subtract an appropriate amount for teeth determined by a clinical history to be missing for reasons other than caries. The total number of decayed, missing, or filled surfaces (DMFS) for the permanent teeth of each subject was determined by means of a clinical examination performed with a mouth mirror and a No. 23 dental explorer, supplemented by a full-mouth series of intraoral roentgenograms. As calculated, the CER represented the total amount of destruction caused by dental caries in the permanent dentition up to the time of the study.

In addition to the CER for the total dentition, ratios were computed separately for different groups of teeth. To obtain these ratios, the appropriate DMFS value was related to the number of surfaces available for attack—that is, 30 in the case of the six anterior teeth (incisors and canines), and 40 for the eight posterior teeth.

A comparison of the mean difference in CER between the two members of dizygotic twin pairs and that between the two members of monozygotic twin pairs was made on the basis of mean intrapair variances. Variance ratios were calculated, and the F distribution was used to obtain the significance level of these ratios (Table 1).

The comparison of the CER of monozygotic with dizygotic twin pairs indicates that there is a measurable genetic component of susceptibility to dental caries. These data confirm the results of the only prior study of caries experience in adult twins (*2*). The discrepancy between the results of studies of adult and juvenile twins suggests that in all probability a hereditary factor in dental caries experience cannot be readily measured until eruption of the permanent teeth is essentially complete.

Comparison of the mean intrapair variances in CER in the four segments of the dentition permits an evaluation of each upon the total difference in CER. The lower anterior tooth group contributes the most to the total difference between monozygotic and dizygotic twins. It has been shown by Knutson *et al.* (*9*) that the lower anterior teeth experience relatively little caries attack, less than any other group. However, this does not permit interpretation of the results of the present study as evidence for a genetic difference in caries susceptibility for the different regions of the dentition, but only indicates that in the area of the lowest caries incidence a genetic component of variability may be measured more readily. Where environmental variation is relatively high, it becomes difficult to make this distinction. It is of interest that the upper anterior teeth, which show the smallest difference between monozygotic and dizygotic twins, normally receive less saliva flow than teeth in the other segments of the mouth. In contrast, the lower anteriors, which provide the largest ratio, are subject to copious saliva flow, being located in an area into which the ducts of the submaxillary and sublingual glands open. Consequently, it appears possible that some genetic characteristic of saliva may be partially responsible for the variations observed in caries experience.

By use of the caries experience ratio (CER), it is possible by study of twins to demonstrate a genetic component of

Table 1. Caries experience ratios. Dz, dizygotic twins; Mz, monozygotic twins.

Group	n (pairs)	Variance	F	P
		All teeth		
Dz	13	78.81	2.74	$< .025$
Mz	22	29.11		
		*Upper anterior teeth**		
Dz	17	58.68	1.57	$> .10$
Mz	27	37.32		
		Upper posterior teeth†		
Dz	17	248.12	2.64	$> .01$
Mz	26	93.92		
		*Lower anterior teeth**		
Dz	19	45.16	5.55	$< .001$
Mz	30	8.13		
		Lower posterior teeth†		
Dz	15	155.53	2.48	.025
Mz	25	62.80		

* Central and lateral incisors, canines.
† First and second premolars, first and second molars.

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variability in the caries incidence of adults in essentially good health. This lends added support to the hypothesis that there is a hereditary factor in susceptibility to caries.

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Life-Shortening by Whole- and Partial-Body X-irradiation in Mice

The fact that ionizing radiations, in whole-body doses which cause little or no immediate morbidity, shorten the life span of animals has been demonstrated in many experiments. With such evidence as a background, it has been argued that *partial-body* exposure, in man as well as in experimental animals, would have a life-shortening effect in strict proportion to exposure dose or integral dose (*1*). Although this concept is misleading for a variety of theoretical reasons, specific experimental evidence relating to life-expectancy after partial-body exposure has not previously been available.

The present data, taken from an experiment designed for another purpose (*2*), illustrate the different potencies of partial- and whole-body x-ray exposure in shortening the life of the mouse. Uniparous female CAF_1 mice (the F_1 generation from the cross, BALB/c females × A/He males) were irradiated at 170 days of age and 26.0 ± 1.4 g body weight. The radiological factors were 250-kvcp x-rays, HVL of 0.55 mm Cu; whole-body exposure dose rate in tissue, 73 r/min; partial-body exposure dose rate in tissue, 53 r/min.

All mice received only a single x-ray dose, with the exception of the 1200-r whole-body treatment which was given as four 300-r fractions 2 weeks apart. All groups (Table 1) were irradiated or sham-irradiated while under moderate Nembutal anesthesia. The whole-body doses were given as described previously (*3*). Mice to be exposed to partial-body irradiation were placed on their backs on 1/16-in. lead sheet, fixed in place with masking tape, and shielded from above (over their ventral surfaces) with 1/8 in. lead sheet. Three different partial-body fields were used: (i) bilateral thorax—from clavicles to tip of xiphoid process; the weight of the tissue irradiated in this field averaged approximately 7.6 g. (ii) Right hemithorax—same as i except for shielding over the left half of the chest; mean irradiated weight, 3.5 g. (iii) Pelvis—the region posterior to a line 1.5 cm anterior to the base of the tail; mean irradiated weight, 5 g.

The exposure doses in tissue given in Table 1 were estimated by placing the sensitive volume of a 100-r Victoreen ionization chamber, surrounded by rice bolus, in a typical exposure field. The dose in the shielded regions was no greater than 4.4 percent of that in the exposed fields, as determined by placing ionization chambers at different points under the shielding while tissue-equivalent bags of rice were being irradiated in the exposure fields. Half the mice receiving 1800 r of partial-body irradiation were irradiated to the right thorax only, the other half to the pelvis as well.

Most of the animals were allowed to die spontaneously, but some (22 percent) were sacrificed when they were moribund. Except the 1200-r whole-body group in which the first death occurred 85 days after the final 300-r fraction, there were no deaths before 169 days postirradiation. Consideration of the data in Table 1 leads to the following comments.

Whole-body exposure. Three hundred roentgens and 560 r both shortened life significantly and to about the same extent. In consequence, the decrement in life span per 100 r (Table 1) is greater at the lower dose. The phenomenon of increased sensitivity of female mice per unit dose, as dose decreases in the range from 600 to 200 r, has been noted previously (*4, 5*). It has been suggested (*5*) that this is somehow related to the peculiarly great sensitivity to x-rays of the mouse ovary.

Partial-body exposure. Per unit of tissue dose, partial-body exposure to the pelvis or chest, or both, was much less effective than whole-body exposure, especially after 300 to 750 r.

Small and large doses. In the whole-body experiments the smallest dose was more effective per unit dose than the larger ones. The smaller partial-body exposures, however, were less effective per unit dose than the larger ones. (The ovaries were not within the fields of partial-body exposure.) Extrapolation of the data for partial-body exposure suggests that, at still lower doses—for example, 100 r—the effectiveness per unit dose may be so reduced as to be negligible. It is of interest to note that doses tested in the present study are hundreds to thousands of times greater than those used in human radiological diagnosis (*6*). Moreover, the total dose built up from repeated diagnostic exposures is fractionated and therefore presumably of diminished effectiveness.

Integral doses. Per unit integral dose, whole-body exposure may shorten life more or less than partial-body exposure, as is shown in the last column of Table 1. In the case of partial-body exposure to one region, the decrement in life span

Table 1. Survival of female mice after whole-body (WB) and partial-body (PB) exposure.

Treatment	Integral dose (kg r)	No. of mice	Mean survival time ± SE (days)*	Decrement in life span per unit dose	
				day/100 r	day/kg r
Control		34	676 ± 25		
300 r, WB	7.8	35	549 ± 24	42	16
560 r, WB	14.6	43	556 ± 23	21	8
1200 r, WB (4 × 300 r)†	31.2	43	429 ± 21	21	8
750 r, bilateral thorax	6.3	34	661 ± 26	2	2
1800 r, right thorax	7.7	20	567 ± 39	6	14
600 r, right thorax + pelvis	5.6	38	660 ± 23	3	3
1200 r, right thorax + pelvis	11.1	40	583 ± 28	8	8
1800 r, right thorax + pelvis	16.7	20	501 ± 37	10	10

* Mean ages may be determined by adding 170 to the mean survival times. SE, standard error of the mean survival time.
† The tabulated figures referring to survival and life span decrement are based on the time elapsed from the beginning of irradiation. Computed from the day on which the final 300-r fraction was given, the last 3 columns of this row would read 387 ± 21 days, 24 days/100 r, and 9 days/kg r, respectively.