liver of hamsters, guinea pigs and rabbits. In the hamsters and the rabbits, liver abscesses were produced which showed the presence of cells morphologically similar to amœbæ; such cells had also diffusely infiltrated the liver.

The passage of the disease from the human to the experimental animals, we believe, gives further support to the view that diffuse amœbic hepatitis exists as a distinct clinico-pathological entity.

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## ANATOMY

### Third Molar Polymorphism and the Timing of Tooth Formation

AGENESIS (congenital absence) of the third molar tooth is a familiar polymorphism in man, with a population frequency of at least 9 per cent in Europeans[1]. Hitherto, congenital absence of this tooth has been considered as an independent trait, or at most associated with reduction in size when agenesis is incomplete. However, there is evidence connecting the presence or absence of the third molar tooth with the timing of formation of the premolar and molar teeth as well.

In a series of 172 white children born in Ohio (excluding monozygotic pairs), agenesis of the lower left third molar was radiographically confirmed in 22 subjects aged fourteen years or older. The time of formation of the remaining lower left premolar and molar teeth was determined from serial, longitudinal oblique-jaw radiographs[2] and expressed as sex-specific normalized $T$-scores[3] so that data on boys and girls could be combined. As shown in Table 1, children lacking the mandibular left third molar were consistently later in premolar and molar tooth formation than 126 'controls' from unaffected sibships.

Table 1. CALCIFICATION TIMING OF THE POSTERIOR TEETH IN CHILDREN LACKING $\overline{M}_3$, THEIR SIBLINGS, AND IN CHILDREN FROM UNAFFECTED SIBSHIPS

| Group | Calcification timing $\overline{P}_1$ | | $\overline{P}_2$ | | $\overline{M}_1$ | | $\overline{M}_2$ | | $\overline{M}_3$ | |
|---|---|---|---|---|---|---|---|---|---|---|
| | $N$ | $T^*$ | $N$ | $T^*$ | $N$ | $T^*$ | $N$ | $T^*$ | $N$ | $T^*$ |
| Affected children | 21 | 53 | 20 | 54 | 18 | 52 | 21 | 59 | — | — |
| Siblings of above | 22 | 56 | 20 | 53 | 17 | 52 | 24 | 52 | 24 | 57 |
| Unaffected children | 111 | 48 | 118 | 49 | 81 | 48 | 126 | 48 | 125 | 49 |

* Normalized sex-specific $T$-scores (ref. 3) for beginning tooth formation, primarily beginning cusp calcification (ref. 4).

Furthermore, 24 unaffected siblings of children with third molar agenesis were compared with the unaffected children from unaffected sibships. These unaffected siblings of the propositi also proved late in tooth formation, especially for mandibular third molar formation which averaged 8 $T$-scores behind the controls (Table 1). For all 150 unaffected children, earliness or lateness of $\overline{M}_3$ formation was significantly associated with the type of sibship from which they came ($\chi^2 = 6{\cdot}4$). Thus, the tendency for delayed tooth formation when the third molar is missing is characteristic of affected lineages and not restricted to affected individuals alone.

Since absence of the mandibular third molar was associated with delayed formation of the molar teeth, it was not surprising to find differences in the sequence of tooth formation[5] between the 22 children with third molar agenesis and the 126 normal controls. Thus, 60 per cent of children in the present work who lacked the mandibular third molar were of the $P_2M_2$ sequence of cusp calcification, while this sequence or order was far less common (22 per cent) in the 126 controls. Third molar polymorphism was unquestionably associated with the $P_2M_2/M_2P_2$ sequence polymorphism ($\chi^2 = 25{\cdot}01$).

Third molar agenesis, therefore, may be viewed as the extreme degree of expression of factors delaying tooth formation over a long developmental period ranging from the first month of life (for $M_1$) to the eighth year or beyond (for $M_3$). Moreover, this common polymorphism is related to the previously described familial $P_2M_2/M_2P_2$ calcification sequence polymorphism[5], indicating a common determinant for the two.

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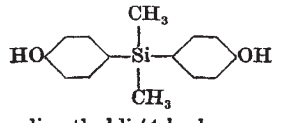
## HISTOCHEMISTRY

### Allergy to a Carbon-Functional Organic Silicon Compound, Dimethyldi-(4-hydroxyphenyl)-silane

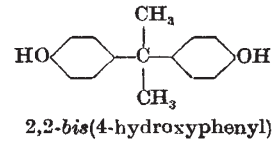
THE noxious effects of certain inorganic silicon compounds are well known[1]. Polymeric organic silicon compounds, on the contrary, are chemically very inert and show a low toxicity[2]. Some organic silicon compounds may, however, produce toxic effects[3].

Inorganic or organic silicon compounds—the latter not present in Nature[4]—are so far not known as allergens.

As a close relationship exists between silicon and carbon atoms, it was assumed that cross-sensitization could occur between silicon and carbon compounds. Therefore the allergenic nature of dimethyldi-(4-hydroxyphenyl)-silane[5] was investigated since the carbon compound, 2,2-*bis*(4-hydroxyphenyl)propane ('Bisphenol *A*'), has been demonstrated as an allergen[6].




dimethyldi-(4-hydroxyphenyl)-silane




2,2-*bis*(4-hydroxyphenyl) propane

Four non-atopic females with hypersensitivity to 2,2-*bis*(4-hydroxyphenyl)propane, demonstrated by positive patch test (1/100 dilution in ethyl alcohol), showed positive reactions to dimethyldi-(4-hydroxyphenyl)-silane (1/100 dilution in acetone). The positive tests were characterized by the presence of erythema, infiltration and papules/vesicles.