Table II. Effect of PCB on ^{3}H-thymidine incorporation (dpm $\times 10^{-3}$) into DNA by mitogen-stimulated lymphocytes

Mitogen		Control		p[a]	PCB		p[a]
		1st bleed	2nd bleed		1st bleed	2nd bleed	
PHA	(1 mg/ml)	25.31 ± 16.58	47.64 ± 12.06	NS	21.11 ± 15.36	70.93 ± 12.90[b]	< 0.005
	(4 mg/ml)	54.73 ± 18.84	60.56 ± 8.17	NS	64.81 ± 32.25	87.26 ± 23.73[b]	NS
PWM	(50 µg/ml)	13.84 ± 6.61	20.73 ± 6.52	NS	14.44 ± 7.66	18.88 ± 7.01	NS
	(250 µg/ml)	14.21 ± 2.23	15.22 ± 4.44	NS	10.29 ± 5.67	12.81 ± 2.82	NS

Each value is the mean ± SD of 5 animals. [a] Student's t-test for paired differences; [b] $p < 0.05$ vs second bleed controls (Student's t-test).

similar in control and treated rats, which tends to rule out non-specific effects related to cachexia or stress. Thymus weight was reduced in the PCB group, in keeping with previous observations[6,7] and suggesting possible immunosuppression.

Lymphocyte responses to PHA and PWM are often used in human and animal studies as indices of cell-mediated immunity and antibody production[10]. In our experiments the response to PHA was unexpectedly enhanced in lymphocytes from PCB-treated rats. At first glance this seems to be at variance with the report by Vos and VAN DRIEL-GROOTENHUIS[7], who showed suppression of cell-mediated immunity in PCB-treated guinea-pigs by delayed hypersensitivity skin reactions to tuberculin. However, our data need not necessarily be contradictory. Allergic skin reactions and lymphocyte transformation in response to PHA need not necessarily correlate[11]. Moreover, in guinea-pigs treated with cyclophosphamide, a well recognized suppressor of cell-mediated immunity, the lymphocyte response to PHA was not suppressed[12] and in some patients treated with cyclophosphamide the proliferative response of lymphocytes to PHA was increased[13]. Two possible mechanisms for this paradoxical effect were suggested[13], first a selective depletion of B lymphocytes not activated by PHA and an increase in the T:B ratio and secondly a non-lethal injury of PHA responsive lymphocytes, with the enhanced proliferative response reflecting chromosomal repair. Similar mechanism(s) may be responsible for the enhanced lymphocyte response to PHA in PCB-treated rats. Thymic atrophy and hypogammaglobulinemia in PCB-treated animals certainly do suggest immunosuppression.

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Familial Association in Serum IgE Levels

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Summary. The serum IgE levels measured by RIST correlated closely with those obtained by the single radioimmunodiffusion method. Values for husband and wife were closely related as were those between father and daughter but in contrast to previous observations no significant relationship existed between IgE levels for mother and son.

The population of Busselton, a small rural town in Western Australia, has been subject since 1960 to mass health examination surveys at three yearly intervals. Since the population is relatively static for most of the time and only resident families are included in the survey this would therefore appear to be an ideal situation in which to examine matters of epidemiological interest.

TURNER, ROSMAN and O'MAHONY[2] reported the prevalence of asthma, hay fever and eczema in the Busselton school children aged 6 to 17 years who were interviewed during the Spring of 1970. They also assayed serum IgE levels in 1,069 of the children and related the serum IgE levels to the prevalence of allergic disease. As anticipated from the results of other studies[3–5] elevated serum IgE levels correlated well ($p < 0.001$) with the incidence of allergic disease. Evidence was presented for an inheritance pattern of both asthma and hay fever and genetic factors were also suggested by correlation of serum IgE levels. It appeared that these were sex-linked in that mothers' IgE values correlated more closely with that of their sons

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Table I. Correlation coefficients between an individual's serum IgE levels in samples taken 3 years apart

Group	No.	Spearman Rank correlation coefficient	Significance (p)
Wife 1969 – wife 1972	258	0.3239	0.001
Husband 1969 – husband 1972	257	0.2995	< 0.005
Mother 1969 – mother 1972	142	0.5325	< 0.001
Father 1969 – father 1972	142	0.5076	0.001
Child 1970 – child 1973	236	0.4471	0.001

Table II. Correlation coefficients between serum IgE values of husband and wife

	Spearman Rank correlation coefficient	Significance (p)
Wife 1969 – husband 1969	0.1218	< 0.005
Wife 1972 – husband 1972	0.315	< 0.001

Data represents 257 married couples.

Table III. Correlatfon coefficients between serum IgE values of parent and child 1969/1970 and 1972/1973

Group	No.	Correlation coefficient	Significance (p)
Mother 1969 – daughter 1970	118	0.1628	< 0.05
Mother 1972 – daughter 1973	118	0.1737	< 0.05
Mother 1969 – son 1970	117	-0.0047	NS
Mother 1972 – son 1973	117	0.1205	NS
Father 1969 – daughter 1970	118	0.2335	< 0.01
Father 1972 – daughter 1973	118	0.2207	< 0.01
Father 1969 – son 1970	117	0.0710	NS
Father 1972 – son 1973	117	0.0786	NS

than with their daughters and conversely fathers' values correlated more closely with daughters than with those of their sons.

An unexpected finding was the significant correlation between serum IgE levels of husbands and wives suggesting non-genetic influences were also operable. TURNER, ROSMAN and O'MAHONY[2] interpreted this data on the basis that micro-environmental factors, particularly those associated with specific antigenic exposure of the home, exerted an influence on IgE synthesis which masked genetic factors. If there was any merit in the latter hypothesis it could be anticipated that the serum IgE levels of husband and wife should approximate more closely as a function of the duration spent in the same micro-environment. The aim of the study reported in this paper was to test this hypothesis and to reaffirm by analysis of sera taken in the recent surveys, 1972 for adults and 1973 for children the parental influence on serum IgE levels reported previously[2].

IgE levels were assayed in the sera of 257 married couples, for whom values were obtained following the 1969 survey, who attended the 1972 adult survey. Assays were also made on the sera of 235 children sampled in 1973 and whose parents represented 141 of the above group of married couples. All of these children were included in the group reported previously[2]. IgE was assayed by the RIST method[6] using commercially available Phadebas kits in contrast to those reported in the previous publication[2] where the single radio immunodiffusion method of ROWE[7] was employed. Since the distribution of serum IgE levels was extremely skew, non parametric statistics were applied to the data.

Table I shows the correlations between an individual's serum IgE and that obtained on serum taken 3 years later for both parents and children. The relationships, which were statistically significant, indicate that no discrepancy could be anticipated by utilizing 2 distinct assay systems for the measurement of serum IgE. Moreover it showed that an individual's IgE value was surprisingly constant over the 3 year span if the blood samples were taken at the same period of the year. Similar correlations between serum IgE values measured on the same sample by 2 different assay systems and with the same assay system on successive serum samples from the same individual have been reported by NYE et al.[8]. The correlation between IgE values for husband and wife reported previously[2] were also found in the later samples (Table II). It is of interest that the IgE values of husband and wife approximate more closely in the 1972 sample than they did in the 1969 samples in support of the postulation made above.

The serum IgE values of fathers (Table III) were more closely related to those of their daughters ($p < 0.01$) than with those of their sons (NS) in accordance with the results of our previous report which was undertaken on a larger number (260–295) of father-child pairs than the present study (117). The present study, however, failed to demonstrate the significant correlation reported previously[2] between IgE values of mothers and their sons, but revealed an unexpected correlation between values found for mothers and daughters. It is possible that this result could be explained on the basis of relative number of parent-child pairs studied in the present compared with past reports. However, in view of this lack of agreement it is apparent that confirmation of the influence of the X chromosome on IgE synthesis must await more extensive investigation.

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