Equus asinus) where abnormalities of synapsis at the primary spermatocyte stage interrupt spermatogenesis. Rearrangements in these viable hybrids include the position of the centromeric heterochromatin[20], but quantitative measurements have not yet been published. In the present case there is a clear difference in the size of the C-bands in the 2 species and quantitative measurements in the hybrid and its parents are being made. At present the significance of centromeric heterochromatin, if any, in connection with hybridization, is not understood.

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High IgM levels in women coincide with reproductive phase

F.J. Grundbacher

Department of Basic Sciences, Peoria School of Medicine, University of Illinois Medical Center, 123 S. W. Glendale Avenue, Peoria (Illinois 61605, USA), 28 January 1980

Summary. Quantitation of IgM in 875 individuals ranging in age from 2 to 95 years revealed that levels in females increased steadily until 15 years of age, remaind high until 40, decreased during the forties and fifties and remained stable after 60. Levels in males remained stable after 2 years of age.

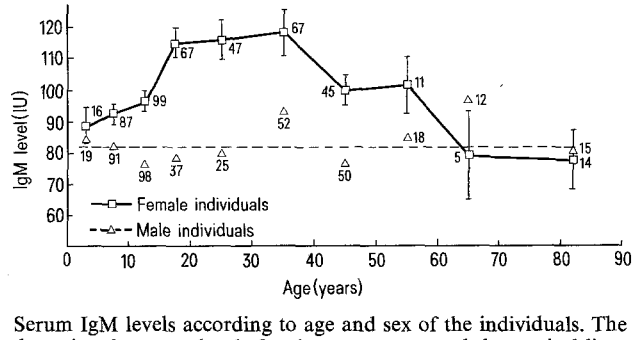
Among the 5 classes of immunoglobulins, IgM is the only one that has repeatedly been shown to be higher in females than in males[1-4]. The number of X chromosomes appears to have an effect because in XO females the IgM level equals that of males and in XXY males it equals that of females[5]. Furthermore, immunoglobulin studies on parents and offspring supported the hypothesis that the X chromosome carries quantitative genes for IgM[4].

Although general agreement seems to exist concerning the higher levels of IgM in women than in men, the duration of this sex difference seems not to have been documented. Our population studies on immunoglobulin levels provide pertinent information on the sex influences on IgM according to age of the individual. This information is summarized in the current report.

The individuals studied were obtained through the Medical College of Virginia Hospital and from the Tecumseh Community Health Study[3,6]. Both sources represented approximately average human populations. In Tecumseh it included the study of a whole community and in Virginia the individuals studied were close relatives of mothers who were in the hospital for delivery during the years 1961-1963 and who lived in the City of Richmond and surrounding counties. A total of 875 individuals, 417 males and 458 females of the black and white populations were included in the study. The individuals ranged in age from 2 to 95 years. To correct for the previously recognized higher mean IgM levels in blacks[6], their levels were multiplied by a factor of 0.89 to bring them to the same mean level as whites. This appeared feasible because blacks and whites displayed practically identical frequency distributions as was shown elsewhere[6]. IgM was quantitated by radial diffusion techniques under standardized conditions with commercially available plates[6].

The IgM levels according to age and sex are shown in the figure. The levels for males fit well to a linear regression line ($Y = 81.96 + 0.0045\ X$) the slope of which is very close to horizontal. None of the means for the various age intervals is significantly different from the grand mean ($p > 0.05$).

IgM levels in girls show a gradual increase up to age group 10-14. The increase is steep from this age group to that 15-19 years of age. A check of the annual means revealed that a rapid increase took place at 14-15 years of age. Each of



Serum IgM levels according to age and sex of the individuals. The data give the mean levels for the age groups and the vertical lines with crossbars represent SEM for females. The group on the far left includes children 2-4 years of age and that on the far right includes all individuals 70-95 years of age. The numbers in the figure represent the numbers of individuals in the age groups.

the means for age groups 15–19, 20–29 and 30–39 is significantly higher than for age group 10–14 ($p < 0.01$). The IgM levels show a gradual decrease after age 40 and remain stable in women 60 years of age and older. For all the age groups between 5 and 49 years of age the means are significantly higher in females than in males ($p < 0.02$ and smaller). However, the means are not significantly different for the children aged 2–4 and also not for the age groups 50 years of age and older ($p > 0.05$). As the figure shows, the sex difference seems to have disappeared completely in individuals 60 years of age and older.

The foregoing results clearly show that IgM levels are elevated in girls and younger women but that this elevation disappears in older women. The time of the highest elevation, from 15 to 40 years, coincides roughly with the reproductive phase in women. The question arises whether the increased IgM levels did evolve to afford the women increased immunologic protection during pregnancy because it is difficult to imagine that such a marked sex difference would have arisen just by chance alone. Girls are more resistant to certain types of infection than boys are[7], indicating that the difference in IgM is of biological importance.

Sex hormones associated with adult gonades have been suggested to be concerned with certain aspects of resistance. The decrease of IgM in women after 40 shows some similarity to the decrease in estrogen secretion[8]. However, in girls the IgM increase is quite different from the rapid preadolescent increase in estrogen secretion. Also, the difference in resistance in children seems to long precede the marked difference in hormone secretions between the sexes[9].

Although the marked changes in females from early childhood to old age have not previously been demonstrated, evidence supporting the present findings does exist in the literature. For children, the data of Allansmith et al.[1] show much higher IgM levels in girls aged 15–16 and 17–18 than younger girls, while IgM levels in males remained stable after 2 years of age. Data showing a significant decrease of IgM in women over 40 years of age were published in 2 reports[10,11]. As in our study, the sex difference had disappeared by about 60 years.

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Morphology of the carnation-light synthetic lethal focus in *Drosophila melanogaster*

A. McCarthy and H. Nickla

Department of Biology, Creighton University, Omaha (Nebraska 68178, USA), 3 March 1980

Summary. A histological study of the carnation-light lethal focus revealed morphological abnormalities in brain tissue. The ratio of core width to total brain width and brain texture consistently differed between lethal *(car-lt)* and their non-lethal sibs.

2 questions fundamental to an understanding of developmental mechanisms relate to the time and anatomical site of gene activity. Because of their high frequency of occurrence, lethal mutations provide a rich source of experimental material since each lethal mutant reveals a specific development action for a mutated chromosomal region. *Drosophila* lethals are generally classified as to phase specificity (larval, pupal, adult) and morphologic abnormality thereby indicating the temporal and spatial requirements for a normal gene product. We used sex mosaic fate mapping[1-3] to determine that nerve tissue is the anatomical site (focus) at which the *carnation* (*car:* position 62.5 on the X chromosome) and *light* (*lt:* position 55.0 on the 2nd chromosome) loci exert their lethal interaction[4]. More precisely, the focus was located in the ventral region of the blastoderm surface approximately 7 sturts from the blastodermal origins of the legs and 17 sturts from the midline.

In this paper we reveal the primary anatomical lesion at the lethal focus as well as other anatomical sites which may represent secondary areas of damage. Focus determination in conjunction with histological studies has been used in *Drosophila* mutants such as *wings-up* and *drop-dead.* The *wings-up* mutant mapped to presumptive mesoderm, and histological examination of flight muscles showed myofibril defects and muscle atrophy. The *drop-dead* focus was located in the brain which showed extensive degeneration upon histological examination[3].

Penetrance of the *car-lt* lethal interaction is complete such that all double mutants *(car/car; lt/lt* and *car/Y; lt/lt)* die before ecolosion. This synthetic lethal is unique in that the lethal phase (3rd instar larva, prepupa, midpupa, late pupa) is specified by the number of maternal car^+ and/or lt^+ genes[5]. All strains of *D. melanogaster* were reared at 25 °C in half-pint milk bottles containing standard cornmeal, yeast, molasses, sucrose, agar medium except that riboflavin was added to the medium to intensify Malpighian tube color and therefore allow a sharper distinction between the lt^+/lt and lt/lt larvae[6]. This intensification occurs even in crosses where a maternal effect influences pigmentation of the Malpighian tubes[7]. Age-controlled larvae were obtained by the method previously described[8]. Larvae were prepared for histological examination by fixation in Dietrich's solution[9], alcohol dehydration, and toluene clearing[10]. 7-µm sections were stained with Delafield's hematoxylin and counter-stained with aqueous eosin[11]. Histological data were collected from tissues deemed significant from