ma mixed with tissue homogenates was incubated at 4 °C for 24 h. This observation, and the results presented, indicate that the differences in organ distribution of HMW-kininogen does not depend directly on the amount of blood remaining, or on different rates of postmortem digestion of this protein. On the basis of this study it could be discussed whether HMW-kininogen may be synthesized in the kidneys, adrenals and thyroid or whether the results reported were due to an effect of accumulation of HMW-kininogen in these organs caused by the disease or processes preceding death, although the protein is produced elsewhere.

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H-Y Evolution

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Summary. The female: male sex ratio in litters born to C57 black mice immunized with male spleen preparations is 2:1 and males were stillborn. In addition *Drosophila busckii* males are H-Y antigen positive. It is argued that H-Y is the primary testicular determinant and that this role is evolutionarily ancient.

H-Y antibodies are produced by inbred female mice after immunization with male cells from the same strain[1]. H-Y antigen is considered to be the primary sexual determinant and the antigen is thought to be the product of the mammalian testis determining gene[2]. H-Y appears to be Y linked in mammals[3], but H-Y cross reaction has been observed in males lacking a Y-chromosome[4]. These findings suggest that progenies of immunized females should be primarily or entirely female. Attempts to alter the sex ratio of mice at birth by immunizing the mother against H-Y antigen were, however, unsuccessful when skin transplantation was the method of immunization[5]. The sex ratio in litters born to C57 black mice immunized with male spleen preparations is 2:1; moreover, all males were stillborn and showed signs of runting and developmental abnormalities (unpublished data). This result supports Wachtel's prediction of a primary sex determining role for H-Y in vertebrates[2].

Most evolutionary biologists see gene exchange as the essence of sex[6], and it has recently been suggested that cellular recognition signals evolved from sexual recognition signals similar to those seen in *E. coli*[7]. Assuming that sex evolved but once and that H-Y is the male determinant, leads to the prediction that cross reaction should be observed in an array of taxonomically diverse organisms. Murine H-Y antiserum gives positive cross reactions with male rat, guinea-pig, rabbit, human, leopard frog[8] and fish[9]. The antigen has also been located in *Xenopus laevis*, White Leghorn chicken[8] and trout females[10]. More recently Pechan (unpublished data) has obtained positive cross reaction with male cells from *Drosophila busckii*. This, in conjunction with the other observations, suggests that H-Y is evolutionarily very old. While Wachtel's[2] suggestion that H-Y stimulates gonadal differentiation of the heterogametic (XY males or ZW females) sex appears to hold in a wide variety of organisms, the suggestion that H-Y directs different developmental pathways in XY and ZW sex determining systems can be questioned. The male determining to female determining switch of H-Y would have to have occurred at least twice, once in the lineage leading to birds and once in that leading to Lepidopterans. Given the apparent evolutionary conservatism of H-Y, this seems unlikely. Agreeing that H-Y is the primary sex determinant, we would like to suggest that the functional H-Y gene exists only in the male of all species. In this scheme heterogametic female positive cross reaction with H-Y murine antiserum is due to spurious cross reaction with cell surface communication components which are closely related to H-Y in that they have a common evolutionary origin. Indeed steric interference between H-2 and H-Y has been reported[11].

In unicellular organisms where mating type is determined by a single gene, the gene product is, most likely, a membrane component which facilitates fertilization or gene exchange. 2 evolutionary schemes relating H-Y antigens to other cell surface communication components may be considered. First, H-Y is derived directly from one of these primitive sex determining alleles, and the spurious cross reacting components developed as cellular recognition signals related to H-Y through gene duplications. Second, the primitive sex determining alleles, *m* and *f*, produced the H-Y gene by duplication. Isogamety requires only single gene sex determination; thus, in arguing for cell surface communication components and H-Y molecular relatedness we start at the evolution of 2 sexes from the point at which isogametes were produced[12]. The advantages accruing to those individuals producing larger than average gametes (female) and to those producing smaller than average gametes (male) were maximized when a small gamete united with a big gamete. In this union the advantages of motility and nutritional resources were combined. Larger × larger and smaller × smaller fertilizations would be selected against by improbability of fertilization and inadequate nutrition for initial development. Heterogamety

clearly requires cell differentiation, and gonads capable of producing these large and small gametes. It seems reasonable to suggest that this differentiation was associated with the evolution of heterogamety from isogamety. In addition, we suggest that the DNA required for this change came from H-Y duplication or *m-f* gene duplication. This mechanism predicts the observed H-Y cross reaction with ZW and XX females, without invoking a switch in the sex determining role of H-Y.

Finally, we would like to suggest that positive H-Y cross reaction in humans lacking a Y is most readily explained by assuming that the H-Y locus is present as part of a heterochromatic rearrangement. This idea may be extended to suggest that balance sex systems are, at base, also H-Y determined. Many Y-autosome rearrangements are known and such rearrangements may account for descriptions of sex determining mechanisms which do not appear to depend on a primary sex determining locus. For example, in *Drosophila* the sex chromosome/autosome ratio describes the sex of an individual. If an X linked H-Y repressor is present in *Drosophila* and H-Y is, indeed, autosomal, then the balance description of sex determination is consistent with an H-Y system. Numerous attempts to locate the sex determining genes in *Drosophila* have been unsuccessful. Pipkin found none in the 2 major autosomes[14] and Bridges[15] eliminated the 4th chromosome. Although Dobzhansky and Schultz[16] reported female-determining genes on the X, these were later shown by Patterson et al.[17] to be non sex determining fertility factors. Now that mapping in the proximal heterochromatin of *Drosophila melanogaster* is more readily done, the H-Y gene should be mapped.

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Effect of some insecticides on cellular lipids of the neurosecretory complex of the red cotton bug, *Dysdercus koenigii*

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Summary: In normal specimens of the bug, *Dysdercus koenigii*, the cells of the pars intercerebralis stain highly positively with Sudan black B and acid haematein, while the corpora cardiaca and allata stain lightly. After the administration of parathion, carbamate and endrin the situation is reversed. The increased level of lipids in the corpora cardiaca and allata coincides with the degree of loss of lipids from the cells of the pars intercerebralis.

The involvement of a number of enzymes and metabolites in the toxic action of organophosphate, carbamate and cyclodien insecticides has been reported by a number of investigators and reviewed by O'Brien[2], but hormonal involvement, direct or indirect, in insecticidal action is still not clear. In this context, it was considered desirable to investigate cytochemical changes that occur in the neurosecretory complex under the influence of various insecticides. Accordingly, the present communication reports the effects of parathion, carbamate, and endrin on lipids in the pars intercerebralis, corpora cardiaca and corpora allata of *Dysdercus koenigii*.

For experiments, 2-day-old adult male and female specimens of the red cotton bug, *D. koenigii*, were separated from the main stock maintained in the laboratory at 27 °C ± 1 and 80% relative humidity. They were divided into 4 groups of 30 each (15 males and 15 females). The animals of groups I-III were treated topically with 2 µl of 2% parathion (dimethyl p-nitrophenyl phosphothionate) 2 µl of 3% carbamate (1, naphthyl-methyl carbamate), and 2 µl of 2.5% endrin in acetone, respectively. The dosages were so adjusted that the insects of each group died after 1 h and 30 min. Animals of group IV, that served as control, were treated with 2 µl of 2% acetone. Each experimental group was further subdivided in to 3 groups of 10 insects (5 males and 5 females) each depending upon the symptoms of intoxication i.e. hyper-excitation, moribundity and paralysis.

The brains, along with the corpora cardiaca and allata of the above mentioned groups of bugs were dissected out, and fixed in formol calcium with post chrome and weak Bouin's. 10-µm gelatine sections were cut and stained in Sudan black B (SBB) for total lipid[3] and acid haematein (AH) for phospholipid[4]. Sections of tissues fixed with weak Bouin's fluid were processed for the pyridine extraction control technique[5]. Paraffin sections of tissues fixed after Helly were stained by Berenbaum's technique[6] for bound lipids.