balance in the medium it is possible to activate a graded series of genetic loci, every Na^+: K^+ ratio inciting different groups of loci to form puffs (Fig. 1). The series of puffing patterns induced by stepwise replacing Na^+ by K^+ is identical with the sequence of puffing patterns which in salivary glands marks the transition from the adult larva to the middle pre-pupa.

In previous experiments[1,2] it was shown that the insect hormone ecdysone, and possibly also juvenile hormone, do not act directly on the genetic loci of a cell but instead exert their effect on a system in the nuclear sap which in turn controls the activity of at least a large fraction of the genetic loci in the chromosomes. It has been concluded that this control system can be present at different levels, each level inciting some loci to become active, others to become inactive. Since the stepwise replacement of Na^+ by K^+ produces the effect of those 'different levels', it seems now that the control system actually consists of the Na^+/K^+ balance in the nuclear sap; both hormones would then act by regulating the ratio between Na^+ and K^+, ecdysone increasing the K^+ concentration, juvenile hormone maintaining a high Na^+ level.

The existence of mechanisms for the extrusion of Na^+ in almost all somatic cells suggests that activation and inactivation of genetic loci by a progressive shift in the Na^+/K^+ balance in the nuclear sap may be a rather universal mechanism.

This work was aided by grants from the Schweizerischer Nationalfonds zur Förderung der wissenschaftlichen Forschung and by the Damon Runyon Memorial Fund (*DRG*-668).

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Differential Ribonucleic Acid Synthesis of *X* and Autosomes during Meiosis

SEVERAL investigations[1-3] of chromosomal RNA synthesis during mitosis have established that such synthesis, maximal in interphase, continues during mitotic prophase. It decreases progressively as chromosome contraction advances, none occurring during the fully contracted stages of metaphase and anaphase. RNA synthesis re-commences with chromosome despiralization at late telophase.

Only two accounts of RNA synthesis throughout meiosis have been published[4,5]. In one, inorganic phosphorus-32, which is incorporated into DNA, RNA and protein, was used, while in the other, ^{14}C-glycine, ^{14}C-orotic acid and ^{3}H-cytidine were used. The relatively RNA specific precursor, uridine, was not utilized, and the results presented are not entirely unambiguous. RNA synthesis was shown to continue into early meiotic prophase but with some precursors a drop was reported at the time of DNA synthesis and at the zygotene pairing stages.

I have sought more information on this question by following the course of RNA biosynthesis autoradiographically through all stages of male meiosis in the locust *Schistocerca gregaria*, using ^{3}H-uridine. All autosomes actively synthesize RNA throughout the whole of first meiotic prophase. Leptotene and zygotene nuclei are evenly labelled after short exposures to the isotope. After longer exposures the two small nucleoli are noticeably more heavily labelled than the remainder of the nucleus. During pachytene and diplotene the truly lampbrush nature of the fuzzy bivalents is evidenced by their continued synthetic activity. Synthesis decreases progressively during diakinesis, as chromosome coiling becomes more and more complete, and has ceased by the time that nuclear membrane breakdown occurs. No RNA precursor is incorporated into any autosome during first metaphase or anaphase. During second prophase RNA is again synthesized by the uncoiled autosomes. Incorporation decreases as chromosome coiling becomes more advanced and it does not occur at the fully contracted stages of second metaphase and anaphase.

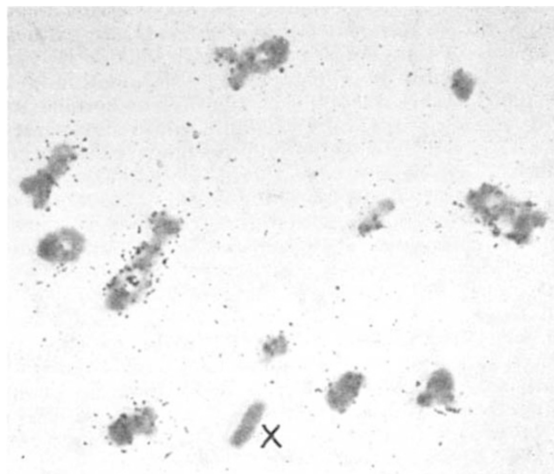

Fig. 1. Diakinesis in *Schistocerca gregaria* showing the distribution of newly synthesized RNA, labelled with ^{3}H-uridine. All autosomal bivalents are labelled, but the *X* univalent is not

Throughout most of meiosis the single *X*-chromosome is allocyclic relative to the autosomes. It is tightly coiled and heavily staining during both first prophase and, in those cells in which it is found, second prophase. In this condition it is synthetically inactive. No RNA precursor is incorporated and the *X*-chromosome is unlabelled throughout its entire length (Fig. 1).

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Effect of *p*H and Temperature on Cell Re-aggregation

THE successful re-aggregation of cells has been used widely as a test for their adhesiveness[1,2]. Moscona[1] observed that the re-aggregation of chick embryonic cells does not occur at temperatures at or below 15° C. Steinberg[2] found that re-aggregated cells did not de-adhere at low temperatures and re-interpreted Moscona's results by suggesting that the initial adhesions of cells alone were prevented below such temperatures. Both authors interpreted their results to indicate that the adhesion of cells in re-aggregation involves a temperature-dependent mechanism. Similarly, Steinberg[3] argued that since amphibian embryonic cells could not re-aggregate at *p*H 4·0, carboxyl ionization and calcium-linked binding between cell surfaces are probably involved in cell adhesion. It remains possible that the failure of re-aggregation in these systems was due to the particular conditions of re-aggregation used, and that low temperature or *p*H values do not of themselves generally impede the adhesiveness of normal viable cells. Successful re-aggregation of cells at low temperatures or *p*H values will demonstrate that low temperature and *p*H do not in themselves necessarily affect cell adhesion. The following tests show that this suggestion is probably correct and that other conclusions can be drawn about the modes of cell adhesion.

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