termination of diapause I. Since the inter-diapausal developmental time is less than 1 week, the embryonic population quickly enters the 2nd stage of diapause before the drought conditions become intense.

Photoperiodism is the only predictable environmental stimulus by which organisms sense the changing of the seasons. The short-day photoperiods experienced by the maternal generation during the later part of the wet season stimulates the production of embryos pre-programed to undergo diapause II[5,6]. Previous studies on the effects of extreme temperatures have shown that diapause II is the most resistant stage in annual fish ontogeny[8]. Preliminary data also indicated that the chorion of diapause II embryos are most resistant to proteolytic digestion by pronase in comparison to all other stages (unpublished). In the light of the present data on the effect of partial desiccation, it is possible that the vast majority of *N. guentheri* embryos may exist in the form of diapause II during the major part of the dry season. The retardation of diapause termination by partial desiccation may complement the photoperiod effect to assure the survival of the embryonic population until the next rainy season.

Studies under controlled laboratory conditions may not necessarily parallel what occurs in nature. Confirmation of the present hypothesis will have to wait until detailed field studies become available. The elucidation of the mechanism of annual fish survival in nature is essential since *N. guentheri* is presently being considered as a possible biological control agent of malarial mosquitoes in nature[13].

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Receptors for thymosin fraction V on rat thymic lymphocytes[1*]

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Summary. Binding by rat thymus lymphocytes of thymosin V, labeled with colloidal gold, was studied. Under the experimental conditions employed at least 2.8% cells exhibited thymosin binding sites.

Peptides of fraction V of thymosin induce expression of T cell surface antigens (Thy, Tl, Ly)[4-6] and differentiation of presursor cells into T_1, and then T_2 immunologically competent cells[7].

Although the mechanism of action of these peptides is still controversial, data from several laboratories indicate that interaction of a target cell population with thymic peptides is mediated by cyclic AMP[8]. Until now, specific surface receptors for thymic peptides have been demonstrated on the following target cell populations; receptor on T cell lymphoblastoid lines for serum thymic factor (FTS)[9], receptors for thymus factor X (TFX, peptides from calf thymus) on rat thymocytes[10].

In this report we present evidence for surface receptors of rat thymic lymphocytes for colloidal gold-labeled peptides of thymosin fraction V.

Material and methods. Thymic lymphocytes were teased from thymuses of Wistar rats, aged 36 days. The suspension of lymphocytes was centrifuged at $2000 \times g$ and washed in PBS. The cell suspension contained approximately 99% lymphocytes, of which 98% were viable, as evidenced by the trypan blue dye exclusion test. Washed cells were fixed with 2.5% glutaraldehyde in 0.5 M phosphate buffer, pH 7.3 for 15 min at 4 °C.

Colloidal gold was prepared by reducing chlorauric acid ($HAuCl_4ICN$, Merck) with trisodium citrate, according to the method described by Georghegan and Ackerman[11]. The gold particles obtained were 20 nm in size. Thymosin was added directly to colloidal gold in a dose of 0.2 mg/ml, at pH 7.4. The gold-protein complex (T-Au) was centrifuged several times to wash the colloidal gold free of traces of nonadsorbed peptides and then was diluted in PBS. Thymic lymphocytes were incubated with T-Au for 60 min at room temperature. Further processing included washing, postfixation in 1% OsO_4, dehydration and embedding in resin according to Spurr, in a routine way. Ultrathin and semithin sections were contrasted in a routine way and examined in JEOL JEM 100 C electron microscope.

The main control reaction involved preincubation of the cells with a solution of thymosin (0.2 mg/ml) prior to incubation with T-Au. For other control reactions the following media for incubation of cells were used: bovine albumin-Au complex prepared in the same way as T-Au and a solution of uncoated colloidal gold.

Results. The site of interaction of thymosin peptides, labeled with colloidal gold, and the cell surface were localized by electron microscopy. The reaction product was detected as electron dense particles on the surface of rat thymic lymphocytes (fig. 1). Occasionally encountered non-lymphoid cells (red cells, macrophages, eosinophils) remained unlabeled. Only 2.8% cells were positive as counted in electron micrographs. The intensity and distribution pattern on the surface of the cells varied markedly. In some thymic lymphocytes gold particles covered only some region of their surface, in others the label in small patches covered a large fraction of cell membrane outline. Therefore, the real percentage of labeled cells may be higher and could be estimated exactly only on analysis of serial sections or by examining smears of labeled thymocytes. Lymphocytes with a narrow rim of cytoplasm, containing scanty organelles and medium sized nuclei, rich in condensed chromatin, were most intensely labeled (fig. 2). Lymphocytes with large nuclei and prevalent euchromatin were found to bind T-Au complexes only on part of their cell surface. Small numbers of T-Au grains were noted also on cell membranes of some dividing cells. The control reactions were negative throughout.

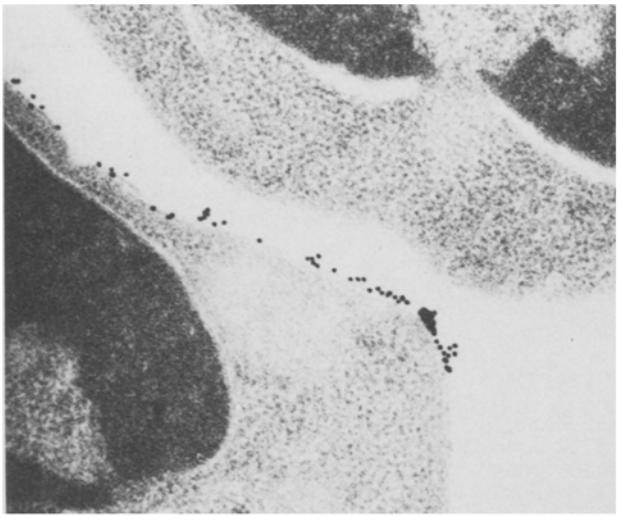

Figure 1. Demonstration of the thymosin-gold-binding sites at the plasma membrane of the thymic lymphocytes. × 33,800.

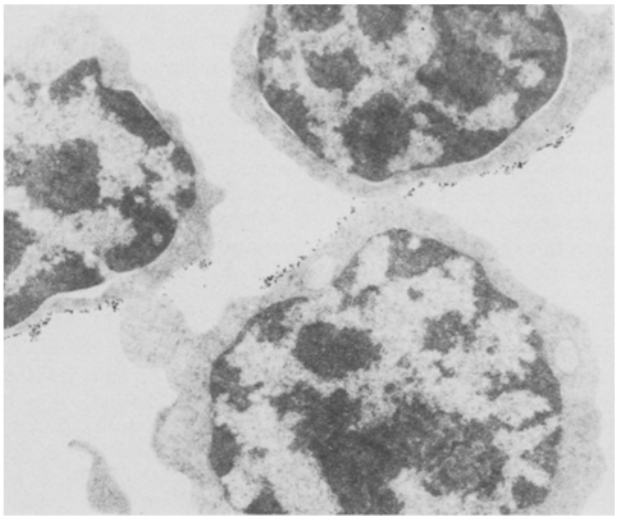

Figure 2. Thymosin-binding lymphocytes. T-Au complex covered some region of cell surface. × 10,000.

Discussion. The experiments showed that the lymphocyte surface labeling, observed in the EM, involved a specific reaction of binding thymosin peptide(s) by cell surface receptors. The reaction could be blocked by preincubating the cells with thymosin before incubation with T-Au complex. Lack of T-Au binding by erythrocytes as well as by other non-lymphoid cells in the thymus seems to exclude T-Au binding by putative free glutaraldehyde groups following glutaraldehyde fixation. Also, no labeling could be obtained when T-Au was substituted by albumin-Au or uncoated colloidal gold while similar labeling results were noted when formaldehyde-fixed or unfixed, cold treated thymocytes were labeled with T-Au. The labeled cells seem to represent a certain subpopulation of lymphocytes with free receptor sites for xenogeneic thymic hormones. If bovine and rat thymic hormones are trapped by identical receptors on rat thymocytes, the unlabeled majority of rat thymocytes may represent cells the receptors of which have been saturated in vivo by the endogenous hormone.

The morphological observations of this paper indicate that thymosin-binding lymphocytes are heterogenous both in ultrastructure and the cell cycle stage. Further characterization of such thymocytes is in progress.

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Night pineal N-acetyltransferase activity in rats exposed to white or red light pulses of various intensity and duration

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Summary. Night N-acetyltransferase activity is suppressed by red and white light; the red light intensity, however, must be 10 times higher. Short light pulses also suppress night N-acetyltransferase; the higher the light intensity, the shorter the pulse is effective.

Rat pineal N-acetyltransferase (NAT) activity increases more than 100 times within a few hours after evening lights off[2]. When lights are kept on, the evening increase does not occur. Dark-induced high NAT activity in rats exposed either to prolonged light[3,4] or to a 1-min light pulse[5] at night declines rapidly and within 20 min it reaches 1/10 of its original value. Photoreception of red by rats is supposed to be poor or absent and hence all handling and killing of rats at night is usually done under red light[6,7]. However, entrainment by red light of running activity rhythm of