flux measurements reported in the literature over similar ecosystems (*12*).

Simultaneous traces of the fluctuations in air temperature, CO_2 concentration, and vertical velocity as measured above these surfaces are shown in Fig. 1. Over the corn crop, upward-moving eddies were warmer than downward-moving eddies by as much as 0.6°C and the CO_2 concentrations of the upward-moving eddies were lower by as much as 6 ppm, signifying the release of heat and the absorption of CO_2 by the corn crop. Fluctuations in the CO_2 concentration were slightly smaller over the forest and much smaller over the water.

Individual and average CO_2 heat flux densities over these surfaces are presented in Table 1. Nonsteady-state conditions may contribute to some of the deviations, particularly for the necessarily short runs over the cultivated fields. In order to evaluate the effect of short sampling time, data from 3-minute measurements over the forest were treated as six 30-second periods. The lack of significant difference indicates that, over extended surfaces, at least 30-second measurements might be adequate. Over short fields with limited fetch, the effects of nonsteady state and spatial inhomogeneity on short-term sampling need to be further investigated, and a displacement equation (*13*) may have to be used. The slight tendency for flux values observed over forest to increase with height might be attributed to the fact that at higher altitudes the contribution of more photosynthetically active surrounding vegetation was also measured.

Cospectral analysis of the time series for fluctuations in CO_2, temperature, and vertical wind can be used to show the range of eddies primarily responsible for the observed fluxes. From the areas under the curves in Fig. 2, which are proportional to the fractional cospectra, it can be seen that at the given altitudes most of the contributions to the flux are from eddies varying in size from 50 to 1000 m. This is well above the minimum eddy size of 10 m that can be resolved by the aircraft's recording system. Peaks in the wind-CO_2 (C_{wc}) cospectra correspond to valleys in the wind-temperature (C_{wt}) cospectra, indicating the inverse directional relationship and scale similarity between the transfer of CO_2 and heat.

The results presented in this report demonstrate the feasibility of studying CO_2 exchange over extensive areas with the use of aircraft-mounted instrumentation. It is expected that future work in this area may well allow large-scale monitoring of biomass production in agriculture and forestry, leading to better yield estimates and to a deeper understanding of the role of the biosphere in the global CO_2 cycle.

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14. We thank A. D. Wood and J. I. MacPherson and other staff members of the National Aeronautical Establishment of the National Research Council, Ottawa, for their assistance in collecting the data. We also thank P. Jones of the Bedford Institute of Oceanography, Nova Scotia, for the loan of a fast-response CO_2 analyzer. This project has been funded by the Crop Information Program of the Research Branch of Agriculture Canada and by the Canadian Biomass Program directed by R. Overend of the National Research Council. Land Resource Research Institute Contribution 130; Engineering and Statistical Research Institute Contribution I-314.

28 July 1981; revised 26 October 1981

Autoradiographic Evidence for a Calcitonin Receptor on Testicular Leydig Cells

Abstract. *Previous studies have indicated that there is a relation between testicular function and adequate concentrations of zinc in testicular cells, and that calcitonin alters cellular zinc transfer in the testis. The present studies provide autoradiographic evidence that calcitonin binds in vivo to the cell membrane of testicular Leydig cells. The data thus confirm the presence of the testicular cell membrane calcitonin receptors that were previously demonstrated indirectly by Scatchard analysis of data collected from binding studies.*

The presence of a homeostatic mechanism for maintaining zinc concentrations in the serum or tissues has been suggested by many lines of evidence (*1, 2*). Recent studies in our laboratory suggested the possibility of a hormonal influence on tissue zinc homeostasis. Calcitonin, a hormone with a definite, but diffuse, role in calcium metabolism, has been demonstrated to have a specific effect on cellular zinc transfer in the testis, a tissue that undergoes atrophic change in the absence of adequate zinc nutrition (*3–5*). Since these effects of calcitonin appear to be biologically specific and significant, it should be possible to demonstrate the presence of testicular cell membrane receptors for calcitonin, with characteristics similar to those of receptors for other peptide hormones. In previous studies (*6*) we did, indeed, demonstrate the presence of such receptors, although we did not identify the cell type responsible for the binding. In this report we describe studies that confirmed those findings and enabled us to identify the cell type whose cell membrane contains the calcitonin receptor.

Male rats of the CD strain (Charles River; 120 to 140 g) were thyroidectomized and parathyroidectomized 5 to 7 days before they were used for the study. One day before the study, we labeled synthetic human calcitonin (Beckman) with ^{131}I using the bead-coupled lactoperoxidase method (Bio-Rad, Inc.) according to the supplier's recommended procedure. The free iodine was separated on a Sephadex G-50 microcolumn. On the day of study, the animals were anesthetized with pentobarbital and both the renal arteries and veins were ligated. One microcurie of either [^{131}I]calcitonin or ^{131}I alone was then injected intravenously into the inferior vena cava. Fifteen minutes after injection, a large-bore needle was placed in the left ventricle and the animal was perfused with normal saline for 2 minutes. The infusate was then changed to a solution containing 2.4 percent glutaraldehyde and 0.4 percent paraformalde-

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hyde for 5 minutes to provide fixation in situ. The testes were removed and a portion of tissue was taken for ^{131}I analysis. The remaining tissue was postfixed for 2 hours in a similar solution and then prepared for autoradiographic study. The tissues were embedded in paraffin, cut at a thickness of 10 μm, coated with NTB-2 liquid emulsion (Eastman Kodak), and exposed for 2 weeks. They were then developed and counterstained with hematoxylin and eosin.

No qualitative differences could be observed between the animals that underwent renal vessel ligation immediately prior to isotope injection and those that did not. The total activity in the testis as reflected by both grain count estimates and direct isotope analysis was higher in the animals that underwent renal vessel ligation. The testicular sections and other data presented were obtained from animals that underwent renal ligation.

When ^{131}I was injected alone it did not bind to any specific cell type. Indeed, the ^{131}I content as measured by qualitative grain count estimate and by scintillation spectrometry was less than 10 percent of that observed when ^{131}I-labeled hormone was used at comparable microcurie dosages.

As shown in Figs. 1 and 2, the grains are almost exclusively associated with cells. There are almost no grains in the intercellular spaces, tubular spaces, or associated with vascular spaces. There is a nonspecific distribution over the tubular tissue and there are cells in the interstitial area over which there is a marked increase in grains. Figure 2, A and B, shows that the Leydig cell is responsible for the specific binding of the [^{131}I]calcitonin in the interstitium. None of the other interstitial or tubular cell types demonstrate binding above the nonspecific level observed in the tubular tissue.

The data reported here indicate that specific testicular cells are responsive to calcitonin. The presence of the calcitonin receptor was demonstrated in vitro by Scatchard analysis of the binding of [^{125}I]calcitonin to membrane fractions isolated from normal rat and normal human testes. The specificity of the binding was confirmed by the demonstration of specific binding in the presence of a 100 to 1000M excess of follicle-stimulating hormone, luteinizing hormone, parathyroid hormone, and insulin. The receptor had a mean dissociation constant of $3.16 \times 10^{-8} M$. High nonspecific binding prevented meaningful measurement of the receptor concentration (*6*). The relatively high nonspecific binding of calcitonin suggested that the cell responsible for the specifically bound calcitonin makes up only a small fraction of the total testicular cell population. Figures 1 and 2 provides direct evidence that in vivo the Leydig cell membrane is responsible for the testicular calcitonin binding. This, coupled with the clinical evidence that severe zinc deficiency, especially in childhood, leads to decreased testosterone production strongly suggests that the Leydig cell may be the site of both zinc and calcitonin action.

These data also suggest that calcitonin may have other, previously unsuspected, effects on multivalent cation metabolism in tissues not previously thought of as target tissues for calcitonin action.

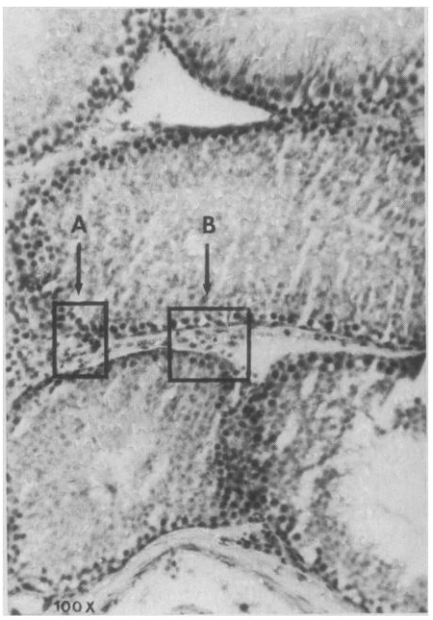
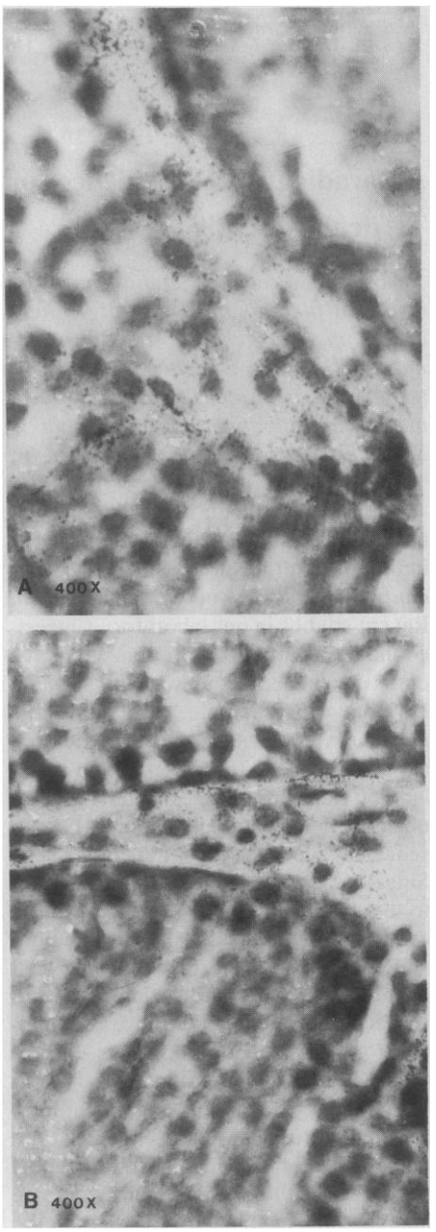


Fig. 1 (left). Low-power (×100) overview of representative section through testicular Leydig cells. Fig. 2 (right). (A and B) High-power (×400) view of areas designated as *A* and *B* in Fig. 1.



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11 February 1982