different from those of 5′ AMP at these wave-lengths[4,7], the millimolar concentrations of 5′ IMP and 5′ ADP may be obtained from the equations:

$$E_{265} = 12{\cdot}7\,x + 6{\cdot}5\,y$$
$$E_{275} = 6{\cdot}2\,x + 1{\cdot}8\,y$$

where E_{265} and E_{275} represent the absorbances of the solution (in 2 N hydrochloric acid) at 265 and 275 mμ, and where x and y represent the millimolar fractions of 5′ ADP and 5′ IMP present in the solution.

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Enzymatic Determination in Serum of Lathyritic and Prednisone-treated Lathyritic Rats

While a remarkably raised serum-level of transaminases and aldolase is found in dermatomyositis, such augmentation, as a rule, is not found in cases of severe muscular involvement of scleroderma and disseminated lupus erythematosus (acutus)[1–3].

With this in mind, we investigated whether, in an acute experimental collagenose, that is lathyrism, the enzymatic activity in serum of glutamic-oxalacetic-transaminase (GOT), glutamic-pyruvic-transaminase (GPT), aldolase (ALD) and lactic-dehydrogenase (LDH) would be changed by an uptake of β-amino-proprionitrile. Using the procedure of Holzmann *et al.*[4], eighteen albino rats on standard altromin diet were divided into three groups. Group 1 (six animals) received 40 mg β-amino-proprionitrile daily; group 2 (six animals) received 40 mg β-amino-proprionitrile plus 0·5 mg prednisone daily; group 3 (six animals) served as a control. After 17 days the serum enzymatic activity was measured, using the 'Biochemica-test' combinations (Messrs. Boehringer, Mannheim).

As can be seen from Table 1, in serum of lathyritic rats, the activity of the transaminases, but not that of ALD or LDH, is raised in comparison to normals. The findings were more significant in the case of GPT than in that of GOT, whereas in the prednisone-treated lathyritic animals the transaminase-levels were again within the normal range. The single and probably non-specific intensification of the transaminases which was detected is probably due to primary lathyrogenic effects of β-amino-proprionitrile on the mesenchyme and may result from generally increased permeability of cells[5,6]. Moreover, an elective augmentation of these enzymes by raised suppliance of substrate[5] as a consequence of enhanced collagen destruction[4,7] can also be assumed. Furthermore, such mechanisms are in agreement with the control of elevated transaminase-values by corticosteroids[8,9].

Table 1. Enzyme-levels in Rats

	Group 1	Group 2	Group 3
LDH			
Average	1,498	1,403	1,518
Standard deviation	±105	±115	±86
Statistical significance		—	
ALD			
Average	17·1	16·9	16·6
Standard deviation	±0·9	±1·0	±1·2
Statistical significance		—	
GOT			
Average	72·1	56·7	59·5
Standard deviation	±7·2	±9·1	±4·0
Statistical significance			
	Significant at the 1 per cent level		Significant at the 1 per cent level
GPT			
Average	27·5	18·0	17·6
Standard deviation	±4·1	±2·5	±1·8
Statistical significance			
	Significant at the 1 per cent level		Significant at the 1 per cent level

Enzymatic activities in mE./ml. serum. Statistical significance was examined using the *t*-test.

The finding of heightened activity of transaminases in lathyritic rats confirms the clinical belief that these enzymatic activities are mainly related to acute disintegration of collagen, for example, in dermatomyositis, and are far less closely related to chronic collagen diseases such as scleroderma.

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High Nucleo-cytoplasmic Concentration Gradient of Chloride in Rat Liver

Previously, a high nucleo-cytoplasmic concentration gradient of sodium was found in rat liver and other tissues[1,2]. Since the concentration of potassium also was higher in the nucleus than in the cytoplasm, about 500 μequiv./g dry weight of anions should exist in the nucleus if the assumption is correct that nucleic acids and basic proteins approximately neutralize each other. This communication presents data which demonstrate a high concentration of chloride in the nucleus of the rat-liver cell.

Nuclei were prepared by the non-aqueous technique[3]. Analyses for chloride were performed, after digestion with 0·1 N nitric acid, by mercurimetric titration[4] in a recording Zeiss spectrophotometer; the end-point was evaluated by graphical means. By this method 104 per cent of the chloride which was added to the tissue fractions was 'recovered'. None of the materials and solvents which were used for the isolation of nuclei raised the blank values above the limit of error. Tissue preparations which were freed from lipids by exposure to the solvents of the non-aqueous procedure did not lose or gain any chloride.

The results (Table 1) demonstrate a concentration gradient of chloride of about 10 between cytoplasm and nucleus. This gradient compares well with that of intra-

Table 1. Concentration of Chloride in Rat-liver Whole Tissue and Nuclei

Tissue fraction	Experimental conditions	RNA/DNA	Cl^- (μeq./g) Dry weight	Cl^- (μeq./g) Fresh weight	No. of analyses
Whole tissue*	1	2·9	135	41	14
Cytoplasm†	1	—	35	10	—
Nuclei*	1	0·21	204	95	18
Whole tissue	2	2·0	42	—	2
Nuclei	2	0·50	68	—	2
Whole tissue	3	2·7	270	—	2
Nuclei	3	0·41	347	—	2

Experimental conditions: (1) analysed as obtained; (2) after perfusion of the livers of 40 animals *in vivo* with 0·30 M sucrose; (3) after perfusion of the livers of 40 animals *in vivo* with 0·15 M lithium chloride.
* These data represent the average of ten different batches of tissue preparations obtained from 40–50 animals each.
† Derived data from analyses of whole tissue and nuclei, after corrections for the chloride content of blood (10 per cent of fresh weight), of extracellular fluid (15 per cent of fresh weight), and of nuclei (6·6 per cent of fresh weight)[2].

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