was resuspended. The enzyme activity was measured a) in the supernatant and b) in the resuspended sediment. In the latter case, incubation was performed in a Dubnoff-shaker with constant agitation.

Results. The enzyme activities found in the 107,000 × *g* supernatants and sediments of lung homogenates from various species are shown in the Table. Much higher activities occur in the sediment, except for rat and mouse. In accordance with our previous studies on the specificity of the assay, the enzyme activities measured could be abolished by dialysis against water and restored by addition of chloride ions.

Discussion. From recent studies[5-7] it appears that the enzyme responsible of the conversion of angiotensin I to angiotensin II is also able to attack other substrates. Thus, it can inactivate bradykinin by removal of the C-terminal dipeptide. The name 'dipeptidyl-carboxypeptidase' is therefore more appropriate than 'converting enzyme' which has been used so far. Availability of purified preparations of the enzyme would certainly help much in improving our knowledge of its properties and function. The present investigation shows that rat lungs represent a particularly rich source of the enzyme. In practice, however, organs of slaughter-house animals are often preferred as a convenient source for large-scale preparations, and in this respect a material such as horse or hog lung may prove more adequate.

A puzzling question is how circulating angiotensin I can be so easily converted to angiotensin II by a non-circulating pulmonary enzyme. This probably implies that the enzyme is located in the very vicinity of the blood flowing through the capillaries. Our studies show that in dog, rabbit, hog, horse, sheep, ox, guinea-pig and man, dipeptidyl carboxypeptidase is not a cytoplasmic enzyme, since it is found in a sedimentable fraction. They confirm the findings of BACKLE[2] and of YANG et al.[7], who found most of the activity of lung to sediment in the microsomal fraction.

We found the enzyme to occur in all 10 mammalian species investigated. The amounts present in each particular case are certainly of importance for the kinetics of angiotensin activation and bradykinin breakdown[8].

Activity of dipeptidyl-carboxypeptidase in lungs from different species

Species	Enzyme activity (mU/g tissue)		
	Supernatant	Pellet	Total
Rat	98.2	46.4	144.6
Rabbit	3.2	106.0	109.2
Mouse	60.4	47.6	108.0
Horse	18.1	47.8	65.9
Dog	9.6	52.2	61.8
Hog	13.0	48.8	61.8
Sheep	2.2	58.8	61.0
Beef	1.6	29.8	31.4
Guinea-pig	3.6	26.4	30.0
Calf	1.2	16.0	17.2
Human	3.6	12.2	15.8

Résumé. On a dosé la dipeptidyl-carboxypeptidase (responsable de la conversion de l'angiotensine I en angiotensine II) dans des préparations de poumons de 10 espèces différentes. Les espèces se classent comme suit par ordre décroissant de teneur en enzyme: rat > lapin, souris > cheval, chien, porc, mouton > bœuf, cobaye > veau, homme. Chez la plupart des espèces, l'enzyme est fixé a une structure subcellulaire sédimentable.

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Effect of Acriflavine on the Radiation Resistance Enhancement Induced by Cysteine Treatment in *Escherichia coli* B/r

Cysteine is a good radioprotector. It has been established that there are at least two kinds of mechanism by which it enhances the radiation resistance of *Escherichia coli*[1]. The physico-chemical mechanisms represent radical scavinging, hydrogen donation, etc[2]. The metabolic or biochemical mechanisms mean that cysteine alters the cell metabolism in several ways and some of these metabolic alterations result in enhanced survival after ionizing radiation[1]. The same is true also for cysteamine[3].

The mechanism of the biochemical radioprotective effect of cysteine has been studied in our laboratory using the following method: *E. coli* cells are treated with cysteine for certain periods, thereafter cysteine is removed (by centrifugation and washing) and the bacteria are irradiated in cysteine-free buffer. Analyzing the changes in radiosensitivity and, at the same time, in cell metabolism induced by cysteine treatment, allowed us to propose a model to explain the mechanism of the metabolic radioprotective effect of cysteine[4]. According to this model, the sharp increase in radio-resistance, obtained by 1 min cysteine treatment, can be explained by the sharp increase in the acid-soluble SH level of bacteria. The further increase in radio-resistance, up to the maximum obtained by 30 min cysteine treatment, can be correlated with the asynchronous synthesis of macromolecula

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incuced by cysteine, i.e. after the addition of cysteine, the net synthesis of RNA and protein stopped immediately, whereas a certain quantity of DNA was synthesized. Theoretically, the alterations of cell metabolism before irradiation may enhance the radiation resistance in either of the following two ways: 1. by decreasing the intrinsic sensitivity of cells to damaging effects of irradiation, or 2. by increasing the recovery after irradiation.

The results presented here show that both of the abovementioned possibilities take part in the enhancement of radiation resistance induced by cysteine treatment in *E. coli* B/r.

E. coli B/r was grown on mineral salts-glucose medium[5]. The cells in mid-log phase of growth were treated with cysteine (2 m*M*) and incubated further at 37 °C. After 1 min or 30 min, the cells were centrifuged, washed 3 times, resuspended in iced phosphate buffer (pH 7.0) and irradiated with X-ray. Surviving fractions were determined by plating bacteria on nutrient agar[6], and on the same agar, containing acriflavine (2 μg/ml), a potent inhibitor of DNA repair[7] (Figure). The 1 min cysteine treatment raised the D_{99} (1% survivals) from 20.5 to 30.6 krad and from 9.3 to 13.5 krad for cells plated on nutrient agar and acriflavine containing agar, respectively (the dose response factors of 1.49 and 1.45). Thus, the radiation resistance induced by 1 min cysteine treatment was not affected by acriflavine, i.e. the intrinsic sensitivity of bacteria was decreased by this treatment. The 30 min cysteine treatment, however, resulted in greater resistance enhancement when the cells were plated on nutrient agar than they were plated on the same agar, containing acriflavine, the dose response factors being 2.1 and 1.7, respectively. If our model is a correct interpretation of the mechanism of biochemical radioprotective effect of cysteine, the results presented here suggest that the asynchronous synthesis of macromolecula induced by cysteine treatment enhances the recovery after ionizing irradiation. This conclusion is in agreement with some recent findings[8,9] that the functional *exr*A gene is required for radiation resistance enhancement induced by the abovementioned type of asynchronous synthesis of macromolecula.

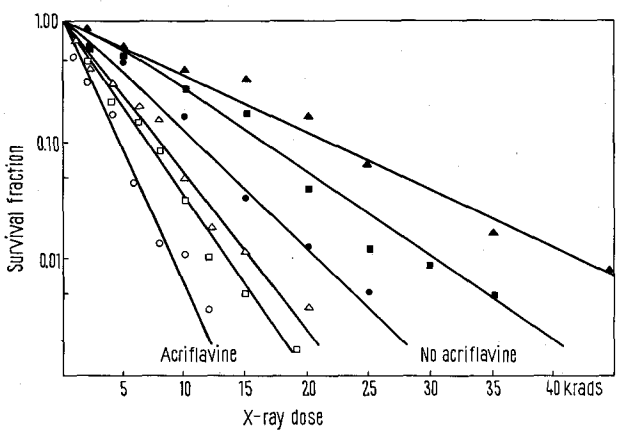

Fig. 1. Effect of acriflavine on the radiation resistance enhancement induced by cyteine treatment in *E. coli* B/r. Surviving bacterial cells were counted by making dilutions in phosphate buffer and plating 0.1 ml samples in triplicate on nutrient agar (closed symbols) or on the same agar containg 2 μg/ml acriflavine (open symbols). The plates were incubated at 37°C before counting. ○ and ●, non-treated cells; □ and ■, cells treated with cysteine for 1 min; △ and ▲, cells treated with cysteine for 30 min.

Zusammenfassung. Zur Prüfung der Strahlenresistenz von *Escherichia coli* nach bewährter Methode wird Acriflavin, ein DNA-Reparaturhemmer, zugegeben und es wird festgestellt, dass die Strahlenresistenz auch unter Cysteinschutz sinkt.

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Changes in the Degree of Orientation of Bone Materials with Age in the Human Femur

In recent years bone has increasingly been considered as a two phase composite of apatite and collagen. Of the two materials apatite has the higher elastic modulii. In a composite of this type the degree of preferred orientation of the high elastic modulii material plays a very important part in determining the physical properties of the composite[1]. A literature survey showed no systematic study of change in the degree of preferred orientation of apatite crystals with age, although the preferred orientation of the apatite crystallites in mature bone and their relationship with collagen fibres had been studied as early as 1936[2]. It was therefore decided to study this aspect of bone.

Material and method. X-ray diffraction diagrams of 20 specimens of femoral bone from 6 day to 76-year-old individuals have been studied. Of these, 6 were below 5 years of age and the rest adults of different ages. X-ray diagrams were taken for both posterior and anterior quadrants of the midshaft of the femoral diaphysis. Specimens for X-ray diagrams were obtained by cleaving wedge-shaped pieces from the middle of the quadrant.

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