from bronchopneumonic changes in the lungs of the 2 cows which developed post-operative pulmonary infection and mild chronic inflammation at the site of implantation of the 3,4-benzpyrene pellets in the kidney, no change of any significance could be detected in any animal.

Subcutaneous implantation of these 3,4-benzpyrene pellets (5 mg) or injections of olive oil suspension (10 mg) of MCA from the same bottle produced sarcomas in nearly 100% Wistar rats within a period of 9 months of exposure to the carcinogens. Our findings, of course, do not demonstrate that cattle are resistant to MCA or 3,4-benzpyrene: observations for longer periods in larger groups of animals or administration of the carcinogens by other routes, and in different doses and schedules may reveal results different from those reported here[4]. Attempts to induce neoplasms in infrahuman primates have also been frequently unsuccessful[5,6].

Zusammenfassung. Fünf jungen Kälbern wurden chemische Karzinogene (20-Methylcholanthren und 3,4-Benzpyren) subkutan oder mittels implantierter Gelatinekapseln verabreicht. Die Tiere wurden 6–36 Monate lang beobachtet. In keinem der Tiere konnten signifikante Veränderungen festgestellt werden.

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Reaction of Pulmonary Macrophages to the Presence of Foreign Protein Material in the Alveoli during Metabolic Acidosis

The reticuloendothelial system in the alveolar walls is represented by a system of alveolar cells placed in the interstitial argentaffin network of the interalveolar septa. These cells enter, on various impulses, into the alveolar spaces and become free alveolar cells with the ability of phagocytosis, i.e. macrophages (BERTALANFFY[1,2]). This delivering process takes place not only after the penetrating of foreign particles into the alveoli but also under other circumstances, e.g. as described by JANSSEN[3] during protracted suffocation. Besides free alveolar cells one sometimes finds giant cells with sudanophilic cytoplasmic inclusions (in otherwise healthy individuals). The longer the interval between the beginning of suffocation and the instant of death, the more often the multinuclear giant cells with sudanophilic cytoplasmic inclusions are found.

JANSSEN[3] believes that the impulse evoking the delivering of alveolar cells and their transformation into giant cells is, in this case, lack of oxygen. This explanation contradicts VALDIVIA's[4] opinion. VALDIVIA[4] kept the experimental animals in a hypobaric chamber with the partial pressure of oxygen decreased to 50% for several weeks; in the histological preparations of the lungs of the killed animals he could not see any free alveolar cells.

In addition to oxygen starvation a number of other changes take place during suffocation: CO_2 accumulation, formation of acid metabolits of anaerobic metabolism and consequently, to a decrease of pH. Even if acidosis itself, as we have demonstrated in another report (MRÁZ et al.[5]) does not produce the delivering of alveolar macrophages, the question is whether it would not effect, in some way, the ability of the alveolar cells to free themselves on different impulses from the tissue unity.

We have studied the influence of acidosis on the delivering of alveolar cells. As the impulse evoking the delivering of the alveolar cells we have used the application of foreign material into the alveoli, in our case denaturated calf plasma (MĚLKA et al.[6]) dissolved in saline.

Material and methods. Rats of Wistar strain weighing 215–265 g were used; all the animals were anaesthetized by i.p. injection of 5% uretane solution, 1.5 ml/100 g body wt. The experimental rats were divided into 3 groups; in the first group complete metabolic acidosis was evoked, the second group was only operated on, and the third group served as controls. Acidosis was evoked by slow infusion of 1 *N* HCl by means of a catheter which was introduced into the vena renalis sinistra. The rate of pH significantly decreased on the average by 0.36 (i.e. in the midst to 7.04). Before the beginning of the infusion, the hili of both kidneys were ligated. The blood for measuring pH was taken from the catheter introduced into the arteria carotis communis sinistra. The pH was measured by means of a microelectrode (by Radiometer, Copenhagen). About 3 h after the beginning of the narcosis (i.e. the time necessary for evoking acidosis in the animals of the first group), the animals were killed by means of an i.v. injection of Pentothal in lethal dose. The lungs of each animal killed were collapsed after cutting the diaphragm. The trachea was opened and a thick transfusion needle introduced into it. 10 ml of denaturated calf plasma dissolved in saline was slowly injected into the lungs through the trachea; thus the lungs were dilated in such a way that they just filled the thorax. The solution was kept in the lungs first for 1 min and then lightly

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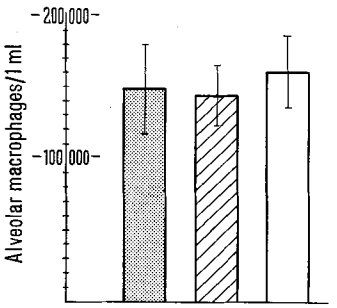
aspirated back into the syringe. The second filling was aspirated back from the lungs after a 10 min interval. After mixing both reaspirated fluids we counted the alveolar macrophages (MAXWELL et al.[7], MYRVIK et al.[8]).

Results. By lavation of the lungs of the first group in which full metabolic acidosis had been evoked (pH 7.04), we obtained on an average 149,813 ± 31,426 alveolar cells/1 ml. From the lungs of the second group, where only the control operation was performed, i.e. ligation of both kidneys, catheterization of the arteria carotis commun. sin. and of the vena renalis sin. (pH 7.40), we obtained on an average 144,500 ± 21,451 of alveolar cells per 1 ml. By lavation of the lungs of the totally healthy animals (pH not measured) we gained on the average 161,500 ± 25,464 alveolar cells/1 ml. As it can be seen from the Figure, there is no statistically significant difference between the amount of delivered alveolar cells gained by lavation from the lungs of animals in the 3 particular experimental groups.



The number of alveolar macrophages obtained by lavation of rat lungs. ■, acidosis; ////, control operation; □, control.

Conclusion. The experiments showed that the entire metabolic acidosis evoked by the infusion of 1 *N* HCl, which produces the decrease of pH on the average to 7.04, could neither increase nor decrease the amount of the cells delivered into alveolar spaces owing to the influence of denatured calf plasma dissolved in the saline. So we can say that metabolic acidosis itself does not influence the ability of the alveolar cells to enter into the alveolar spaces.

Zusammenfassung. Tierexperimentell wurde festgestellt, dass die metabolische Azidose keinen Einfluss auf die Fähigkeit der Alveolarzellen hat, in die Alveolen auszuwandern.

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Radioprotective Effect of Phytohemagglutinin in Mice

Phytohemagglutinin (PHA) has been found to induce blastoid transformations, mitoses[1], and radioresistance[2] in human blood lymphocytes in vitro. Similar morphologic transformations have also been observed in suspensions of blood and tissue lymphocytes obtained from laboratory animals[3]. MEKORI et al.[4] hold the view that phytohemagglutinin (PHA) acts in vivo in a manner similar to its established action in vitro, i.e., by stimulating the change of lymphocytes into dividing blast cells which can initiate lymphoid colonies in the spleen of an irradiated host. Moreover, HUMBLE[5] observed regeneration of bone marrow function in 6 patients with aplastic anemia, treated with repetitive i.v. injections of PHA. HUMBLE holds the view that the small lymphocytes of the blood normally enter the bone marrow and become transformed into precursor cells of the red and white series, as Maximow claimed. Based on these observations, a series of experiments was designed to test whether PHA had any protective effect on mice exposed to dosages of irradiation which normally cause death from bone marrow failure.

Methods. Twelve-week-old CF_1 mice (Carworth, Inc.) weighing 20–25 g were used in these experiments. Vials containing 100 mg of phytohemagglutinin-P (Difco) were diluted with 33.3 ml of isotonic saline. This resulted in a final PHA-P concentration of 3000 μg/ml. For the serial dosage experiments, dilutions were made from this preparation. Two groups of control and PHA-treated animals received i.p. 1 ml of saline or diluted PHA, respectively. Two other groups with the same number of animals in each, treated with either saline or PHA alone, were retained as non-irradiated controls. Whole body irradiation was performed by a 300 KeV, 20 ma X-ray machine with target-mouse distance of 50 cm, HVL 2 mm Cu and 150 R per min dose rate.

Results. Table I presents the percent survival of the mice exposed to 600 R whole-body irradiation as a function of PHA dose. The drug was injected 30 min before irradiation. The degree of radioprotection appears to be a linear function of the PHA dose used. At the highest dose of 3000 μg or approximately 120 mg/kg, 53.3% versus 16.7% survival was observed in favor of the experimental group. This dose was used in the additional experiments. Subsequently tested was the radioprotective effect of PHA as a function of time between drug injection and irradiation (600 R). Table II shows that the protection

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