tubules décrits par LEDBETTER et PORTER[7] et récemment observés dans la glande mammaire normale (SANDBORN et al.[8]). De telles formations ont déjà été signalées dans les lymphomes de souris en relation avec les particules de type A (DALTON et al.[9], KAKEFUDA et al.[10]). La présence de structures filamenteuses dans les lumières acineuses est en tous cas un fait très particulier.

Le dernier point intéressant à souligner dans les tumeurs mammaires examinées ici est la coexistence assez remarquable de virus et de phénomènes sécrétoires. Dans la cellule tumorale l'élaboration de grains de protéine s'effectue d'une manière analogue à celle qui se voit dans une glande mammaire lactante (HOLLMANN[11]). Cela suggère manifestement l'intervention d'un facteur hormonal dont le rôle dans la génèse des tumeurs mammaires de la souche PS n'est certainement pas négligeable[12].

Summary. In the recently isolated Mouriquand strain of mice, virus-like particles of the A and B type are abundant in mammary tumours, and fewer particles of the C type are present in leukaemic and non-leukaemic organs. In one animal all three kinds of particles have been shown. Striking cytological features of the mammary tumours are described.

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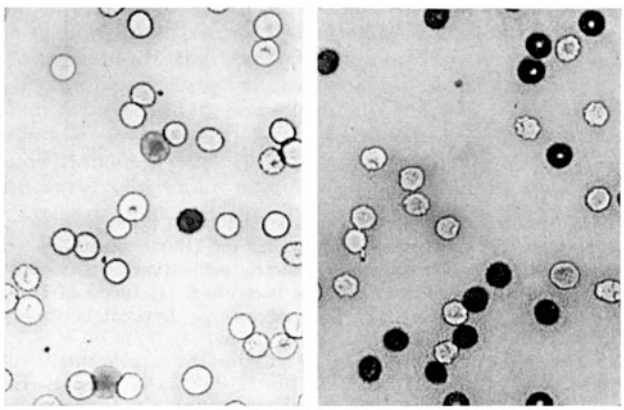
Cellular Distribution of Catalase Activity in Red Cells of Homozygous and Heterozygous Cases of Acatalasia

Attempts have been made to determine whether Lyon's concept of gene inactivation, which is well established for X-chromosomes, could be extended to autosomes[1]. Acatalas(aem)ia, an enzyme defect of autosomal recessive inheritance, seemed to be suitable for analysis in this respect: heterozygous individuals in Acatalasia families exert an intermediate catalase activity in red cells and other tissues, varying between 15-85% of the normal level[2]. This may be either due to the presence of two cell populations, one of them being virtually free of catalase, or to one single cell population of intermediate enzyme activity. In order to decide between these possibilities, two types of experiments have been performed. Both of them make use of the fact that – under suitable experimental conditions – sensitivity of red cells towards H_2O_2 (or X-rays) largely depends on their catalase content[3,4].

First, a simple technique for the distinction of normal and acatalatic red cells in blood smears has been devised. A suspension (1.5 mg Hb/ml) of washed red cells is incubated in an isotonic, buffered medium containing glucose (5 mg/ml) and glucose oxidase (30 μg Deoxin 'Nagase'/ml) at 25°C for 30 min. Under these conditions, i.e. at an H_2O_2-generation rate of $3.0 \cdot 10^{-9}$ Moles H_2O_2 min^{-1} ml^{-1} normal human red cells remain unaltered, whereas in acatalatic cells hemoglobin is completely oxidized to methemoglobin by H_2O_2[5]. After centrifugation of the suspension, the cells are washed, smeared and stained as described by KLEIHAUER and BETKE[6]. This differential staining technique is based on the fact that oxyhemoglobin but not cyanmethemoglobin is active as a peroxidase. In smears obtained by this procedure, erythrocytes of approximately normal catalase activity (normal human blood) are stained red; erythrocytes virtually free of catalase (acatalatic human blood) are eluted and appear as ghosts (Figure 1a). Erythrocytes from heterozygotes essentially show the same result as normal human red cells.

As a control, experiments with a mixture consisting of equal parts of acatalatic and normal blood (imitating



a b

Fig. 1. a, Red cells of a homozygous case of acatalasia (M.V.). About 99% of all cells are eluted, $^1/_2$–1% are not. b, 'Artificial' mixture of normal and acatalatic red cells in equal amounts (normal cells = not eluted; acatalatic cells = eluted). The smears have been deliberately made relatively thin.

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mosaicism) have been made. By this technique, any heterogeneity in respect to catalase activity can be detected, as shown in Figure 1b. Evidently this is not the case in blood of heterozygotes. However, it has been observed that in blood smears from homozygous acatalatics one out of 100–150 red cells (i.e. 0.6–1.0%) behaves as if it contained approximately the same amount of catalase as a normal red cell (Figure 1a). The percentage of apparently normal red cells detectable in blood smears of acatalatic individuals of the families V., B. or G.[7] roughly corresponds to the level of residual catalase activity as measured by Feinstein's perborate method (0.1–1.3% of normal level). This figure, however, also falls within the range of reticulocyte counts of the samples analysed (0.3–1.0%). Therefore it cannot be decided whether residual catalase activity in blood of homozygotes is due to its presence in reticulocytes or to a small fraction of apparently normal red cells.

Additional evidence for even distribution of catalase in blood of heterozygotes has been obtained by chemical measurements of methemoglobin formation at varied rates of H_2O_2 generation. In suspensions of acatalatic red cells, H_2O_2 is used almost quantitatively for hemoglobin oxidation; but if catalase is present in the system, only a small fraction of hemoglobin is oxidized. Therefore the amount of methemoglobin formed at a given H_2O_2-production rate is a function of catalase activity[4,5]. By plotting methemoglobin formation against the rate of H_2O_2-generation, curves of different slope and shape are obtained (Figure 2). The difference between curve 2 and 3 is in agreement with the finding mentioned above that in blood of heterozygotes no evidence could be obtained for the existence of two cell populations differing in catalase activity.

The observations presented in Figure 1 and 2 suggest that in blood of heterozygotes (= 'Hypocatalasemia') all red cells exert approximately the same level of catalase activity. The homogeneous distribution of catalase in erythrocytes of heterozygous individuals from all three Swiss acatalasia families is in agreement with the concept that mosaicism in heterozygotes has been found so far only in enzyme defects, which are inherited by the X-chromosomes[1,9,10].

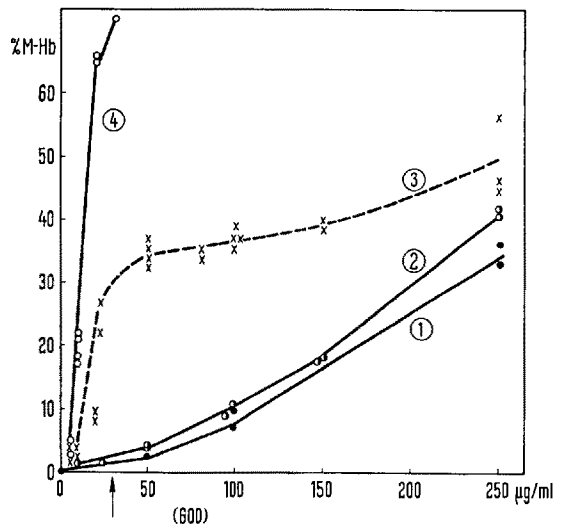

Fig. 2. Methemoglobin formation as a function of the rate of H_2O_2 production. Cell suspensions (approx. 1.5 mg Hb/ml) incubated for 30 min at 25°C. For spectrophotometric estimation of methemoglobin the method of FLEISCH[8] has been used. (1) Blood of H.A. (normal), (2) blood of P.V. (heterozygote = hypocatalasaemia; mother of M.V.), (3) 1:1 = mixture of (1) and (4), (4) blood of M.V. (homozygous acatalasia). The arrow indicates the experimental conditions used in the standard procedure (i. e. 30 µg/ml Glucose-oxidase = GOD).

Zusammenfassung. Es wird ein Verfahren beschrieben, welches im Blutausstrich katalasehaltige und katalasefreie Erythrocyten zu unterscheiden erlaubt. Dieses beruht auf der sehr ungleichen Methämoglobinbildung bei Inkubierung der Zellen mit Glucose und Glucoseoxidase. Es wird nachgewiesen, dass im Blut homozygoter Defektträger 0,5–1% aller Erythrocyten einen anscheinend normalen Katalasegehalt aufweisen. Im Blut heterozygoter Defektträger konnten keine Anhaltspunkte für das Bestehen zweier Zellpopulationen von verschiedener Kataloseaktivität gefunden werden.

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Medizinisch-chemisches Institut der Universität Bern (Switzerland), September 17, 1965.

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Renal Function Following Complete Ligation of the Renal Artery

It is well known that after complete ligation of the renal artery some blood still leaves the kidney through the renal vein (HARTWICH[1]; WOLF and HEINSEN[2] etc.). Anatomists maintain that collateral vessels reach the kidney through various branches of the suprarenal and ureteral arteries and the arcus exorenalis, respectively (CORNING[3]). The patchy character of the necrosis after complete ligation supports the functional significance of these collaterals (SHEEHAN and DAVIS[4] etc.). There are, however, no data concerning renal function after occlusion. We endeav-

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