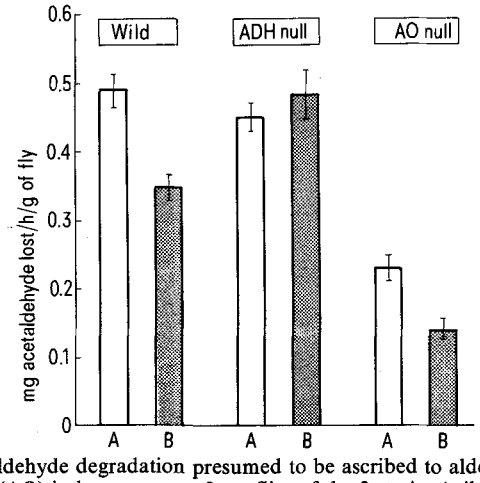
0.05 M Tris buffer (pH 7.5). The values thus obtained were used as control.

After incubation, the aliquots received 10 µl of 40 mM isopropanol, which is used as a reference standard in the gas-liquid chromatography test, as its retention time is slightly superior to the retention time of acetaldehyde or ethanol. 1 µl of this mixture was injected into the gas-liquid chromatography column.

Quantitative assessment of both acetaldehyde and ethanol in the incubation medium, following the 1 h incubation, was achieved by use of a Perkin-Elmer gas-liquid chromatograph.

In the absence of pyrazole, which is a specific inhibitor of ADH activity[11], the acetaldehyde concentration decrease observed can be partially ascribed to a reverse reaction restoring some ethanol back from the initially present acetaldehyde. Such a 'return to ethanol' is lacking in homogenates from the 'ADH null' strain flies, which do not have any active ADH enzyme. For these 'ADH null' flies, addition of pyrazole changes nothing. But the results are quite different for the 'AO null' and the wild flies (fig.). For the wild ones, pyrazole suppresses any restoration of ethanol; the acetaldehyde concentration loss observed in these conditions seems to be attributable almost exclusively to AO. A slight but significant restoration of ethanol subsists in the case of the 'AO null' flies (could it be due to some enzyme other than ADH?). Even if one takes into account this slight 'return to ethanol', an important degradation of acetaldehyde is evident.

These last results are in good agreement with David's and our own previous observations in vivo. For David the strains which do not produce an active AO enzyme show about the same tolerance to alcohol as do wild strains[15]. In our experiments the 'AO null' flies are relatively tolerant of ethanol (although to a lower degree than our wild ones) and they can even tolerate some acetaldehyde in their environment, whereas the 'ADH null' flies are much less tolerant of both ethanol and acetaldehyde[11]. In our present state of knowledge it is, however, impossible to conclude that this acetaldehyde degradation by the 'AO null' flies is, without any doubt, due to the action of an AO produced by some gene other than *Aldox;* the action of some other enzyme cannot be excluded.




Acetaldehyde degradation presumed to be ascribed to aldehydeoxidase (AO) in homogenates from flies of the 3 strains 'wild', 'ADH null', 'AO null'. It is the difference between the total acetaldehyde concentration loss observed and the part of this concentration diminution which is probably due to the retransformation of acetaldehyde into ethanol, as estimated from the ethanol concentration increase observed at the same time. This reversed reaction seems to be catalyzed by alcoholdehydrogenase (ADH) which is known to be responsible for the dehydrogenation of ethanol into acetaldehyde; indeed, it is almost completely suppressed by pyrazole, which is a specific inhibitor of ADH (however, not in homogenates from the 'AO null' strain flies). The mean values are given with confidence intervals (95%). A. Incubation with pyrazole. B. Incubation without any inhibitor.

1 This work was supported in part by a grant from the FNRS of Belgium.

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## Asymmetric distribution of male and female foetuses in the pregnant rabbit uterus

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*Summary.* The sex distribution of foetuses in the uterine horn of the pregnant rabbit was found to be asymmetrical, with more males being present in the left uterine horn and more females in the right ($p < 0.05$).

It has been suggested that in general more males than females are born in the rabbit[1-5], but no statistical data were presented. Brambell[6] recorded 506 males to 534 females from 226 litters of wild rabbits. In a study of the biological effects of active immunization of female rabbits against testosterone, it was observed that when pregnancy ensued more male foetuses were present in the left uterine horn than in the right[7]. It was therefore of interest to

Number of foetuses in the rabbit uterus (mean ± SEM)

| | Total foetuses | | Percent males | | Combined | |
|---|---|---|---|---|---|---|
| | Left uterine horn | Right uterine horn | Left uterine horn | Right uterine horn | Males | Females |
| Number | 4.11 ± 0.26 | 3.87 ± 0.28 | 56.5 ± 4.6 | 42.4 ± 4.5 | 157 | 154 |
| Student's 't' statistical value | 0.61 | | 2.22 | | | |
| Degrees of freedom | 37 | | 37 | | | |
| Level of significance | N.S. | | < 0.05 | | N.S. | |

determine whether a similar asymmetry occurred during normal pregnancy.

*Materials and methods.* New Zealand white rabbits were housed individually with food and water available ad libitum. 38 does were mated between November and July using 2–4 fertile bucks each time. Pregnant does were sacrificed at days 22–31 of pregnancy and the foetuses removed from each uterine horn. The sex of each foetus was determined by microscopic examination of haematoxylin-eosin stained sections of one gonad, and the testosterone content of the other. Data were analyzed by the Student's paired t-test.

*Results and discussion.* The number of males was expressed as a percentage of the total number of foetuses in each uterine horn. A larger percentage of males was present in the left horn ($p < 0.05$, table). Similarly more females were present in the right horn ($p < 0.05$). There was no significant difference in the number of foetuses present in each horn or in the total number of male and female foetuses. These data confirm that there is no sex difference in the number of offspring produced by the domestic rabbit (157 males:154 females) and the normal 1:1 sex ratio is present. Sex selection or controlling the sex ratio could have a number of benefits especially in animal husbandry where one sex may be more desirable.

As far as we are aware there has not been a study of the sex distribution of foetuses in the uterus of animals with large litter size. Buchanan[8] has observed that there is an asymmetrical distribution of foetuses in the rat uterus but the sex of the foetuses was not recorded. Slob et al.[9] have tried to increase the probability of litters of one sex by hemihysterectomy of rats prior to mating. They found 11 out of 30 pregnancies with males greater than females and 14 out of 30 with females greater than males. Overall, the ratio was not different from 1:1.

The possibility of unequal loss of embryos from one sex can be ruled out since the overall sex ratio was 1:1, the average litter size (7.98) was similar to that (7.39) reported by Hagen[10], and the average number of foetuses per horn was approximately the same.

The significant difference in the sex ratio between the uterine horns of the rabbit suggests that hemihysterectomy may be a method for regulating the sex of rabbits. However, the number of rabbits required to achieve this difference may preclude this procedure being a feasible one.


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## Mutagenic effects of sulfur dioxide on *Saccharomyces cerevisiae* diploid strains


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*Summary.* In resting cells of diploid *Saccharomyces cerevisiae* strains sulfur dioxide induces at very high frequency: a) respiratory deficient mutants; b) mutants with altered methionine metabolism. In growing cells the following kinds of mutants appear: a) revertants for respiration; b) mutants altered in the methionine metabolism; c) $SO_2$-resistants. It is suggested that sulfur dioxide acts as a selective agent through the induction of $SO_2$-resistant mutants.


Sulfur dioxide is commonly used in wine-making; this compound selectively inhibits the growth of acetic and lactic bacteria, allowing desirable yeast strains (*S. cerevisiae* and related species) to dominate the fermentation[2]. These strains are generally different from the wild-type for methionine biosynthesis and particularly in sulfate reduction. Studies on the mutagenic effects of sulfur-dioxide have been reported for phage[3,4] bacteria[5] and yeast[6,7].

The primary biochemical lesion of sulfur-dioxide is reported to involve deamination of cytosine to uracil[8,9], transamination[10], free radical damage to DNA[11] or an indirect effect on RNA[12] and on protein metabolism. (For an extensive review see Shapiro[13]). Recently, the effects of sulfite on resting cells of *S. cerevisiae* were investigated by Schimz[7]. The author has studied the influence of various factors, including the extracellular pH, and has determined that