The olfactory epithelium was superfused with either Ringer's solution or Ringer's solution containing 5 mg of concanavalin A/ml, as described in the text. The results are expressed as the response parameter R.

Concanavalin alone, when applied in Ringer's solution, did not affect the response to the standard odorant isopentyl acetate. However, we have found previously that concanavalin A reduces the R value of the EOGs to isovaleric acid (Polak, E., Shirley, S., & Dodd, G. H. (1982) in Abstracts 5th ECRO Congress, Regensburg, p. 59) and we have investigated the selectivity of this effect using a large number of odorants. The results in Table 1 show that potent reduction of the EOGs, as defined by R < 0.4, is found only with aliphatic carboxylic acids. The R value for the acids is correlated with the structure and is suggestive of a recognition site for isovaleric acid.

**Table 1. The effect of concanavalin A on the EOG responses from frog olfactory mucosa**

<table>
<thead>
<tr>
<th>Odorant type</th>
<th>n</th>
<th>R</th>
<th>n with R &lt; 0.4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acids</td>
<td>16</td>
<td>0.35–0.81</td>
<td>5</td>
</tr>
<tr>
<td>Aldehydes</td>
<td>9</td>
<td>0.60–0.96</td>
<td>0</td>
</tr>
<tr>
<td>Alcohols</td>
<td>13</td>
<td>0.71–1.02</td>
<td>0</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>17</td>
<td>0.73–1.02</td>
<td>0</td>
</tr>
<tr>
<td>Thiols</td>
<td>2</td>
<td>—</td>
<td>0</td>
</tr>
</tbody>
</table>

* Abbreviations: EOG, electro-olfactogram.

1983). This selective inhibition by concanavalin A has been studied only in the rat. Since volatile fatty acids are effective odorants for all vertebrates so far tested we would expect that concanavalin A would bring about a pattern of selective odorant inhibition in other vertebrates similar to that found in the rat. We report here the effects of concanavalin A on frog olfactory mucosa.

The animal preparation and the odour delivery system have been described previously (Menevse et al., 1978). The experimental protocol involved perfusing the olfactory cavity with a solution of concanavalin A in frog Ringer's solution. After a period of washing with Ringer's solution the response of the olfactory mucosa was measured by determining the EOG* to a sequence of odorants. Every fourth odor pulse was the standard odorant isopentyl acetate and the results are expressed as R, the survival of the mean EOG amplitude normalized with respect to the EOG amplitude for the standard odorant. The stability of the preparations was excellent, with little change in the EOG amplitude to isopentyl acetate during the course of an experiment.

Concanavalin A gives a differential inhibition of odor responses in frog olfactory mucosa (Table 1). The concentration required for a strong effect, 5 mg/ml, was about an order of magnitude greater than that required in the rat. The mucus layer overlying the olfactory receptors in the frog is much thicker than the mucus layer in the rat and the high concentration of concanavalin A required for the frog may reflect considerable adsorption of the protein to the mucus. The EOG responses to the small, sweaty-smelling carboxylic acids were preferentially inhibited. No significant inhibition of the EOG signals to most of the non-carboxylic acid odorants tested was found. The exception was the t-butane thiol. Interestingly, in the rat the EOG signals to two thiols was also inhibited to some extent by concanavalin A.

The pattern of selective inhibition of EOG to fatty acids brought about by concanavalin A has now been found for two vertebrates and may be interpreted as evidence for a specific receptor system for sweaty smelling fatty acids.
Deposition of elemental selenium in various parts of organs from rats given toxic doses of sodium biselenite

REGINA SCHOENTAL
Department of Pathology, Royal Veterinary College, University of London, Royal College Street, London NW1 0TU, U.K.

and SHEILA VAN DORST
Department of Biological Sciences, Chelsea College, University of London, Hortensia Road, London SW10 0QX, U.K.

Selenium, an essential micronutrient, is toxic at higher concentrations. Yet certain plants and micro-organisms are remarkably resistant to selenium (Rosenfeld & Beath, 1964). When the resistant micro-organisms (such as yeasts or Escherichia coli) are grown in media containing sodium selenite they reduce this salt to the biologically inert elemental selenium, recognizable by its characteristic brick-red colour (Falcone & Nickerson, 1963).

Particles of the elemental selenium could be seen at the cell walls of E. coli by electron microscopy (Gerrard et al., 1974).

Animals can also become resistant to selenium. It has been reported that, during the travels of Marco Polo in Central Asia, the beasts of burden which accompanied him became ill and lost their hooves, but those bred locally did not show ill effects from eating plants rich in selenium (quoted by Rosenfeld & Beath, 1964). In rats Cameron (1947) induced experimentally resistance to selenium. The rats were given subcutaneous injections of sodium selenite twice weekly, starting with doses corresponding to 1.6 mg of selenium/kg body wt. (less than half of the LD50, 3.5 mg/kg body wt., when given as a single subcutaneous dose), then increasing the doses over 140 days till they reached 12 mg of selenium/kg body wt., more than 3-fold its LD50. The mechanism of this induced resistance was not explained (Cameron, 1947).

We obtained evidence that mammalian tissues can also reduce selenium salts to its elemental form. White rats were treated with aqueous solutions of sodium biselenite, some by skin applications, and others in drinking water. At various times some of the rats were given increasing doses of it intragastrically until the animals died.

At autopsy, the stomach and certain other organs of some of the rats showed striking brick-red areas or streaks. Such red parts contained much higher concentrations of selenium (measured by the method of Hall & Gupta, 1969) than the adjacent, not specifically coloured, parts of the organs (Table I). The selenium content of the red part of one liver was about 7 times that in other parts of the same liver. To our knowledge, in mammalian tissues deposition of elemental selenium has previously not been recorded (see, e.g., Spallholz et al., 1981).

The mechanism of the reduction of sodium biselenite and the deposition of elemental selenium is not known in detail. On interaction with glutathione (or with other thiol compounds) selenium sulphides are formed, which break down according to the scheme:

\[ 4 \text{R-SH + SeO}_{3}^{2-} \rightarrow 2 \text{RS-Se-SR} + 2 \text{H}_{2} \text{O} \downarrow \]
\[ \text{RS-Se-SR} + \text{Se} \]

Of particular interest is the preferential deposition of elemental selenium in specific parts of rat organs, and its possible implications to the contradictory results encountered in experimental and epidemiological studies into the role of selenium in relation to cancer (Schoental, 1983).

Table I. Selenium content in various parts of tissues from white rats treated with sodium biselenite

<table>
<thead>
<tr>
<th>Rat no.</th>
<th>Sex</th>
<th>Tissue</th>
<th>Se-coloured (μg/g)</th>
<th>Naturally coloured (μg/g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>Stomach</td>
<td>105.65</td>
<td>65.31</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>Liver</td>
<td>147.98</td>
<td>20.48</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>Stomach</td>
<td>69.98</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>F</td>
<td>Skin</td>
<td>11.36</td>
<td></td>
</tr>
</tbody>
</table>


This work was supported, in part, by the Medical Research Council.