Drug resistant mutants

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Abstract
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eliminated when sulfur compounds required for growth are added to the minimal medium. The method therefore seems useful for obtaining a large class of sulfur-requiring auxotrophs with partial losses of enzymatic function.

**Hsu, K. S. Drug resistant mutants.**

Drug resistant mutants have been isolated and tested for their mode of inheritance, whether Mendelian or non-Mendelian. The aim was to add a new class of Mendelian markers besides the visible and nutritional ones, and to search for cytoplasmic determinants. No cytoplasmic mutants have been found, but gene controlled resistance to the drugs acriflavine, actidione, and caffeine has been demonstrated. The results obtained are summarized in the following table:

<table>
<thead>
<tr>
<th>Isolation No.</th>
<th>Drug</th>
<th>Resistance level (μg/ml)</th>
<th>Origin</th>
<th>Locus and linkage group</th>
</tr>
</thead>
<tbody>
<tr>
<td>KH1</td>
<td>acriflavine</td>
<td>2</td>
<td>spontaneous</td>
<td>acr-1 (II)</td>
</tr>
<tr>
<td>KH2</td>
<td></td>
<td>10</td>
<td>spontaneous</td>
<td>acr-2 (IIIL)</td>
</tr>
<tr>
<td>KH3</td>
<td></td>
<td>10</td>
<td>U.V.</td>
<td>acr-2 (IIIL)</td>
</tr>
<tr>
<td>KH4</td>
<td></td>
<td>50</td>
<td>U.V.</td>
<td></td>
</tr>
<tr>
<td>KH5</td>
<td></td>
<td>50</td>
<td>spontaneous</td>
<td>acr-2 (IIIL)</td>
</tr>
<tr>
<td>KH6</td>
<td></td>
<td>50</td>
<td>spontaneous</td>
<td>acr-2 (IIIL)</td>
</tr>
<tr>
<td>KH7</td>
<td></td>
<td>50</td>
<td>U.V.</td>
<td></td>
</tr>
<tr>
<td>KH51</td>
<td>actidione</td>
<td>10</td>
<td>U.V.</td>
<td></td>
</tr>
<tr>
<td>KH52</td>
<td></td>
<td>10</td>
<td>U.V.</td>
<td>act-1 (I)</td>
</tr>
<tr>
<td>KH53</td>
<td></td>
<td>10</td>
<td>U.V.</td>
<td></td>
</tr>
<tr>
<td>KH101</td>
<td>caffeine</td>
<td>2500</td>
<td>spontaneous</td>
<td></td>
</tr>
</tbody>
</table>

All in St. Lawrence background: A dash indicates that analysis is in progress.

With the exception of KH1 and KH2, which were picked up during serial subculture on acriflavine-containing minimal medium, all of the mutants were recovered by plating conidia on Vogel's minimal medium plus the drug. The concentration of the drug was such that no growth was observable when $10^6$ to $10^7$ conidia from the sensitive strain were plated. U.V.-irradiation giving 50-75% killing was applied in some cases. Strains displaying colonial growth and conidiating well were selected for use in the experiments. These included cr; cot; cr; cot; yle; cr; bal; and rq cr, all in a background of St. Lawrence stocks 74A and 73a.

Scoring for resistant vs. sensitive is clear-cut for all of the three categories of mutants. The gene acr-2 is the first gene to be located in the left arm of linkage group III. In a three-point cross acr-2 (allele KH2) x sc tryp-1, where sc is the centromere marker, the following phenotypic classes were observed:

\[
\begin{align*}
(\text{acr} + +) & 74 \\
( + \text{ sc tryp}) & 76 \\
(\text{acr} + \text{ tryp}) & 28 \\
( + \text{ sc} + 3) & 31
\end{align*}
\]

( + + + +) 1
( acr sc tryp) 6
( + + tryp) 0
( acr sc +) 0

**Roves, J. The production of mosaic mutations in Neurospora crassa.**

Nitrous acid, ultra violet light and X-rays have been used for producing recessive lethals in conidia of a balanced heterokaryon between arginineless and methionineless amycelial. A modification of Atwood's method showed that the