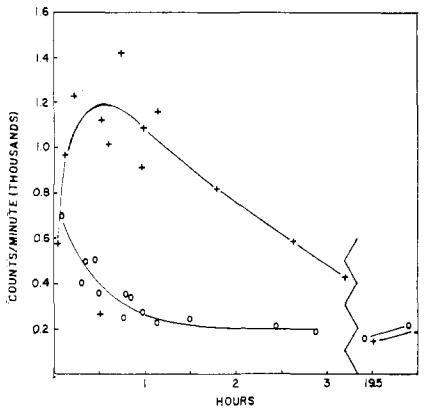


Figure 1. Time distribution of selenium in whole blood after injection of selenite and selenite plus arsenite

+ Values for rats injected with selenite plus arsenite

○ Values for rats injected with selenite

Each symbol represents the activity and time of sacrifice for one rat. Selenium-75 injected at zero time

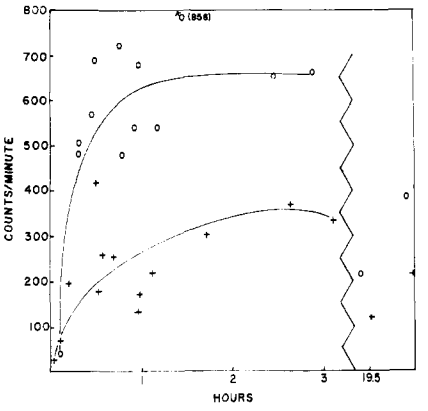


Figure 2. Time distribution of selenium in the liver after injection of selenite and selenite plus arsenite

+ Values for rats injected with selenite plus arsenite

○ Values for rats injected with only selenite

Each symbol represents the activity and time of sacrifice for one rat. Selenium-75 injected at zero time

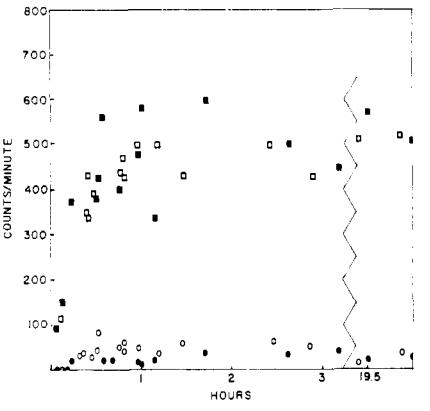


Figure 3. Time distribution of selenium in the kidney and in trichloroacetic acid extracts of the liver after injection of selenite and selenite plus arsenite

■ Activity in kidney of rats injected with selenite plus arsenite

□ Activity in kidney of rats injected with selenite only

● Activity in trichloroacetic acid extract of livers of rats injected with selenite plus arsenite

○ Activity in trichloroacetic acid extract of livers of rats injected with selenite only

The unique reaction of selenite would then be a rapid irreversible one antagonized by arsenic, involved in the formation of volatile selenium and probably initiated at the membrane of liver cells. Another reaction, toxic at a higher level of injection, although not necessarily at a higher concentration at the reaction site, would be a rapid reversible one in which its toxicity and that of arsenite are additive. This could be the reaction that Barron and Kalnitsky (*1*) found to be reversible by dithiols. A third reaction would be a rather slow, nonspecific, complex formation with proteins. This third reaction would account in some measure for the report by Klug, Lampson, and Moxon (*6*) that arsenite had no effect on distribution of selenium in rats that were protected from liver necrosis in a long-term experiment.

If these assumptions are reasonably accurate, the requirements for the blocking of acute selenite toxicity can be postulated rather simply. Enough arsenite must be present to block the first reaction without there being enough arsenite and selenite present to make the second lethal. If these conditions are met for an hour or two, the third reaction and excretion will render the selenite incapable of taking part in the unique reaction which leads to selenium exhalation and death.

**Table I. Mortality in Rats Produced by Injecting Indicated Amounts of Selenium as Selenite and Arsenic as Arsenite per Kilogram of Body Weight**

(Five rats in each group)

| Se, Mg./Kg. | As, Mg./Kg. 0 | 3 | 4.5 |
|---|---|---|---|
| 4 | 4 | 0 | 0 |
| 5 | 4 | 0 | 0 |
| 6 | 5 | 0 | 0 |

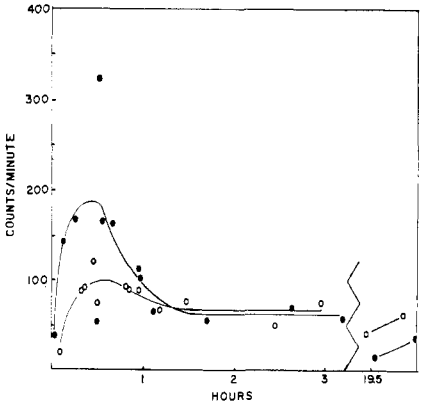


Figure 4. Time distribution of selenium in the spleen after injection with selenite and selenite plus arsenite

● Values for rats injected with selenite plus arsenite

○ Values for rats injected with selenite only

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## Alcoholic Fermentation of Blackstrap Molasses—Correction

On page 610 [J. AGR. FOOD CHEM. **5,** 610 (1957)], the author credit should be Walter Borzani, Universidade de São Paulo, São Paulo, Brasil. On page 612, the title, "Continuous Fermentation," should read, "Molasses Fermentation."

WALTER BORZANI