Comments and Communications

Hydrolysis of Steroid Esters on Activated Alumina

R. H. Levin, *et al.*, in a paper read before the meeting of the American Chemical Society, held in New York City on September 17, 1947, described the deformylation of the 3-formoxide derivatives of steroids when chromatographed on activated alumina (Fisher). This appears to be a special case of a more general hydrolytic splitting undergone by 3-hydroxy-steroid esters when passed through activated alumina.

A report from this laboratory (W. Dasler and C. D. Bauer. *J. biol. Chem.*, 1947, **167**, 581) has previously pointed out that steroid-3,5-dinitrobenzoates are partially hydrolyzed when chromatographed on activated alumina (Alorco). Thus, free calciferol could invariably be isolated from the eluates of alumina columns upon which calciferyl-3,5-dinitrobenzoate had been adsorbed.

W. C. Hess (*J. lab. clin. Med.*, 1947, **32**, 1163) has reported that when known mixtures of cholesterol and cholesteryl stearate were chromatographed on alumina (according to Brockman, Merck) prior to analysis, the values for free cholesterol tended to be about 5% high, and those for esterified cholesterol, about 5% low. Although other explanations can be advanced to account for these results, it seems not unlikely that this may be another instance of the same phenomenon, viz., the hydrolytic splitting of the steroid ester by the adsorbent.

It seems likely that partial hydrolysis of steroid esters during chromatography has been frequently encountered without being recognized. Some of our more recent work seems to indicate that the *p*-phenylazobenzoates of sterols are more resistant to hydrolysis than some of the other esters when chromatographed on activated alumina.

The hydrolytic splitting of 3-formoxide derivatives of steroids reported by Levin, *et al.* is noteworthy in that the deformylation in the 3- position was apparently complete, whereas formoxide residues at other positions in the molecule were unaffected.

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Crayfish in Southern Nevada

Faxon (*Mem. Mus. comp. Zool.*, 1885, **10**(4), 178), in his Revision of the Astacidae, stated that "the genus *Cambarus* [=Cambarinae] ranges from Lake Winnipeg to Cuba and Guatemala, from New Brunswick to Wyoming Territory (in Mexico to the Pacific Ocean)." Since that time there have been, to our knowledge, no published records which extend appreciably the known range of the Cambarinae. In the United States the most western records are those from Texas, New Mexico, Colorado, and Wyoming [*Procambarus simulans* (Faxon) from Mexico and Chaves Counties, New Mexico, and Boulder County, Colorado; *P. clarkii* (Girard) from Kinney County, Texas; and *Orconectes virilis* (Hagen) from Laramie County, Wyoming].

In August 1944 the junior author collected several crayfish in the Las Vegas River at Las Vegas, Nevada, and forwarded them to the senior author for identification. Upon comparing these with specimens of *Procambarus clarkii* from Texas and Louisiana we find that none of the variations exhibited are greater than those present in a single collection of this species from any given locality in either of these states.

It seems improbable that this species has arrived in the Las Vegas River by normal migration; however, all attempts to find out when and by whom it was introduced have proved futile.

Several years ago Waldo L. Schmitt, of the U. S. National Museum, informed us that *P. clarkii* had been introduced with success in the Santa Rosa, California, region, and we strongly suspect that their presence in the Las Vegas River in numbers may be explained by their finding here, after introduction, another congenial habitat.

It is hoped that we will be able to determine if and when crayfish were introduced in the Las Vegas River.

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The Effect of Rutin on Anaphylactic and Histamine Shock

Raiman, Later, and Necheles (*Science*, October 17, 1947, p. 368) have reported that rutin, a flavonol derivative, protected guinea pigs from anaphylactic shock, but afforded no protection against histamine shock. These authors concluded, therefore, that liberated histamine is not the direct cause of anaphylactic shock, or that rutin prevents liberation of histamine rather than protecting the sensitized animal against liberated histamine.

These conclusions would not be justified if it could be shown that flavonols or their derivatives protect against histamine shock. Such evidence was presented earlier in 1947 by Wilson, Mortarotti, and DeEds (*J. Pharm. exp. Therap.*, 1947, **90**, 120), who showed that under carefully controlled conditions rutin had a slight, though definite, protective action against histamine shock. Still earlier, Parrot and Richet (*C. R. Soc. Biol. Paris*, 1945, **139**, 1072) published data showing that death from histamine could be prevented by compounds closely related to flavonols. These authors demonstrated an increased sensitivity to histamine in scorbutic guinea pigs and reported that administration of a mixture of *d*-catechin isomers counteracted this increased sensitivity.

To permit demonstration of the protective action of rutin we found that the amount of intravenously injected histamine was critical. An approximately LD_{50} dose of 0.25 mg of histamine base/kg of body weight proved to be satisfactory. Raiman, Later, and Necheles used a mini-