through the fact that the galactosan, but not the glucosan, condenses easily with acetone. Acetone-D-galactosan yields D-galactosan (m. p. 223–224 cor., $[\alpha]^{20}D$ −22.0 in water, agreeing with Micheel's[4] data) by the hydrolytic conditions that were used in making D-mannosan from its acetone compound.[2] Periodate oxidation shows that the ring configurations for D-galactosan are $<1,5>\beta$-$<1,6>$. Acetone-D-galactosan $<1,5>\beta<1,6>$ possesses only one free hydroxyl group, the position of which is limited to one of three carbon atoms (2, 3 and 4).[5] The substance, now so readily available,[6] offers possibilities for syntheses, especially of disaccharides. The work is being continued.

(5) The recent research of McGreath and Smith, *J. Chem. Soc.*, 387 (1939), indicates that the free hydroxyl group is one carbon atom 2, the acetone having condensed on the *cis* hydroxyls of carbon atoms 3 and 4, as originally supposed by Micheel.

(6) Micheel's synthesis of D-galactosan starts from acetobromogalactose and trimethylamine.

NATIONAL INSTITUTE OF HEALTH
U. S. PUBLIC HEALTH SERVICE RAYMOND M. HANN
WASHINGTON, D. C. C. S. HUDSON

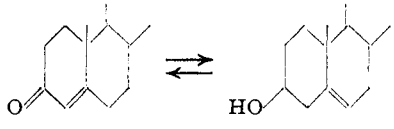
RECEIVED MARCH 25, 1941

STEROLS. CXXII

Sir:

Recently, Wolfe, Fieser and Friedgood [THIS JOURNAL, **63,** 583, 1941] raised a question as to our hypothesis on the formation of the Δ^5-3-hydroxysteroids as reduction products of a Δ^4-3-ketosteroid as lacking any foundation of analogy and as unlikely because "the process would require the migration of the double bond at 4,5, presumably after reduction of the carbonyl group, away from its position of conjugation with the oxygen atom."

We wish to point out that this type of reduction in the animal has been accomplished by Schoenheimer, Rittenberg and Graff [*J. Biol. Chem.*, **111,** 183 (1935)]. These authors fed coprostenone, a Δ^4-3-ketosteroid to a dog and found that it was eliminated as cholesterol, a Δ^5-3-hydroxysterol in which reduction of the ketone group had taken place with a migration of the double bond at 4,5.



They concluded that the formation of cholestenone from cholesterol is a biologically reversible process. Their work was discussed in our article on the theory of the formation of the various steroids [THIS JOURNAL, **60,** 1725 (1938)]. Although it is possible that the above reduction may be bacterial, it should be noted that it has never been proven that the various urinary steroidal reduction products are formed by glandular reduction and not by bacterial reduction.

In addition, it should be pointed out that according to our hypothesis of the formation of the various steroids, dehydroisoandrosterone need not be a transformation product of testosterone, or of androstenedione, but can arise directly as a degradation product of many of the numerous cortical steroids, by mechanisms described in our paper, without going through testosterone as an intermediate. The same is true for isoandrosterone, androsterone, etiocholanolones, etc. The numerous routes through which these products could be formed from the cortical compounds were omitted from our original paper, for the sake of brevity.

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STATE COLLEGE, PENNA. RUSSELL E. MARKER

RECEIVED FEBRUARY 21, 1941

ORIGIN OF DEHYDROISOANDROSTERONE IN URINE

Sir:

The observation of Schoenheimer, Rittenberg and Graff cited by Marker in the accompanying communication does not seem to us to constitute a valid reason for believing that the dehydroisoandrosterone secreted in urine arises from a Δ^4-3-ketosteroid precursor. The feeding of cholestone to a dog on a biscuit diet resulted in an increase in the fecal cholesterol over that noted in control periods; when the dog was put on a meat diet, cholestenone feeding increased the output of the principal fecal sterol, which in this case was coprosterol. The cholestenone used carried no indicator element, and no proof was adduced that the excess excretory sterols were transformation products of the material administered. Over 80% of the administered material remained unaccounted for, even on the supposition of a conversion, and the ketone may have stimulated normal sterol excretion, supplanted a normal transformation product of cholesterol, or influenced the sterol excretion in some other indirect manner. The experiment, therefore, cannot be considered to have established that cholestenone is capable of undergoing reduction to cholesterol