LIPID MOBILIZATION IN ADIPOSE TISSUE

By

LUCINDA GAYLE KEMPER CARR

Submitted to the faculty of the Graduate School
in partial fulfillment of the requirements
for the degree Master of Science in
the Department of Pharmacology,
Indiana University

June, 1963

Accepted by the faculty of the Graduate School, Department of Pharmacology, Indiana University, in partial fulfillment of the requirements for the Master of Science degree.

James Ashmore
Director of Thesis

Committee: James Ashmore, Chairman
Francis W. Hughes
Ward W. Moore
Joseph H. Stern

CHAPTER V

SUMMARY

1. The lipolytic response of adipose tissue *in vitro* is stimulated by catecholamines, lipolytic blocking agents at low concentrations, and polypeptide hormones. This stimulation can be modified by insulin, 3'4'-dichloroisoproterenol (DCI) and (2'4'-dichlorophenyl)2-t-butylaminoethanol (DCB).

2. DCI has a dual action on adipose tissue which is dose dependent. At lower concentrations of DCI *in vitro* which are approximately the same as the concentrations used *in vivo*, FFA release is stimulated; at higher DCI concentrations which inhibit the lipolytic response of catabolic hormones, the release remains at control levels.

3. The mechanism for the stimulation of lipolysis persists in the absence of the lipolytic hormones.

4. The inhibition of lipolysis produced when 3'4'-dichloroisoproterenol (DCI) is present in the medium is reversed when 3'4'-dichloroisoproterenol is removed; therefore, the inhibition is due to the molecule itself and not destruction of the lipolytic mechanism.

5. Insulin has two actions in regulating the fatty acid balance in adipose tissue: 1. to increase esterification, and 2. to directly inhibit the lipolytic mechanism.

6. The changes in carbohydrate metabolism due to lipolytic agents are undoubtedly influenced by FFA levels. However, since (2'4'-dichlorophenyl)2-t-butylaminoethanol (DCB) does not induce FFA release but does alter carbohydrate metabolism, it appears that these lipolytic agents also have a direct action on carbohydrate metabolism.

7. 2-deoxy-D-glucose blockade of re-esterification does not increase FFA release which suggests that the lipolytic mechanism and therefore its blockade is independent of glucose metabolism.

TABLE OF CONTENTS