Glucose Metabolism in Subjects with Behavior Pattern A and Hyperlipemia

By Ray H. Rosenman, M.D., Meyer Friedman, M.D., and Sanford O. Byers, Ph.D.

Seemingly healthy persons possessing a behavior pattern (type A) earlier observed to bear an associative and more recently, a predictive relationship to increased incidence of coronary heart disease also have been found to exhibit as a group, abnormal preprandial and postprandial values of serum triglyceride. Moreover, the general pattern of their serum lipid abnormalities is similar to that found in patients already suffering from coronary heart disease.

Because of the possible relationship of this lipid derangement to the pathogenesis of coronary heart disease, we have interested ourselves in further investigation of the possible mechanism(s) responsible for its presence in the majority of type A subjects studied. Our earlier studies indicated that it is not due to any deficiency in circulating lipoprotein lipase, nor is it due to the relatively greater consumption of alcohol on the part of some subjects with this type of behavior pattern.

Hyperlipemia is often associated with either an overt or covert hyperglycemia. This is particularly apt to be true in cases in which postabsorption turbidity, despite sufficient quantities of circulating lipoprotein lipase, is a prominent feature of the lipemia. In subjects with type A pattern, however, the hyperlipemia usually becomes significant only after ingestion of a fat meal. We thought it desirable, therefore, to determine in such subjects whether they too exhibited signs of abnormal glucose metabolism before and after an oral glucose challenge. The results herein reported indicate that the hyperlipemia observed in these subjects is not accompanied by any overt abnormality in handling of glucose.

Methods

Selection of Subjects

Nineteen subjects were selected for this study from a total group of 57 volunteer men whose vocation was the selling or processing of life insurance policies. The selection was done in the following manner:

All 57 men were interviewed, and the type of behavior pattern (that is, type A or type B) was determined according to previously described methods. Thirty-nine were adjudged as exhibiting pattern A (that is, they showed the signs of, or admitted the presence of, excessive competitive drive, sense of time urgency, or some semifrustration of considerable intensity). The remaining 18 men were thought to exhibit the converse behavior pattern B. From the 39 type A men, we selected 14 who, we believed, most intensely exhibited this pattern. We similarly selected 12 of the 18 type B men who, we believed, were the least aggressive and least harried by time pressure and other conditions of this group. This selection of men exhibiting either pattern in its most extreme form was done to facilitate detection of differences which might be less readily perceived in a short period in men differing only moderately in behavior pattern.

The 14 type A and 12 type B subjects were given a standard test meal containing approximately 85 g of dairy fat, after a fast of 13 hours. Serum triglyceride determinations were obtained before, and then 4 and 9 hours after the ingestion of the meal. Preprandial serum cholesterol determinations also were obtained.

On employing the previously described standards, 10 of the 14 type A men exhibited hyper-

From the Harold Brunn Institute, Mount Zion Hospital and Medical Center, San Francisco, California.

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lipemic, and nine of the 12 type B men
normalipemic, responses following the ingestion of
the test meal. These 19 men were recalled ap-
proximately 1 month later. At this second visit,
they were given a single load of 75 g of glucose
by mouth. Serum was obtained before and again
1 and 2 hours after the glucose intake and
analyzed for glucose.¹⁷

Results
As might be expected from the mode of
selection of these type A and type B subjects,
the former exhibited (table 1) markedly high-
er serum cholesterol and triglyceride values
than the type B subjects did. As previously
found,⁶ a strong correlation also was observed
in this study between the serum cholesterol and
the 4-hour postprandial serum triglyceride of
both type A and type B subjects (r = +0.93
in the type A subjects, and +0.73 in the
type B subjects).

Despite the marked difference, in the serum
cholesterol and triglyceride responses of these
two groups, their average serum glucose val-
ues either at the fasting or 1 or 2 hours after
glucose intake were essentially similar (table
1). Only one type A subject (Ho) showed
abnormal fasting and postalimentary serum
blood glucose values.

Discussion
The present results strongly suggest that
a defect in glucose metabolism does not
accompany the type of hyperlipemia that
occurs so frequently in subjects who exhibit
behavior pattern A in its most overt or ex-
treme form. Since the hypertriglyceridemia

Table 1

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<th>Preprandial and Postprandial Serum Triglyceride and Glucose of Hyperlipemic and Normalipemic Subjects</th>
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<td><strong>Subject</strong></td>
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<td><strong>Hyperlipemic subjects with pattern A</strong></td>
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found in these subjects is similar in some, but not all, respects (for example, absence of postabsorption turbidity) to the variety found by other investigators\textsuperscript{12, 13} to be associated with hyperglycemia, it is possible that hyperglycemia may occur later in some of our subjects. If it does, however, it would appear preferable to attribute it to the prior hyperlipemia—an order converse to that generally thought at present.

At this juncture, we still have not succeeded in identifying any of the possible mechanisms involved in the abnormal lipid responses of so many of the type A subjects. Even the discovery of its temporary abolition by administration of commercial preparation of corticotropin\textsuperscript{18} has introduced more problems than it has solved. Guided presently by Nestel's studies,\textsuperscript{19} we are inclined to believe that our subjects primarily lack the capacity of ridding their blood of chylomicra which circulate chiefly in the form of so-called primary fat particles.\textsuperscript{13} We are further inclined to suspect that this presumed failure in clearing capacity may reside in a major organ such as the liver, as well as in the adipose tissue. In this connection, although the liver is the only organ demonstrated to supply endogenously synthesized triglyceride into the blood,\textsuperscript{20} the rise and fall of this lipid in blood has generally assumed to be under the control of lipolytic activity in adipose tissue.\textsuperscript{12} The rather widespread acceptance of this assumption cannot help but evoke some surprise when it is considered that no quantitatively secure evidence attesting to the soundness of this assumption has yet been obtained, or for that matter, even searched for. The possible fallacy of thus equating increased peripheral discharge of fatty acid with increased serum triglyceride levels was demonstrated clearly by the recent observation\textsuperscript{21} that the administration of epinephrine, which increases the discharge of fatty acids into the circulation actually effects a decrease in the postprandial serum triglyceride of the fat-fed adrenalectomized rat.

\section*{Summary}
Men exhibiting an extreme form of behavior pattern type A and preprandial and postprandial hyperlipemia were not found to suffer from any discernible defect in glucose metabolism.

\section*{Acknowledgment}
We gratefully acknowledge the technical assistance of Ashley Tam.

\section*{References}
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13. \textsc{Bierman, E. L., Porte, D., Jr., O'Hara, D. D.}, 

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In clinical medicine the difficulties of securing objective proof by exact observation of identically recurrent events are very great. This tends to make the observer rely on intuition rather than on established conclusions and to make him impatient of the labour of proving or disproving the truth of opinions arrived at by intuition. The result is that the current clinical medicine of any given period is apt to be loaded with 'authoritative' opinions often contradictory of one another, many of which are actually capable of, but have never been submitted to, definite proof or disproof.—Wilfred Trotter: *The Collected Papers of Wilfred Trotter*. London, Oxford University Press, 1941, p. 120.
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