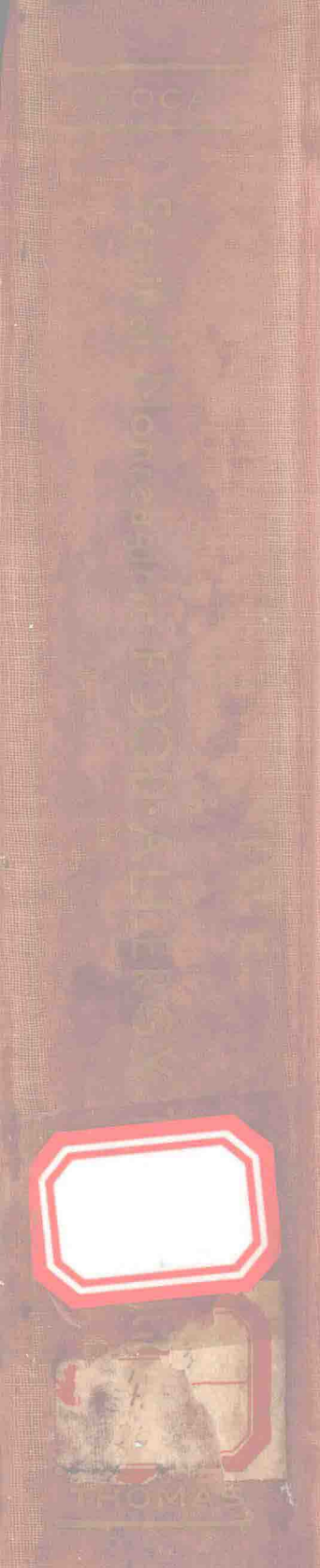

Familial Nonreaginic FOOD-ALLERGY

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TO

ELLA F. GROVE

skillful collaborator, wise counsellor,
friend to all in need,
my admired wife

Introduction

THE CURRENTLY orthodox concept of the pathogenesis of allergic disease is that of von Pirquet and Schick.[1] In its elementary form this theory stated that antigen and antibody, in their mutual reaction in or upon a susceptible tissue, may irritate that tissue, thus causing the injurious effects that are revealed in allergic symptoms.

The theory of von Pirquet and Schick has been fully realized in only two of the several known categories of allergic disease that are described in the next chapter; that is, in the atopic category and in serum sickness. Only in these two categories have antibodies been identified with certainty as the specific cause of the allergic phenomena, and it seems most probable that the antibodies in the two categories, that is, the atopic reagins and the antibodies of serum disease (Karelitz[2]), are qualitatively different from each other and that both are different from precipitin.

In the three other categories (contact dermatitis, allergy of infection and familial nonreaginic food-allergy) the hypothetical antibodies postulated by the theory have not been demonstrated and the search for them is somewhat discouraged by the circumstance that some of the excitants of these allergic conditions are not antigenic; for example, metals, alkaloids and synthetic chemicals. Nevertheless, the assumption of antibody-like substances as the specific mechanism of all allergic disease seemed justified by the *specificity* of the allergic reactions.

However, recent observations, which are discussed in this edition (Chapter XIX), have indicated that antibodies, as we know them, can have no part in the specific reactions of the fifth category of allergic disease, idioblapsis.

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[1] von Pirquet and Schick: *Die Serumkrankheit.* Deuticke, Leipzig & Wien, 1905.
[2] Karelitz, S. and Stempien, S. S.: *J. Immunol.*, *44*:271, 1942.
Karelitz, S.: *J. Immunol.*, *44*:285, 1942.
Karelitz, S. and Glorig, A.: *J. Immunol.*, *47*:121, 1943.

Preface to the Third Edition

KNOWLEDGE OF THE theory and practical use of the principles of idioblaptic allergy has been substantially extended since the publication of the second edition of this monograph.

Eight items seem worthy of special mention:

1. The effective control of allergenic house-dust with Dust-Seal, a product devised by Leonard S. Green.
2. The diagnostic and prognostic use of the stellate ganglion block.
3. Locke's demonstration of the predispositional relation of idioblaptic allergy to poliomyelitic paralysis.
4. Milo G. Meyer's report; including his 24 cases of hypertension, and his two cases of multiple sclerosis, since increased to 13 and fortified by the later cases of Alan Johnston and the writer.
5. The reports on the high incidence of tobacco-sensitivity by the writer and that by Granville F. Knight.
6. The demonstration that idioblaptic allergy does not depend on an antigen-antibody mechanism.
7. The report of Conrad Berens and his associates on Allergy in Glaucoma, studied with the pulse-diet method.
8. The adoption of the routine plan of a series of single-food tests at 1 or $1^1/_2$ hour intervals through the day.
9. The message to gynecologists and pediatricians in the report by Alan Johnston and the author; "Concerning the Special Problems of Idioblaptic Disease as It Affects Women."
10. John H. Irwin's description of his simplified technic in "Conservative Sympathectomy as an Antiallergic Measure," (Medical Record, Vol. 163, Dec., 1950).

Among those who have mastered the now fully developed pulse-dietary procedure, the impression grows that it cannot well be fitted into the heavy program of the practicing physician.

Its proper personal application to one or two patients may require the almost constant presence of the physician in his office for most of one or two days.

On the other hand it is easily possible to train nonmedical college-graduated nurses in the art of pulse-dietary interpretation. With the assistance of a sufficient staff of such personnel a few experienced medical specialists could organize a service which would accommodate a relatively large number of patients at a cost within the reach of practically all working people.

The establishment of such a diagnostic center with adequate facilities for the usual clinical laboratory examinations will greatly advance the cause of the new preventive medicine.

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Preface to the Second Edition

IN THE PREFACE of a publication an author is permitted, by custom, to intrude his more personal thoughts.

For taking advantage of that custom in this second edition I plead a considerable provocation at the hands of many personal acquaintances among experienced allergists and other medical specialists, from whom I could reasonably expect at least an unprejudiced hearing, if not a generous coöperation.

The attitude of most of these towards the first edition of this monograph has been that of a skepticism so uncompromising that I have not even been invited to demonstrate the new method of examination described therein.

It is quite out of the question to attribute this attitude to any personal prejudice; no, the reason for it is that the medical profession is again faced with scientific findings and their consequences that are so far out of line with settled concepts as apparently to represent the impossible.

The following preliminary conclusions drawn from my own study and the reports of Locke and of Price will illustrate the wide divergence of these findings from accepted medical dogma:

1. The level and range of the normal pulse-rate is a physiological constant in each individual, varying widely in different individuals.

2. The most common cause of variations in the individual from this normal constant is familial nonreaginic food-allergy (idioblapsis).

3. Upwards of 80 per cent of the white population are hereditarily affected with idioblaptic allergy.

4. Idioblapsis is probably a lethal character; the most important primary cause of a noninfectious disease, and a predisposing cause of some infectious disease.

These four major conclusions are of a sufficiently revolutionary nature to explain the hesitation of experienced medical specialists

to waste much time with the described method of diagnosis and treatment. A few tell me that they have tried it unsuccessfully but only two* have seriously asked me to help them with their difficulties in its use, much as I should like to do so.

However, I know that an interest in this matter is stirring here and there and confirmation of the fundamental facts is already beginning to appear.

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* One of these was the late Warren T. Vaughan, who sent me a beautifully complete pulse-diet record, from which it could be seen that the patient was intermittently exposed to an environmental pulse-accelerating allergen.

Preface to the First Edition

THERE IS A large body of published clinical evidence contributed by many observers and much more unpublished observation that points to an allergic nature of many clinical symptoms and syndromes, some of which are thought to be independent pathological entities. Such interpretation has been impressed upon the thought of these numerous professional and lay observers by the repeated association of the symptoms with the eating of particular foods that were ordinarily taken at relatively long intervals of time. In some instances the belief in this causal relationship has been supported by the demonstration of a cutaneous sensitivity to the incriminated foods in the affected persons.

But the very acceptance of this last point of evidence as supporting the allergic theory with respect to the few cases in whom specific cutaneous sensitivity could be shown has actually served to prevent the acceptance of the theory with respect to the much larger group of clinically identical cases in whom the cutaneous tests have failed to support it.

Fortunately, the new diagnostic method described in this report provides a highly accurate, practical means of identifying the excitants of food-allergy and thus enables us at last to determine which symptoms and syndromes are food-allergic.

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CHAPTER I

Classification of Allergic Disease

IN THIS BOOK, chief attention is paid to a certain category of allergic disease.

It may be helpful, therefore, to some readers who are not conversant with the classification of allergic diseases that is now generally recognized, if this is briefly set forth at the outset.

Five categories of allergic disease are distinguishable:

(1) *Atopy.* This group comprises bronchial asthma, hay-fever, and the condition sometimes called infantile eczema (atopic dermatitis), which are subject to a common hereditary cause. It is, indeed, this hereditary character of these clinical conditions which unites them in one group, and separates them from the other categories.

Many of the subjects of these atopic conditions exhibit another quality, which is lacking in the other four categories, namely, the presence of demonstrable allergic antibodies, "atopic reagins," which are sometimes responsible for the clinical sensitivity of the atopic individual. The existence of these atopic antibodies, as differing qualitatively from all the classical antibodies, especially those referred to as precipitin, was first reported in 1925.[1] The differences first noted were the following: (1) Precipitin passively sensitizes the unstriped muscle of the guinea-pig but not the human skin, whereas atopic reagin sensitizes passively the human skin but not the guinea-pig's unstriped muscle; (2) mixtures of the atopic antibody with the related antigen show no visible precipitate, as is the case with similar mixtures of other antibodies with the respective antigen; nor does partial desensitization of atopically sensitive tissue by the specific antigen follow the law[2,3] controlling this process in the case of anaphylactically

[1] Coca, A. F. and Grove, E. F.: *J. Immunol., 10:*445, 1925.
[2] Coca, A. F. and Kosakai, M.: *J. Immunol., 5:*297, 1920.
[3] Walzer, M. and Grove, E. F.: *J. Immunol., 10:*483, 1925.

sensitive unstriped muscle of the guinea pig; (3) the atopic antibodies are much more susceptible to heat than are the classical antibodies.[4,5]

Other differentiating peculiarities of the atopic antibodies are: (4) The easy reversibility of their specific union with the related antigen as shown by their inability to inactivate the latter.[5,6,14] A mixture of pollen-extract or extract of rabbit-dander with the respective atopic serum causes the same reaction in a sensitive skin as a mixture of the extract with the same volume of saline solution.[6] (5) Mixtures of egg-white with precipitating immune serum versus egg-white from a nonatopic child who has had parenteral contact with egg, fix complement permanently in a quantitative zone of the antigen ranging between 1–30 and 1–100,000, whereas, with the serum of a highly egg-sensitive child, the fixation is only transient and in a zone of characteristically much higher antigenic dilutions, 1–240,000 to 1–10,000,000.[7,8] (6) The atopic antibodies possess a high affinity for human fixed tissues,[9,10,11,12] which is lacking in the classical antibodies.[13]

A third important feature of the atopic illness is the independence of its organic localizations. Thus, although the atopic sensitivity in two individuals may be mediated by atopic reagins specific for the same excitant, the clinical symptom in one may be asthma, in the other rhinitis. This fact alone suggests that these so-called "shock-organs" possess some abnormality other than their quota of allergic antibodies; this assumption is needed to account for their evidently independent susceptibility to the antibody-antigen reaction that takes place equally on both organs in the two individuals.

This principle of the independent susceptibility of the allergic

[4] Coca, A. F. and Grove, E. F.: *J. Immunol., 10:*445, 1925.
[5] Loveless, M. H.: *J. Immunol., 38:*25, 1940.
[6] Levine, P. and Coca, A. F.: *J. Immunol., 11:*411, 1926.
[7] György, Moro, and Witebsky: *Klin. Wchnschr., 9:*1012, 1930.
[8] Katzin, E., cited by Coca, A. F.: *Ergeb. d. Hyg. u. s. w., 14:*538, 1933.
[9] Coca, A. F. and Grove, E. F.: *J. Immunol., 10:*445, 1925.
[10] Gay, L. N. and Chant, E.: *Bull. Johns Hopkins Hosp., 40:*270, 1927.
[11] Bell, S. D. and Eriksson, Z.: *J. Immunol., 20:*447, 1931.
[12] London, McKinley: *J. Allergy, 12:*244, 1941.
[13] Freund, J.: *J. Immunol., 16:*515, 1929.
[14] Reddin, L., Jr.: *Am. J. Vet. Res., 6:*60, 1945.