THE SIGNIFICANCE
OF
PHENYLKETONURIA IN AUSTRALIA.

by

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February, 1960.
STATEMENT OF THE THESIS.

PART I.
A Review of the History of Medical Knowledge of Phenylketonuria, and of the Contribution made by this Thesis.

PART II.
An Essay on the Epidemiology of Phenylketonuria in Australia.

PART III.

PART IV.
The False Precise of the Symptomatology of Phenylketonuria.

PART V.
An Aspect of the Physiology of Phenylketonuria: Reactivity to Adrenaline.

PART VI.
The Public Health Problem, with Recommendations for Australia.


ACKNOWLEDGMENTS.

REFERENCES.

AUTHOR'S DEDICATION.

DEDICATION BY THE AUTHOR SUPPORTING THE THESIS.
The prevalence of phenylketonuria that has been observed in Australia is exceptionally low, and the disease has attracted correspondingly slight attention. Yet many of its features - including the low prevalence itself - are of considerable medical interest and wide epidemiological, economic and social significance. Investigations of the epidemiology, social psychology, symptomatology, physiology and public health problems permit the prediction that the disease is about to become more conspicuous in this country.

HYPOTHESES COMPLEMENTARY TO THE MAIN THESES.

Complementary to the main thesis are five hypotheses (pertaining respectively to epidemiology, social psychology, symptomatology, physiology and public health), demonstration of which affords a strong inference of the truth of the main thesis.

First Hypothesis (epidemiology): The disclosure is made that there is a remarkably low known prevalence of phenylketonuria in the Australian population. A systematic study of the epidemiological factor suggests that this is illusory, and reveals the probable source of the deception. A projection into the future reveals that the prevalence is likely to rise.

Second Hypothesis (social psychology): The unconsciously determined behaviour and attitudes of the people having to do with a disease may have a regulating effect on its prevalence. (This hypothesis is referred to as "participant-regulation"). A systematic discussion is presented of evidence which affords a reasonable inference of its effect on phenylketonuria.
Third Hypothesis (symptomatology): A presentation is given of the symptomatology of the known phenylketonuric population in Australia. From the variability of the symptoms, it is probable that they are of no real use in diagnosis, nor will they form a basis for estimating the success achieved by therapy.

Fourth Hypothesis (physiology): In addition to the involvement of central nervous system and skin, the aberrant arachidonic acid metabolism in phenylketonuria affects biosynthesis in the adrenal medulla. Phenylketonuric individuals show increased reactivity to adrenaline, and this abnormality in the internal environment may play a part in the mental deficiency.

Fifth Hypothesis (Public health): An appraisal of the public health aspects of phenylketonuria in Australia leads to the conclusion that mental hygiene departments should consider establishing programmes for mass detection and treatment in infancy.