THE INFLUENCE OF HEREDITY IN PHTHISIS.

BY

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THE question of Heredity in Phthisis is one of great importance and of some difficulty. The importance of the subject cannot be doubted, for there is hardly a family in the country in which phthisis has not claimed a victim in one or more generations. If heredity be a potent cause of the disease, how few are the individuals who could hope to escape! But is the disease hereditary? The difficulties of deciding the question are numerous. Not only is the collection of the necessary *facts* far from easy from the want of knowledge or the want of accuracy on the part of patients in giving their family histories, but the *deductions* from these facts when obtained are by no means clear.

It is difficult also to approach the question with an unbiassed mind, in the face of the almost universal acceptance of the hereditary nature of consumption handed down from the past. The universality of the belief in the hereditary nature of consumption is easily explained when we know how frequently phthisis is found

attacking several members of the same family in successive generations ; and anyone who wished to uphold the doctrine of heredity in phthisis would have no difficulty in amassing facts in support of his thesis. It is the apparent self-evidence of the matter which makes it difficult to conceive that there is any room for discussion. As Wilson Fox remarks, "the influence of this cause (heredity) in the production of phthisis is unquestioned." That phthisis is more common in some families than in others is not open to question ; but the same is true of almost any disease, or indeed of any occurrence. If we were to investigate the statistics of scarlet fever or of malarial fever, we should find apparent support for a contention that these are hereditary ; for undoubtedly we should find these diseases occurring repeatedly in some families, and never recorded in others. A family in which for generations the sons have served in the army and navy would possibly show malarial disease in some of its members for several generations ; but this would not prove any hereditary transmission of malarial taint. The occurrence of ague for generations in families living in the Fens is no proof of the hereditary nature of malaria ; nor is the frequency with which pediculi attack the children of successive generations of some poor families any evidence of heredity in this occurrence. In the above instances the mode of life, place of residence, and surrounding conditions are easily seen to exert so powerful an influence in determining the morbid condition as to furnish a complete explanation of its causation ; and we at once agree that the greater incidence of these conditions on certain families is due to the members of them following the same profession, or living amidst similar surroundings. With phthisis, however, we are less able to see clearly the relative value of hereditary influence on the one hand, and of occupation and surroundings on the other. It is only within the present generation that the nature of pulmonary consumption has been determined, and the influence of

the surroundings and occupation of the individual on its causation more accurately studied.

Beliefs which have been held for ages are not easily relinquished, even when the contentions upon which they were founded have been disproved. It requires a more ample array of facts and more accurate reasoning to disprove an old theory than to successfully launch a new one. In the present case my inquiry has led me to consider the old theory of heredity in phthisis to be fallacious. I did not propose to myself, in preparing this paper, the task of trying to disprove the theory that phthisis is an hereditary disease. I set myself to analyse, as far as possible, the notes of 1000 cases, chiefly drawn from my hospital records; and to see how the facts, as I find them given in these ward notes, fit in with the theory of heredity.

In the foregoing preliminary remarks I have merely endeavoured—with the object of clearing the ground and counteracting prejudice in favour of the heredity theory—to urge that the question of hereditary influence is still open. I may at once state that, though I think my figures warrant a conclusion that direct heredity plays a less important part in the causation of phthisis than has sometimes been supposed, there appears to be no doubt that a *predisposition to become phthisical*—if exposed to tubercular infection—is often dependent upon inherited constitutional weakness.

Before proceeding further it will be well to define more exactly what we understand by heredity as a factor in disease causation.

(A) There is first *direct hereditary transmission* of a disease, the development in the offspring before birth of a morbid condition derived from the parent. The child is born suffering from the disease. This is true hereditary transmission, and is exemplified in congenital syphilis; it also appears in some inherited deformities, such as supernumerary fingers or toes. The disease is then properly called “congenital.” This kind of heredity is seen in

those cases, comparatively few in number, in which tuberculosis has been found in the young of animals *in utero*, or in the infant at birth.

(B) Then, secondly, the disease may be implanted in the body before birth, but does not manifest itself until some time after, *e. g.* infantile syphilis.

It is almost impossible to prove the occurrence of this mode of transmission for tuberculosis; for there are so many ways in which the causative bacillus may get access to the body of the infant after birth. It is, therefore, difficult to say whether a tuberculosis, showing itself some weeks after birth, is inherited (congenital) or acquired; in the majority of cases I should suspect the latter mode of origin.

In both the above categories a definite germ of disease is implanted; they differ thus essentially from the next two.

(c) Of these, the most common way in which heredity acts in disease causation is that there is transmitted a peculiar liability or susceptibility to develop a certain disease if the individual be exposed to the exciting cause of that particular disease; there is a diminished resisting power with regard to some special ailment. Here a predisposition is inherited, not the disease itself, and we may call this "direct hereditary predisposition." Examples of this form are numerous, as in gout and insanity. This susceptibility may be transmitted by parents who, though themselves inheriting the predisposition, have escaped the disease,—"*atavism*." It may be noted that this escape of the susceptible person shows that the inherited tendency may be combated and the disease itself avoided by avoiding the determining or exciting cause.

(D) In the last form the inherited susceptibility to disease may be general, not special, *i. e.* a liability to take any of a wide range of diseases to the exciting cause of which the person may subsequently be exposed. There is a diminution of resisting power against disease in

general. We may term this general inherited predisposition to disease, or delicacy of constitution.

Direct hereditary predisposition is what is now generally implied when consumption is said to be inherited.

As we are not now discussing tuberculosis in general, we may dismiss, for the purposes of this paper, the two first-mentioned forms of heredity, *i. e.* congenital diseases. Phthisis, or primary pulmonary tuberculosis, is essentially a disease of adult life, and it is difficult, with our present knowledge of the causative micro-organism of tuberculosis, to imagine the bacillus remaining latent in the body from birth to maturity.¹

We have, then, to consider—

- (1) Direct hereditary predisposition to phthisis.
- (2) General inherited delicacy of constitution in relation to phthisis.

The first being a definite tendency to a particular disease, transmitted from parent to offspring, would be a true heredity, and as such is a matter of considerable importance. It behoves us, therefore, carefully to inquire whether it can be proved to exist for phthisis, and if so to what extent.

The second, though having an important bearing on the future health of the individual, hardly comes pro-

¹ Cases frequently occur which tend to show that the bacillus of tuberculosis may remain potentially active in the body for a long time without causing symptoms, and may yet eventually light up fatal disease. Encapsuled masses of tubercular disease, containing bacilli, may remain for considerable time in the lung without any apparent effect on the health, until in some way the bacilli escape and infect other parts of the lung, the disease then rapidly spreading. An example of the escape of bacilli from such an encapsuled mass was shown by me at the Pathological Society last session. Those cases in which the individuals in early life suffered from strumous glands—tuberculosis of the superficial cervical lymphatic glands—and then in later life develop pulmonary tuberculosis, may be examples of a similar event; though it is impossible to exclude the influence of a fresh infection of the lungs from without. The comparative immunity of the sufferers from strumous glands in childhood from subsequent phthisis is against the theory of auto-infection in these cases.

perly under the designation of an hereditary predisposition to phthisis, since it might equally plausibly be considered an hereditary tendency to scarlet fever or to yellow fever, or to almost any disease to which the individual might become exposed. This is in fact the essential point to which I wish to draw attention, and which I have here set myself to examine. Is the inherited predisposition, which is at present considered so important and so potent a factor in determining the incidence of consumption, a true heredity, or is it merely an illustration of the fact that those of delicate constitution, from whatever cause, are more liable to contract diseases to which they become exposed than those who are constitutionally robust?

Again we find it necessary to define terms somewhat more exactly. What do we understand by "delicate" and "robust" constitutions?

The breeders of animals know well how much of the usefulness and capacity for work depends upon the stock from which the animal springs. They recognise that only selected animals should be utilised for breeding purposes, and that these stud animals must be kept under certain conditions as to food, air, and exercise, in order that their offspring may turn out valuable. They endeavour to ensure that whatever peculiarities of shape, colour, or disposition the young animals may inherit from their parents, they shall have no blemish and no inherited liability to illness. The offspring of healthy parents who have been living in accordance with hygienic requirements will probably be themselves healthy, and if kept under similar hygienic conditions will in all probability grow up strong and capable of a full measure of work. Such animals, if exposed to unfavourable (unhygienic) conditions, such as extra strain of work or unhealthy surroundings, are less likely to break down, or will withstand the evil effects of these conditions for longer, than animals less carefully bred. They have "robust" constitutions—the strength which lies in

healthy tissues and healthy organs performing their functions naturally and harmoniously. Divergence from this desirable condition, if habitual, constitutes "delicacy," the ever-present liability to break down under a strain, or to become *invalid*.

Suppose the constitutionally robust animal to be afterwards placed under conditions unfavourable to health, his inherent strength may prevent a break-down. But although not suffering from any definite disease, the animal, whose tissues and organs are now unable perfectly to perform their several functions, would, in all probability, produce offspring similarly deficient in functional vigour—*i. e.* constitutionally delicate. A stage further in the downward course, and the animal would be incapable of producing offspring at all. This, then, illustrates the well-known fact that unhealthy offspring may come from parents who have no actual disease.

The same is true for human beings: if the parents are not strong and healthy, the children will be delicate in constitution, less able to bear the strain of work and anxiety, and more easy victims to disease than the children of perfectly healthy parents. The debility of the parents is not necessarily due to disease; it may be dependent upon unhygienic surroundings, yet the result is similar in the children—they have less power of resistance against disease. The Londoner of the fourth generation is said not to exist; and even if this statement oversteps the bound of literal accuracy, it is undoubtedly true that the conditions of life in the crowded slums of a large city tend to diminution of vitality in the individual. The children of parents thus debilitated, and themselves brought up in the same unhygienic surroundings, are weakly and delicate, and have small resisting power against disease, whether it be tuberculosis or some other malady. The particular disease which attacks them depends upon the liability to exposure to one or another of them. The debility of the parents may, on the other hand, be due to their being consumptive; and if this

cause a greater liability to the children becoming phthisical—if there be a special or specific hereditary predisposition—we should find the children of such parents phthisical in greater proportion than the children of parents whose debility arises from other causes. Let us then carefully examine our figures to see how far this is the case (see Table I). We find here that out of 1000 cases of phthisis, one or both parents were consumptive in 325, or 32·5 per cent. In 474 of these cases I have obtained a fuller account of the family, and of these 199 (nearly 42 per cent.) give a history of phthisis in the parents. In the families of the non-phthisical parents, 24·87 per cent. of the children became phthisical, against 33·71 per cent. of the children of the phthisical parents.¹ This shows a difference of only about 9 per cent.—a very small influence in favour of hereditary predisposition.

This additional 9 per cent. of phthisical children in those families in which the parents are consumptive might be explained by the increased exposure to the exciting cause, to which those living with consumptive parents are necessarily liable. Dr. Philip, of Edinburgh, in an analysis of 1000 cases of phthisis, put the percentage of cases in which the disease seemed to him to be due to direct infection at seven, or very nearly the whole proportion which, in the figures given above, represents the possible influence of hereditary predisposition.

It is argued that the parents may transmit this specific predisposition from their parents, without being themselves consumptive; so that to exclude the possibility of hereditary transmission in any case we should know the history of earlier generations. We might grant this, and yet take a very hopeful view of the effect of hereditary influence in phthisis. It seems probable that inherited peculiarities become weakened in each successive

¹ It is remarkable how closely the figures obtained by two different modes of inquiry approach to one another. The proportion of phthisical patients who give a history of phthisis in their parents is 32·5 per cent., whilst the proportion of the children of phthisical parents who became consumptive is 33·71 per cent.

generation, and tend to die out unless strengthened by intermarriage with a family in which the same peculiarity is strongly marked. If, therefore, the offspring of phthisical parents escape the disease, they transmit the predisposition with a diminished potency, and their children should be even more able to escape. I would lay stress upon this point, that however frequent we may find the hereditary influence to appear in statistics, it is not so strong as to cause alarm; though it may be strong enough to serve as a caution, and to indicate the necessity for extra care in avoiding the determining cause of the disease.

We will now proceed to a fuller examination of our figures, and see what we can glean from them. In the 1000 cases a history of phthisis in one or both parents was obtained in 325 cases, giving a proportion of 32·5 per cent. of the total number of cases; 604 were males, and of these 175 had a history of phthisis in the parents, *i. e.* 28·97 per cent. The 396 females gave a history of phthisis in the parents in 150 cases, or 37·87 per cent.

So far we see that in 1000 families in which one or more members of one generation became consumptive, one or both parents also had phthisis in about a third of the whole number. If we were to include uncles and aunts and grand-parents, the proportion of families in which phthisis was present in previous generations would be increased to about 35 to 40 per cent. This would seem to show a fairly strong hereditary influence, but on the other side we must put the not inconsiderable number of families in which phthisical individuals have had children and grandchildren free from the disease, and which necessarily do not appear in statistics taken from the records of hospitals for consumption. We are at once face to face with one great difficulty in attempting to decide the influence of heredity in consumption. To obtain reliable results we should commence with phthisical individuals, and obtain the history of their children and grandchildren, or their offspring for several generations;

In illustration of this statement let us take a supposititious family with a very bad phthisical history. We have seen that of the children of phthisical parents, only about one third become consumptive. In the following scheme capital letters indicate phthisical individuals, the small letters those who remain free.

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graph TD
    A --> B
    A --> b
    B --> c
    B --> C
    C --> c
    C --> c
    c --> a
    c --> D
    c --> d
    c --> d
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    c --> d
    b --> c
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    c --> a
    c --> d
    c --> d
    c --> d
    c --> d
    b --> C
    b --> c
    C --> c
    C --> c
    c --> a
    c --> D
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If we trace back to grand-parents the proportion showing heredity is 50 per cent., and going back one more generation we find it to be 100 per cent. Even if we only go back one generation and take the collateral

branches (uncles and aunts) the hereditary influence appears as high as 83·3 per cent., that is five out of six consumptives (*D*) show phthisis in parents or uncles and aunts (*O*). If, however, we commence with the phthisical patient *A*, we find the influence of heredity represented by 33·3 per cent. in the children, by only 25 per cent. in two generations, and by 25 per cent. in three generations, in spite of the large number of consumptives in the last generation.

We might, under unfavourable conditions of life, find quite as large a proportion of phthisical persons in three generations derived from a perfectly healthy stock.

Seeing, then, how fallacious will be the results obtained in the ordinary way, I have endeavoured to find out how far the children of phthisical parents are affected. I have not the details of the whole number of children in all the 1000 families, but only in about half of these, viz. 474. In these 474 families there were 275 in which neither of the parents was phthisical. These families comprised 1745 children (an average of 6·34 to each family); 935 were males and 787 females, and 23 who died in infancy, of whom the sex is not mentioned. If we divide these 23 proportionally between the sexes we should have 947 males and 798 females. Of the *male children* 111 died in infancy (11·87 per cent.), and of the remaining 836, 244 became phthisical, or 29·06 per cent. (see Table II).

Of the *female children* 82 died in infancy (10·41 per cent.), and of the remaining 716, 142 became phthisical, or 19·81 per cent. The proportion of phthisical invalids of both sexes is 24·87 per cent.

The above figures tend to show the influence of occupation in determining phthisis where there is no special predisposing cause in the family; for the males suffer out of all proportion to the females.

In 204 out of the above 275 cases I have noted the grand-parents and uncles and aunts of the patient, and in 179 there was no phthisis at all known in the family,

whilst in 25 cases there was phthisis in grand-parents or collaterals. I have endeavoured to find whether the proportion of individuals affected with phthisis is modified by this presence of consumption in the remote members of the family. The numbers are too small to be of much value, but my figures show that in the families where phthisis has been previously known, though the total incidence of phthisis is less than where no phthisis has been present (19·39 per cent. against 20·48 per cent.), the proportion of females attacked by phthisis is much greater in those families where previous examples of the disease are noted (males 16·25 per cent., females 22·35 per cent. as against males 27·78 per cent., females 18·69 per cent.) (Table III).

In 84 families (out of the 474) the father was phthisical.

These families comprised 511 children (an average of 6·08 to each family), of whom 267 were male and 244 female; 67 (or 13·11 per cent.) died in infancy, and of the remaining 444, 38 became phthisical, or 31·08 per cent.

In these families the incidence of phthisis is greater on the female children than on the males (35·18 per cent. as against 27·19 per cent.).

Delicacy of constitution is as potent a cause as the effect of occupation.

We see, also, the result of weakness in the father shown in the greater proportion of infantile deaths, especially amongst the male infants. The weakly sons having died off, the proportion of phthisis amongst the surviving male children is less than in the previous class, but the girls are more prone to phthisis than in families where there is no hereditary predisposition. We must remember, however, that the girls stay more at home than the sons, and are in closer contact with the phthisical parent, especially when the disease causes him to give up work and lie up.

In 82 families the mother was phthisical. These comprised 506 children (6·17½ to each family), of whom 246

were male and 260 female. Fifty-six (or 11·06 per cent.) died in infancy, and of the remaining 450, 155 became phthysical, or 34·4 per cent.

Here, as in families where neither parent was phthysical, the male children suffer proportionally more than the female (39·43 per cent. against 29·95 per cent.). And a greater proportion of male children die in infancy (13·41 per cent. male, female 8·84 per cent.). The proportion of females affected is actually smaller than in the families where the father was phthysical. It might be urged that the male children inherit the mother's characteristics, whilst the female children are most influenced by the male parent. This is the reverse of what Dr. Reginald Thompson gathered from his figures. When the mother is delicate the proportion of male children is deficient, whilst those that are born are wanting in stamina, as shown by their liability to phthisis.

In 33 families both parents were phthysical; and in these there were 165 children (5·0 to each family). Eighteen died in infancy (10·9 per cent.), and of the remaining 147, 58 became phthysical, or 39·45 per cent.

The female children were only slightly more affected with phthisis than the males (38·8 per cent. of males to 40·0 per cent. of females). The infantile deaths are numerous amongst the male children, but few amongst the girls. In the survivors phthisis is common.

In the above families where phthisis is noted as having attacked one or both parents, it cannot be stated whether the disease was present before the birth of the patient. In the majority of cases it is probable that at the patient's birth the parent was non-phthysical.

To sum up the preceding remarks, we see that if we take 1000 cases of consumption, we get a history of phthisis in one or both parents in 325 cases, or 32·5 per cent.

We see also that in families of which one or both parents are phthysical, 33·71 per cent. of the children suffer from the disease; whereas in families with no consumption in the parents, 24·87 per cent. of the children become

phthisical. Phthisis in the mother results in a higher percentage of phthisis in the children than when the father alone is phthisical. This might be expected, without conceding any hereditary influence, from the more intimate relation between mother and children. When both parents are phthisical 39·45 per cent. of the children become consumptive.

It seems, then, that the influence of heredity may be represented by about 9 per cent.—the difference between the percentage of phthisis in the children of non-phthisical parents and those whose parents were consumptive. Even when both parents were phthisical the influence of heredity cannot be seen to result in more than fifteen children in every hundred becoming phthisical.

I have collected the following figures from various sources, so as to get the percentage in which phthisis occurred in the parents of consumptive patients from a sufficiently large number of cases.

		Cases.		Percentage heredity.		Number of cases in which the parents were phthisical.
Brompton (1st report)	...	1010	...	24	...	242
„ (2nd report)	...	4482	...	28	...	1255
Edward Smith	...	1000	...	21	...	210
Wilson Fox	...	884	...	33	...	127
Theodore Williams	...	1000	...	12	...	120
Philip...	...	1000	...	23	...	230
Müller	...	988	...	21·8	...	215
Koranyi	...	900	...	20	...	180
Wahl	...	745	...	26·4	...	197
My own cases	...	1000	...	32·5	...	325
Total	...	12,509	...	24·79	...	3101

We have here 12,509 cases, of which there was phthisis in one or both parents in 3101 instances, or 24·79 per cent. We may take it from the above figures that 25 per cent. is as a fair average for the extent of apparent heredity from parents.

Naturally when phthisical grand-parents and collaterals

are included we have a larger percentage of apparent heredity :

				Cases.			Percentage heredity.			Number of cases in which the parents were phthisical.
Brompton (2nd report)				...	4482	...	87	...		3899
Edward Smith				...	1000	...	56.2	...		562
Theodore Williams				...	1000	...	48	...		480
Wilson Fox				...	384	...	48	...		184
Cotton				...	1000	...	36	...		360
Müller				...	988	...	50.4	...		498
Bochdalek				...	3292	...	48	...		1580
Total				...	12,146	...	62.34	...		7563

The only comment I have to make on this second list is that the figures seem to me quite unreliable (especially as regards the Brompton report), for all who have much to do with London out-patients know how wide a term "consumption" is when employed by them in their family histories; it includes, amongst other diseases, bronchitis, asthma, anæmia, and alcoholism.

I have previously pointed out that we have only to carry the inquiry sufficiently far back in order to bring up the apparent proportion of heredity to 100 per cent.

We may note in the first of the above lists how closely the percentage of apparent heredity in Dr. Wilson Fox's cases coincides with my own; in both of these series the patients were drawn from the same class of people, chiefly London poor living under unhygienic conditions.¹

It is also worthy of note that in the cases gathered from the London hospitals the percentage of apparent heredity is in excess of that for the total number of cases here shown. This might be taken to indicate that where the people are living under conditions favorable to the development of phthisis the apparent influence of heredity is greater than elsewhere. To my mind it tends rather to

¹ Having been house physician to Dr. Fox at University College Hospital, I am able to make this statement with confidence.—J. E. S.

show that mode of life and unhygienic surroundings, not inheritance, caused the disease in the children as in the parents.

The percentage of phthisis in the children of *non-phthisical* parents amongst my patients is as great as that representing the hereditary influence in the above list; this, again, tends towards the conclusion that the value of the influence of occupation and surroundings in the causation of phthisis is very much greater than that of any inherited tendency. The relative value, as a cause of phthisis, between heredity and the influence of occupation and surroundings is well shown in the difference between the percentage of cases where the parents were phthisical in Dr. Theodore Williams' 1000 cases from private practice and that of 1000 cases of hospital patients. Dr. Williams' cases give a proportion of apparent heredity of only 12 per cent. My hospital cases show a percentage of 32·5. Is not this almost sufficient in itself to negative the theory of direct heredity? For if hereditary influence were a potent factor in the causation of phthisis we should expect to find it showing nearly equally amongst the rich and the poor.

It has been stated that each sex has a tendency to transmit directly, fathers to sons, and mothers to daughters.

In the majority of cases, as far as I can gather, by which this statement has been supported, the figures are obtained by taking a single case in a family and finding out if either parent was consumptive. For example, a male patient presents himself with phthisis and states that his father was phthisical; without further inquiry this might lead to error, for he might have two sisters phthisical, and be the only consumptive son out of several in the family. To show that this way of taking the manner of transmission would lead to such an error, I have worked out this point in the way I have above alluded to for some of my cases. In 500 male patients the father was phthisical in forty-eight cases, the mother

in forty-six cases, and both parents in eighteen cases—showing transmission from father to son to be slightly in excess of cross-transmission, instead of as seen below.

By getting the history of all the children of a phthysical parent, as I have done in the present series of cases, we get a far more reliable result. It will be seen (Table II) that my figures are directly opposed to the theory of "direct" transmission.

The figures are—Father to sons	. 27·19 per cent.
Father to daughters	. 35·18 per cent.
Mother to sons	. 39·43 per cent.
Mother to daughters	29·95 per cent.

This shows a cross-transmission, as do also the figures arrived at by Dr. Wilson Fox from his 384 cases.

In order to see that my figures might not be misleading from dealing with an undue proportion of one sex amongst my cases, I have compared the proportion of males to females in my 1000 cases with the known proportion between the sexes amongst phthysical patients. I find my 1000 cases to consist of 604 males and 396 females. In 1000 cases recorded by Dr. Theodore Williams there were 625 males and 375 females.

The first Brompton report gives a proportion of 61 males and 39 females per cent.

The mortality from phthisis in London in 1880 gives for every 100 deaths, 57 males and 43 females.

Finally, amongst the 737 children in my tables who were phthysical, I find 418 males and 319 females, giving a proportion of 56·72 males and 43·28 females per cent. The proportion between the sexes in the 1000 cases dealt with in this paper is, therefore, about the relative proportion in which the two sexes are attacked with phthisis.

Of the 474 cases from which my tables as to children are drawn up, 309 were male patients and 165 female, a proportion of 65·19 males and 34·81 females per cent., or, again, not a long way from the proper relative proportion between the sexes.

CONCLUSION.

The analysis of these cases warrants the opinion that the hereditary predisposition in the case of phthisis is not a direct predisposition to this malady, but merely the general predisposition to disease which belongs to the offspring of weakly parents; a predisposition which the children of phthisical parents have in common with those of parents whose health is lowered by other conditions. The families of consumptives have, however, the disadvantage, not shared by the delicate children of non-phthisical parents, of living in constant association with a source of infection. They have a general predisposition to take any disease to the infection of which they may be exposed, and being constantly exposed to the infection of tuberculosis they are prone to become consumptive. This circumstance may well account for these families showing an incidence of phthisis only 9 per cent. in excess of the incidence of the disease amongst the children of non-phthisical but weakly parents.

I believe that the present criticism of the influence of heredity by the examination of the total number of children of one generation in a phthisical family deals with a larger number of cases than has as yet been published.

The results of the examination of the figures may not go very far in deciding any of the various questions which surround or are included in the theory of inheritance in phthisis. The figures themselves may, however, be of some interest and value even to those who cannot agree with the deductions I have drawn from them.

STATISTICS.

Of 1000 phthisical patients 325 gave a history of phthisis in one or both parents. Apparent heredity = 32·5 per cent. Male patients 604 ; apparent heredity 28·97. Female patients 396 ; apparent heredity 37·87.

TABLE I.—*Showing the incidence of phthisis in the children of non-phthisical and of phthisical parents respectively.*

474 CASES.	CHILDREN.		DIED IN INFANCY.		PHTHISICAL.	
	Total number.	Proportion to each family.	Total number.	Per cent. of whole.	Total number.	Per cent. of children, omitting infantile deaths.
A. 275 families. With <i>no phthisis</i> in parents }	1745	6·34	193	11·06	386	24·87
B. 84 families. <i>Fathers phthisical</i> }	511	6·08	67	13·11	138	31·8
C. 82 families. <i>Mothers phthisical</i> }	506	6·17	56	11·06	155	34·4
D. 33 families. <i>Both parents phthisical</i> }	165	5·0	18	10·9	58	39·45
199 families. Phthisis in one or both parents (Summary of B, C, D) }	1182	5·93	141	11·92	351	33·71

TABLE II.

APPARENT HEREDITY IN 474 CASES.	SEX OF CHIL- DREN.	CHILDREN.		DIED IN INFANCY.		PHTHISICAL.	
		Total number.	Proportion to each family.	Total number.	Percentage of whole.	Total number.	Percentage of children, omitting infantile deaths.
A. Nil. 275 families	M.	947	3.44	111	11.87	244	29.06
	F.	798	2.90	82	10.41	142	19.81
B. Paternal. 84 families	M.	267	3.05	39	14.60	62	27.19
	F.	204	2.90	28	33.3	76	35.18
C. Maternal. 82 families	M.	246	3.00	33	13.41	84	39.43
	F.	260	3.17	23	28.04	71	29.95
D. Double. 33 families	M.	84	2.54	12	14.28	28	38.8
	F.	81	2.45	6	7.47	30	40.0
Summary of B, C, D. 199 families	M.	597	3.00	84	14.07	174	33.91
	F.	585	2.93	57	9.74	177	33.52

TABLE III.—*Showing a further analysis of 204 (from Cases A in the previous table) in which the history of grandparents, uncles, and aunts could be determined.*

204 CASES WITH NO PHTHISIS IN THE PARENTS.	SEX OF CHIL- DREN.	CHILDREN.		DIED IN INFANCY.		PHTHISICAL.	
		Total number.	Proportion to each family.	Total number.	Percentage of whole.	Total number.	Percentage of whole, omitting infantile deaths.
179 cases with no phthisis previously known in the family	M.	581	3.24	70	12.04	142	27.78
	F.	522	2.91	62	11.87	86	18.69
25 cases, phthisis in grandparents or collaterals . .	M.	84	3.36	4	4.76	13	16.25
	F.	89	3.56	4	4.49	19	22.35

Table showing the proportion of phthysical children to 100 non-phthysical in 80 cases from Dr. R. Thompson's table of heredity (p. 45 of his book), compared with a similar proportion in 199 cases of my own with phthisis in one or both parents. (See totals in the first two tables.)

		Phthysical.	Non-phthysical.
Dr. R. Thompson.	{ Males	116.1	100
	{ Females	137.14	100
My own cases	{ Males	51.32	100
	{ Including all infantile deaths	76.10	100
	{ Females	50.42	100
	{ Including all infantile deaths	66.6	100

Table showing the number of males and females who developed phthisis out of 474 families, in 199 of which heredity was suspected.¹

Heredity.		Phthysical.	Died in childhood.	Non-phthysical.
Nil 275 .	{ Males	244	111	592
	{ Females	142	82	574
Paternal, 84 .	{ Males	62	39	166
	{ Females	76	28	140
Maternal, 82 .	{ Males	84	33	129
	{ Females	71	23	166
Double, 33 .	{ Males	28	12	44
	{ Females	30	6	45
Total of 199 hereditary cases	{ Males	174	84	339
	{ Females	177	57	351

Table showing the number of males and females who developed phthisis out of 80 families in whom heredity was suspected (from Dr. R. Thompson).

Heredity.		Phthysical.	Died in childhood.	Non-phthysical.
Paternal, 24 .	{ Males	20	2	17
	{ Females	30	5	19
Maternal, 30 .	{ Males	38	9	55
	{ Females	34	4	40
Double, 14 .	{ Males	17	9	8
	{ Females	30	5	8
Atavism, 12 .	{ Males	23	1	4
	{ Females	15	2	3
Total (80 hereditary cases)	{ Males	98	21	84
	{ Females	96	16	70

¹ For comparison with Dr. R. Thompson's Table III, p. 45 of his work.

² Table III of Dr. R. E. Thompson (p. 45).

**(For report of the discussion on this paper, see ‘ Proceedings of the
Royal Medical and Chirurgical Society,’ Third Series, vol. vii, p. 29.)**