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Can the causal model approach contribute to the study of the epidemiology and the control of sleeping sickness?

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1. Introduction

In spite of sustained efforts at reducing African trypanosomiasis, foci of the disease remain active, and quite a number of foci that were believed to be extinct are reviving. The maintenance or recrudescence of trypanosomiasis foci depends on a large number of factors, some of which are well known - even if their role is not always clearly quantified - while other factors are as yet unknown (9, 11, 13, 17, 18, 19).

Since it is impossible to include all known or presumed factors in an epidemiometric model*, the builders of such models have the choice between either (1) discarding factors which they consider less important, (2) aggregating the variables that represent certain factors into composite parameters which will eventually appear in the mathematical model, (3) ignoring potentially important factors, or (4) combining two or more of these possibilities.

It may happen in certain situations that a factor, which was not taken into account, is nevertheless important in explaining the maintenance of the phenomenon of interest (persistence of transmission, failure of control, etc.) Also the different variables that constitute a composite parameter may vary unconsistently, or even in opposite directions, thereby concealing the real meaning of the parameter value.

In the building of mathematical models of an epidemiological phenomenon there also seems to be an inherent risk of committing logical and/or semantic errors and of omitting important confounding factors, which may affect the validity of the model.

It is one of the basic hypotheses of this paper that the chances of such events occurring unnoticedly can be reduced if the building of a mathematical model is preceded by a previous step, that is an exhaustive causal analysis of the phenomenon of interest.

Actually the building of epidemiometric models is necessarily based on a set of causal hypotheses that are not always explicit. The approach proposed below leads to a systematic enunciation of all causal hypotheses taken into consideration, and their linking and hierarchizing in a logical and easily understandable manner.

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* For definitions, please see the "glossary", page 15

2. The causal analysis

The causal analysis, as described in this paper, proceeds through the following steps: (A) the establishment, by a group of experts in all fields related to the study of the transmission and control of tryponosomiasis, of an exhaustive list of all factors known or presumed to play a role, (B) the identification of causal chains, that is of sets of hypotheses linking two or more (real or presumed) variables, and the ordering and combination of such chains into a causal framework.

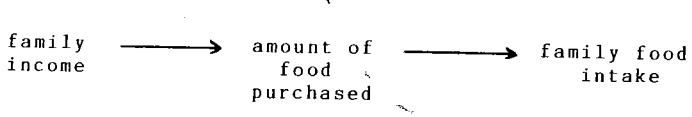
The technique of modelbuilding obeys simple and well-tested rules and conventions (4). Operationally it consists of a series of brainstorming sessions during which the model is progressively built.

Nutritionists were among the first systematically to analyse causality and to build causal models presumably because the causes of malnutrition are so multiple and complex. This is why we shall use examples from the nutrition field to illustrate, in a simplified manner, the procedures used to build causal models. An additional advantage of such an example is that no special subject matter knowledge is needed to understand the technique.

Initially, models of this type were developed for rationalizing the selection of variables to be used in nutritional assessment of populations (2, 3, 15). The method for constructing such models was further developed under the umbrella of GRIMM, an interdisciplinary research group in Belgium, an it is now used in an increasing variety of fields : epidemiology, health services utilization, demography, etc. (1). GRIMM stands for "Groupe de Recherche Interdisciplinaire sur la Malnutrition et la Mortalité" - See Beghin et al (3).

3. Building of a causal model

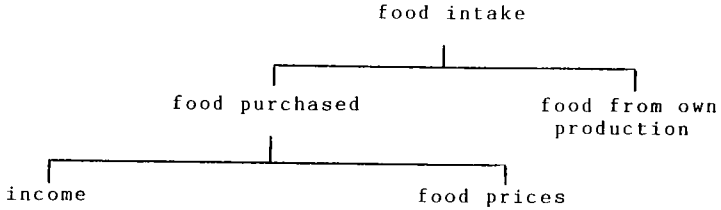
3.1. Procedure. The basic element is a causal chain :



This example can be read in two ways : "income influences the amount of food purchased, which in turn influences food intake", or in the passive form "food intake is influenced by the amount of food purchased, which in turn is influenced by income". Both manners are interchangeable for simple causal chains, but the passive form is the only one to be used when building a model.

Each link between two factors represents a causal hypothesis. Yet most factors are influenced by more than one determinant, and it would be more correct to read the example as follows : "food intake is influenced, among other factors, by the amount of food purchased, which in turn is influenced by income,

amongst other factors". The identification of the "other factors" is a major task of model building. Our example would then become



This is a simple causal model of food intake at the family level. It is a set of causal hypotheses, linking the variable of interest - food intake - to some of its determinants. The hypotheses are ranked in a hierarchical order, which is easily understood by the reader.

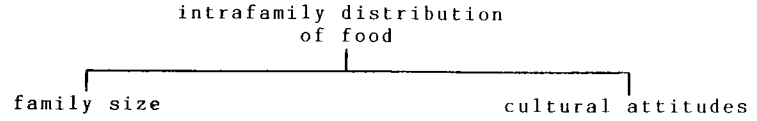
Another, and more accurate reading of the model is to state that "all the determinants of food intake can be grouped into two categories, those that influence the intake of food which was purchased, and those that influence the intake of food produced by the household. Factors affecting food purchases can in turn be divided into two large categories", and so on. The model is therefore a succession of submodels, imbricated like a Russian doll. Figure one is an example of a more elaborated model (Fig. no. 1, from Beghin et al, 1988, 4).

3.2. Rules. Building a causal model obeys a few simple rules :

(1) The construction is retrospective : it starts from the dependent variable. The construction goes against the flow of causality (from effect to most immediate cause), and is expressed in the passive form.

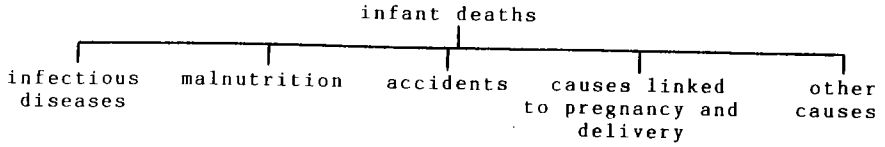
(2) Each box is broken down into at least two boxes. Decompositions are of two kinds (11)

- reflecting an influence relationship, attempting to provide an explanation : this is the case when the boxes in the line immediately below are true determinants. Example:

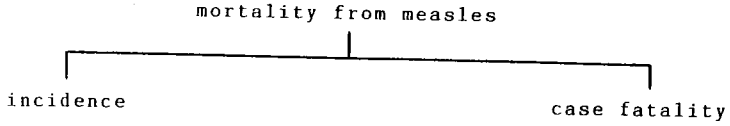


- decomposition in a logical sum or a logical product. This is merely a breakdown that enriches the information, and

guides towards the identification of more determinants. Example of a sum :



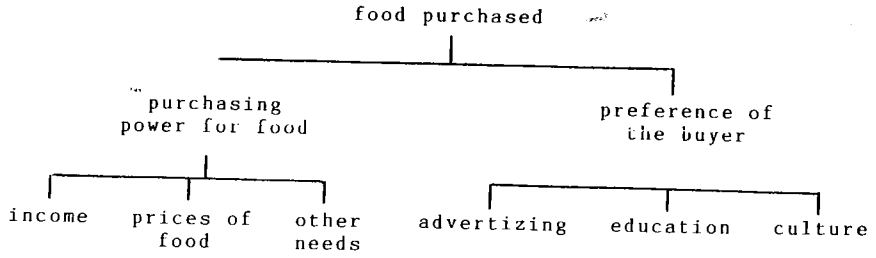
Example of a product :



It should be a mistake, however, to try to look systematically for sums and products. Product-like relations, or "quasi-products" are very common. Also common are a certain number of stereotype structures such as supply vs. demand; factors linked to the individual vs. environmental factors; propensity vs. possibility etc.

(3) The majority of boxes in the model can be reflected quantitatively through one or more indicators. There is however a category of boxes, containing "conceptual" factors, which are difficult to measure. They generally express a capacity or a propensity. Appetite is a good example.

(4) Efforts must be made to identify any factor that might be intermediate between two neighbouring boxes. It is an essential rule in the building of such models not to omit intermediary links. In the example above, an additional link between "family income" and "amount of food purchased" should be introduced, i.e. "purchasing power for food". The model would then become



A new set of potential determinants is identified in this manner, and new chains are built. The systematic search for intermediary links and their subsequent breakdown is the most effective way of recognizing confounding factors, and preventing omissions.

(5) If such search of intermediary links is not conducted properly, side branches, which may correspond to an important causal factor, might be ignored.

(6) Mutual influence of two factors on the same horizontal line, or influence of one of them on the other, is not represented in the model, by convention.

(7) Similarly, by convention, when a given factor acts at different places in the model, no lateral links are used in the graph, but the factor is preferably repeated.

(8) Feedback loops are not represented. This last point often causes surprise : let us remind the reader that a causal model is non-mathematical and non-systemic. The simplification consisting in eliminating horizontal and lateral links as well as feedback loops, is probably the most significant single characteristic of causal model building, and it probably also is the reason why such models find wide acceptance.

(9) A recurrent difficulty is that participants to the model building exercise tend to move from boxes at the individual level to aggregates, and vice versa. When analysing food intake, are we considering an individual or the group as a whole ? The decision must be made at the start, and the builders will have to stick to it.

(10) A compromise must be reached between rigorous logic and exhaustive incorporation of factors (which increases time consumption, aggravates complexity, and decreases the distinction between important and secondary factors) on the one hand, and parsimony on the other. There is a trade-off between a natural inclination towards completeness, and the advantages of a rather simple model, with not too many boxes, and easy to understand and to handle. The level of compromise will depend on the purpose. Judgment must be exercised throughout.

(11) In practice a model of this type must be designed by a group representing different disciplines, selected in such a manner that their aggregate fields of competence cover most or all areas of the phenomenon under study.

In actual practice, the exercise starts with the building of an exhaustive list of all factors known or presumed to play a role in the causality of the phenomenon. This listing exercise may be repeated, if necessary, whenever a new box is coming under scrutiny. The actual making of such a list is useful for preparing the actors, intellectually and psychologically, for the building exercise, and for eventual use as a checklist.

Then the building of the model can start. Experience has repeatedly shown the advantages of a series of brainstorming sessions over the lonely efforts of one scientist thinking in an office.

4. Uses of causal models. Pros and cons

The initial, and so far still widest application of causal models is the nutritional diagnosis or assessment of population groups. The model helps to identify relevant variables, thereby eliminating useless data collection, saving time, and allowing for faster analysis. It also provides the initial framework for data analysis and interpretation (4).

Other uses include the determination of the relevance of interventions to be selected in rural development or public health projects, evaluation of interventions, the study of health service utilization, etc. (1, 3, 8, 11, 16).

A particularly important and promising development is the use of such models as an organizational tool in the design of complex research programmes (6).

Yet one must be cautious and not expect more from those models than they can offer. Model builders and users (ideally the same individuals !) should at no moment lose sight of the fact that causal models, or conceptual frameworks as understood in this paper, are constructions which attempt to represent, in a rational and comprehensive manner, the complex and multiple mechanisms linking social, economic and cultural determinants to a biological phenomenon. They are nothing more than a set of hypotheses. It is the representation - as best as we can formulate it - of how we, at this moment and with our very incomplete knowledge, understand the phenomenon. It is subject to continuous improvement as new facts emerge, as postulated relations become better understood, or as new hypotheses are born from experience, observations, simulations or reasoning.

It has been objected against such models that they ignore the time dimension. Durations, flows, velocities, time lags, etc. are not clearly taken into account. The intensity of certain phenomena, or the relative strength of relations, are not represented although it is possible (20); the dynamics of the phenomenon is not sufficiently illustrated. Actually, such models are not meant to represent the dynamics of the phenomenon. They are not statistical or systemic models. They are a tool for organizing our thinking about mechanisms. Their major advantages - largely due to the simplifying assumptions of ignoring horizontal links and feedback loops, and by allowing repetitions of the same factor, are (1) to make explicit and comprehensive all our hypotheses about the mechanisms leading to the observed outcome, (2) to make this set of hypotheses easy to communicate, and(3) if the construction rules listed above are rigorously respected, to reduce the chances of omitting important factors and confounders (commonly of a behavioural or environmental nature). Basically

the model helps identify gaps in knowledge.

Another frequently-made objection is that model building is time consuming, fastidious, and too theoretical, and that it does not lead to practical results.

Model building, if done properly, is time consuming indeed (as a rule of the thumb, about five sessions of 2 to 3 hours are needed, spread over at least five days, occupying from 6 to 15 people, besides the time spent by a coordinator, on the content definition of boxes and links, which may be weeks or even months). Experience shows, at least in the case of nutritional assessment, that the time eventually saved in questionaire development, data collection and analysis, largely compensates for the initial investment. So do the communication and learning aspects. In our experience this criticism is seldom expressed by people who do have experience in a model building exercise.

A different kind of resistance, sometimes very strong, is more intellectual and/or ideological. A significant number of persons, who consider themselves pragmatists, disbelieve global approaches, resist intellectual constructs, give precedence to facts, look for concrete results, distrust theories. Ideologically, they are close to the supporters of "interventionist" approaches. Another category of people is unsympathetic to such type of analysis because it almost inevitably leads to questioning the established state of affairs, something they are not inclined to do.

This modeling approach is sometimes criticized as not valid, because it does not produce predictions. This model is not meant to be an alternative for mathematical ones. It can give insight in the choice of the variables when a mathematical model construction is considered. Without such a causal model construction the choice of the basic variables for a mathematical model might be more arbitrary and reflect more personal biases of the author.

Yet the major and real drawback of a causal model is that as such, it is not verifiable. To be verified (or "falsified", in Popper's sense) such model needs to undergo a "reduction" into a statistical model, as Palloni has suggested, (14). This point is taken in the next section.

5. Causal models and the epidemiology of infectious diseases

Whether causal models are useful for the study and control of infectious diseases is, so far, conjectural. Yet we have adopted the view that they are, as the central hypothesis of this paper.

The hypothesis can be formulated as follows :

"We would have better epidemiological models if we built them from an earlier causal model.

"Better" means (1) more correct, logically; (2) fitting reality more closely; and (3) more useful for the analyst, the decision maker and the field worker".

We believe three reasons justify the adoption of this hypothesis.

a) In the early 1980's, GRIMM, the Belgian group of students of malnutrition and mortality already mentioned, was identifying determinants of young child mortality. The group was faced with serious methodological difficulties in trying to build a causal model for infectious diseases. At that time the group reviewed a number of existing models, generally mathematical (1). It came up with two types of outcomes.

In the first place we arrived at a tentative break-down for infectious diseases relevant to young child mortality in developing countries (Figures 2 and 3 from Bartiaux et al, 1983, 1). A second outcome was the impression that logical flaws and semantic confusion sometimes existed in some of those models - for example the models proposed by Cvjetanovic et al (7) - that would have been avoided with a prior causal analysis. Because of other priorities and lack of funds this work remained unfinished.

b) In 1985, the Institute of Tropical Medicine and GRIMM organised an International Colloquium on Young Child Mortality (5). An American demographer, Alberto Palloni, who had been doing theoretical and practical work in the same direction, participated in this meeting. Palloni, in a dense and substantial contribution, (14), argued that a well-constructed and comprehensive conceptual model is necessary in all causative research. He further argued that such a model should be an intermediary step between theory and statistical models, and that statistical models not based on a thoroughly built causal model may at times serve to verify different theories. For Palloni, a conceptual model is a "reduction" of theory, and the statistical model a further reduction from the conceptual model - although the latter may be built in the absence of theory. This point was particularly interesting because it was made by a worker who had extensive experience in using statistical models.

c) More recently, two of us became involved in research in collaboration with the University of the Philippines at Los Banos, in testing the general hypothesis that this kind of modeling is of practical use in the evaluation of complex rural development projects. A second model was derived from the causal model : i.e. a set of hypotheses linking project inputs to outputs and outcomes. In other words the second model does not merely explain the natural history of malnutrition - the dependent variable - but also takes into account the interference of interventions. Still it is not yet a "statistical model" as understood by Palloni.

d) Two questions have been raised by Dr. P. De Raadt regarding the application of this approach to the study of infectious diseases*. Are there as many unknown variables in the study of an infectious disease as in the case of malnutrition ? And : the use of a model serves mainly to identify questions; do we really not know the questions, or is it the answers that we don't know ? (In the case of trypanosomiasis, the essential question is, so far, the amount of bites per person per day. In that case a modeling exercise would be useless).

On the other hand, if there still are unidentified questions, new questions to raise, or questions that are believed to be known, a model might be useful.

6. Relevance to the study and control of trypanosomiasis

In January 1988, a Colloquium on "Mathematical Models in the Study and Control of African Trypanosomiasis" called together in Antwerp a group of researchers and practitioners of African trypanosomiasis from a number of countries and disciplines. This meeting was called at the initiative of, and sponsored by TDR, the Tropical Diseases Research programme of WHO. Our contribution to that meeting was based on the two assumptions that (1) it might be worthwhile to try to build a causal model of trypanosomiasis, and (2) that it seemed that the causality of the disease was less complex than that of malnutrition. There probably also were fewer determinants, and maybe some of them could be reaggregated into a lesser number of variables.

Both assumptions were strengthened after a first brainstorming session, with a number of volunteer participants to that Colloquium, and more generally by the discussions and outcome of the meeting.

During the following months, further work was done in the group, with the participation of students and staff from the Institute of Tropical Medicine. The results will be presented in a forthcoming paper.

A first difficulty faced in attempting to build a causal model of trypanosomiasis was the precise identification of the dependent variable. A number of possibilities were initially considered :

(1) Prevalence of disease. The number of existing cases can be reduced by either lowering the incidence, or by shortening the duration of the disease through treatment, or by both. The problem, however, is that diseased persons are only a fraction of all the people who are sources of infection.

(2) Prevalence of infection, i.e. the sum of sick persons plus symptomless carriers, in other words, the "human reservoir".

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* P. De Raadt, 1988. Personal communication. See also references 9 and 13

(3) Incidence : dynamic. Is the basic parameter to assess the impact of the control efforts.

(4) New cases detected. This would depend on the incidence and on the effectiveness on detection, and could be an operationally suitable starting point. This parameter reflects better the transmission.

(5) Number of infective bites per person per day. This number represents the risk of real disease transmission.

Actually and whatever the choice of the dependent variable, if the model is comprehensive all other significant factors will inevitably appear in the course of model elaboration.

Since the major purpose is to be comprehensive and to avoid omitting any important factor, and since anyhow such models do not include feedback loops, it is more reasonable to proceed to build the model, to develop submodels, and eventually to select that dependent variable which, operationally, seems most suitable.

The actual serious limitations came from two basic reasons : gaps in our knowledge, and the inclusion of the time element.

a) Major gaps in knowledge : A major difficulty was our insufficient knowledge of what appeared, during the building exercise, as potentially important causal chains. Presumably significant determinants are insufficiently known (or at least insufficiently known by the group who did the exercise), particularly factors linked to the behaviour and to the environment. Some of such factors, it should be pointed out, had indeed been identified during the recent symposium. Examples are :

- perception of symptoms, attendance at screening sessions, attitude towards the disease, attitude towards the health providers
- determinants of such behaviours
- mobility of the population
- effective utilization of health services and the reasons therefore
- vector behaviour
- role of the animal reservoir
- land management and its effect on human behaviour, on mobility, and on the vector and animal reservoir
- determinants of "density dependence"
- etc.

b) Consideration of the time element : As already discussed above (in section 4) a causal model - certainly not in the present stage of development of this tool - does not satisfactorily express the time element. Yet, in the case of trypanosomiasis transmission and control, time is a crucial element, in at least three aspects

- As an element of flows and intensities. The latter are not, or are only poorly, reflected in a causal model, but would be essential in a mathematical model

- As an expression of duration. For example the time between onset and detection, or between infection and disease, or between detection and treatment (12) or between primary infection and late complications. The duration of the clinical disease may be unimportant as a determinant of the reservoir's size, but it may be significant as a factor affecting effectiveness of treatment, acceptance of treatment, chances of being detected, mobility, and other behavioural aspects of the epidemiology of trypanosomiasis. Further work is needed to know whether duration can be introduced into a causal model of this kind.

- Because of the dynamics of the system : some determinants change over time (infectivity, perception of the disease, population density, etc., either spontaneously or as the result of interventions).

The phenomenon of transmission - which did not exist in our example of malnutrition - adds an important aspect, and an additional difficulty, to use such models for a disease such as African trypanosomiasis.

7. Directions for the future

Work done so far suggests two alternatives for the near future :

- a "minimalist" procedure consisting in continuing the elaboration of a causal model; then reducing it - or parts of it - to statistical models using secondary or primary data; improving the causal model; etc. Such a procedure would verify (or "falsify") parts of the causal model, and therefore help improving it.

- a "maximalist" procedure, which would end up with an operational epidemiometric model of trypanosomiasis.

7.1. The minimalist alternative

In broad terms, it would consist in pursuing the present efforts. Three priority aspects are clear

(1) to explore alternative points of departure, corresponding to alternative uses of the causal model

(2) to do more work on selected submodels through looking into the literature and consulting with specialists in the submodel subject areas.

(3) to reduce the causal model or selected submodels to

statistical models, as indicated by Palloni (14)

It is strongly recommended that the construction of any such submodel should start with the listing of all known determinants of the dependent variable which is at the top of the submodel. Then, as new facts become available, from observations suggested by the model or from other sources, the model would be periodically adjusted and improved.

The output objective would then be a model that would be

- rigorous, logical and comprehensive
- accepted by all participants
- easily understandable by non-participants

The more general outcome objective would be the identification of new research priorities. Indeed, one of our hypotheses - which will be developed and illustrated in another paper - is that causal models can be used in identifying priority topics in a broad research programme about a complex problem, among which epidemiology and control of trypanosmiasis is one.

According to the minimalist procedure, we consider that the model, AND the very process of building it within an expert group, would at least

- provide new insights into some of the subtle relationships between factors
- identify new or hitherto neglected potential determinants
- clarify the meaning - and sometimes refine the semantic and logical accuracy - of some terms, definitions or relationships
- point to areas of insufficient knowledge
- more generally organize and integrate information generated by different disciplines.

In practice, the "minimalist" alternative would result in partial verification of fragments of the causal model with data from the literature or from new research to be undertaken in improving the causal model, and at the same time increasing relevant knowledge through an iterative process. In a note commenting on causal models designed to study the compliance of antenatal care by migrant women in Belgium (8), Wilson* suggests using cross tabulations to study the relevance of the boxes (and of the eventual questionnaires) approach and makes a number practical suggestions, to move from a conceptual to a statistical model.

He also suggests that such simplification would pave the way for an eventual, later move to mathematical modeling.

7.2. The maximalist alternative

Ideally, after getting at a reasonably satisfactory causal model, having identified most relevant determinants and potential

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* Wilson, 1988. Personal communication

confounding factors, and having enough knowledge of the nature and characteristics of the relationships between such factors, one would move towards building a useful mathematical model. Such a model would argueably in the first place be verifiable, and in the second place enable us to quantify our understanding of the epidemiology of trypanosomiasis, and to predict in an operationally useable way the effect of interventions on the maintenance or control of the disease.

This hypothesis, so far, is purely conjectural. Two crucial issues remain completely unanswered at this moment.

In the first place it is not proven that the prior building of a causal model would indeed assist in the preparation of mathematical models or in the improvement of existing ones. Possibly it is merely an elegant, intellectually attractive, yet costly exercise in relation to the time invested in it. Secondly, even if the output would be the right choice of variables and parameters to put into an acceptable mathematical model of trypanosomiasis, we still don't know whether it would be of real use in the effective control of the disease in Africa. We must face the fact that previous experience with earlier epidemiometric models does not leave room for excessive optimism.

In either of the two alternatives, whatever the output of the exercise, either a verifiable statistical model (or set of partial submodels), or a genuine mathematical model, a field-testing stage would be needed, such testing would assess, in varying degrees,

- the relevance, accuracy and predictive role of either model
- its practical usefulness as perceived by decision makers, evaluators, and/or research scientists.

8. Conclusion

Causal models do not, and cannot be expected to, substitute for mathematical models. In this paper such models are seen not as an alternative to mathematical models, but as a prior step towards the building or improvement of mathematical models. It seems that, in any case, they would help into understand better the meaning and implications of mathematical models.

In the opinion of the authors the preliminary work reported here should encourage students of African trypanosomiasis to build ad hoc causal models or parts of models, adapted to either the particular disease focus they are studying, or to the specific problem they are trying to solve, with good chances that such an exercise would provide them with new insights into the mechanisms of transmission and of its interruption, suggest to them relevant topics for research, assist them in reanalyzing existing data, and generally increase communication between places and between disciplines.

Acknowledgments

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Glossary of terms as used in this paper

Causal model : A simplified representation of the complex multicausality of a phenomenon. More specifically, a set of hypotheses about the causes of, and the mechanisms leading to a given phenomenon, linked together in an orderly and hierarchized manner.

Density dependence : The influence of the infection rate of the vertebrate host on the infection rate of the vector, or vice versa.

Dependent variable : A variable, the value of which is dependent on the effect of other variables (independent variables) in the relationship under study. A manifestation or outcome whose variation we seek to explain or account for by the influence of independent variables.

Epidemiometric model : Mathematical model of an epidemiological phenomenon.

Falsifiable : That can be shown to be false. In Popper's view, knowledge is being built more by demonstrating propositions to be false, and eliminating them, and than by demonstrating propositions to be true. Statistically analysis is systematically used to discard, through "falsification" the hypothesis that is being tested.

Indicator : a quantitative expression reflecting a selected aspect of the phenomenon under study. In a small number of cases a measurement of the phenomenon itself.

Mathematical model : A representation of a system in mathematical form, in which equations are used to simulate the (dynamics of the behaviour of the system under study.

Systemic model : A didactic, simplified representation of a process (for example the set of actions and interactions determining the value of the dependant variable), in which the process is seen as a system.

System : A set of (1) elements linked to each other through functional relationships, and (2) the relationships themselves.

References

1. Bartiaux, F., I. Beghin, I. Borlée, P.M. Boulanger, G. Masuy-Stroobant, D. Nzita, M. Sala-Diakanda, D. Tabutin, M. Vanderveken, W. Van Lerberghe, J. Vuylsteke. La mortalité aux jeunes âges : un essai d'approche explicative interdisciplinaire. In : Programme de recherche coopérative intercentres, projet no. 1, rapport final, pp. 161-176, CICRED-ARSOM, Paris, 1983

2. Beghin, I. Emploi d'un modèle causal simple dans le diagnostic de la situation nutritionnelle au niveau d'un pays. Académie Royale des Sciences d'Outremer de Belgique, séance du 27 février 1979, compte rendu de l'ARSOM, p. 244, 1979

3. Beghin, I. L'approche causale en nutrition. In : Lemonnier, D. & Y. Ingelbeeck. La malnutrition dans les pays du Tiers-Monde, INSERM, Série Colloque 1986, 136:615-629, 1986

4. Beghin, I., M. Cap et B. Dujardin. Nutritional Assessment Guide. OMS, 1988 (in press)

5. Beghin, I., Masuy-Stroobant, D. Tabutin & W. Van Lerberghe (Eds.), Santé et mortalité aux jeunes âges dans les pays en voie de développement - Health and mortality of young children in developing countries. Colloque international/International Colloquium. Ann. Soc. belge Méd. trop., 67, suppl. 1, pp. 142, 1987

6. Beghin, I. Comments on the paper by A. Palloni : Theory, analytical framework and causal approach in the study of mortality at young ages in developing countries. In : Beghin et al, pp. 47-50, 1987

7. Cvjetanovic, B. and K. Uemura. Dynamics of acute bacterial diseases. Bull. WHO, 56 (suppl. 1), 1978

8. Da Silveira, V.C., A. De Muynck, C. Timmerman and P. Van der Stuyft. Development and uses of a conceptual model in the study of antenatal services utilization by migrant women in Belgium. Health and Community, Working Paper no. 19, Institute of Tropical Medicine, Antwerp, 1988

9. De Raadt, P. Lutte intégrée contre la maladie du sommeil. Dans Cavalloro, R. (ed.). Integrated tsetse fly control : methods and strategies. Proceedings of the CBC international Sÿmposium ISPRA, 1987

10. Jordan, A.M. Trypanosomiasis control and African rural development. Langman. London, 1986

11. Masuy-Stroobant, G. et D. Tabutin. L'approche explicative en matière de mortalité des enfants. Réflexions et perspectives, Genus, 38(3-4), p. 19-37, 1982

12. Mercenier, P. Potentiel des services de santé polyvalents pour la détection des malades trypanosés. Ann. Soc. belge Méd. trop., 57:323-329, 1977

13. Molyneux, D.H., De Raadt, P. and Seed, J.R. African human trypanosomiasis. Recent advances in Tropical Medicine, ed. H.M. Gills, Churchill Livingstone. London, 1984

14. Palloni, A. Theory, analytical frameworks and causal approach in the study of mortality of young ages in developing countries. Ann. Soc. belge Med. trop., 67 (supplement), 1987

15. Pradilla, A., I. Beghin, J. del Canto, V. Bent and M.T. Menchu. Modelos interpretativos para la seleccion de prioridades en nutricion (nivel nacional). Arch. Latinoamer. Nutr. 27(2):104, 1977 (Suppl. No. 1)

16. Van Lerberghe, W. and I. Beghin. The evaluation of large scale health and nutrition projects : an outline of a methodology. A report to WHO. 19 pp. mimeo. Institute of Tropical Medicine, Antwerp, May 1985

17. Wéry, M. Conclusions au Colloque international sur la trypanosomiase humaine africaine. Ann.Soc.belge Med. trop., 57: 533-538, 1977

18. Wéry, M., D. Le Ray and A. Makumyavira M'Pondi. La maladie du sommeil : un défi authentiquement africain. Revue Zool. afr. 100 : 121-135, 1986

19. WHO Expert Committee. Epidemiology and control of African Trypanosomiasis. Technical Report Series, no. 739, 1986

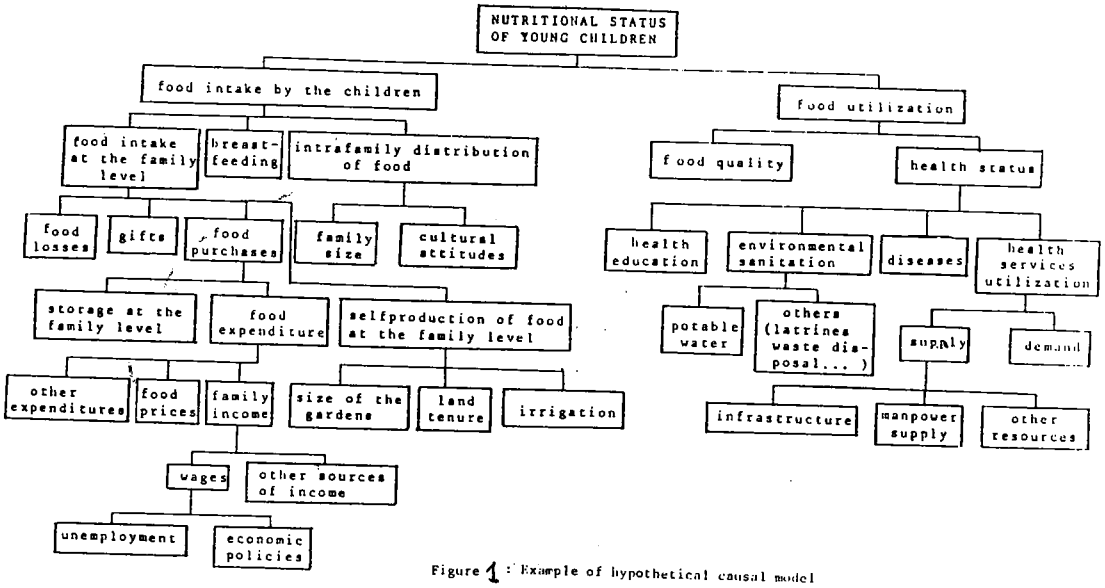
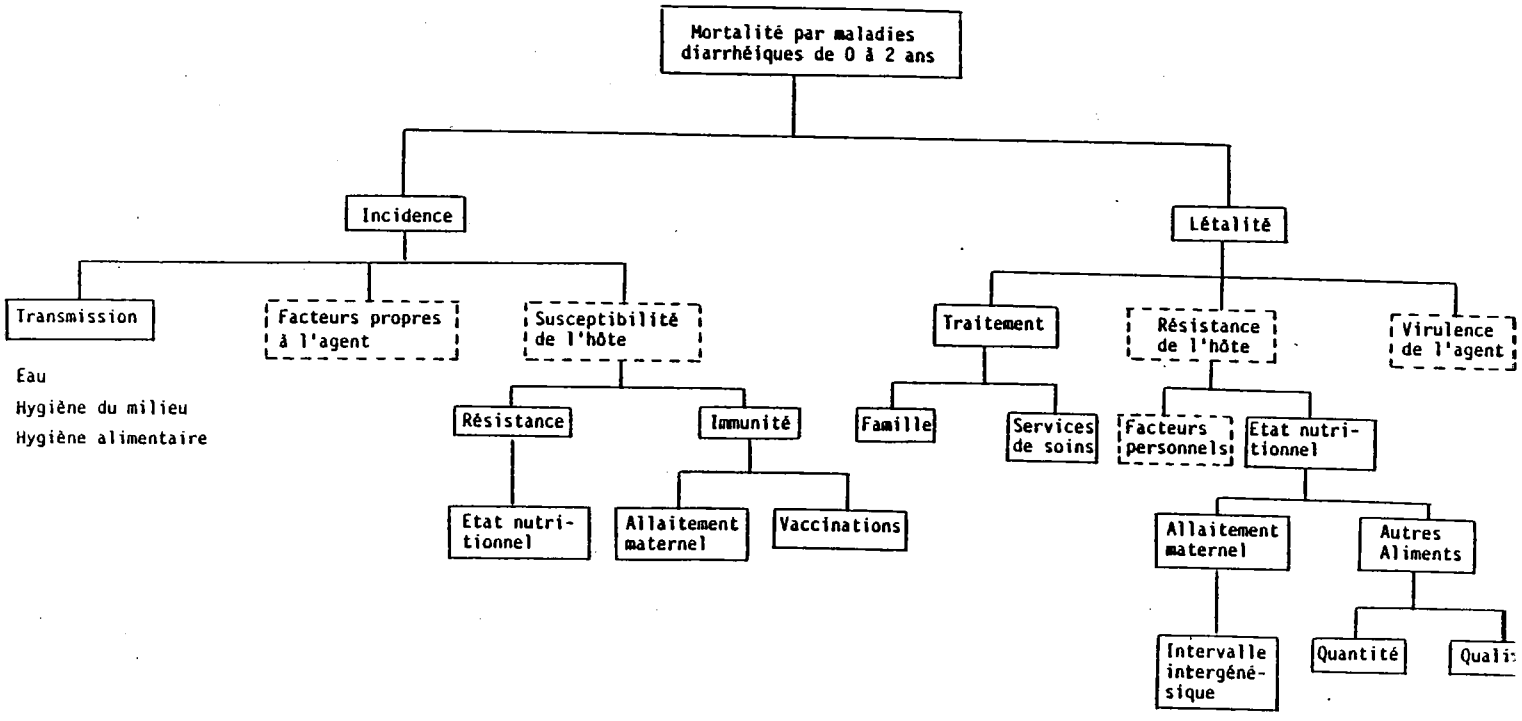


Figure 1 : Example of hypothetical causal model



- en traits pleins : variables mesurables et retenues
- en traits pointillés : variables non mesurables et non pertinentes dans notre cas

Figure 2 : Exemple d'utilisation du cadre analytique général pour la construction

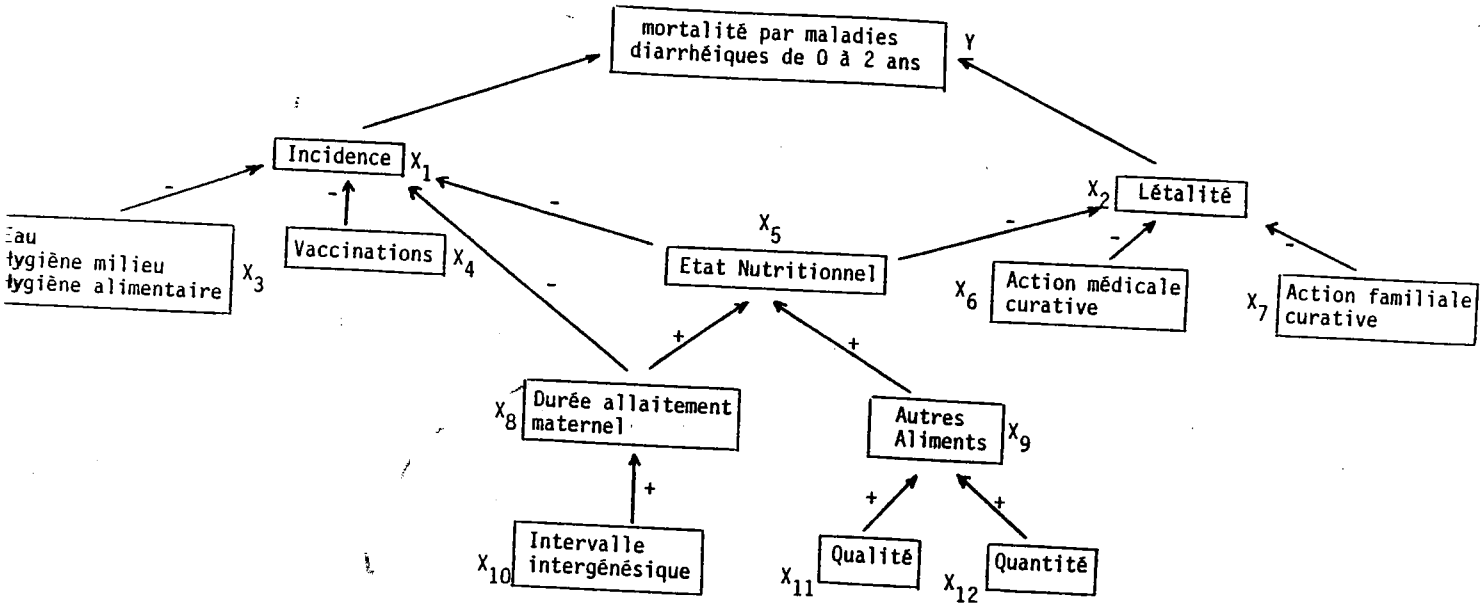


Figure 3 : exemple de modèle de mortalité par maladies diarrhéiques