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RECURSIVE *VS.* NONRECURSIVE SYSTEMS: AN ATTEMPT AT SYNTHESIS

(PART I OF A TRIPTYCH ON CAUSAL CHAIN SYSTEMS)

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This paper, which in part serves as a common introduction to the two papers following in this issue, attempts to define the meaning of the "causal interpretability of a parameter" in a system of simultaneous linear relationships. It attempts, moreover, to expound a basis for interpreting the parameters of a nonrecursive or interdependent system causally. This is done in terms of an underlying causal chain system to which the interdependent system is either an approximation or a description of the equilibrium state.

OVER THE PAST fifteen years there has been an extended discussion of the meaning and applicability of nonrecursive as distinct from recursive systems in econometrics, and throughout this discussion there has been a marked divergence of views as to the merits of the two types of models. It is not the purpose of this note to extend that controversy further, but rather to attempt a constructive statement of the relationship between the two approaches and the circumstances under which each is applicable.

We assume that the reader is generally familiar with the past discussion[1] and that it will suffice here simply to recall that a recursive, or causal-chain, system has the formal property that the coefficient matrix of the non-lagged endogenous variables is triangular (upon suitable ordering of rows and columns) whereas a nonrecursive, or interdependent, system is one for which this is not the case. While the triangularity of the coefficient matrix is a formal property of recursive models, the essential property is that each relation is provided a causal interpretation in the sense of a stimulus-response relationship. The question of whether and in what sense nonrecursive systems allow a causal interpretation is the main theme of this paper.

1. FUNDAMENTAL PRINCIPLES

Much controversy can, in our opinion, be resolved once there is agreement on some initial points of principle.

(1) The first thing to consider when constructing an economic model is its purpose, that is, how it is to be applied in dealing with economic facts. We want to distinguish in this connection between descriptive and explanatory models. A descriptive model simply sets forth a set of relationships which have "bound together" different variables in situations in which they have

[1] See references appended at end. An extensive bibliography is included in [**8**].

previously been observed. More generally, these relationships may be described in probability terms, certain terms in these relationships representing the "disturbances" which in fact occurred. One can in this way describe given observations as a random drawing from a joint conditional probability distribution. Methodologically, the estimation of such a distribution is an exercise in n-dimensional "curve fitting." A descriptive model is thus cognate to the notion of a vector *function* such as (in the linear case)

$$\mathrm{A}x' = u' \tag{1}$$

where A is a (not necessarily square) matrix of constants, x' is a column vector of the variables in question, and u' is a vector of zeros in the exact case or of stochastic variables in the case of a probability model. Whatever the validity of such a specification, the validity of any other model obtained by applying any linear transformation is the same. If a priori restrictions are imposed upon the sort of distribution which is to be used for this descriptive model, this may, of course, circumscribe the acceptable transformations.

Explanatory models, by contrast, are causal. This means that each relation (equation) in the model states something about "directions of influence" among the variables. (But see Section 3(b) below.) In the case of explanatory models, then, the theorist asserts more than functional relationships among the variables; he also invests those relationships with a special interpretation, that is, with a causal interpretation. But what is a "causal interpretation" to mean?

(2) No one has monopoly rights in defining "causality." The term is in common parlance and the only meaningful challenge is that of providing an explication of it. No explication need be unique, and some may prefer never to use the word at all. For us, however, the word in common scientific and statistical-inference usage has the following general meaning.[2] z is a cause of y if, by hypothesis, it is or "would be" possible by *controlling* z indirectly to control y, at least stochastically. But it may or may not be possible by controlling y indirectly to control z. A causal relation is therefore in essence asymmetric, in that in any instance of its realization it is asymmetric. Only in special cases may it be reversible and symmetric in a causal sense. These are the cases in which sometimes a controlled change in z may cause a change in y and at other times a controlled change in y may cause a change in z, but y and z cannot both be subjected to simultaneous controlled changes independently of one another without the causal relationship between them being violated.

The asymmetry of causation in any instance of its realization has the following probability counterpart. It may make sense to talk about the

[2] H. O. A. Wold has elaborated his views in [**6**, **7**].

probability distribution of y as being *causally conditional* on z, but not make sense to talk about the probability distribution of z as being *causally conditional* on y. This asymmetry is classical in statistical theory. It appears in the difference between a sample statistic and a population parameter. We speak of the probability that the sample frequency of successes will be 0.5 conditional upon the population frequency being 0.4. We do not speak of the probability that the population frequency is 0.4 conditional upon the sample frequency being 0.5.[3] Thus if we wish to estimate (by the maximum likelihood method) a population parameter knowing a sample of observations, we write the likelihood function as the conditional probability distribution of the *sample.*

Suppose we were to estimate by the maximum likelihood method the nth value of a causal variable (population parameter) $z(n)$ on the basis of the nth value of a resultant variable (a sample observation) $y(n)$ by use of a regression fitted to $n-1$ previous observations in all of which z has been causal. We should use the regression of y on z—not of z on y—over the previous observations, although this point is occasionally misunderstood.[4] Causality as used here is an essential notion in the statistical inference of population parameters by the maximum likelihood method. We must hypothesize how the sample observations are *generated* (i.e., caused) in order to proceed.

The concept of causality presented here is intended to be that of the everyday usage in the laboratory and emphasizes mainly the notion of control. Now, others may present a different explication of causality. Other versions may involve strange and seemingly unnatural notions, two of which are of particular interest to us. (a) The first involves accepting simultaneously the two statements: (1) "y has the value 100 because of (by cause of) z having the value 50" and (2) "z has the value 50 because of (by cause of) y having the value of 100." (b) The second involves accepting simultaneously the two statements: (1) "z causes y in accordance with the function $y = f(z)$" and (2) "z causes y in accordance with the function $y = g(z) \neq f(z)$." Usage (a) we shall describe as a "causal circle" and discuss in Section 3(c). Usage (b) we describe as "bicausality" and discuss in Section 3(d).

Whether such notions of causality seem weird or not, whether or not

[3] "Probability" is used here in the "relative frequency," not in the "degree of belief" sense.

[4] For what we regard as the correct treatment, see A. Mood, [**2**, Sec. 13.4]. For the contrary view see F. V. Waugh [**5**]. A qualification is needed: one must not have any constraining a priori knowledge about the possible values of z, or, if z is itself a random variable, about its probability distribution. The model we have in mind is given by $y = \alpha + \beta z + u$, with u and z statistically independent. Otherwise, the likelihood function is $f(y(1), \ldots, y(n)|z(1), \ldots z(n)) \cdot g(z(n))$, rather than the f function alone, where f and g are probability density functions.

they conform to usage in the scientific workshop, there is nothing to prevent their use in theory construction. Argument for the interpretation of causality presented by us is not an argument for "strait-jacketing" the freedom of the theorist and econometrician to use other interpretations. But we would (and will) argue that the notion of causality in economics is the one we have presented. Examples are: "Income is the cause of consumer expenditure (the consumption function)," "Price is a cause of quantity demanded," and "Price is a cause of quantity supplied." Most of the problems in assessing this claim arise in equilibrium models.

2. THE CAUSAL INTERPRETATION OF A RECURSIVE SYSTEM

It was not our purpose in the previous section to provide a precise definition of "causation." The term enters our discussion essentially as a "primitive," and what efforts we have made at definition have been ostensive: we have pointed to the familiar usage of the word in the laboratory. With reference to this primitive meaning of "causation," however, we wish to define the concept of "the causal interpretability of a parameter." This is what will occupy us next.

Suppose a recursive system is written in the form

$$
(2)\qquad
\begin{aligned}
y_1 + \beta_{11}y_2 + \dots + \beta_{1g}y_g + \beta_{1,g+1}y_{g+1} + \dots + \beta_{1G}y_G + \sum_k \gamma_{1k}z_k &= u_1\,,\\
y_2 + \dots + \beta_{2g}y_g + \beta_{2,g+1}y_{g+1} + \dots + \beta_{2G}y_G + \sum_k \gamma_{2k}z_k &= u_2\,,\\
\dots\dots\dots\dots\dots\dots\dots\dots\dots\dots\dots\dots\quad&\\
y_g + \beta_{g,g+1}y_{g+1} + \dots + \beta_{gG}y_G + \sum_k \gamma_{gk}z_k &= u_g\,,\\
\dots\dots\dots\dots\dots\dots\dots\dots\dots\dots\dots\dots\quad&\\
y_G + \sum_k \gamma_{Gk}z_k &= u_G\,,
\end{aligned}
$$

where the y's are causally dependent variables, the z's predetermined variables, and the u's stochastic variables statistically independent of the z's. Each u_g is assumed, moreover, to be statistically independent of $y_{g+1}, y_{g+2}, \dots, y_G$.[5] In each equation the y variable with unit coefficient is regarded as the resultant variable and the other y variables and the z's are regarded as causal variables. We now consider the possibility that we gain direct control over y_g, that is, we can manipulate y_g by use of variables

[5] Although Wold has imposed this specification in his definition of a causal chain system, Strotz feels it may be too restrictive and would classify causal chain systems under two headings: those that are causal chains in their stochastic form (Wold's case) and those that are causal chains only in their exact part. In the latter case the covariance matrix of the u_g's need not be diagonal. For further discussion of this, see Strotz, *infra*, p. 430, fn. 6.

other than the z's appearing in the model. In this case we now need a new model. It can be obtained, however, by a single change in the old one. We merely strike out the gth equation and reclassify y_g as an exogenous (predetermined) variable rather than as a dependent variable. The coefficients of y_g in the G — 1 equations of the new model will be the same as they were before, namely, $\beta_{1g}, \ldots, \beta_{g-1,g}$, and zeros. It is in this sense that each non-unit coefficient in a recursive system has a causal interpretation. It describes the influence of the variable whose coefficient it is on the resultant variable, irrespective of whether the causal variable is dependent or exogenous in the system. Such a parameter has causal interpretability.

3. THE CAUSAL INTERPRETATION OF NONRECURSIVE SYSTEMS

We now turn our attention to nonrecursive systems. What is the possibility of causal interpretation in these systems?

(a) *No causal interpretation.* It may be that no causal interpretation of a nonrecursive system is intended. The relations in the system may be asserted only to define the joint probability distribution of the dependent variables conditional upon the predetermined variables. The coefficients to be estimated are then simply parameters in the joint conditional distribution of y given z. With nothing further claimed, there is no objection to such a model or to efforts to estimate the parameters of the distribution.

(b) *Vector causality.* It may be asserted for the *nonrecursive* model

$$\mathrm{B}y' + \Gamma z' = u' \tag{3}$$

that the vector z *causes* the vector y. Causality in this sense goes beyond the definition of "causality" given in Wold [**6**, **7**]. It may readily be accepted, however, as an abstract terminological extension of the more usual notion of causation and may be employed in the everyday sense of the statement, "The food supply causes the fish population." An example may be useful. Suppose z is a vector whose elements are the amounts of various fish feeds (different insects, weeds, etc.) available in a given lake, and that y is a vector whose elements are the numbers of fish of various species in the lake. The reduced form $y' = -\mathrm{B}^{-1}\Gamma z' + \mathrm{B}^{-1}u'$ would tell us specifically how the number of fish of any species depends on the availabilities of different feeds. The coefficient of any z is the partial derivative of a species population with respect to a food supply. It is to be noted, however, that the reduced form tells us nothing about the interactions among the various fish populations—it does not tell us the extent to which one species of fish feeds on another species. Those are causal relations among the y's.[6]

[6] Indeed, even though $\partial y_g/\partial z_k > 0$, this does not imply that fish species g consumes food supply k. It may be that species g consumes species h which consumes food supply k.

Suppose, in another situation, we continuously restock the lake with species g, increasing y_g by any desired amount. How will this affect the values of the other y's? If the system were recursive and we had estimates of the elements of B, we would simply strike the gth equation out of the model and regard y_g, the number of fish of species g, as exogenous—as a food supply or, when appearing with a negative coefficient, as a poison. It will be the purpose of subsections (c) and (d) to determine whether if the model is not recursive the problem can be dealt with in this same way. The nonrecursive model does, in any case, enable us to predict the effects on the y's of controlled variations in the z's.

Herbert Simon in developing a sense of causality for econometric models [3] has used this notion of vector causality. He defines causal relations among *subsets* of dependent variables by using a model recursive in these subsets. Partition y' into three subsets, that is, into three column vectors, y_1', y_2', and y_3', so that $y' = (y_1, y_2, y_3)'$ and partition B conformally. Consider the system (3) in which B may be written as

$$\text{(4)} \qquad \mathrm{B} = \begin{pmatrix} \mathrm{B}_{11} & \mathrm{B}_{12} & \mathrm{B}_{13} \\ 0 & \mathrm{B}_{22} & \mathrm{B}_{23} \\ 0 & 0 & \mathrm{B}_{33} \end{pmatrix}$$

consisting of nine submatrices. Then y_3' is caused by z'; y_2' is caused by y_3' and z'; and y_1' is caused by y_2', y_3', and z'. In the previous sentence the word "caused" is used in the sense of vector causation, and B is "block triangular." No causal relations among the variables *within* a subset are defined. Press this logic further. If each subset consists of but a single endogenous variable, and a causal sequence is established among subsets, B is triangular and the system is recursive.

If, by way of contrast with vector causality, each effect variable is given as an explicit function of only variables that are its causes, we may speak of *explicit causality*.

(c) *Causal Circles, Mutual Causation, and Equilibrium Conditions.* By a "causal circle" we shall mean a system such as

$$\text{(5a)} \qquad p(t) = \alpha_1 + \beta_1 q(t) + \gamma_1 z_1(t) + u_1(t)\,,$$

$$\text{(5b)} \qquad q(t) = \alpha_2 + \beta_2 p(t) + \gamma_2 z_2(t) + u_2(t)\,,$$

where $z_1(t)$ and $z_2(t)$ are exogenous and for which the following two statements are asserted: (1) In equation (5a) $q(t)$ is a cause of $p(t)$; (2) In equation (5b) $p(t)$ is a cause of $q(t)$. Causation is here used in a sense not allowed by the operative meaning that causation has in an experimental laboratory. To accept a causal circle is, in the laboratory meaning of the word "cause," to suppose that the value of one variable is determined by the value of another variable whose value cannot be determined until

that of the first has been determined. To assume that the values of the two variables determine each other makes sense only in an equilibrium system, and such a system provides no explanation of how the equilibrium comes about (of change or of causal connections among the endogenous variables of the system).[7]

The familiar illustration of the three balls in the bowl mutually *causing* one another's location, which has been advanced as an example of mutual causation,[8] might be considered in this connection. For the steady state (equilibrium) there are certain mutual conditions which must be satisfied; but if the balls are displaced and then roll towards equilibrium they are either in mutual contact and roll (or slide) as a single mass or the position of each can depend on the positions of the others only when the latter are lagged in time. Indeed, mutual causation in a dynamic system can have meaning only as a limit form of the arrow schemes shown in Figure 1 where the time lag θ is reduced towards zero.[9]

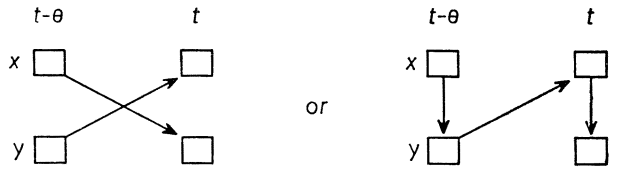

FIGURE 1

There is, however, a sense in which the coefficients of an equilibrium system may be given a causal interpretation, even though the relations in an equilibrium system may not themselves be causal relations. Let us take first an imaginary case in ecology: the balanced aquarium. Suppose there are two species of fish in an aquarium, big, b, and small, s. Their populations are y_b and y_s. The big fish feed on the small ones and on weed type a, available in quantity x_a. The small fish feed only on weed type c,

[7] If, for example, an entrepreneur wishing to produce a given amount of product at minimum cost decides simultaneously on how much of each of two factors of production, x_1 and x_2, to employ, it might be said that his decision as to x_1 causes his decision as to x_2 and his decision as to x_2 causes his decision as to x_1. We do not believe this conforms to the "laboratory" meaning of causation and we therefore reject this usage. On this, see [**1**].

[8] This illustration, due to Marshall, has been referred to recently by Stone [**4**] in this connection. Marshall spoke of mutual "determination" rather than "causation" and it is not clear whether these words are to be regarded as synonyms.

[9] In equilibrium when $x(t) = x(t-\theta)$ and $y(t) = y(t-\theta)$, these structures may be collapsed into one of apparent "mutual causation," but what are simultaneous equilibrium conditions ought not to be confused with causal relations.

available in quantity x_c. It takes time for the big fish to catch the small ones. The model is linear and stochastic, thus:

$$y_b(t) = \alpha_1 + \beta_1 y_s(t - \theta) + \gamma_1 x_a(t) + u_1(t)\,, \tag{6a}$$

$$y_s(t) = \alpha_2 + \beta_2 y_b(t - \theta) + \delta_2 x_c(t) + u_2(t)\,. \tag{6b}$$

Suppose now that every time we observe the aquarium it is in equilibrium. Moreover, we wish to estimate two numbers—are they β_1 and β_2?—so that we can answer these questions:

If we controlled the population of the big (small) fish, y_b (resp., y_s), by adding them to the aquarium or taking them out, and thereby held y_b (resp., y_s) at some arbitrary level, what would the expected value of the population of small (resp., big) fish, y_s (resp., y_b), be at the new equilibrium level, conditional upon the values of x_a and x_c?

Suppose we next formulated the model:

$$y_b(t) = \alpha_1 + \beta_1 y_s(t) + \gamma_1 x_a(t) + u_1(t)\,, \tag{7a}$$

$$y_s(t) = \alpha_2 + \beta_2 y_b(t) + \delta_2 x_b(t) + u_2(t)\,. \tag{7b}$$

To control the population of big fish is to wipe out or invalidate relation (6a) and to regard $y_b(t - \theta)$ as exogenous. To control the population of small fish is to wipe out (6b) and to regard $y_s(t - \theta)$ as exogenous. In the absence of such intervention, we certainly cannot say that $y_s(t)$ *causes* $y_b(t)$ and that $y_b(t)$ *causes* $y_s(t)$, i.e., we cannot use model (7) as a *causal* model telling us what happens through time in the uncontrolled aquarium. Nevertheless, the values of β_1 and β_2 appearing in model (7) do tell us what a second fish population will be conditional upon our specifying (controlling, manipulating) a first fish population—provided that the aquarium is then brought back to an equilibrium situation. Equilibrium models do tell us (enable us to predict) something about equilibrium values under *control.*

A comparable example in economics might well be the cobweb model. The model is:

$$p(t) = \alpha - \beta q_h(t) + \varepsilon z_1(t) + u_1(t) \quad \text{(demand)}\,, \tag{8a}$$

$$q_h(t) = \gamma + \delta p(t - 1) + \eta z_2(t) + u_2(t) \quad \text{(supply)}\,, \tag{8b}$$

where $p(t)$ is price at time t, q_h is quantity harvested, z_1 and z_2 are exogenous variables, and u_1 and u_2 are stochastic shocks. This system is recursive, but if observed in equilibrium may, by use of the equilibrium condition

$$p(t - 1) = p(t)\,,$$

be written as

$$p(t) = \alpha - \beta q_h(t) + \varepsilon z_1(t) + u_1(t) \quad \text{(demand)}\,, \tag{9a}$$

$$q_h(t) = \gamma + \delta p(t) + \eta z_2(t) + u_2(t) \quad \text{(supply)}\,, \tag{9b}$$

and will be subject to the same causal interpretation as given in the previous example. The question of how β and δ are best to be estimated is left open.

(d) *Bicausality.* Suppose we confront a demand-supply model of the following sort

$$q(t) = a_{10} + a_{11}p(t) + a_{13}z_1(t) + u_1(t) \text{ (demand)}, \tag{10a}$$

$$q(t) = a_{20} + a_{21}p(t) + a_{24}z_2(t) + u_2(t) \text{ (supply)}, \tag{10b}$$

where $q(t)$ is quantity, $p(t)$ is price, $z_1(t)$ and $z_2(t)$ are exogenous variables, and $u_1(t)$ and $u_2(t)$ are stochastic shocks.

Now suppose this system is given the following causal interpretation: $p(t)$ causes $q(t)$ in accordance with equation (10a) and $p(t)$ also causes $q(t)$ in accordance with equation (10b). This notion of causality is certainly out of accord with the usual laboratory or control notion which we find so natural. Those who write such systems do not, however, really mean what they write, but introduce an ellipsis which is familiar to economists. What is meant is that

$$q_d(t) = a_{10} + a_{11}p(t) + a_{13}z_1(t) + u_1(t), \tag{11a}$$

$$q_s(t) = a_{20} + a_{21}p(t) + a_{24}z_2(t) + u_2(t), \tag{11b}$$

$$q_d(t) = q_s(t), \tag{11c}$$

where $q_d(t)$ is quantity demanded and $q_s(t)$ is quantity supplied. This is an equilibrium model (nonrecursive), and (11c) is not an *identity*, but an *equality* which is assumed to hold in fact over the observations.[10]

A somewhat far-fetched example, but in a surer context, is the following. Suppose there are two crops, d and s, whose yield is measured in bushels, and that (over the relevant range) the yield of one, q_d, is a positive linear function of rainfall, p, while the yield of the other, q_s, is a negative linear function of rainfall, and that these functions intersect within the relevant range. An amount z_1 of fertilizer 1 is applied to crop d and an amount z_2 of fertilizer 2 is applied to s. Suppose for each of N years we conduct an experiment for each crop, applying different amounts of fertilizer. Rainfall is uncontrolled. Imagine now the amazing result that each year of the experiment Nature chooses a rainfall that makes the two yields equal. We may then represent the experiment by system (11) and reduce this to system (10) simply by dropping the subscripts on q and keeping track of which equation is for which crop.

Now, while it would be remarkable for Nature to choose rainfall so as

[10] Especially for one not familiar with the economist's ellipsis, difficulty may result from the careless use of symbols in this reduction. Strictly speaking, the q in (10a and b) should be either a q_d or a q_s, or an additional equation such as $q = \min(q_d, q_s)$ or $q = q_d$ should be added to model (11). Otherwise, identity (and not simply equality) of quantities demanded and supplied is technically implied.

to give us this strange result, it may be not so remarkable for the market to choose price so that quantity demanded equals quantity supplied, at least approximately. This is because, while rainfall is independent of past crop yields, price may well depend on past quantities demanded and supplied, and, if the system is not subject to violent change, the price adjustment relation may work with great efficiency. Whether it will or not is, of course, an empirical question: the answer may vary from market to market and time to time; but this is a matter of realism which need not concern us here. When the equality holds the theoretical system (11) may be represented by system (10), although if (11c) holds only approximately, system (10) is one with errors in the variables.

The causal system which underlies the equilibrium model (11) is then one in which (11c) is replaced by some function such as

$$(11d) \qquad p(t) = f[q_d(t-\theta), q_s(t-\theta), p(t-\theta), z_1(t), z_2(t), z_1(t-\theta), z_2(t-\theta)] + u_3(t)$$

and the causal (and, in this case, dynamic) model is recursive. If $p(t)$ were now to be subject to direct control, (11d) must be abandoned and $p(t)$ must be regarded as exogenous. Equations (11a) and (11b) would then answer questions regarding the causal effect of controlled variation in $p(t)$ on $q_d(t)$ and $q_s(t)$, and α_{11} and α_{12} would be causally interpretable coefficients. They are, moreover, the same coefficients which enter the "bicausal" system (10).

What is to be concluded from all this is that equilibrium systems may appear to entail "causal circles" or "bicausality," but that this is not what is intended. The causal interpretation of a coefficient in either of these types of equilibrium models is to be found in the underlying dynamic model which, *if the laboratory notion of causality is to be sustained*, will be recursive in character.[11]

Two major questions remain: (1) Can a stochastic shock model of the sort commonly considered—i.e., equation (1)—ever be assumed to be always in equilibrium whenever observed?[12] (2) If not, must it not be said that either there are measurement errors introduced by the assumption of equilibrium (for example, ought not (11c) be regarded as an *approximate* equality) or there is a specification error? If so, this raises questions as to appropriate estimation procedure and as to the properties of estimates that ignore these model qualifications. The subsequent paper by Strotz deals with this problem.

[11] Incidentally, differential equation systems are regarded as recursive with respect to infinitesimal time intervals. See [**8**].

[12] For (11a, b, and c) to hold exactly, (11d) would need to include $u_1(t)$ and $u_2(t)$ as arguments and exclude $u_3(t)$.

Our contribution ends with a paper by Wold, who gives a brief presentation of conditional causal chains, a new type of model which is designed as an extension of ordinary (pure) causal chains in the direction of independent systems.

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