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I Introduction: the Need for a Theory of Family Formation

In recent years practitioners in many disciplines, most notably perhaps demography, economics and sociology, have been much concerned with the explanation of changes in fertility and with the problems of predicting changes in birth rates. Accordingly, much empirical work has been carried out in terms of the aggregate characteristics of populations and reinforced by detailed empirical investigations at a micro level of individual groups or communities, both contemporary and historical.[1] Interest has been intensified by the urgent population problems of developing countries. A main focus of attention has been the descriptive framework of the 'Demographic Transition', the essence of which is to postulate three stages in demographic development: (1) High fertility offset by high mortality with little or no population growth, (2) Decreasing mortality accompanied after a time lag by decreasing fertility with rapid population growth, (3) Low fertility and low mortality with slow population growth.[2]

Unfortunately as Tabarrah has argued "...since the theory does not specify what major factors will be responsible for the decline in fertility, it does not shed light on how these factors could be manipulated by policy makers. Lacking this important theoretical requirement, population policy has become increasingly aimed at the direct prevention of pregnancies rather than the basic factors underlying high or low fertility."[3] Not surprisingly, therefore, an economist has recently argued that "the most important question (demographers) are asking is: What is the explanation of the demographic transition?"[4]

The empirical studies typically yield data relating to observable statistics such as the crude birth rate or gross reproduction rate

at the macro level and actual family size or intervals between births at the micro level. The link between detailed studies of family reproductive behaviour and aggregate population movements is not, however, just a straightforward matter of direct inference from a sample to the parent population. The observed statistics reflect family formation processes and decisions which are themselves impinged on by a variety of factors. If we are to explain the empirical results and links between them it is about these decisions that inference has to be made. Even if the decisions are sampled by questionnaire methods the reliability and interpretation of such data is questionable and in any case fertility is by no means wholly determined by factors of which the respondents are aware. If the investigation is of a historical community no such method is possible. Thus deductions have to be made from observable statistics.

Many discussions of changes in fertility have been based on presumed empirical association[5] although as Coale has pointed out this approach has not been particularly successful; "Fertility reduction seems to be a nearly universal feature of the development of modern secular societies, but its introduction and spread cannot yet be explained by any simple universally valid model or generalised description."[6] The difficulties of this "statistical empiricist" approach not based on a priori reasoning are well known but its unsuitability in this particular field was made particularly clear by Becker. Starting from the economic theory of consumer behaviour he argued that whilst "The traditional view, based usually on simple correlations, has been that an increase in income leads to a reduction in the number of children per family"[7]..."The secular decline in fertility may also be consistent with a positive relationship since the secular decline in child mortality and the secular rise in both contraceptive knowledge and child costs could easily have offset the secular rise in income."[8] Similarly at the micro level the

paradoxical positive correlation between size of family and the adoption of family planning is an example of the need for theory. Once it is realised that families have differing natural fertility, then if contraception is used on reaching target family size, a situation attained only by those of relatively high fecundity, the paradox is explained.[9]

An appropriate line of approach to fertility change seems to be to consider the results of micro level studies in the context of a theory of family formation. In this way, a change in the observable statistics can be related to a change in behaviour or factors influencing behaviour, and the significance of the latter changes for the aggregate population then examined. This way of doing things contrasts with much of the recent literature where the problem has been tackled by regression analysis based on cross section data of an aggregate type. These studies have produced somewhat conflicting and confusing results.[10] This is not altogether surprising since many of these studies have at best a very tenuous connection with theories of the determination of fertility, which inevitably are concerned with individual family units and furthermore they generally ignore important differences WITHIN countries.[11]

Many of the important issues CANNOT actually be examined satisfactorily at a macro level. For instance, if we consider the important work of Carlsson which asks whether an "innovation" or an "adjustment" approach is a more accurate way of looking at the adoption of contraception we find that he poses two main questions; (1) Did birth control within marriage exist on a considerable scale before the secular decline of fertility began, or did the decline end a phase in which control was not practised by married couples at all, or very rarely at most? (2) Does the pattern of spread of fertility decline in fact conform to the lag assumption so prominent in current theory?"[12] Attempts to answer these questions require micro level approaches. Moreover, as

Carlsson points out not only are "the lag assumptions very important for the diagnosis and prediction of population trends"[13] but also "No theory of fertility and fertility change in Sweden can be regarded as satisfactory unless it accounts for the variations in marital fertility which existed before the secular decline. And Sweden is no isolated case."[14] Similarly a micro approach would be essential in any attempt to discriminate between Becker's explanation of fertility changes in terms of quantity and quality elasticities of demand[15] and Leibenstein's theory based on the different preference structures of social status groups.[16]

In the next Section we will present our view of the present state of the theory of family formation. Following on from this we argue in Section III that a simulation model can be an essential link between theory and observation. In Section lV we present an example of such a model and discuss the associated data problems in Section V. We report some illustrative experiments in Section VI and finally present our conclusions.

II The State of the Theory of Family Formation

Recourse to a theory of family formation behaviour, however, is rarely without problems. It must connect observable statistics to non-observable or difficult to observe factors such as preferences for family size, which may be target family size as opposed to ideal family size[17], criteria for using and effectiveness of contraception, fecundability etc. The difficulties may be illustrated by reference to the use of birth intervals as indicators from which inferences can be drawn.[18] For instance it has been argued that an increase in the last closed birth interval of "completed" families[19] where the wife married below thirty years of age is indicative of the adoption of contraception.[20] Put crudely the argument is that the smaller target family is quickly constituted with birth control over total size rather than spacing.

Long last intervals occur because of "accidents" or "changes of mind" with a tendency to raise the mean last birth intervals for the group concerned. This argument is more closely examined in section VI but we may notice now that anything which tends to change the sample balance between "accidents" and "non-accidents" makes the last birth interval an ambiguous indicator. For example a general decrease in fecundability will mean less people have "accidents" but those who do reach target family size will be more concentrated in the upper deciles of the fecundability distribution. Thus the cause of a change in the last birth interval is not uniquely defined. Similar problems occur in the assessment of the effects of birth control programmes.

The difficulties of constructing an adequate theory of family formation and testing it become more obvious when we compare the approaches to fertility change made by different disciplines. As Easterlin has pointed out "Real world problems of fertility explanation rarely involve only one fertility determinant."[21] This has the corrollary that there is no general presumption that actual fertility corresponds to target fertility if only because of the intervention of biological (supply-side) factors. We have indeed already identified this as a major problem in interpreting sets of observed outputs.

When we turn to the literature, however, we find that the economists concentrate on the demand for children to the almost entire exclusion of supply considerations; "there is a noticeable scarcity in the economic analysis of references to physiological or biological factors that may influence fertility."[22] Moreover, their general approach to the problem of fertility determination has been in the context of a world of perfect certainty via a static model of lifetime utility miximisation; for instance one of the most recent models is constructed on the following basis. "A one-period comparative static framework will be

used in which a husband and wife of given ages and characteristics are considered to adopt, at the outset of marriage, a utility-maximising lifetime plan for child bearing, for expenditure of time and money on children, and for other sources of parental satisfaction not related to children... The couple will be assumed to have perfect and costless control over their fertility and to possess perfect foresight concerning all relevant demographic and economic variables over the course of their marriage, so that the lifetime plan adopted ex ante at marriage coincides with ex post observations of their completed fertility."[23]

Any attempt to interpret the observed statistics on the basis of such a theory immediately runs into at least two interrelated problems: (1) important conceptual variables may not be directly observable; (2) proxy variables which are available may well be impinged on by a variety of factors including supply-side ones and hence be subject to a sort of identification problem.

Sociologists on the other hand have generally been interested in the supply ("production") aspects of fertility; Davis and Blake classified factors affecting exposure to intercourse, factors affecting exposure to conception and factors affecting gestation and successful parturition.[24] In attempting a synthesis of these approaches Easterlin has brought out clearly the further difficulty for the "economist's view" that the demand for children may actually be related to the potential output; in his formulation the "target family size" (C) is influenced by the "motivation to restrict fertility", $C_n - C_d$, where C_d is "ideal family size" and C_n is "natural fertility", interpreted as the number of surviving children forthcoming in the absence of voluntary control of fertility.[25] In some circumstances this produces new and counter-intuitive results such as "an increase in natural fertility, rather than raising observed fertility as would be predicted if the possibility of interdependence were overlooked

may actually lower it by inducing the adoption of fertility control."[26] It must also be recognised that some factors may impinge both on potential fertility and on demand for children (e.g. income) and that this may cause difficulty in discriminating between demand and supply relationships.

However, Easterlin in his analysis confines himself to the static perfect certainty framework of the economists and in particular assumes that the decision-makers have perfect information concerning natural fertility. In common with these models the possibility of adjustment to changed circumstances or expectations is excluded although, as Schultz has argued, "To cope intelligently with rapid rates of population growth in low income countries one needs an understanding of precisely this process of behavioural adjustment to demographic and economic change and the disequilibrium that it introduces into the family-formation process."[27]

This perfect certainty assumption contrasts sharply with the work of many demographers. Whilst, like the socioligists, they concentrate on the supply side in doing so they explicitly emphasise the stochastic nature of human reproduction as well as differences in fecundity etc. They have applied probability models to fertility change and in particular to the effectiveness of birth control programmes.[28] An early result was to demonstrate that in general the positive relation between contraception and family size would only partly be a result of the subfecundity of non family planners and would partly be simply through chance.[29] Their perspective makes even more clear the likelihood of discrepancies between changes in target family size and changes in actual family size.

So far these models have considered very crude types of contraceptive behaviour and have borne little relation to the demand-based theory of family formation. Their predictions of the effects of birth control programmes are essentially sophisticated numerical calculations, leading to results such as "Relatively inefficient contraceptive methods used by a large part of the reproducing population would have less effect on the asymptotic fertility (and presumably on a resultant birth rate) than would highly efficient methods adopted by a smaller portion of the same population. This conclusion, of course, would hold only if there were no selection in favor of less fecund couples for the more effective method."[30] Whether there would be such selection is exactly the sort of prediction one might hope to obtain from a theory of family formation to improve the assessment of family planning programmes in this stochastic framework.

III Simulation a a Link between Theory and Empirics

It seems an obvious conclusion that it is inefficient to consider one of a number of observed statistics in isolation. If one considers reproductive experience as a system having inputs of non-observable behavioural parameters and distributions and outputs of observable family activities, then one must consider the relationship between the sets of inputs and the sets of outputs. We can identify the following questions:

(1) Can we say what happens to the set of outputs if the set of inputs changes?

(2) Can we identify 'plausible' changes in inputs that create observed changes in outputs, taking into account the stochastic nature of the system? Can we identify a unique cause of the observed changes?

(3) Can we quantify such changes in inputs and are the quantities 'plausible'?

It is our thesis that an aid to seeking answers to these questions is the construction of a simulation model. Particularly it should be viewed as a link between the unobserved decisions predicted by theory and the observation of empirical research.

We have attempted to list below some particular, although interrelated merits of the use of a simulation model for these purposes.

(1) We know that in general the observed outputs will be impinged on by the interplay of a variety of non-observed inputs; evidently this limits the usefulness of analysis based on observed data which are the result of non-controlled experiments. Simulation allows controlled experiments - although in an artificial environment - and this focuses attention on and permits an assessment of these interactions.

(2) Because there are variables intervening between actual and target fertility performance, it is necessary to examine very carefully whether available (proxy) indicators adequately reflect changes in the appropriate conceptual vaiables; yet most changes in the set of inputs can be expected to have both direct and indirect consequences frequently involving offsetting effects. The relative quantitative importance of these direct and indirect effects can be sought by a simulation approach.

(3) The use of probability models can not yield explicit analytical results where an acceptable degree of "realism" is involved because the parameters involved are not in general constant over time. Not only does a woman's fate this month depend on what happened last month, it also depends on her age. A simulation model does not need to have an analytic solution, and therefore does not require stochastic elements 'simplified' out, to the detriment of realism.

(4) Simulation models offer the possibility of allowing a cohort of women to <u>adjust</u> their contraceptive behaviour for instance to accommodate a change in their perception of their natural fertility.

(5) There is opened the possibility of compensating for missing data at least in the sense of performing experiments to examine what kind of impact the variables in question may have had and thus throw some light on the vulnerability of any results. This may be important as a micro level approach is likely to run into small samples problems. In Section V we will consider the use of 'high' and 'low' values of some variables to assess the sensitivity of the model to these variables.

(6) Some of the most promising indicators are subject to truncation effects. In particular changes in mean birth intervals may be biased by the finiteness of the observation period.[31] Simulation offers a chance of estimating the seriousness of such problems.

It should be noted that any quantitative analysis of family formation behaviour is also made more difficult by the fact that the investigator is dealing only with sample data. The extent of a change in target family size thought necessary to produce observed changes in actual family size is in this sense only a point estimate and rarely unbiased at that. Furthermore it is necessary to distinguish changes in family behaviour which account for all the observed changes from those which account for enough to make the residue statistically insignificant.

IV The Model

In this section we describe briefly the main features of the simulation model that we have used in our experiments. Not all of its provisions were necessary for all the experiments. We would like to emphasise two features of the model:

(1) A probability distribution of the monthly chance of conception (MCC) is used so that women of the same age are not 'homogeneous'. This

contrasts with much other simulation work in this area. (See section V).

(2) The model is of the stochastic control type and can be analysed with emphasis on the form of control used.

The model is first considered in detail under various headings and then discussed with the aid of the diagram on page 15 . Where parameters or distributions are described as 'known' it implies they are data inputs. (See Section V)

(i) Length of Marriage

In this paper we deal only with marital fertility and susceptibility to conception is assumed to commence at marriage. There is no possibility of either divorce or remarriage. Each woman's age at marriage is a random sample observation from the known population probability distribution. "Effective marriage" ends as soon as either (a) the wife or husband dies or (b) the wife becomes permanently sterile. All these events are timed by means of random sample observations from known independent population probability distributions. We also assumed that each partner had the same age at marriage. For our purposes the length of marriage or "effective marriage" is the period in which the family can conceive children.

(ii) Monthly Chance of Conception (MCC)

We regard "effective marriage" as <u>susceptible</u> to the conception of a child in a given month if the woman is neither pregnant nor in the amenorrhea period following the termination of pregnancy. The probability of conceiving a child in a given month given that the marriage is susceptible and that no contraception policy is active is defined as MCC. MCC is assumed to be age-specific. MCC differs from the notion of "natural fecundability"[32] which we can conceptually regard as the biological potential of a woman in terms of a monthly probability of conception given that the cycle is ovulatory and "normal" sexual relations are occurring. This is believed

by many demographers to be approximately invariant with age although this view is not universally held.[33] In practice it is difficult to separate "natural fecundability" changes from temporary sterility (anovulatory cycles) or changed coital frequency. MCC subsumes these latter considerations as well as fecundability and is thus regarded as age specific whether or not fecundability itself is constant throughout marriage. MCC is distributed as approximate Beta distributions, the distribution shifting with the age of the reproductive cohort. Each woman is treated via a random observation x on a rectangular distribution defined on $\{0 \leqslant x \leqslant 100\}$. This observation is considered to be the percentile ranking of the woman in terms of MCC and is assumed to be constant over the life of the marriage. From this ranking actual levels of MCC are found for each woman at different ages by reference to the appropriate Beta distribution.

(iii) The Period of Amenorrhea

The amenorrhea period is a random observation on a known probability distribution. In the event of a miscarriage, stillbirth or the death of an infant before the end of the amenorrhea period it is assumed that susceptibility is restored after one month.

(iv) Outcome of Pregnancy

It is assumed that a conception may fail to produce a live birth in two ways. Firstly a miscarriage may occur after three months of pregnancy and secondly the child may be stillborn after nine months. Each of these events occur with known independent probabilities. It is assumed that there are no multiple births and that the probability of a male child is fifty percent.

(v) Family Size

The ages at death of the children born are random independent observations from known mortality distributions. The mortality conditions applying to children are assumed to be consistent with those applying to their parents in the sense of being based on the same model life-tables.

The family size at a point during marriage is the difference between the number of children born and the number who have died.

(vi) Contraception

Contraception is assumed to have a known effectiveness $E(0 \leq E \leq 1)$ such that if n is the MCC $(0 \leq n \leq 1)$ then the probability of becoming pregnant in a given susceptible month is $n(1 - \lambda E)$ where $\lambda = 1$ if contraception is used (policy on) and $\lambda = 0$ if contraception is not used (policy off).

(viii) Control

The family has a degree of control over its size by using or not using contraception. The family is in a stochastic world. It does not know with certainty the length of effective marriage, waiting times for future children, whether present or future children will survive, whether 'errors' will occur during periods of 'policy on' etc. In a world of perfect certainty the family would find an optimal control at the outset of the marriage in the form of a trajectory of on/off controls terminating at the end of the marriage subject only to the feasibility of its requirements. With the large number of stochastic elements and the age specificity of the reproductive process present in the model, this is of course not possible and instead we have considered four separate control systems. All the controls were to achieve a target family size (T) in terms either of live births (B_1 and B_2) or surviving children (S_1 and S_2) at the end of effective marriage. Most of the experiments centre on the simpler ones, B_1 and S_1, to save computing time.

The control systems are outlined below

B_1 : Each month if the number of live births so far is greater or equal to T then <u>policy on</u>, otherwise <u>policy off</u>.

S_1 : Each month if the number of surviving children is greater or equal to T then <u>policy on</u>, otherwise <u>policy off</u>.

B_2 : Every 12 months the family estimates its terminal number of live births on the basis of a simulation of the future with

policy on. If this estimate is less than T then policy off for the next 12 months.

S_2 : Every 12 months the family estimates its terminal size in terms of the number of surviving children on the basis of a simulation of the future with policy on. If the estimate is less than T then policy off for the next 12 months.

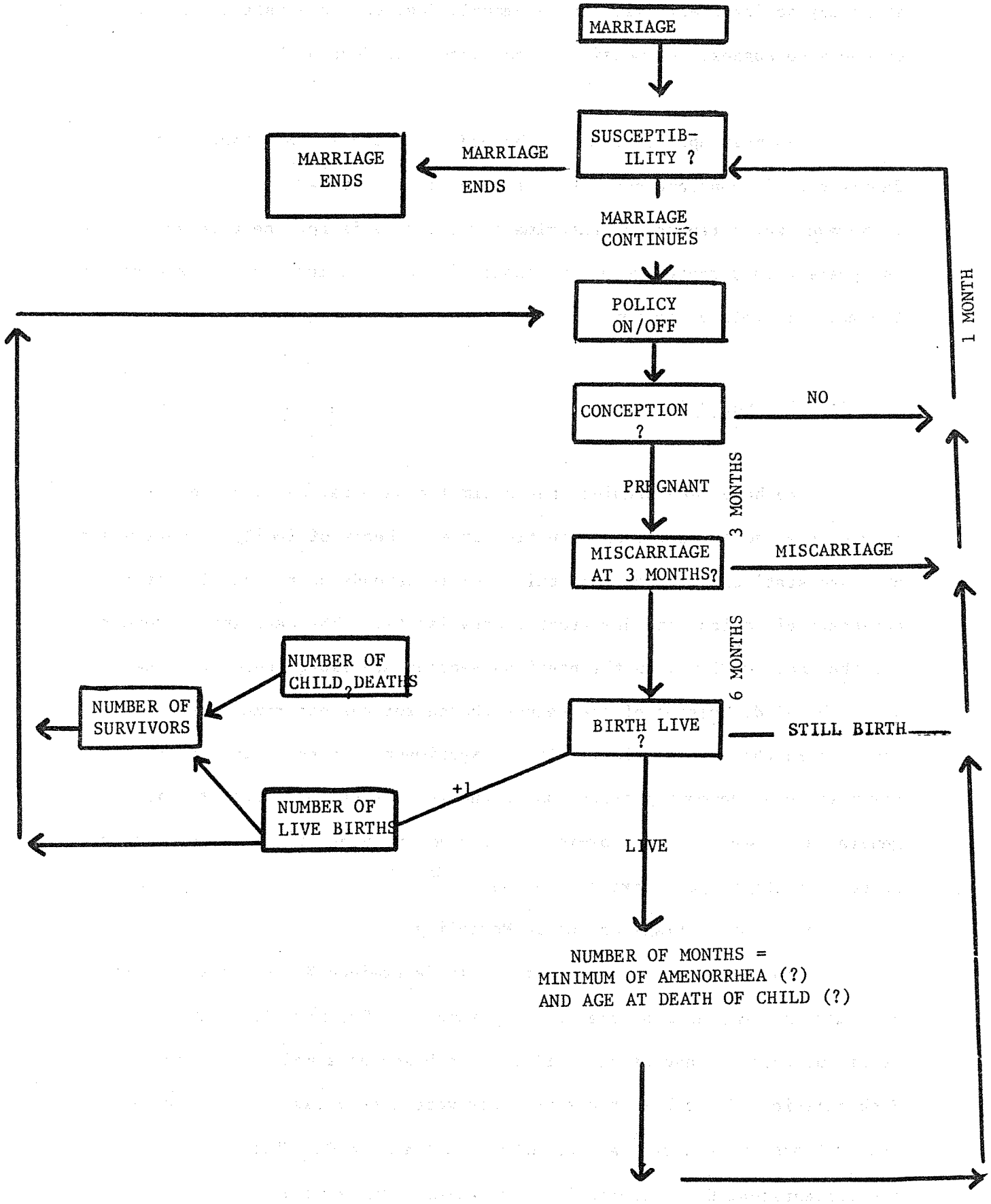
The working of the model is illustrated in the diagram. Each woman enters the model upon marriage and her reproductive experience is dealt with sequentially. The length of her effective marriage and her rank order position in MCC are generated and she then remains in the system until the marriage ends through death or sterility. Once susceptible policy on or off influences the probability of conception which itself may end in miscarriage or stillbirth. There is then a delay before the process can be repeated. A running total of births and deaths of children is fed into the control system to determine policy on/off.

The number of women considered is the required size of the sample. The programme to compute the model outputs the ages of women when births occur, the number of births and of surviving children, age at beginning and end of effective marriage and the intervals between births.

(viii) Limitations of the Model

Many of the restrictions of this model we do not regard as likely to be very serious (e.g. no multiple births, all miscarriages at three months etc.). However it is worth drawing attention to two particular omissions which could have important consequences for the generality of our results. In common with other work in this area we have so far assumed that women's biological characteristics are independent of each other, e.g. we have not related her MCC to the expected value of her amenorrheic period or the expected chance of a miscarriage, although a priori we might expect that within a cohort of women there would be some degree

A Diagrammatic Representation of the Model



? indicates choice by random process

of association between such inputs. We have also not included at this stage any parity-specificity in the model, despite the existence of some evidence to suggest its possible importance (See Section V).

In most applications of the model we have assumed that target family size is constant over the lifetime of the marriage. In Section VI however we do report one experiment where this is not the case and it is apparent that there are no intrinsic difficulties involved in generalising the model in this direction.

V Data for the Model

We have been arguing that a simulation model can prodive a link between the family decisions resulting from a theory of family formation and observed statistics. How good this link is depends on the quality of the necessary biological and demographic data inputs. The data inputs required for the model outlined in the previous section are listed below together with a brief discussion of the values chosen for the experiments to be reported in the next section. These experiments are not meant to reflect any particular community situation, although data has been selected in general accordance with a low-income economy and none of the assumed values is outside the range of existing evidence.[34]

(i) Length of Marriage and Child Mortality

The length of actual marriage is determined by the time between marriage and the death of one of the partners. The mortality conditions obtaining both for adults and children are based on a model life-table. Probabilities of death in the first year were interpolated on the basis of observations by Wrigley[35] and Stoeckel and Alauddin Chowdhury[36] rather than the conventional U.N. figures.[37] In Tables 1 and 2 we set out the mortality rates relevant to our experiments and two marriage distributions which are

based on data from Wrigley[38] and Gautier and Henry.[39] One of the marriage distributions considers families where age at marriage was less than thirty years. Experiments with this latter category should produce an increased incidence of cases where supply factors do not dominate all other factors in determining family size.

The effective length of marriage may be shorter than the actual length due to the onset of permanent sterility. We assumed that for all women 45 years is the age at which this occurs, despite the existence of several possible age distributions[40], because of the difficulty in distinguishing permanent sterility from the decline of the probability of conception with age (see (ii) below).

(ii) Monthly Chance of Conception

The measurement of MCC is difficult. The attempts made to calculate fecundability have been particularly controversial and quite a wide range of values have been suggested as "typical" for various age groups.[41] In practice measures have to be made of "outputs" in the sense of observed waiting times and this makes it difficult to distinguish changes in "natural fecundability" from sterility or changed coital frequency. We assume two fundamental characteristics of MCC, (a) MCC varies between different marriages (b) MCC is age specific. Some writers, e.g. Roy and Venkatacharya[42] considered data with characteristic (b) but not (a). Others such as Barrett[43] estimated a distribution of MCC, but assumed this constant at all ages, thus incorporating characteristic (a) but not (b). We have used Barrett's estimated distribution, a Beta distribution but shift the distribution with age by multiplying the percentiles of the distribution by successive constants. The pattern of increase to a plateau at age 20 - 34 followed by decline is regarded as being broadly in accord with the evidence of Döring[44] on anovulatory cycles and Bourgeois-Pichat[45] on coital frequency. The deciles of the distribution at different ages are shown in Table 3. Each marriage obtains a ranking

in the MCC distribution which is maintained over the effective lifetime of the marriage. In this way it is hoped that the model has captured both characteristics (a) and (b) of MCC.

(iii) Number of Ovulatory Cycles

We have assumed that there are twelve such cycles a year when the woman is "susceptible" and have preferred to deal with the incidence of anovulatory cycles via the estimate of MCC. An alternative could be to use a distribution of the number of ovulatory cycles which is possibly marginally preferable but has not yet been included in the model.

(iv) Length of Gestation Period

Pregnancy which does not end in miscarriage is assumed to last for the equivalent of nine ovulatory cycles, i.e. $\frac{3}{4}$ of one year.

(v) Length of Amenorrhea

There are also some difficulties in estimating amenorrhea. We have taken our data from Roy and Venkatacharya[46] and tabulate the probability of ending the amenorrhea period in the t^{th} month following a live birth in Table 4. Amenorrhea is affected by nutritional standards and lactation practices and the values used are in accordance with the experience of a low rather than a high income country.[47] Amenorrhea may be age-specific[48] but we have not yet included this in the model, nor have we related amenorrhea to MCC.[49]

(vi) Miscarriages and Stillbirths

The outcomes that a conception ends in either a miscarriage or a stillbirth are independent random variables. We have taken the probabilities to be .2 for a miscarriage and .02 for a stillbirth.[50] These values are much higher than those the UN report[51] thought typical but much lower than have sometimes been reported in poor countries.[52] Though there is evidence to suggest that there is a positive association between miscarriage and age and with a previous history of miscarriage[53] such considerations have been neglected.

(vii) Target Family Size

In general this may be either a result of or a prediction of a theory of family formation. Alternatively, as in most of our experiments reported here, it can be assumed and the implications of that assumption found from the simulation model. Where this is the case the values chosen are not inconsistent with the survey findings on "ideal family size" in developing countries reported by Berelson.[54]

(viii) Contraceptive Effectiveness

Values are assumed and the sensitivity of the model to changes in these values analysed, particularly by contrasting a 'high' value with a 'low' value. The values assumed vary within the range 0.7 to 1.0; in practice E is particularly difficult to measure but values recently reported by Michael include 0.84 for the rhythm method, 0.94 for the condom and 0.996 for the pill.[55]

(ix) Sample Size

The sample size is constrained by the shadow cost of computer time required for the simulation experiments.

Table 1 - Assumed Mortality Rates : Probability of death before age t.

t	Prob.	t	Prob.
1 month	.0725	2 years	.162
2 "	.079	3 "	.177
3 "	.0855	4 "	.192
4 "	.092	5 "	.207
5 "	.0985	10 "	.225
6 "	.105	15 "	.240
7 "	.1115	20 "	.261
8 "	.117	25 "	.286
9 "	.1235	30 "	.312
10 "	.13	35 "	.338
11 "	.1375	40 "	.366
12 "	.145	45 "	.398

Table 2 - Assumed Marriage Distributions : Probability of woman being married by age t, given that the woman is married by age (a) 45, (b) 30

(a)				(b)	
t	Prob.	t	Prob.	t	Prob.
15	0	31	.79	15	0
16	.02	32	.81	16	.01
17	.04	33	.83	17	.03
18	.07	34	.85	18	.05
19	.13	35	.87	19	.07
20	.19	36	.90	20	.09
21	.25	37	.92	21	.18
22	.31	38	.94	22	.28
23	.37	39	.95	23	.38
24	.44	40	.96	24	.48
25	.5	41	.97	25	.58
26	.56	42	.98	26	.67
27	.62	43	.99	27	.75
28	.68	44	.99	28	.83
29	.74	45	1.00	29	.92
30	.77			30	1.00

Table 3 - Deciles of M.C.C. Distribution

Decile	Age : 15-19	Age : 20-34	Age : 35-40	Age : 40-45
0	0	0	0	0
1	.046	.069	.023	.0115
2	.06	.09	.03	.015
3	.075	.1125	.0375	.0188
4	.085	.1275	.0425	.0213
5	.1	.15	.05	.025
6	.11	.165	.055	.0275
7	.12	.18	.06	.03

Table 3 - continued

Decile	Age : 15-19	Age : 20-34	Age : 35-40	Age : 40-45
8	.14	.21	.07	.035
9	.17	.255	.085	.0425
10	.3	.45	.15	.075
Mean	.106	.159	.053	.026

Table 4 - Amenorrhea : Probability of having regained susceptibility t months after a live birth, given the child survived until at least month t+1

t	Prob.	t	Prob.
1	.04	16	.82
2	.08	17	.85
3	.13	18	.87
4	.18	19	.89
5	.23	20	.90
6	.29	21	.91
7	.35	22	.92
8	.42	23	.93
9	.49	24	.94
10	.55	25	.95
11	.61	26	.96
12	.66	27	.97
13	.71	28	.98
14	.75	29	.99
15	.79	30	1.00

VI Some Illustrative Experiments

Here we report some experiments that we have conducted with the model described in Section IV and the data tabulated in the last Section. We report only a few of many experiments that we have performed, and these have been selected to reflect a variety of different lines of research. Firstly we show in Table 5 the connection between the monthly chance of conception and the average number of births per family given no control (i.e. no contraception practised). Then we consider the efficiency of the last birth interval to signal changes in contraception practice. The third experiment compares the four controls B_1, B_2, S_1 and S_2 in terms of their performance in relation to two target objectives. Finally we consider a utility function approach in a simplified form of the model.

Experiment 1

In the last Section we tabulated the distribution of MCC which we have used in the model. A change in each woman's MCC at any age by a factor λ will produce a change in the average 'maximum' (no contraception) number of births per family. In Table 5 we report these averages for $\lambda = \frac{1}{2}, \frac{3}{4}, 1, \frac{5}{4}, \frac{3}{2}$ together with the variances of the number of births, for samples of size 50. The experiment is performed with both marriage distribution (a) and (b), and in both cases premature ending of the marriage due to death of either partner is ignored.

Table 5 - Average and Variance of Births for different MCC

Marriage Distribution (a)

λ	$\frac{1}{2}$	$\frac{3}{4}$	1	$\frac{5}{4}$	$\frac{3}{2}$
Corresponding Mean MCC at age 20-35	.0795	.119	.159	.199	.2285
Average Number of Births	4.16	4.14	5.00	5.90	6.86
Variance of Number of Births	7.29	9.88	8.88	8.81	7.36

Marriage Distribution (b)

λ	$\frac{1}{2}$	$\frac{3}{4}$	1	$\frac{5}{4}$	$\frac{3}{2}$
Corresponding Mean MCC at age 20-35	.0795	.119	.159	.199	.2285
Average Number of Births	5.10	5.66	6.24	7.22	7.08
Variance of Number of Births	4.25	5.38	6.94	6.17	7.87

A functional relationship between λ and the average number of births (Y) was fitted for both sets of data. The form chosen was:

$$Y = \alpha(1 - e^{\beta\lambda}) + u$$

where u = error term

and α and β are parameters with α interpreted as the upper bound on Y, and a priori we expect $\alpha > 0$ and $\beta < 0$. The function was fitted by finding values of α and β which minimised the sum of squared errors. The parameters found were:

With Marriage Distribution (a) : $\alpha = 7.90$

$\beta = -1.15$

With Marriage Distribution (b) : $\alpha = 7.42$

$\beta = -2.12$

R^2 statistics were calculated at 0.8 and 0.9 respectively but little significance can be attached to these measures due to the non-linearity of the model. However, we were reasonably encouraged by the performance of this functional relationship, despite the fact that the larger α should be associated with marriage distribution (b). Larger samples (i.e. greater than 50) and simulations with more values of λ would we are convinced improve the plausibility of the relationship.

The most striking result of the experiment is the low response of average births (Y) to changes in MCC (λ), that is the low value of β. It is observed that increasing λ from $\frac{1}{2}$ to $\frac{3}{2}$ only increases Y by 2 or 3 births. The reason for this may lie partly in the fact that we are considering a distribution of MCC. For those families at the bottom end of the distribution, the change in λ will not imply anything but a very small absolute change in MCC yielding on average little increase in fertility. However, those at the top end of the distribution only have (on average) short periods of susceptibility but failure to conceive. Thus little increase in their fertility is possible. Also a rise in MCC partly offsets itself by increasing the number of no-risk periods associated with pregnancy and its aftermath.[56]

It should be noted that variances in number of births are generally lower with marriage distribution (b) and that coefficients of variation, if calculated, would decrease as λ increased. Standard errors of the averages

range from about 0.3 to 0.45.

The possibility of obtaining such an expression to relate changes in MCC to the expected number of children with no contraception may prove useful in developing further analysis using the C_n, C_d approach of Easterlin[59] to take account of the existence of factors leading to changes on both the supply and demand sides of reproductive behaviour. Our results might be taken as an indication that the most important sources of changes in C_n will in general be via mortality conditions rather than MCC. However until more is known about the nature, extent and causes of variability in MCC such a conclusion must be at best tentative. In particular we might expect different results from alternative types of variation in MCC, for example an equal absolute change for each woman.

Experiment 2

In Table 6 we report last closed birth intervals of families with 4 or more births with different targets and controls B_1 and S_1, with two levels of contraceptive effectiveness (E) 0.8 and 0.9. The sample size in each case was 200 and marriage distribution (a) was used. Averages and variances of numbers of births (all families) are also given. For instance, with control B_1 and a target of 5 births and a contraceptive effectiveness of .8, 100 of the 200 families in the sample had 4 or more births. For these 100 families the mean last birth interval was 45.3 months, and the variance of last birth intervals was 702 months. For the whole 200 families the average number of births was 3.31, and the variance of births was 4.64.

The 'no-control' mean number of births of 4.02 is less than the value found in Experiment 1 for E = 0. Apart from sampling variation, this can be explained by the ending of marriages due to the death of wife or husband before the marriage has become sterile.

Table 6 - An Analysis of the Last Birth Interval

Control B_1

Target in Births	E	Last Birth Interval			Number of Births	
		Number	Mean	Variance	Mean	Variance
3	.8	70	63.1	1615	2.84	2.95
3	.9	50	72.9	2137	2.47	2.44
5	.8	100	45.3	702	3.31	4.64
5	.9	101	43.3	679	3.30	4.51

Control S_1

Target in Survivors

3	.8	80	56.3	1251	2.95	3.84
3	.9	75	48.0	935	2.83	2.72
5	.8	102	51.5	963	3.58	6.50
5	.9	103	44.9	764	3.57	5.83
No Control		103	47.3	617	4.02	9.36

It is to be noted from Table 6 that a decrease in the target is accompanied by a decrease in the mean number of births and a rise in the length of the last birth interval which would accord with intuitive explanations and the theory discussed in Section II above. However, increasing E generally reduced both these statistics. The obvious exception is when the control is B_1 and the target is 3. Here the last birth interval will increase as E improves since fourth-born and later children are in all cases genuine accidents. Otherwise where E is increased the proportion of 'accidents' would decline and the expected direction of change of the statistic is ambiguous.

The possibility of the "perversity" of the last birth interval as an indicator is raised by the fact that MCC is a distribution. With the controls B1 and S1 only the more fecund (on the average) will reach T - at least if this is high - and even with contraception their following birth intervals may be no longer than the general population average for earlier birth intervals.

The identification problem of there being less genuine accidents discussed in Section II is also reflected in Table 7 and is once again related to the distribution of MCC. Intuitively we would expect falls in MCC to bring about rises in all birth intervals including the last one. This is a result we have generally observed in our experiments where no contraception is employed as reported in Table 7. However, after T is achieved there will be less people having accidents but those who do will be more concentrated in the upper deciles of the MCC distribution. The results in Table 7 suggest as expected that the behaviour of the last birth interval is more likely to be "perverse" the higher are T and E.

Table 7 - The Relation of the Last Birth Interval to MCC

λ	1	$\frac{2}{3}$	1	$\frac{2}{3}$	1	$\frac{2}{3}$
T	-	-	3	3	5	5
E	-	-	.7	.7	.9	.9
Mean LBI	48.2	54.4	59.4	59.4	52.0	46.5

The results reported in Tables 6 and 7 emphasise the possible difficulties of making inferences from observed outputs and seem to us to make clear the potential usefulness of simulation experiments to clarify by means of controlled experiments the plausibility of alternative hypotheses purporting to explain the observed changes. Furthermore reliance on the last birth interval to imply anything about contraceptive practice, at least in isolation, seems in general to be far from satisfactory, especially as our results indicate that in many cases it is not a very sensitive indicator which is particularly unfortunate when faced with problems of small samples or observed statistics containing a mixture of contraceptors and non-contraceptors.[58]

Experiment 3

In this experiment we compare the performance of the four controls B_1, B_2, S_1 and S_2 in achieving given targets. The results are presented in Table 8. They are based on samples of size 50. Marriage distribition (b) was used in this experiment and marriages were not prematurely terminated by the death of either partner.

Each control was assessed with regard to two different targets and with two levels of contraception effectiveness. The performance of the

controls is summarised by the average number of births or survivors (which is appropriate to the target) and the mean squared error (MSE) defined as:

$$MSE = \frac{\Sigma(A - T)^2}{n}$$

where A = actual number of births (or survivors)

T = target number of births (or survivors)

n = sample size (50)

Two results can be seen immediately from Table 8. Firstly, the more sophisticated controls B_2 and S_2 perform better with only one exception, than the simple controls B_1 and S_1, in terms of mean squared error. Secondly B_2 and S_2 produce a lower average number of births (survivors) than B_1 and S_1, again with only one exception. Both these results are to be expected, as some thought to the future should increase target accuracy, especially in terms of preventing exceeding the targets.

The results bring out the problem of the likely lack of correspondence between desired and actual fertility even with relatively sophisticated controls. They also suggest that changes in actual fertility in both proportional and absolute terms may tend to be lower than the changes in desired fertility which brought them about, thus stressing the dangers of an exclusively demand oriented approach to fertility.

Table 8 - Relative Performance of Different Controls

Control	E	Target (Births)	M.S.E.	Mean Number of Births
B_1	.7	3	6.54	4.46
	.7	5	4.18	5.14
	.9	3	1.82	3.54
	.9	5	2.44	4.72

Table 8 - continued

Control	E	Target (Births)	M.S.E.	Mean Number of Births
B_2	.7	3	4.40	3.72
	.7	5	2.98	4.78
	.9	3	1.52	3.16
	.9	5	2.88	4.56
		Target (Survivors)		Mean Number of Survivors
S_1	.7	3	2.20	3.72
	.7	5	3.74	4.40
	.9	3	0.82	3.26
	.9	5	2.62	4.18
S_2	.7	3	2.14	3.50
	.7	5	3.00	4.00
	.9	3	0.76	2.88
	.9	5	1.92	4.44

Experiment 4

So far experiments have been based on a once-and-forever desired target. However it seems reasonable to consider that individuals change their minds over the lifetime of their marriage. This might be caused by the following:

(i) A change in tastes possibly because of the additional information on the utility and cost of having further children gained from the experience of the first child.

(ii) A change in information concerning MCC as evidence becomes available (i.e. waiting-to-conceive times are observed). This has implications concerning the opportunity cost of having a

given number of children, in terms of lost wife's earnings in the case of an intertemporally disposed set of births, rather than a 'bunched' one.

(iii) Changes in prices, incomes, etc.

(iv) Changing views about what is and what is not feasible due to the pessimism or optimism of the family's continuous evaluation of the uncertain future.

In this experiment we consider a reevaluation of the family's target after every birth in the light of a different view of future events. The views differ only by sampling variation from the particular probability distributions that apply to future events. Perfect knowledge of all these distributions and the family's own MCC is assumed, and there are assumed to be no changes in tastes or price/income parameters. We are therefore restricting ourselves to (iv) above. For simplcity we will assume marriage distribution (b), a contraception effectiveness of one (i.e. no accidents), and no premature ending of the marriage through death of one of the marriage partners. Also the family will be maximising the utility function:

$$U = n - bn^2 + cY - .001C$$

where n = number of births

Y = lifetime family income stream which is assumed to be given by

$$Y = Y_H + \alpha Y_F \quad \text{where}$$

Y_H = the husband's income plus wife's income before marriage and after 50 years of age.

Y_F = the maximum possible wife's income from employment between marriage and 50 years of age.

α = the proportion of the time interval between marriage and 50 years of age that the wife can work, given that she can only work when she has no children under five years old.

C = number of months contraception is practised. The coefficient .001 was chosen arbitrarily, and represents the cost in utility terms of a month's supply of contraceptives.

The utility function chosen has several implicit assumptions which should be pointed out. Firstly there is an obvious but uncertain trade-off between n and α; the family will choose that n which maximises U given its belief as to this trade-off. Suppose this is n^*. Then n^* will not be affected by changes in Y_H. Thus, in a limited sense, children are a neutral commodity in the sense of being neither inferior nor superior. We have incorporated this assumption as we have no a priori reason for believing that n^* should react positively or negatively to changes in Y_H in a general context. The marginal utility of births diminishes if $b > 0$ for constant Y. One could say that in the absence of any opportunity cost in wife's earnings, the extra utility from having the r^{th} child is negative if $r > (b + 1)/2b$. We report experiments involving two values of b, $b_1 = 0$ and $b_2 = \frac{1}{12}$, implying $r = \infty$ and 6 respectively.

Apart from b, the only other parameter of the utility function that has to be assigned a value is $c\ Y_F$ which is the utility derived from the income of a wife in the period between marriage and 50 years of age when she has no children. This can be compared with the unit of measurement of the utility function (if $b = 0$), which is the utility (net of actual cost) derived from the first child given no loss of income due to that child. We considered values of 10, 5 and 2 for this parameter.

The model allows the family to choose an initial target (T_1) by comparing the different (simulated) utility levels from contracepting after the birth of successive children. The highest utility level defines T_1. The process is repeated after the birth of each child, and a sequence of targets is thus produced. The final target (T_2) either implies that

the actual number of births (A) is equal to T_2 or that the target was not in fact achieved at the termination of the marriage. Thus $T_2 \geq A$.

The results of this experiment can be summarised as follows. Each case was considered on the basis of a sample of 50 families with identical utility functions. With C = 10, $b = \frac{1}{12}$, no births occurred whatsoever. With C = 10, b = 0, 33 families had no births and with C = 5, b = 0, 19 had no births. With C = 2, b = 0 and C = 2, $b = \frac{1}{12}$, the average T_1, T_2 and A are given in Table 9, together with the mean squared deviation (MSD) of T_1 from T_2 and of both T_1 and T_2 from A.

Table 9 - Initial and Final Targets and Actual Numbers of Births

C = 2, b = 0

mean T_1	= 7.24	mean $(T_1 - T_2)^2$	= 4.36
mean T_2	= 8.48	mean $(T_1 - A)^2$	= 2.70
mean A	= 6.84	mean $(T_2 - A)^2$	= 4.00

C = 2, $b = \frac{1}{12}$

mean T_1	= 4.66	mean $(T_1 - T_2)^2$	= 0.76
mean T_2	= 4.36	mean $(T_1 - A)^2$	= 1.30
mean A	= 4.12	mean $(T_2 - A)^2$	= 0.32

Where b = 0, the marginal utility of children net of opportunity cost was frequently viewed to be increasing, as the additional period of lost earnings from having another child was often expected to be much less than 60 months, particularly for families with large MCC. Thus, in this case families were rarely satisfied with the number of births they had had,

and usually wished to revise targets upwards. Thus here T_2 is much larger on average than T_1, and the mean squared deviations are all quite large, particularly those involving T_2. Where $b = \frac{1}{12}$, the situation is seen as being considerably different. Here targets would sometimes be revised downwards and hit the actual values thus signalling contraception. In this case the mean squared deviations were all smaller.

Thus the sequence of targets will vary in their pattern according to the chosen value of b, that is according to the family size which implies negative marginal utility for further births. Obviously the expected impact of contraception becoming available is extremely sensitive to the assumed form of the utility function, in particular with the function used in this experiment the value of b would appear to be critical. Also the measurement of the impact would not be invariant to which a sequence of targets is chosen, as for instance T_1 and T_2 are not generally the same.

The adoption of a utility function approach and the generation of a sequence of targets is an obvious and interesting extension. However, one crucial question that this experiment raises is that of the intervals between reevaluation. In the model used here, for instance, no reappraisal takes place after some target has been reached.

This experiment has shown that T_1 is in generalneither equal to T_2 nor A. This is despite full information concerning MCC on the part of the families, no changes in tastes or exogenous parameters and completely effective contraception. Also as births rather than survivors are considered as the appropriate argument in the utility function no reassessment in the light of child mortality is required. The necessity of a model producing a sequence of targets from a sequence of decisions has been demonstrated with only the family's changing view of the future being additional to the conventional static lifetime utility maximisation model described in

Section II. Evidently the more general the model in terms of incorporating non-full information and changing parameters, the less possible is an analytical model of the conventional kind and the more appropriate are approaches based on simulation.

VII Conclusions and Implications

We have considered in this paper the role of simulation in the analysis of family formation. In particular we have argued that a simulation approach is an essential link between theories of family formation and the interpretation of observable data. To ignore either theory or data is inefficient and can lead to spurious conclusions as suggested in Section II. To demonstrate the validity of a simulation approach we presented in Section IV a simple model of family formation and considered in Section V questions of data input including possible sources. Of course, any simulation is only as representative of the real system as the quality of the available data with allow. However there are two mitigating points. Firstly a simulation approach creates an explicit need for better and new forms of data, the collection of which is self-rewarding. Secondly simulation allows a reasonable enquiry into the sensitivity of the system to errors in data inputs.

In Section VI we reported various experiments to illustrate applications of the approach. The first was concerned with the connection between the input - MCC - and the output - average 'maximum' fertility. The second experiment investigated the ability of the last birth interval to signal changes in contraceptive practise. Both these examples show the advantages of conducting 'controlled experiments' under 'laboratory' conditions. The last two experiments considered variations in the central part of the model - the decision-making section. Experiment 3 contrasted different stochastic control processes and demonstrated that the method of achieving

a given target (i.e. the choice of control) would effect performance. The final experiment considered just one of many mechanisms which result in 'changes of mind'.

We would like to put forward two implications of the analysis in this paper. Firstly, simulation of a model with a simple control but careful choice of data could be profitably used in conjunction with empirical data such as obtained by methods of family reconstitution in, for instance, an anlysis of the causes of the Demographic Transition. Secondly, it seems undeniable that a model incorporating the stochastic elements of the model in Section IV and perhaps 'reassessment of target' opportunities as discussed in Experiment 4 of Section VI could not yield results other than by simulation.

The work reported here is the initial stage of an on-going research project. It is hoped, however, that this paper might provoke research and discussion in the use of simulation of models of family formation.

FOOTNOTES

1. Valuable summaries of much of this work are to be found in J. Bourgeois-Pichat, 'Les facteurs de la fecondité non dirigée', Population, 20 (1965) pp 383-424. G. Hawthorn, The Sociology of Fertility (London 1970). R.A. Easterlin, The Economics and Sociology of Fertility: A Synthesis (mimeographed University of Pennsylvania 1973).

2. A more complete statement may be found in F.W. Notestein, 'The Economics of Population and Food Supplies', in Proceedings of the Eighth International Conference of Agricultural Economists (London 1953).

3. R.B. Tabarrah, 'Towards a theory of demographic development', Economic Development and Cultural Change, 19(1971) p. 359.

4. T.W. Schultz, 'The value of children: an Economic perspective', Journal of Political Economy, 81(1973)p. 54.

5. A.J. Coale, 'Factors associated with the development of low fertility: an historic summary', in Proceedings of the World Population Conference vol II (United Nations 1967) pp. 205-209.

6. A.J. Coale, op. cit., p.209.

7. G.S. Becker, 'A theory of the allocation of time', Economic Journal, 75 (1965).

8. G.S. Becker, 'An economic analysis of fertility', in Demographic and Economic Change in Developed Countries (N.B.E.R. Princeton 1960) p.231.

9. See for example J.M. Stycos and K.W. Back, The Control of Human Fertility in Jamaica (Ithaca 1964). This "explanation" ignores the stochastic nature of reproduction; see below p. 7.

10. For brief summaries and references to this literature see T.P. Schultz,' Explanation of birth rate changes over space and time: a study of Taiwan', Journal of Political Economy,81 (1973) pp. S238-274. B.S. Janovitz, 'An Econometric analysis of trends in fertility rates' , Journal of Development Studies, 9(1973) pp. 413-425.

11. This has been pointed out forcibly by L. Phillips, H. Votey and D. Maxwell, 'A Synthesis of the Economic and Demographic models of fertility: an Econometric test', Review of Economics and Statistics, 51 (1969) pp. 298-308.

12. G. Carlsson, 'The decline of fertility: innovation or adjustment process', Population Studies, 20 (1966) pp. 149-174.

13. G. Carlsson, op. cit., p. 151.

14. G. Carlsson, op. cit., p. 159.

15. G.S. Becker, Demographic and Economic Change... op. cit.

16. H. Leibenstein, 'The Economic Theory of Fertility Decline, (Harvard Institute of Economic Research Discussion Paper no. 292 1973).

17. "Ideal" family size is that chosen in a completely costless environment, "target" family size is based on preferences when costs exist. For a full discussion see R.A. Easterlin, loc. cit.; "ideal" corresponds to his C_D and "target" to his C.

18. This technique has become quite popular both in historical studies e.g. E.A. Wrigley, 'Family limitation in pre-industrial England', Economic History Review, 19(1966) pp. 82-109, and examinations of birth control programmes e.g. R.G. Potter, 'Birth intervals: structure and change'. Population Studies, 17(1963) pp. 155-162.

19. i.e. where the marriage survived until the wife was 45 years of age.

20. E.A. Wrigley, loc. cit.

21. R.A. Easterlin, op. cit., p.61.

22. R.A. Easterlin, op. cit., p.3.

23. R.J. Willis, 'A new approach to the economic theory of fertility behaviour', Journal of Political Economy, 81(1973),p.S17

24. K. Davis and J. Blake, 'Social structure and fertility'. Economic Development and Cultural Change, 4(1956), pp.211-235.

25. R.A. Easterlin, op. cit., pp.31-59.

26. R.A. Easterlin, op. cit., p.56.

27. T.P. Schultz, op. cit., p.S239.

28. See for example J.C. Ridley and M.C. Sheps, 'An analytic simulation model of human reproduction with demographic and biological components', Population Studies, 19 (1966), pp.297-310. M.C. Sheps and E.B. Perrin, 'Changes in birth rates as a function of contraceptive effectiveness : some applications of a stochastic model', American Journal of Public Health, 53(1963),pp.1031-1045.

29. J.C. Ridley, M.C. Sheps, J.W. Lingner, J.A. Menken, 'On the apparent sub-fecundity of non-family planners', Social Biology, 16(1969), pp.24-28.

30. M.C. Sheps and E.B. Perrin, op. cit., pp.1042-3.

31. See for example K. Venkatacharya, 'Some problems in the use of open birth intervals as indicators of fertility change', Population Studies, 26 (1972)pp.495-505.

32. This is not the same as natural fertility or fecundity which are concepts based on the whole of a woman's reproductive career including periods of insterility, pregnancy and amenorrhea.

33. For differing views see L. Henry, 'La fécondité naturelle: observation-théorie-resultats', Population, 16(1961),pp.625-634. R.G. Potter and J.M. Sakoda, 'Family planning and fecundity', Population Studies, 20 (1967),pp.311-328.

34. Much of the available evidence is summarised in J. Bourgeois-Pichat, loc. cit.

35. E.A. Wrigley, 'Mortality in pre-industrial England: The Example of Colyton, Devon over three centuries', Daedalus, 97 (1968), pp. 546-580.

36. J. Stoeckel and A.K.M. Alauddin Chowdhury, 'Neo-natal and postnatal mortality in a rural area of Bangaladesh', Population Studies, 26 (1972) p.115.

37. United Nations, Foetal, Infant and Early Child Mortality (New York 1954)

38. E.A. Wrigley, 'Family limitation...', op.cit., p.88.

39. E. Gautier and L. Henry, La Population de Crulai: Paroisse Normande (Paris 1958).

40. See for example L.Henry, op. cit., p.630, J.C. Ridley et al., op. cit., p.26, E. Gautier and L. Henry, op.cit.,p.117

41. See the discussions in R.G. Potter and J.M. Sakoda, loc. cit. J. Bourgeois-Pichat, loc.cit.

42. T.K. Roy and K. Venkatacharya, 'An application of analysis of variance technique to Monte Carlo data of human reproduction', Sankhya B, 33(1971), pp. 293-304.

43. J.C. Barrett, 'Use of a fertility simulation model to refine measurement techniques', Demography, 8(1971),p. 481.

44. G.K. Döring, 'The incidence of anovular cycles in women', Journal of Reproduction and Fertility, Supplement 6(1969)pp.77-81.

45. J. Bourgeois-Pichat, op.cit., pp. 407-410.

46. T.K. Roy and K. Venkatacharya, loc.cit., consider three amenorrhea distributions with means of 10.8, 14.7 and 20.1 months. We used the 10.8 months mean in our experiments.

47. See R.G. Potter, loc.cit.

48. See L. Henry, Anciennes Familles Genevoises (Paris 1956).

49. Interrelations such as these are difficult to identify. It can be argued that nutrition, for instance, affects both MCC and amenorrhea and so these will be correlated. For evidence of some effects of nutrition on amenorrhea see G.A. Saxton jr. and D.M. Serwadda, 'Human birth interval in East Africa', Journal of Reproduction and Fertility, Supplement 6 (1969), pp.83-88.

50. Similar to the values reported in F.E. French and J.M. Bierman, 'Probabilities of fetal mortality', Public Health Reports 77(1962), pp.835-847.

51. United Nations, loc. cit.

52. A Retel-Laurentin, 'Fecondité et syphilis dans la region de la Volta Noire', Population, 28 (1973) pp. 7931815.

53. See S. Shapiro, E.W. Jones and P.M. Densen, 'A Life-table of pregnancy terminations and correlates of fetal loss, 'Millbank Memorial Fund Quarterly, 40(1962),pp.7-45.

54. B. Berelson, 'KAP Studies on fertility', in B Berelson (ed), Family Planning and Population Programs (Chicago 1968).

55. R.T. Michael, 'Education and the derived demand for children', Journal of Political Economy, 81(1973),p.S142.

56. This effect has been noted previously, see for example R.G. Potter and J.M. Sakoda, op.cit., p325.

57. R.A. Easterlin, loc.cit.

58. This is especially important in an historical context and is more fully discussed on our forthcoming Warwick Economic Research Paper,'Family Limitation and the U.K. Demographic Revolution: A Simulation Approach.'