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This paper is circulated for discussion purposes and its contents should be considered preliminary.

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* The authors are lecturers in Economics at the University of Warwick. They are grateful for the research assistance of Mrs. D. Ellwood. They would also like to express their debt to participants of seminars at the Universities of Pennsylvania, Cambridge and Oxford. Responsibility for errors of omission and commission remain their own.

(b) R. E. Jones : Moreton Say, Shropshire

	Mean Birth Interval 1-2,2-3,3-4 (months)			Mean Last Birth Interval (months)			Mean Age at First Marriage (Women)(years)			Mean Completed Family Size (AFS)		
	Farmers	Labourers	ALL	Farmers	Labourers	ALL	Farmers	Labourers	ALL	Farmers	Labourers	ALL
1681-1720	28.9(85)	32.8(68)	30.6(153)	40.5(35)	46.6(29)	43.2(64)	-	-	-	4.7(15)	4.6(11)	4.7(26)
1721-1760	28.0(73)	28.4(130)	28.1(203)	41.7(28)	36.8(46)	37.3(74)	26.0 (13)	28.1 (33)	27.4 (46)	4.6(13)	4.9(33)	4.8(46)
1761-1800	26.5(64)	30.4(223)	29.5(287)	30.6(21)	44.4(79)	41.5(100)	26.9 (22)	29.0 (52)	28.4 (74)	6.2(22)	5.0(52)	5.4(74)

Notes: Figures are taken from Jones, op. cit.
Figures in brackets are numbers of observations kindly supplied privately by the author. We have altered his periodisation in line with his textual argument to reduce the small samples problem; this does obscure the apparent decline in labourers AFS after 1760.
Last birth interval figures are for women of all family sizes, no observation occurs in both categories.
"Farming families" are those which farmed ten or more acres, "labourers" are all others.

(c) D. J. Loschky and D. F. Krier : Over Kellet, Gressingham and Claughton, Lancashire, 1648-1812

Occupation	Mean Age at First Marriage (Women)	Expected Births (F_e)	Mean Actual Births (F)	Probability that $F \neq F_e$
Labourers	22.60 (10)		5.67 (9)	
Gentry	24.57 (7)	4.6	3.21 (14)	t = 1.58
Clergymen	25.00 (8)	4.4	4.69 (13)	
Craftsmen	25.00 (36)	4.4	3.57 (62)	t = 2.48
All Poor	25.11 (9)	4.4	4.61 (31)	
Farmers	27.31 (52)	3.6	3.80 (94)	t = 0.69
Tradesmen	28.86 (14)	3.0	2.69 (13)	

Notes : Figures are taken from Loschky and Krier op. cit.
Figures in brackets are numbers of observations.

The hypothesis of family limitation is in each case based, as it has to be, on the use of indirect evidence. Investigators must rely on proxy indicators to reflect non-observable family planning decisions. However, to infer that birth control occurred we need to isolate the contribution of contraception from that of the many other variables which are likely to affect the course of fertility itself and our chosen indicator. In general this is difficult not only because actual fertility is likely to differ from target fertility but also because many important factors such as changes in fecundability, miscarriage rates etc. are impossible to measure wholly satisfactorily in today's advanced countries and are simply not known in historical populations. Further, in order to use indirect indicators we require a theory to relate them to the underlying decision process.

These problems can be illustrated with relation to the use of the last birth interval (LBI) as an indicator of family limitation. Its use by Wrigley and Jones is based on a theory of Henry.[1] The hypothesis is that control would be over total size rather than spacing of families ; long last intervals occur through "errors" or "changes of mind" with a tendency to raise the mean last birth interval for the group concerned. It is clear that the usefulness of LBI as an indicator depends heavily on the validity of this model for the group under investigation. Moreover, birth intervals are comprised of several components - pregnancy, pregnancy wastage, amenorrhea, anovulatory cycles and waiting to conceive (which will depend on whether and how effectively contraception is used). In general each of these components may be variable both between groups of women and over time. Any inference concerning changes in family limitation practices must take into account the possibility that in historical reality these other factors may not have remained constant, in other words that like other potential indicators interpretation of the LBI is subject to identification problems.

Both our knowledge of demographic change during the industrial revolution and the theory of family formation[2] would suggest that the appropriate level of approach is a disaggregative one. We rely therefore on the analysis of individual parish registers. In practice, as is the case with the evidence summarised in Table 1, this tends to imply that in order to avoid

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1. See L. Henry, Anciennes Familles Genevoises (Paris 1956), esp. pp. 93-110 .

2. See the discussion in N.F.R. Crafts and N.J. Ireland, "The Role of Simulation Techniques in the Theory and Observation of Family Formation," Warwick Economic Research Paper 39 (1974), Coventry.

dealing with samples having very few observations we have to be prepared to assume that these villages' fertility history can be analysed satisfactorily by collecting observations over long periods.[1]

Our discussion of the determinants of fertility change has made it clear that any attempt to explain differences in fertility performance is faced with the difficulty of discriminating between a large number of alternative hypotheses. It is perhaps not surprising therefore that doubts have been expressed over the acceptability of the "pre-industrial" family limitation evidence. The literature does seem to leave some important issues in need of clarification.

(a) Is the last birth interval a good indicator of birth control?

(b) Are there any plausible alternative hypotheses which are consistent with the data of Table 1?

(c) What contraceptive effectiveness would be required to produce the observed changes?

(d) What sort of changes in target family size (T) would be implied?

(e) If family limitation should properly be thought of as "induced", what were the important independent variables?

If one considers reproductive experience as a system having <u>inputs</u> of non-observable behavioural parameters and distributions and <u>outputs</u> of observable family activities, then to answer these questions it is necessary to consider the relationships between the <u>sets</u> of inputs and <u>sets</u> of outputs. It is our thesis that this can be aided by the construction of a simulation model. There are several ways in which simulation can help.

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1. Wrigley's periodisation was "chosen to maximise the difference between the main periods of Colyton's demographic history". 'Family Limitation' op. cit. p. 87.

(1) In general, as we have argued above, observed outputs are affected by the interplay of a variety of non-observed inputs. This limits the usefulness of analysis based directly on observed historical data, itself the result of non-controlled experiments. Simulation allows controlled experiments and permits the assessment of magnitudes of changes in inputs which would be consistent with observed changes in outputs.[1]

(2) There is also 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.

(3) Most changes in the set of inputs can be expected to have both direct and indirect consequences involving offsetting and sometimes counter-intuitive effects. The use of probability models cannot, however, yield explicit analytical results where any acceptable degree of "historical realism" is involved because the parameters involved are normally time-specific. A simulation model does not need to have an analytic solution and therefore does not requre the "unrealistic" simplifying out of stochastic elements.

In the next section we present an example of a simulation model and discuss its limitations as a technique in historical demography. In Section IV we consider data problems, in Section V we report some experiments designed to explore the strength of the family limitation evidence and in Section VI present our conclusions.

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1. As will be seen it is necessary to distinguish between those changes in family behaviour which would account for all the observed changes from those which account for enough to make the residue statistically insignificant.

III

The simulation of family reproduction behaviour in the societies that we have cited requires both a satisfactory model and reasonable data. The model that we have used has many provisions not all of which are necessary for the consideration of the major hypotheses investigated in Section V. We confine our attention here to just those provisions that we have used. A more detailed exposition of the model particularly in terms of its stochastic control nature can be found elsewhere.[1] It should be noted that where parameters or distributions are described in the discussion here as 'known' it is implied that they are data inputs.

(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 the wife becomes permanently sterile which is assumed to occur at age 45 years.[2] 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.

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1. See Crafts and Ireland op. cit. pp. 10-16.

2. Note that the evidence from family reconstitution studies is gained by a similar concept of effective marriage implicit in the use of the "completed family".

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, and to be 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 $\{0 < x < 100\}$. 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 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. Otherwise it is assumed that there are twelve ovulatory cycles per year.

(iv) Outcome of Pregnancy

It is assumed that a conception may fail to produce a live birth in two ways. First, 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 assumed independent probabilities of 0.2 and 0.02 respectively. It is assumed that there are no multiple births, that the probability of a male child is 0.5, and that pregnancy lasts for nine months.

(v) Contraception

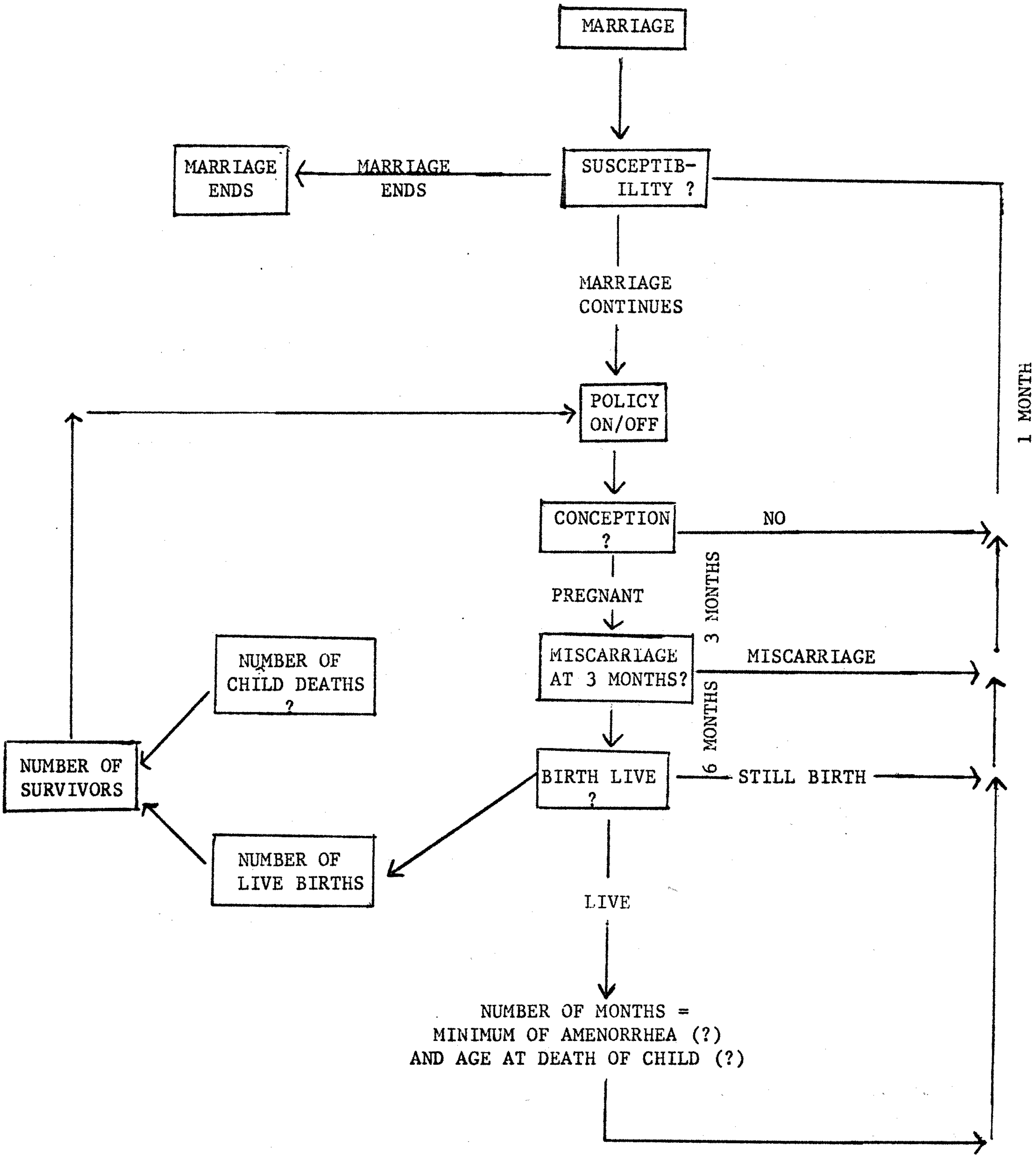
Contraception is assumed to have a known effectiveness $E(0 \leqslant E \leqslant 1)$ such that if n is the MCC $(0 \leqslant n \leqslant 1)$ then the probability of becoming pregnant in a given susceptible month is $n(1 - \mu E)$ where $\mu = 1$ if contraception is used (policy on) and $\mu = 0$ if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

(vi) Control

The family has a degree of control over its size by using or not using contraception. 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 a simple control mechanism: Each month if the number of surviving children is greater or equal to T (target family size) then policy on, otherwise policy off. We have assumed for the purposes of the experiments reported here that T is constant over the lifetime of the marriage. The ages at death of the children born are random independent observations from known mortality distributions.

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 number of women considered is the required size of the sample. All experiments reported here are based on a cohort of 100 women.

A Diagrammatic Representation of the Model



? indicates choice by random process

Our aim is to use this model to clarify possible changes in inputs which could be consistent with the changes in outputs reported in the studies which hypothesise family limitation. The suitability of simulation for this purpose depends on the quality of both the models and the data in terms of their ability to permit the performing of experiments relevant to the historical circumstances in question.

As we have argued, fertility is influenced by a number of non-observable inputs. It is in general the case that any given set of observed outputs could be consistent with a number of combinations of inputs. Further both the outputs resulting from simulation experiments and the evidence resulting from reconstitution studies are subject to sampling variation. Hence we could never claim to have definitely "recreated" the villages being examined, the best we can say is that our set of inputs is capable of reproducing the observed historical outputs, subject to sampling variation.

Inference of family limitation from the available indicators relies on a theoretical model, whether implicit or explicit; our simulations incorporate a "naive" theory of family formation in terms of the control strategy assumed. Obviously if birth control was used it need not necessarily have been practised in this way. We have adopted this control mechanism because it typifies the one envisaged by Wrigley and Jones[1] whose plausibility we wished to examine.

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1. Wrigley, 'Family Limitation', op. cit. pp. 93-5; Jones op. cit. pp 19-22.

These problems, especially the importance of non-observables and the need for theory, are indeed formidable, but they apply to all attempts to "explain" historical fertility change. For example, Wrigley in putting forward his hypothesis had to base his arguments on the plausibility of putative changes in an implicit set of inputs[1] which may or may not correspond with the historical reality of Colyton's non-observable system of behavioural parameters. Rather than encountering new difficulties our method of approach makes apparent some which have previously been overlooked.

IV

The propositions that we consider in the next section are concerned with the examination of the effects on observed fertility behaviour of changes in the data inputs. We feel that exphasis on the analysis of changes rather than attempts to recreate exact circumstances in absolute terms is less likely to be erroneous and is capable of yielding insights without requiring such recreation. However, we have attempted to ensure that the data used is compatible with existing evidence for low income communities in the cases where it is not obtainable from the original village studies and that it is able to approximately reproduce the observed demographic characteristics of eighteenth century rural England as reported by Wrigley and Jones. Loschky and Krier's work does not provide sufficient detail for us to know whether this criterion would be satisfied, but our simulations do allow us to make some comments on the robustness of their results.

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1. See the arguments in Wrigley, 'Family Limitation,' op. cit., especially pp. 100-101.

We describe this data below.

(i) The age at marriage of a woman is a random observation from cumulative probability distributions given in Table 1. Distribution (b) is the distribution (a) when only considering a sample of women who all married before the age of 30. Distribution (d) is similarly related to distribution (c). All distributions are constructed by interpolation from information relating to Colyton;[1] distributions (a) and (b) relate to the period 1560-1646, distributions (c) and (d) to 1647-1719.

(ii) A mortality distribution is required to translate births into survivors for the consideration of proposition (1) in the next section. This distribution is given in Table 3; it is an amalgamation of information relating directly to Colyton and a U.N. model life table.[2]

(iii) The probablity distribution of amenorrhea is taken from Roy and Venkatacharya [3] and is based on recent Indian experience. This distribution is summarised in Table 4.

(iv) The measurement of MCC is difficult. In practice measures of this "input" have to be inferred from "outputs" such as time of waiting to conceive and this makes it difficult to distinguish changes in "natural fecundability" from sterility or changed coital frequency as well as leading to the strong possibility of biased results.[4] Not surprisingly

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1. Wrigley 'Family Limitation,' op.cit. tables 2 and 3 pp.87-8; Jones does not supply information on the distribution of age at marriage but the means are very close to Wrigley's, (see above Table 1), and the differences are unlikely to be important, see below pp. 23-25.

2. Wrigley, 'Mortality,' op. cit. pp.568-71 and U.N. model life table level 60. These values are slightly higher than the suspiciously low rates reported by Jones but are themselves somewhat low compared with the mortality characteristics frequently assumed to have prevailed in such societies. A slightly higher or lower level would not change the tenor of our results.

3. P. K. Roy and K. Venkatacharya, "An Application of Analysis of Variance Technique to Monte Carlo Data of Human Reproduction," Sankhya B XXXIII (1971) 295.

4. For recent discussions of the problems of estimating fecundability, see W. H. James, "The Fecundability of U.S. Women," Population Studies XXVII (1973) 433-500 and D. Wolfers, "Determinants of Birth Intervals and their Means," Population Studies XXII (1968) 253-262.

quite a wide range of values have been suggested as "typical" for various age groups.[1] We assume two fundamental characteristics of MCC, (a) MCC varies between different marriages and (b) MCC is age-specific. We have used variations of the fecundability distribution, a Beta distribution, estimated by Barrett from data contained in the Irish census of 1911.[2] Each variation consists of a distribution defined for a given age of women plus a transformation of the percentiles of the distribution to yield cross-sectional distributions of MCC for any age of women. Two particular variations are basic and are reproduced in Table 5. Distribution A incorporates a pattern of increase to a plateau at age 20-34 followed by decline whereas in distribution B the plateau is replaced with a gradual decline.[3]

Family limitation parameters (target family size and contraceptive effectiveness) and registration failure parameters are discussed in the relevant parts of the next section.

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1. See for example the discussions in Wolfers op. cit., R. G. Potter and J. M. Sakoda, "Family Planning and Fecundity," Population Studies XX (1967) 311-328 and J. Bourgeois-Pichat, "Les Facteurs de la Fécondité non Dirigée," Population Studies XX (1965) 383-424.

2. J. C. Barrett, "Use of a Fertility Simulation Model to Refine Measurement Techniques," Demography VIII (1971) 481.

3. MCC differs from the notion of "natural fecundability" 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. In practice it is difficult to separate changes in "natural fecundability" from temporary sterility (anovulatory cycles) or changed coital frequency. MCC subsumes these latter considerations as well as fecundability. There are differing views among demographers as to whether fecundability varies with age; the thesis that it is approximately constant with age is expounded by L. Henry, "La Fécondité Naturelle: Observation - Théorie - Résultats," Population XVI (1961) pp. 625-634, whilst the opinion that it declines continuously from age 20 is to be found in James, op. cit. Distribution A is intended to be broadly in accord with Henry's views modified to accomodate the evidence on coital frequency in Bourgeois-Pichat op. cit. and on anovulatory cycles in G. K. Döring, "The Incidence of Anovular Cycles in Women," Journal of Reproduction and Fertility, Supplement 6 (1969) 77-81. Distribution B represents a James-type view.

Table 2: Assumed Marriage Distribution: probability of a woman being married by age t years

	t	15	20	25	30	35	40	45
Marriage distribution	a	0	.19	.50	.77	.87	.96	1.00
	b	0	.25	.67	1.00			
	c	0	.06	.36	.63	.78	.92	1.00
	d	0	.09	.58	1.00			

Table 3: Mortality Distribution: probability of death by age t.

t	1 month	12 months	5 years	10 years	20 years	30 years
Prob.	.0725	.145	.207	.225	.261	.312

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

t	6	12	18	24	30
Prob.	.29	.66	.87	.94	1.00

Note this distribution has a mean of 10.8 months and a standard deviation of 6.8 months.

Table 5 Deciles of MCC Distribution

Distribution I	Decile	Age 15-20	20-35	35-40	40-45
	0	0	0	0	0
	1	.058	.087	.029	.015
	2	.075	.113	.038	.019
	3	.094	.141	.047	.024
	4	.106	.159	.053	.027
	5	.125	.188	.063	.032
	6	.138	.207	.069	.035
	7	.150	.225	.075	.038
	8	.175	.263	.088	.044
	9	.213	.320	.107	.054
	10	.375	.563	.188	.094
	Mean	0.132	0.198	0.066	0.033

Distribution II

Let x be a randomly chosen observation from Distribution I age 15-20 yrs above.

Then MCC will vary according to the equations:

$$MCC = \left[2 - 2\,(240 - t)/540\right] x \qquad \text{if } t \leq 240$$

$$MCC = (2/3 + 4(480 - t)/720)x \qquad \text{if } 240 < t \leq 480$$

$$MCC = (2(540 - t)/180)x \qquad \text{if } 480 < t \leq 540$$

$$MCC = 0 \qquad t > 540$$

where t is the age of the woman in months

The mean MCC of women at 20 yrs, 30 yrs and 40 yrs of age are 0.262 0.176 and 0.088.

Note

These distributions are regarded as BASE versions. These base versions can be adapted to reflect changed levels of MCC ; proportional changes in each woman's MCC by a factor λ is the method generally chosen. For a base version $\lambda = 1$; increases in MCC imply that $\lambda > 1$, decreases that $\lambda < 1$. Absolute changes in MCC are also calculated as deviations from these base versions.

V

In this Section we will report simulation experiments designed to assist the interpretation of family reconstitution evidence as discussed in Section II. We must stress that we cannot reproduce the evidence exactly. Nor do we wish to do so as this evidence is only sample evidence and simulation will produce results which are themselves subject to sampling variation. Obviously, for this reason and also because of the possibility of modelling and data errors we cannot prove or disprove any explanation of the family reconstitution evidence. Nevertheless we believe that we can explore the plausibility of alternative explanations to the extent of estimating the order of magnitude of various changes in inputs capable of producing the changes in demographic conditions reported in the village studies.

The use of statistical tests will be limited to ad hoc remarks concerning broad confidence limits, etc. This is because we have taken the family reconstitution evidence as being 'point-estimates' of the true state of affairs, and have thus been concerned with examining all evidence and not just that which is statistically significant. Also the independence of the statistics that are considered is doubtful and the number of degrees of freedom questionable.

Of the three studies cited in Section II, Wrigley's has the strongest evidence for the proposition that family limitation existed in the pre-industrial economy.

The other papers seem less satisfactory both in terms of the data they report and their procedure in analysing it. Jones reports last birth intervals for completed families of <u>any</u> size (rather than for those of 4 or more children) which are susceptible to a number of disturbing influences [1] and there are difficulties in dealing with the periodisation of the article which tend to obscure the relationship between changes in completed family size and last birth intervals.[2]

Loschky and Krier in predicting expected family size assume that all groups would expect to have the same birth intervals in the absence of family limitation and that the mean family size of the 9 labourers observed is the population mean for those married at age 22.6 years.[3] Because of these assumptions and the absence of a systematic analysis of birth intervals there are problems for a direct assessment by simulation.

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1. Most notably changes in length of effective marriage.

2. This is principally due to the adoption of cohort analysis of completed family size but period analysis of the last birth interval coupled with the problem of small numbers inherent in the periodisation chosen by Jones. See Table 1 and Jones op. cit. pp. 11 and 18-19.

3. See Table 1 and Loschky and Krier op. cit. p. 441. Our simulations suggest this procedure is very doubtful, see below p.37 . There are also serious doubts over the validity of the significance tests used as the composition of the samples from which average age at marriage and average family size are calculated is not identical.

We will therefore confine our attention mainly to Wrigley's results, but we hope our conclusions regarding the legitimacy of the various propositions put forward as explanations can be considered as applicable to all the studies.

Before we consider family limitation as an explanation of the observed evidence, we will compare the different marriage distributions given in Table 2 in terms of their effects on birth intervals and mean number of births (AFS). The results are presented in Table 6. The upper part of the Table presents these results in the absence of any contraception. The lower part presents them when contraception is present with parameters E and T. E is the proportion by which MCC is reduced if contraception is used and T is the target family size such that if and only if the number of surviving children is not less than T contraception is used.

Table 6 permits a consideration of the results of a move from marriage distribution (a) to (c), or from (b) to (d) if attention is confined to women married before 30 years of age. The moves from (a) to (c) and (b) to (d) approximate the changes in marriage distributions in Colyton between the period 1560-1646 and 1647-1719 as reported by Wrigley; they are also similar in terms of changes in mean age at marriage to Jones' evidence.[1] The results in Table 6 suggest for example in the absence of contraception that a move from (a) to (c) would produce a slight (but not statistically significant) increase in LBI; a move from (b) to (d) on the other hand leads to a similarly small decrease in LBI. Also our simulation

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1. See above Tables 1 and 2.

Table 6 Comparison of 4 Marriage Distributions using M.C.C. Base Distribution I

MD	E	T	LBI			ABI			AFS	
			N_L	$\bar{X}_L$	S^2_L	N_A	$\bar{X}_A$	S^2_A	$\bar{X}_F$	S^2_F
a	0	-	79	41.9	502.1	231	28.0	171.5	6.03	11.23
b	0	-	91	46.6	654.5	269	27.6	160.7	7.27	8.96
c	0	-	68	44.6	682.7	198	27.8	173.0	5.43	8.84
d	0	-	89	44.2	554.1	269	27.6	181.7	6.95	7.41
a	.8	3	70	62.7	1542.0	184	30.6	317.9	4.27	5.46
b	.8	3	82	59.7	1416.0	218	30.0	317.3	4.74	3.53
c	.8	3	57	55.8	1285.3	151	27.1	258.9	3.85	5.37
d	.8	3	81	53.0	1124.7	214	28.1	295.7	4.67	2.20

Note MD, E, T, are as defined in the text. LBI, ABI, AFS are as described in Table 1, i.e. as used by Wrigley. N, $\bar{X}$ and S^2 are relatively the number of observations, mean and variance of the statistics presented. This notation is used throughout the rest of the tables.

predicts a fall in average family size of between one third and three-quarters of a child.

This contrasts with the changes in fertility observed by Wrigley in Colyton, where there was a 'significant' increase in LBI coupled with a much larger decrease in family size. We therefore conclude that changes of this type in marriage distribution are unlikely to provide a sufficient explanation of the changes in fertility observed by Wrigley. This conclusion would seem also to apply to the changes in fertility observed by Jones.

It is possible that a particularly odd-shaped marriage distribution might imply that our constructed distributions are unrepresentative. Even so, we cannot find an argument that will relate the LBI, Wrigley's chosen indicator of family limitation, to the marriage distribution in the absence of contraception.

In view of the importance placed on the LBI as an indicator of family limitation, we will precede our discussion of our attempts to simulate the evidence of fertility changes with a consideration of the sensitivity and suitablity of the LBI in this role. Then we will consider the proposition that changes in family limitation practice were indeed responsible for the observed changes in fertility, and finally we will consider two alternative explanations: that of a change in the MCC distribution and that of a change in registration behaviour.

In order to reduce the number of simulations, we will report each experiment for two of the four marriage distributions, one of the distributions being that of all marriages (distribution (a)) and the other of marriages before the wife reached 30 years of age (distribution (d)). Most experiments reported are performed with MCC distribution I. However the qualitative outcome of the experiments is not affected by changing to MCC distribution II. The last birth intervals found in experiments with MCC distribution I are generally higher; and those with MCC distribution II generally lower (see Table 9) - than those observed by Wrigley and Jones. A mix of the two distributions would thus produce LBIs of the appropriate magnitude. As it is the changes in these indicators rather than the absolute values that is of interest we will concentrate on MCC distribution I.

Proposition 1 : The observed fertility changes can be explained by the onset of family limitation.

The evidence for this proposition is centred on two main statistics: the magnitude of the fall in fertility and the increase in the LBI. Unfortunately, the reliability of the LBI as an indicator of family limitation practice is subject to an identification problem. The theory of family limitation adopted by Wrigley and Jones suggests that in the event of contraception "accidents "or "changes of mind" tend to increase the mean last birth interval (see above Section II). However, where MCC is a distribution there occurs the possibility of "perverse" changes in LBI. For example a general decrease in MCC will mean less people have "accidents" but those who do will be concentrated in the upper deciles of the MCC distribution. Even with contraception their following birth intervals may be no longer than the general population average for earlier birth intervals. In fact anything which tends to change the sample balance between "non-accidents" and "accidents" ("ordinary" and "extraordinary" LBIs) makes

Table 7 Family Limitation and LBI using M.C.C. Base Distribution I

MD	E	T	LBI N_L	LBI $\bar{X}_L$	LBI s^2_L	ABI N_A	ABI $\bar{X}_A$	ABI s^2_A	AFS $\bar{X}_F$	AFS s^2_F
a	.5	3	79	51.6	775.7	224	29.4	270.8	5.47	5.60
a	.8	3	70	62.7	1542.0	184	30.6	317.9	4.27	5.46
a	.9	3	54	57.2	1617.5	138	27.8	240.4	3.64	3.21
a	.8	6	77	46.8	671.6	223	30.7	227.1	5.44	6.69
a	.9	6	76	44.0	953.7	219	27.7	204.2	5.12	7.22
d	.5	3	82	47.9	651.8	231	29.3	228.1	5.35	7.39
d	.8	3	81	53.0	1124.7	214	28.1	295.7	4.67	2.20
d	.9	3	62	53.9	894.0	155	30.5	338.4	3.92	2.17
d	.8	6	90	47.9	749.0	258	29.3	199.1	5.77	4.52
d	.9	6	92	43.8	576.4	272	26.4	137.5	6.08	3.71

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the expected direction of change of LBI uncertain and hence renders it an <u>ambiguous</u> indicator.

Some illustrations of this problem are reported in Table 7. We present results of experiments concerning the impact of changes in target family size (T) and contraceptive effectiveness (E) on LBI. We see that a decrease in T with E held constant is accompanied by a decrease in mean AFS and an increase in LBI. This accords with intuitive predictions and the theory discussed in Section II above. Increases in E with T held constant generally produce falls in mean AFS as expected. However, the effects on LBI are less clear cut. We find that an increase in E from .5 to .8 would produce an increase in LBI, but from .8 to .9 generally to a decrease, the "perverse" case. With the better contraceptive technology, there would be less "accidents" in relation to "replacements" although LBIs for accidents would in general be longer. In the case where T = 6 the "ordinary" LBIs would become a greater proportion of the sample of LBIs. This is an example of the identification problem discussed above.

The results in Table 7 also allow an assessment of the magnitude of changes in target family size and/or contraceptive effctiveness which would be required to produce the fertility changes observed in Colyton. We can compare those results relating to marriage distribution d with what is observed when there is no contraception as reported in Table 8.

Table 8 Fertility with no Contraception: a base for comaparison with MCC Base Distribution I

MD	LBI			ABI			AFS	
	N_L	$\bar{X}_L$	S^2_L	N_A	$\bar{X}_A$	S^2_A	$\bar{X}_F$	S^2_F
d	89	44.2	554.1	269	27.6	181.7	6.95	7.41

It seems that fertility changes (in terms of LBI and AFS) of a similar order of magnitude to those observed by Wrigley for Colyton between 1560-1646 and 1647-1719 (see Table 1) could be consistent with various changes in behaviour. For example we could envisage a move from no contraception to contraception with a target of 3 and effectiveness of .8 approximately or a change in target from about 6 to about 3 with effectiveness constant at .9. However, the introduction of contraception with effectiveness of only .5 seems unlikely to fit the observed changes. Improvement in contraceptive effectiveness from a very low level such as .5 to .9 might account for the change in AFS but because of the eventually perverse effect on LBI would not be very likely to account for the observed changes in that variable. As we have already seen a change in effectiveness from .8 to .9 would not fit.

On the assumption that birth control was employed in the manner envisaged in the model, as Wrigley and Jones suppose, then ceteris paribus it would seem that proposition 1 could not be rejected given the existence of a contraceptive technique with $E \geq .8$ coupled with the imposition of a target of about 3 as compared with a previous situation of no target or a target ≥ 6, provided that this behaviour was adopted by (practically) all in the sample.[1] If we were prepared to take the evidence at face value similar remarks would apply to the most convincing cases in the other studies, the landlords in Moreton Say, and the craftsmen in Lancashire (see Table 1).

The question immediately arises as to how likely these conditions are to have been fulfilled.

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1. An alternative could be some sort of dispersion in behaviour with some having very low targets, at or about zero.

(a) Contraceptive Effectiveness

Evidently in practice E is very difficult to measure and the small amount of available evidence refers to effectiveness in use in advanced countries. This work strongly suggest that use effectiveness depends not only on contraceptive technology, but also on the determination and care with which the technique is used. Michael[1] has recently presented estimates for E with various techniques relating to the modern U.S.A. which suggest that, with efficient use, even poor techniques are capable of achieving $E > .8$ and the value of E for withdrawal, the method Wrigley supposes was used,[2] is put at .93.

(b) Target Family Size

We have no way of knowing what this was or whether villages or groups of people were in fact capable of achieving such flexibility in fertility goals. Recent survey evidence has suggested that "ideal family size" does not vary a great deal within developing countries on a cross-section basis, but the relevance of such evidence is at best very tenuous.[3]

The possibility of a family limitation explanation of the observed changes seems to remain open and within the scope of an efficient use of the likely contraceptive technology. This would seem to narrow the range of speculation somewhat but whether these conditions were fulfilled remains a matter of historical judgement.

We believe that the results have other important implications.

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1. R. T. Michael, "Education and the Derived Demand for Children," Journal of Political Economy 81 (1973), S142.

2. Wrigley, 'Family Limitation..' op. cit. pp. 104-5.

3. See for example the studies in B. Berelson (ed.), Family Planning and Population Programs (Chicago 1968). However there is no reason to suppose that "ideal family size" is necessarily equivalent to "target family size".

(i) Even if these conditions were not met, it may be that a different type of control was used e.g. involving control over spacing as well as size. Such a control might also be capable of explaining the rises in ABI observed by Wrigley which the Wrigley type control does not do.

(ii) LBI may not only be a "perverse" indicator in some circumstances, it may also not be a very sensitive one. This is suggested by the fact that rather a large change in target adopted by nearly all the observed sample is required for a clear effect on LBI. Contraception aiming at higher target family size adopted by a few could easily be indistinguishable from sampling variation and overlooked. This problem is illustrated by the divergent experience of the village, Moreton Say, and groups within the village. (See Table 1).

(iii) We believe that if family limitation was practised it is more likely to be an example of the 'adjustment' rather than the 'innovation' case of Carlsson.[1] We assert that in the framework of our model, 'adjustment' would take the form of a change in target (T) with given contraceptive technology represented by a constant value of E, whereas 'innovation' implies a change in E but no change in T. Obviously this is a somewhat oversimplified view.[2]

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1. See Carlsson op. cit. and above p.3.

2. 'Adjustment' in terms of a reduction in T might imply a greater return from more careful use of the given technique, thus increasing E. Also in the limit an arbitrarily high T and a zero E would produce the same outcome.

However, the large change in E required to reproduce the change in LBI is unlikely to have occurred in the communities considered. There is considerable evidence that techniques such as withdrawal were known at much earlier times than the periods under discussion. We therefore consider changes in targets (adjustment) as more likely than changes in effectiveness (innovation).

Proposition 2.: The observed fertility changes can be explained by a shift in the MCC distribution.

We report in Tables 9 and 10 average family size and birth intervals for some proportionate and absolute changes in MCC applying to all women at all ages. We regard the investigation of the effects of changes in MCC largely as an examination of the alternative hypothesis put forward by Chambers to explain events in Colyton. He argued that in the second half of the 17th century Colyton was probably subjected to a "new kind of epidemic history" which may have "included besides smallpox, typhus, influenza, diptheria, malarial ague, spotted fever, relapsing fevers, dysentery." [1] He maintains this led to "a fall in fertility reflecting, it would seem, psychological as well as biological factors." [2]

We can interpret this argument in terms of a two-fold impact on MCC. First that there was a change in health standards leading to lower fecundability and more anovulatory cycles. Second that there was a lower coital frequency, due both to the state of physical health and also to changes in mental and social attitudes.

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1. J. D. Chambers, "Some Aspects of E. A. Wrigley's 'Population and History,' " Local Population Studies 1 (1968) 21.

2. J. D. Chambers, Population, Economy and Society in Pre-Industrial England, (1972) p. 75.

In Table 9 results are reported for proportionate changes in MCC involving both base distributions and cases with and without contraception. Comparisons can be made between the original base distributions ($\lambda = 1$) and experiments involving $\lambda = 0.4$ and 0.8. In general we find that as λ decreases LBI and ABI increase. However the sensitivity of both AFS and LBI to changes in λ is small. This is particularly true for the LBI and is even more apparent in Table 10 which deals with absolutes rather than proportionate changes in MCC.

The low sensitivity of LBI with respect to changes in MCC is not surprising since a decrease in MCC would mean that families in the upper deciles of the MCC distribution would become a bigger proportion of the sample. Indeed in the experiments involving MCC distribution 1 with contraception reported in Table 9 some "perverse" results are observed.

Our experiments suggest that the reduction in MCC sufficient to reproduce approximately the decline in average family size generally fails to induce an increase in LBI of the magnitude reported in the village studies. However, the difference is not so great that it could not be explained by sampling variation. The fall in MCC would be capable of producing the rise in ABI observed by Wrigley. The magnitude of the change in MCC in the absence of contraception required to approximately reproduce the fertility evidence would indicate a change in mean MCC for women at age 20 years of from 0.198 to 0.079 (distribution I, proportionate change), from 0.264 to 0.106 (distribution II, proportionate change) and from 0.248 to 0.148 (distribution I absolute change).

Given the difficulty of measuring MCC there must be some uncertainty

Table 9 Proportionate Changes in MCC

MCC					L B I			A B I			A F S	
Distn.	λ	MD	E	T	N_L	X_L	S^2_L	N_A	X_A	S^2_A	X_F	S^2_F
I	0.4	a	0	-	59	51.7	1239.0	157	34.6	573.4	4.04	6.58
I	0.8	a	0	-	69	46.54	584.6	203	30.9	228.3	5.48	10.67
I	1	a	0	-	79	41.9	502.1	231	28.0	171.5	6.03	11.23
I	0.4	d	0	-	66	49.1	905.1	179	31.9	323.0	4.36	5.51
I	0.8	d	0	-	91	46.38	646.2	265	29.7	220.6	6.55	6.31
I	1	d	0	-	88	45.59	768.0	260	27.7	202.5	6.40	6.86
I	0.4	a	0.8	3	32	58.9	1363.0	79	28.2	492.7	2.78	3.15
I	0.8	a	0.8	3	62	58.1	1190.7	157	31.7	336.2	3.78	3.49
I	1	a	0.8	3	70	62.7	1542.0	184	30.6	317.9	4.27	5.46
I	0.4	d	0.8	3	49	71.3	1394.3	125	32.5	505.1	3.54	2.33
I	0.8	d	0.8	3	71	47.7	684.8	190	30.4	296.6	4.20	2.42
I	1	d	0.8	3	81	53.0	1124.7	214	28.1	295.7	4.67	2.20
II	0.4	a	0	-	55	48.2	560.6	156	34.0	423.3	4.07	8.16
II	0.8	a	0	-	81	34.9	318.3	231	29.6	174.3	6.31	10.65
II	1	a	0	-	77	34.1	231.9	225	26.7	168.8	6.57	13.87
II	0.4	d	0	-	73	40.7	462.1	205	24.5	439.2	5.03	5.81
II	0.8	d	0	-	88	38.8	331.8	254	30.6	238.7	6.33	7.62
II	1	d	0	-	96	34.0	307.1	284	28.0	167.3	7.57	6.13
II	0.4	a	0.8	3	48	62.5	984.3	124	24.0	570.9	3.39	3.92
II	0.8	a	0.8	3	68	56.2	1396.8	178	30.4	290.8	4.21	4.41
II	1	a	0.8	3	66	50.4	891.2	183	28.8	244.1	4.36	5.51
II	0.4	d	0.8	3	52	60.0	1044.4	135	34.5	650.4	3.68	1.98
II	0.8	d	0.8	3	75	56.0	1254.1	190	30.1	323.7	4.35	3.11
II	1	d	0.8	3	82	52.7	1151	221	30.9	298.9	4.49	2.93

TABLE 10 Effects of changing MCC by an absolute amount Δ .

Δ	M.D.	E	T	LBI N_L	LBI $\bar{X}_L$	LBI s^2_L	ABI N_A	ABI $\bar{X}_A$	ABI s^2_A	AFS $\bar{X}_F$	AFS s^2_F
-.05	a	0	-	65	48.2	667.8	185	31.1	403.8	4.87	9.55
+.05	a	0	-	82	43.4	479.7	241	26.6	131.4	6.99	11.85
-.05	d	0	-	78	44.3	493.7	220	30.5	230.0	5.41	6.84
+.05	d	0	-	100	39.8	317.7	300	26.8	133.9	8.02	5.00
-.05	a	0.8	3	46	58.5	995.6	121	32.7	543.2	3.25	4.39
+.05	a	0.8	3	76	51.3	761.9	205	26.6	213.6	4.72	3.60
-.05	d	0.8	3	58	59.7	1477.0	151	31.0	394.0	3.83	2.60
+.05	d	0.8	3	94	50.8	900.4	263	26.1	212.2	5.47	1.99

as to the likelihood of such changes taking place. Furthermore little is known about the causes of changes in intrinsic fecundability. There are suggestions in the literature that in some cases fecundability has varied over time [1] and it is known that natural fertility rates vary considerably between countries.[2] Certainly estimates of fecundability differ considerably. For instance Wolfers found means of around 0.1 - 0.15 for samples of Oriental women at age 20,[3] whilst Potter and Sakoda argued that for American women of that age "the most credible estimates of mean fecundability fall mostly within a range of 0.25 - 0.35," [4] How far this discrepancy is due to different methods of estimation rather than real differences in fecundability is not clear.

Demographic evidence does suggest that if changes of this magnitude in MCC were a result solely of changes in coital frequency this would imply a change in average frequency of intercourse from about 12 to 5 times a month.[5] Quantification of the impact of the diseases mentioned by Chambers is not available, though evidence from modern Africa suggests that an epidemic of venereal disease could certainly produce such changes in MCC.[6]

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1. See for example Henry op. cit.
2. Bourgeois-Pichat op. cit.
3. Wolfers op. cit. pp.407-9.
4. Potter and Sakoda op. cit. p. 317.
5. See P. Lachenbruch, "Frequency and Timing of Intercourse : Its Relation to the Probability of Conception," Population Studies 21 (1967), 23-31.
6. A. Retel-Laurentin, "Influence de Certaines Maladies sure la Fécondité: Un Exemple Africain," Population 27 (1972) 841-860.

Our experiments also suggest the following points.

(i) Whilst a fall in MCC by itself has very limited effects on LBI more pronounced effects could be forthcoming if it were hypothesised that disease affected amenorrhea and/or the miscarriage rate in an age or parity specific fashion. This is not altogether implausible. For this reason and the existence of sampling variation we cannot definitely reject the Chambers hypothesis, although, as we have suggested, the changed MCC proposition on its own is not particularly convincing.

(ii) The variances in birth intervals and average family size recorded in Table 9 provide further reasons for believing that Loschky and Krier's method of making a point estimate of F_e and therefore their test for birth control are of dubious validity.[1]

(iii) It has been frequently argued that rises in the expected number of surviving children, through rises in fertility or falls in child mortality may be an important cause of family limitations in developing countries. This does not seem to have been the case in Colyton. Wrigley himself has noted that the period saw an increase in child mortality.[2] Table 9 shows that rises in MCC together with contraception tend to reduce ABI which does not fit the Colyton experience.

Proposition 3 : The observed fertility changes can be explained by a change in the reliability of birth registration statistics.

Parish registers can only record registered births, and therefore

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1. See above Table 1 and p.

2. Wrigley, 'Family Limitation' op. cit. p. 101.

the evidence cited in Table 1 is subject to errors due to unregistered births. The registration failure rate may indeed change sharply over time. It has therefore been argued, notably by Hollingsworth,[1] that fertility change as observed by Wrigley may in fact be largely accounted for by varying reliability of the register.

Simulation allows us to try different assumptions about the nature of registration errors to see whether they would produce the same kind of changes in demographic statistics as given in Table 1. Below we list four possible assumptions.

1. Each birth has the same given probability of not being registered.
2. A birth in a particular family has a probability of not being registered which is a function of the number of births previously registered and the number of births previously not registered in that family.
3. Some families register all births ; others register none.
4. Families generally omit to register their penultimate birth but would register their last birth.[2]

Assumption 4 would certainly produce both a decrease in average (registered) family size, and an increase in LBI. However, there appears little justification for this assumption, particularly as in the absence of family limitation practice a birth cannot be recognised as either 'penultimate' or 'last'. Assumption 3 would not affect the LBI, as this would only be observed for families who registered all births.

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1. Hollingsworth op. cit. pp. 193-5. See also P. E. Razzell, "The Evaluation of Baptism as a Form of Birth Registration through Cross-Matching Census and Parish Register Data : A Study in Methodology," Population Studies, 26 (1972) 130-1.

2. This is the case suggested by Hollingsworth, op. cit. p. 194.

We have therefore confined our simulation experiments to consideration of assumptions 1 and 2. Our results are reported in Table 11. For assumption 1 the probability of non-registration was 0.25 and for assumption 2 this probability was α when $\alpha = 0.25 + 0.1(K-I)$ where K is the number of previous children not registered and I the number of previous children registered in that family.

TABLE 11 Birth Registration Failure with known probability, with MCC Base Distribution I.

Probability	M.D.	E	T	LBI NL	LBI $\bar{X}L$	LBI S_2^2	ABI N_A	ABI $\bar{X}_A$	ABI S^2_A	AFS $\bar{X}_F$	AFS S^2_F
.25	a	0	-	65	53.2	782.3	175	34.5	595.1	4.40	8.24
.25	a	.8	3	46	62.4	1403.5	114	35.3	744.9	3.13	3.41
.25	d	0	-	80	46.4	707.2	222	33.4	454.8	4.81	4.51
.25	d	.8	3	54	61.3	1330.3	140	33.1	555.9	3.64	1.89
$\hat{P}$	d	0	-	69	43.9	446.6	399	31.2	289.9	5.35	11.09

with $\hat{P} = \alpha$ if $0 \leq \alpha \leq 1$

$= 0$ if $\alpha < 0$

$= 1$ if $\alpha > 1$

In both these cases change of about the right magnitude occurs in AFS. However, it is seen that the LBIs in the absence of family limitation and with marriage distribution d are 46.4 and 43.9 respectively. No tendency for the LBI to increase over the comparison level of 44.2 (Table 8) is revealed in either case.

This is not surprising (given assumption 1) for two reasons :

(i) Either the last or the penultimate birth may fail to be registered.

(ii) There is an identification problem. The 0.25 registration failure probability would mean that on the average a family would now need 5 children to be in the sample and hence again the more fecund would be more heavily represented.

These problems are accentuated with assumption 2.

VI

In this paper we have attempted to investigate by the use of simulation techniques the relative merits of different explanations of fertility changes observed in family reconstitution studies of parish registers. We believe this approach to be novel and we hope that our analysis has served both to increase understanding of the demographic processes reflected in the evidence and to illustrate the potential of the approach as a technique in historical demography.

Simulation cannot prove or disprove particular hypotheses, but it can reduce the area of doubt and give estimates of the quantities by which various inputs must change in order to account for given observed changes. We would also argue that simulating the system of family reproduction increases our understanding of the relative strengths and weaknesses of any behavioural theory.

For the purposes of exposition we have considered each of three possible explanations individually. Obviously, however, they are not mutually exclusive and nor are they exhaustive. In particular a possible explanation could be any combination of the three we have considered. Also if demographic conditions were changing rather than constant during the periods isolated by the reconstitution studies, then the same explanation would not necessarily be valid for the whole of the period.

Our results are no doubt subject to various interpretations. However, restricting our comments to the individual propositions as these relate to Wrigley's study we would like to emphasise the following points.

(i) A change in family limitation practice as advocated by Wrigley remains a plausible hypotheses - probably the most plausible of the propositions we have investigated. Our analysis suggests that the change would have been of the 'adjustment' rather than 'innovation' type. It adds weight to the significance of the cause of any such adjustment (particularly if the required change in T is regarded as large).

(ii) Changes in MCC within the range of human experience can explain the observed fertility change with the aid of sampling variation. However these changes would be large, and as in (i) above the cause of such changes needs to be identified. In fact to establish the implications of both propositions 1 and 2 for generalisations about pre-industrial English society we need to indentify reasons for such changes.

(iii) We believe that no acceptable model of registration failure can account for the evidence.

Wrigley's important reinterpretation of demographic-economic interactions in English economic development still seems very attractive. However particularly in view of the difficulties of analysing demographic data, it seems essential to increase the number of relevant studies which provide a systematic and clearly defined set of demographic statistics, and to subject these statistics to all techniques of analysis including simulation.