OVERCROWDING AND INFANT MORTALITY: A TALE OF TWO CITIES

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[Overcrowding and infant mortality]

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ABSTRACT

Using detailed historical data for the cities of Glasgow and Edinburgh, evidence is found in support of the hypothesis that overcrowding is a significant cause of infant mortality. We distinguish between voluntary overcrowding (due to budgetary choices of poorer families) and involuntary overcrowding (due to market failure in the provision of an adequate supply of appropriate housing). We found that Glasgow's infant mortality rate was significantly higher than that of Edinburgh, despite their close geographical proximity, and that a large part of the difference can be attributed to involuntary overcrowding prior to World War II. We argue that this was due to the distinctly different housing policies adopted by the two cities, with lessons for present day public authorities.

1. INTRODUCTION

"The mortality of infants depends upon so many factors, that the effect of overcrowding may readily be masked by other things; that overcrowding plays a part, particularly in the one-room populations, there can be no reasonable doubt."[1]

Earlier this century, public health experts the world over accepted the view that the overcrowding of rooms multiplies the chances of infection to an unknown degree and contributes to higher levels of infant mortality. Today, however, the impact of overcrowding tends to be under-played, especially with respect to other economic and social factors. Thus, it is possible to argue that the question of whether overcrowding is to be called a 'causative factor' of infection, or merely a concomitant factor, reduces itself to a purely verbal difference. Although we agree that overcrowding is but one of several factors predisposing to or causing infant mortality, we argue in this paper that it is possible to provide clear-cut statistical evidence that overcrowding can become a highly significant factor when certain conditions prevail.

High infant mortality rates are normally associated with wretched living conditions brought on by poverty, poor health standards and/or care, and overcrowding. We argue that overcrowding can raise the infant mortality rate significantly, in certain circumstances, even in relatively affluent countries. We investigate the cities of Glasgow and Edinburgh over a

fifty year period (1911-1961), during which time Glasgow experienced an infant mortality rate significantly higher than other industrial cities in Britain, despite comparable levels of disposable household income and access to healthcare. Ironically, prior to 1911, Glasgow's infant mortality rates, although high by today's standards, were amongst the lowest in industrial Britain.[2]

Overcrowding was more severe in Glasgow than Edinburgh because of its role as an industrial city, attracting influxes of population at a rate faster than housing could be constructed. Crucially, however, new arrivals did not overcrowd evenly across the range of house sizes but concentrated in the city's large and ageing stock of one-roomed and two-roomed houses. In the literature overcrowding has typically been approximated by a quantitative measure, namely, numbers of persons per room; however, such a summary measure masks a critical qualitative dimension, namely, the proportion of small houses in a district which, as we shall demonstrate, was crucial in Glasgow's case.

The difference between the extent and nature of overcrowding in Glasgow and Edinburgh was a reflection of each city's housing policy. Edinburgh introduced urban planning at an earlier stage, providing public land to private developers at a reasonable price. Edinburgh's planning policies resulted in both higher public and private housing construction rates than Glasgow's. Even as late as 1961, nearly half of the population of Glasgow lived in houses of two or fewer rooms. This degree of concentration continued in spite of the fact that the 1917 Royal Commission on Housing of the Industrial Population of Scotland condemned both one and two-roomed houses. Its condemnation of the one-room house was particularly strong, concluding, ". . . it may be said that life in one room is incompatible with family decency; it is incapable of affording conditions for a healthy or moral family life; it involves an overwhelming burden on the occupants; it is marked by a higher disease-rate, a higher general death-rate, a higher infantile death-rate, and a higher tuberculosis death-rate".[3]

Throughout the developing world, people have drifted from the countryside into shanty towns and overcrowded urban conditions in order to obtain employment, just as they did in Glasgow in the nineteenth and early twentieth centuries. The lessons that we can learn from this episode are pertinent to all communities today where overcrowding in small houses persists. Furthermore, the effect of this type of overcrowding on infant mortality is not confined to densely populated urban areas. For example, in Australia, infant mortality rates in Aboriginal communities are much higher than the Australian average. Many factors are responsible for this, but it is striking how many Aboriginal families are overcrowded into small houses, lacking the open ventilation and space of traditional settlements. We hope that our historical findings might help to inform current thinking on aspects of both housing and urban policy.

II.   EXPLANATIONS OF HIGH INFANT MORALITY RATES

At the turn of the twentieth century it was recognised that infant mortality (deaths under one year of age) was an important health issue for Britain, in spite of industrial maturity and an assumed overall improvement in economic well-being. Numerous government reports were issued on the subject prior to 1920.[4] All held similar views: there was considerable regional variation, with no clear direct relationship between population levels and infant mortality rates; summer months generally yielded higher rates due to an increase in diarrhoeal infections—this was particularly the case wherever sanitary conditions were inadequate; infant mortality rates were not as responsive to sanitary improvements as was the general death rate; poverty was not a direct cause, although it was the case that the poor with larger

families experienced higher infant mortality rates; the incidence of deaths was higher with bottle-, rather than breast-fed infants; a third of all infant deaths, due primarily to prematurity and congenital defects, occurred within the first month of life. All reports recognised that the causes of infant mortality were complex, though most suggested that programs should be established to increase pre- and post-natal care.

A number of studies during the 1950s attempting to determine the impact of housing conditions upon mortality rates generally concluded that it is "abundantly clear that the higher mortality springs not simply from the closeness with which people live together but from a whole complex of social and economic factors which may be associated with such crowding".[5] These other factors include medical services, occupation, personal habits, educational background, nutrition, and level of income. However, the studies also concluded that when housing conditions did affect mortality it was because of the incidence of transmissible diseases. In all cases examined the morbidity rates of these diseases were higher in crowded houses, especially one- and two-room houses, and where washing facilities and water closets were shared.[6]

Over the last decade a number of regional studies on nineteenth-century England have examined the causes of infant mortality.[7] These studies arrive at similar general conclusions. First, average infant mortality rates were higher in urban than in rural areas. Second, considerable regional variation was present. Third, within urban areas and counties the rates varied widely. Fourth, there was a dramatic secular decline in infant mortality rates in the late-nineteenth and early-twentieth centuries. Fifth, climatic changes and population density influenced the pattern of infant mortality rates. The most noted of these studies was that undertaken by R.I. Woods, P.A. Watterson, and J.H. Woodward. Two of their findings are significant for our study. Firstly, they concluded that the direct influence of poverty on infant mortality is ambiguous and that it is possible to demonstrate that infant mortality declined irrespective of poverty.[8] Moreover, they argued that "income and social class do have a significant influence on the level of infant mortality, *but they do not appear to have been of critical importance in influencing either the timing of decline or the rate of change of infant mortality* [italics added]".[9] Secondly, they questioned the impact of breast-feeding by stating "it is doubtful whether improvements in this particular form of nutritional status could have had a significant bearing on the initial stages of the secular decline of infant mortality".[10]

Michael Haines came to the same overall conclusions with respect to income. He argued that "variations in income did not explain any significant change [in infant mortality]".[11] Furthermore, "income was virtually unrelated to the pattern of mortality decline".[12] Haines placed major emphasis upon overcrowding, measured by room density, observing that in England, as in Stockholm, there "existed an inverse relationship between crowding and child survival".[13] He demonstrates that the mortality index fell as the number of rooms in a house increased, and that infant mortality rates declined at a faster rate for families living in smaller dwellings than those in larger dwellings, given the high initial inequality between the rates for both groups. He found that inequalities in infant mortality rates increased between 1921 and 1949/50, and that the evidence showed that child mortality was lower for families living in houses of many rooms.[14]

In 1991 C.H. Lee argued that the sluggish decline in infant mortality rates for Scotland, compared to England and Wales, was a result of the high room density in Scottish houses, which persisted during the twentieth century. Professor Lee noted that Scotland's infant mortality rates shifted from being lower than England's prior to 1900 to being higher after 1900, and argued that the only regionally significant difference was the proportion of the

population housed in houses of two or fewer rooms. Furthermore, the statistics reveal a slower decline in housing room density for Scotland than for England and Wales. According to Lee, "Almost half the variation in infant mortality between regions in 1921 can thus be explained in terms of housing density".[15] In other words infant mortality was directly related to high-density living in terms of people per room, and inversely related to low-density patterns. J.G. Williamson came to a similar conclusion.[16]

In 1995 Paul Huck published a paper that examined the pattern of infant mortality rates for nine parishes in the industrial North of England between 1813 and 1846.[17] These parishes were a mixture of towns, villages, and rural districts. Huck shows that infant mortality rates rose between 1813 and 1846 for his sample parishes. He isolates three variables as being mainly responsible: population density, feeding practices, and climatic variation. Huck estimates that increased population density accounted for about 43.4 per cent of the increase in infant mortality. The balance, he argues, was "a reflection of reduced breast feeding as higher wages induced more work effort by mothers". Thus, he explains the rise in infant mortality "as a consequence of compensated trade-offs made by families in response to a changing economic environment".[18] His overall conclusion is that the pattern of infant mortality in his nine parishes does not demonstrate a substantial increase in living standards in the industrial towns.

In 1994 R.A. Cage examined the pattern of infant mortality for Glasgow from 1860 to 1970, including a comparison with seven other major British cities.[19] A clear pattern emerges from the data. First, there was a noticeable fall in infant mortality rates between 1870 and 1880. This was a decade in which many cities and towns worked towards the provision of a clean water supply and adequate sewer systems, in addition to the introduction of slum clearance and public housing programs. The next major drop occurred between 1900 and 1910, when greater emphasis was placed upon pre- and post-natal care, the importance of breast-feeding, and the establishment of Child Welfare Schemes. The final major fall was after 1940, primarily as a result of a more extensive use of incubators and the widespread availability of penicillin and other antibiotics. These factors were common to all of the cities considered. However, in the case of Glasgow, other factors were clearly at work since, prior to 1900, Glasgow's infant mortality rate was the lowest of the selected British cities but, by 1925, it had become the highest.

Cage provides a plausible explanation as to why this shift occurred. He undertook a detailed analysis of 1911 census data for Glasgow, showing that there was little correlation between geographic location or population density per acre and infant mortality. What did appear to matter was overcrowding in houses, measured by room density, which was found to be correlated strongly with infant mortality across Glasgow districts in that year. As a rule of thumb, if a district's average room density was one per cent higher than another, then its average infant mortality rate was also one per cent higher. The two major causes of infant deaths at that time were respiratory problems and infectious diseases, which are both sensitive to the degree of overcrowding in buildings. Cage also found some evidence that the proportion of one-roomed houses in a district contributes to a higher infant mortality rate. Using this evidence for 1911, Cage goes on to suggest that Glasgow's infant mortality rate may have become the highest amongst the cities examined in the early twentieth century because of the high room densities in small houses.[20] Given that both infant mortality and room density are both used as measures of well-being, and that these were higher in Glasgow than elsewhere, Cage concluded that the working classes in Glasgow experienced a slower rate of improvement in their standard of living relative to those in other British cities.

Cage's cross district findings for 1911 suggest an explanation as to why Glasgow's infant mortality rates and associated standard of living worsened, in a relative sense, over the next half century. However, it is necessary to examine several years of comparable data and to make an explicit comparison with another city, before strong support for the hypothesis that room density effects were important in Glasgow can be claimed. We selected Edinburgh for comparison because of its geographical and cultural closeness, its different housing policies and the availability of comparable data. Before we move on to the statistical analysis, it is worth providing a historical comparison of conditions prevailing in our two chosen cities over the period in question.

III.   THE HISTORICAL SETTING

In 1900, both Glasgow and Edinburgh experienced housing problems, because the supply of housing in both cities had not kept pace with demand. The problem, though, for Glasgow was more intense, given its role as Scotland's major industrial centre and the preponderance of small houses in its housing stock. In 1901, 26.1 per cent of Glasgow's families lived in 1 room, with a further 43.6 per cent living in 2 rooms; the comparable figures for Edinburgh were 13.1 per cent and 37.3 per cent.[21] During the twentieth century, until at least 1951, Glasgow's housing shortage relative to Edinburgh's intensified, as people moved into Glasgow during the wars seeking employment. Table 1 illustrates the relative problem of overcrowding for the two cities in 1936; twenty-nine per cent of Glasgow's houses were overcrowded, compared to seventeen per cent for Edinburgh in 1936. Of Scotland's estimated 269,758 overcrowded families in 1936, 30.4 per cent lived in Glasgow and 7.5 per cent in Edinburgh.[22] In 1950, 50.6 per cent of Glasgow's families were still living in houses of two or fewer rooms. In 1951 the number of persons per room for Glasgow was 1.27, compared with Edinburgh's 0.94.[23]

TABLE 1

Number of Overcrowded Houses, by room size, 1936

City	Number of Houses Surveyed	Number and Size of Houses Overcrowded							Number of families overcrowded
		1 room	2 rooms	3 rooms	4 rooms	5 rooms	6+ rooms	Total	
Edinburgh	99,608	1,494	11,147	1,790	582	94	84	17,101	20,244
Glasgow	257,421	15,494	44,958	12,858	1,548	69	25	74,952	82,109

A 1935 survey of housing in Edinburgh found that 18.4 per cent of the housing stock was overcrowded and that an estimated 13,594 new houses were needed immediately. Most of the overcrowding was located in a few Old City wards, such as St Giles and St Leonard's. When new housing standards were introduced in 1944, the overcrowding problem was even more severe, with an estimated 32.7 per cent of the stock overcrowded, requiring 50,000 new houses.[24] Bad as this was, the Glasgow experience was much worse. In 1931, 153,503 houses, or 59.9 per cent of the total stock, were overcrowded, if the standard definition of 1944 was applied; in 1951 this figure had fallen only to 130,435 houses or 44.2 per cent of the total. In terms of people, the survey of 1935 was a telling indictment of inter-war policy: 82,109 families (over 31 per cent of all families in Glasgow) were overcrowded under the not particularly exacting standards established in the Act of 1935.[25] Even though Glasgow authorities were committed to increasing and improving the housing stock, the response of private sector property developers was muted because of the high cost of scarce land and the

prospect of low rental income because of rent controls. Table 2 indicates the relative scale of the overcrowding problem in 1951.[26] Moreover, not only was Glasgow unable to build a sufficient number of houses, compared to Edinburgh, but, critically, the housing was generally of a lower standard, with fewer rooms and shared sanitary facilities.

TABLE 2

Housing Occupancy Rates, 1951

City	Persons per household	Rooms per household	Persons per room
Glasgow	3.44	2.71	1.27
Edinburgh	3.12	3.33	0.94

The 1917 Royal Commission on the Housing of the Industrial Population of Scotland provides much evidence on the ill effects of overcrowding, not just for infant mortality, but for a host of other infectious diseases. It was well-known that of the children that died in Glasgow before the age of five, 32 per cent had lived in one-room houses, while less than two per cent lived in five or more rooms.[27] Dr A.K. Chalmers, Glasgow Medical Officer of Health, claimed that, in 1901, male children under the age of one year in one-room houses died at the rate of 210 per thousand living, in two-room houses at the rate of 164 per thousand, in three-room houses at the rate of 128 per thousand, and in four-room houses at the rate of 103 per thousand.[28] From his evidence the Royal Commission concluded that:

"For a nation that needs to conserve its population, the first problem in housing is to prevent the one-room house from increasing the death-toll of the children. For every ten male children that die in houses of four rooms and upwards, forty die in houses of one room. For every forty that die in one-room houses in Glasgow, only thirty die in houses of two rooms, and eighteen in houses of three rooms. It is relevant to say that, in this wholesale destruction of children from one to five, the house is not the only factor–a comment made already in another connection; but it is shown by Dr Chalmers to be a real factor of undetermined magnitude. From the cold facts he adduces, it is legitimate to conclude that, when the family passes into the one-room house, the children enter the valley of the shadow of death."[29]

Furthermore, the Commission stated that it is possible to "establish the broad general proposition that the incidence of all diseases due to the over-aggregation of people within a limited amount of space is higher in the one-room houses than in the houses of more than one room. But the *two-roomed* house is a close second, so close that in many cases the same criticism would apply to it".[30]

The Commissioners then acknowledged that a previous Glasgow Medical Officer of Health, Dr Russell, in the 1880s made similar observations. Indeed, his comments led to stricter rules regulating the number of people allowable for a given space. The intention was that the single-room house be the preserve of elderly couples or younger couples without children. Nonetheless, this type of housing became the homes of an excessive proportion of children under the age of five, forcing the Commissioners to conclude that "the selection of a one-room house is determined by forces too strong to be controlled by any form of superintendence yet established".[31]

Dr Chalmers made another very important observation. He concluded that "the general drift shown to be taking place in Glasgow from the one-room houses into the two-, three-, and four-room houses, indicates that the one-room population is very largely a floating population, *and that the identical people, when they go from the one-room houses into larger houses, show a lower death-rate* [italics added]".[32] Dr Williamson, Edinburgh's Medical Officer of Health, presented statistics to the Royal Commission supporting Dr Chalmer's conclusions.[33] Dr Hay, the Medical Officer of Health for Aberdeen, presented statistics showing that the infant mortality rate for bottle-fed infants in one-room houses was five to six times higher than for the same group of babies in houses of five rooms and upwards.[34] He also showed that the mortality rates for a range of infectious diseases were highest in one-room houses and declined steadily as the number of rooms increased.
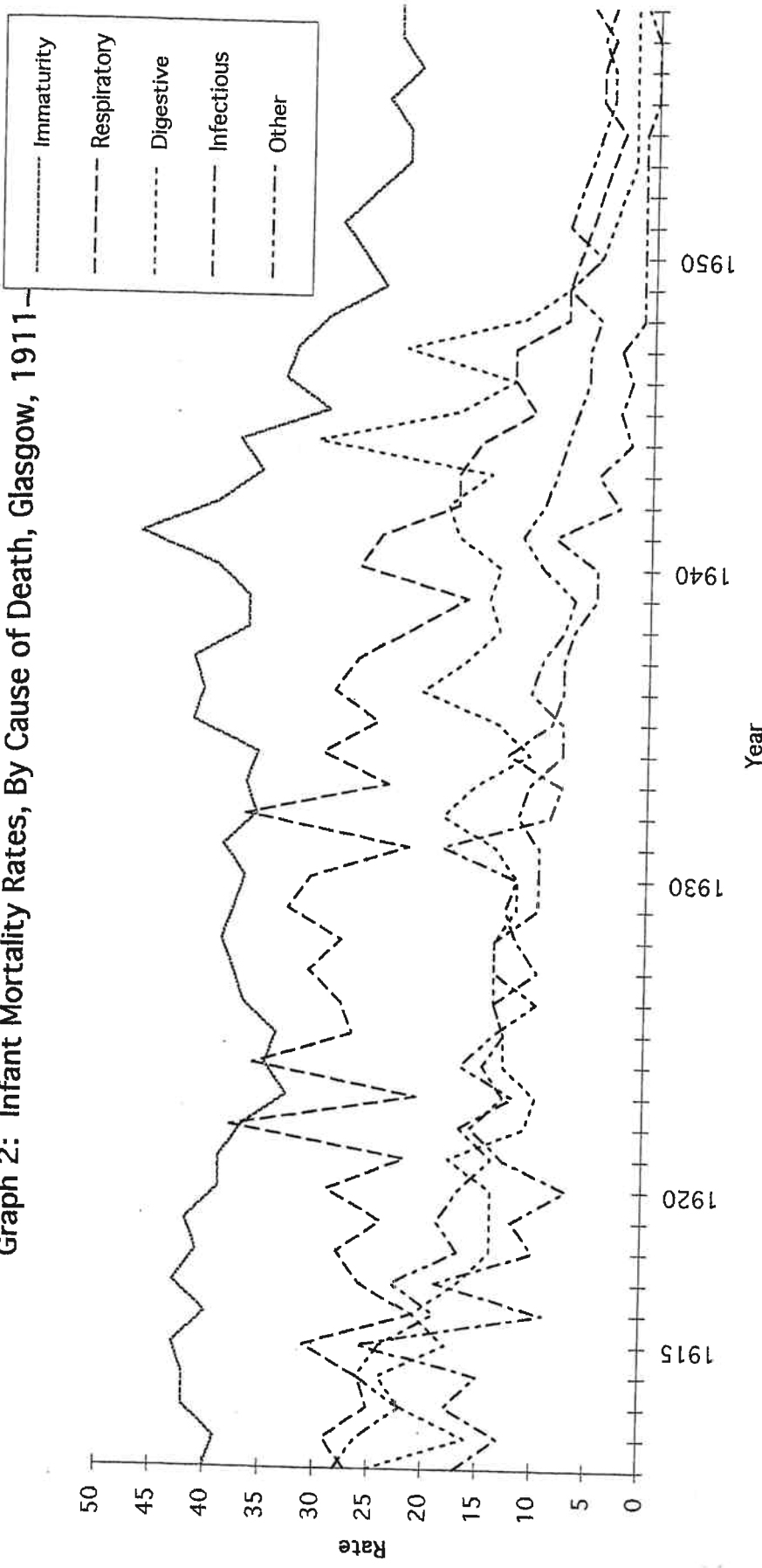
Another way of looking at the relative experience of the two cities is to examine vital statistics data in the twentieth century. Graph 1 illustrates the infant mortality and birth rates for Glasgow and Edinburgh from 1901 to 1972. For both cities the birth rate follows the same pattern, though with Glasgow's always slightly higher than Edinburgh's. Moreover, from about 1930 the birth rate for both cities was nearly constant, experiencing a moderate fall from the pre-1930 rates. Infant mortality rates, on the other hand, exhibit a radically different pattern. For both cities the infant mortality rates fell throughout the period, however, for Glasgow during the interwar years, the rate of decline was significantly lower than Edinburgh's, causing the infant mortality rates for the two cities to diverge until the mid-1950s. An analysis of cause of infant deaths provides some indication as to why Glasgow's infant mortality rates deteriorated.

Graphs 2 and 3 show infant mortality rates by cause of death groupings (immaturity, respiratory, digestive, infectious, and other) for Glasgow and Edinburgh for the years 1911 to 1958. For both cities immaturity (mainly premature birth and congenital defects) is the major cause of death, though the patterns for the two cities are different, with Glasgow's rate higher and failing to fall as rapidly as Edinburgh's after 1940. Since the pattern of immaturity as a cause of death is roughly similar for both cities until 1940, immaturity cannot account for the interwar divergence in infant mortality rates. However, the categories "respiratory" and "digestive" do diverge during the interwar years for the two cities. Indeed, respiratory (primarily bronchitis and pneumonia) disease rates increased for Glasgow after 1920 until about 1935, whereas for Edinburgh they fall. Digestive (diarrhoea) diseases fell for the cities until 1920, after which they remained relatively stable until 1948. Even during this period, the rates for Glasgow were substantially higher than those for Edinburgh, showing a slight increase for Glasgow and a slight decrease for Edinburgh. Ironically, for Glasgow after 1940, respiratory diseases as a cause of death began a sustained fall, while digestive diseases rose. Morbidity rates for both respiratory and digestive diseases tend to be affected by overcrowding.

Graph 1: Birth and Infant Mortality Rates, Glasgow & Edinburgh, 1901-1972

Legend:
- Birth—Glasgow
- Infant Mortality—Glasgow
- Birth—Edinburgh
- Infant Mortality—Edinburgh
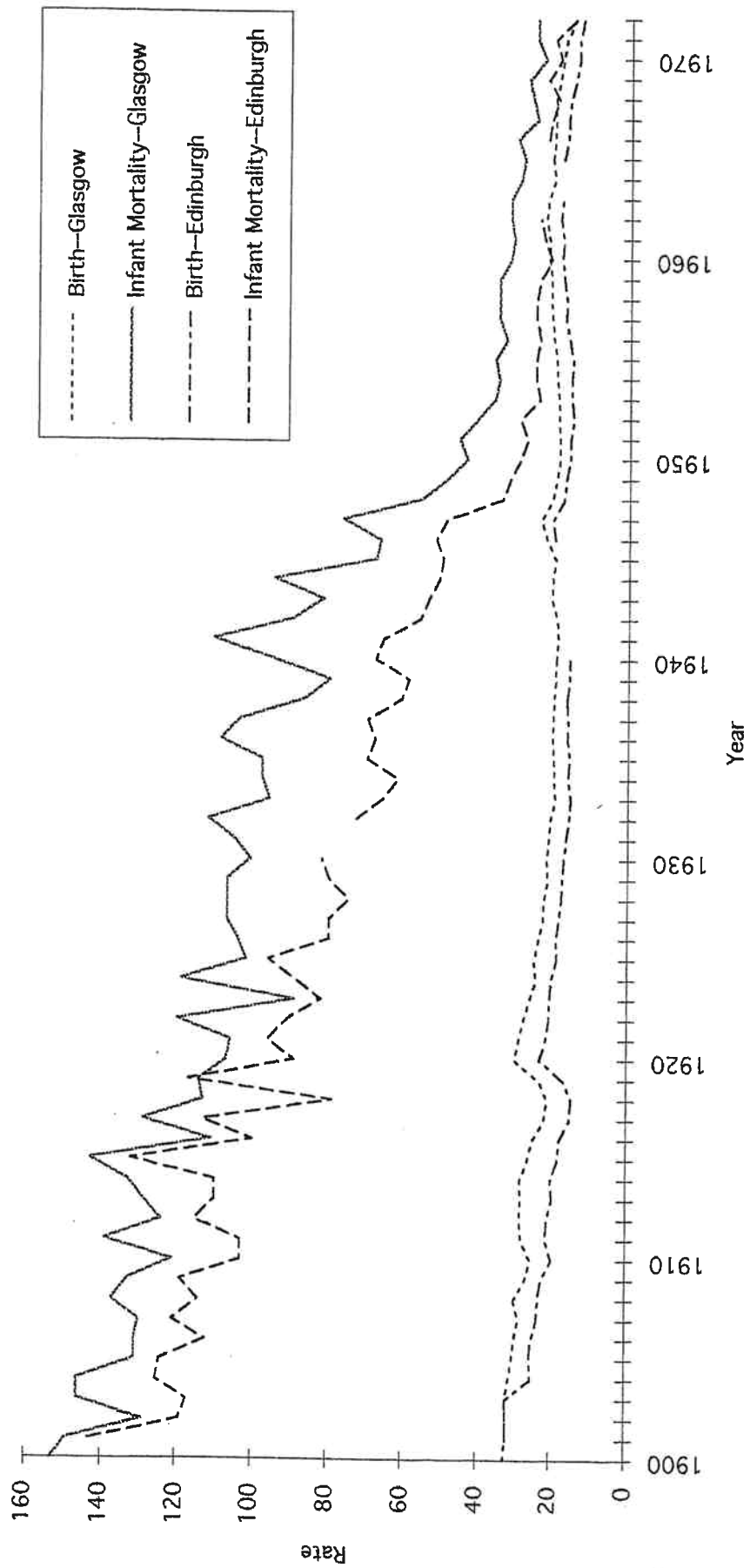
Graph 2: Infant Mortality Rates, By Cause of Death, Glasgow, 1911

Legend:
- Immaturity
- Respiratory
- Digestive
- Infectious
- Other

Rate axis: 0, 5, 10, 15, 20, 25, 30, 35, 40, 45, 50
Year axis: 1915, 1920, 1930, 1940, 1950

Graph 3: Infant Mortality Rates, by Cause of Death, Edinburgh, 1911—1958

Immaturity

Respiratory

Digestive

Infectious

Other

Rate

50

45

40

35

30

25

20

15

10

5

0

1920

1930

1940

1950

Year
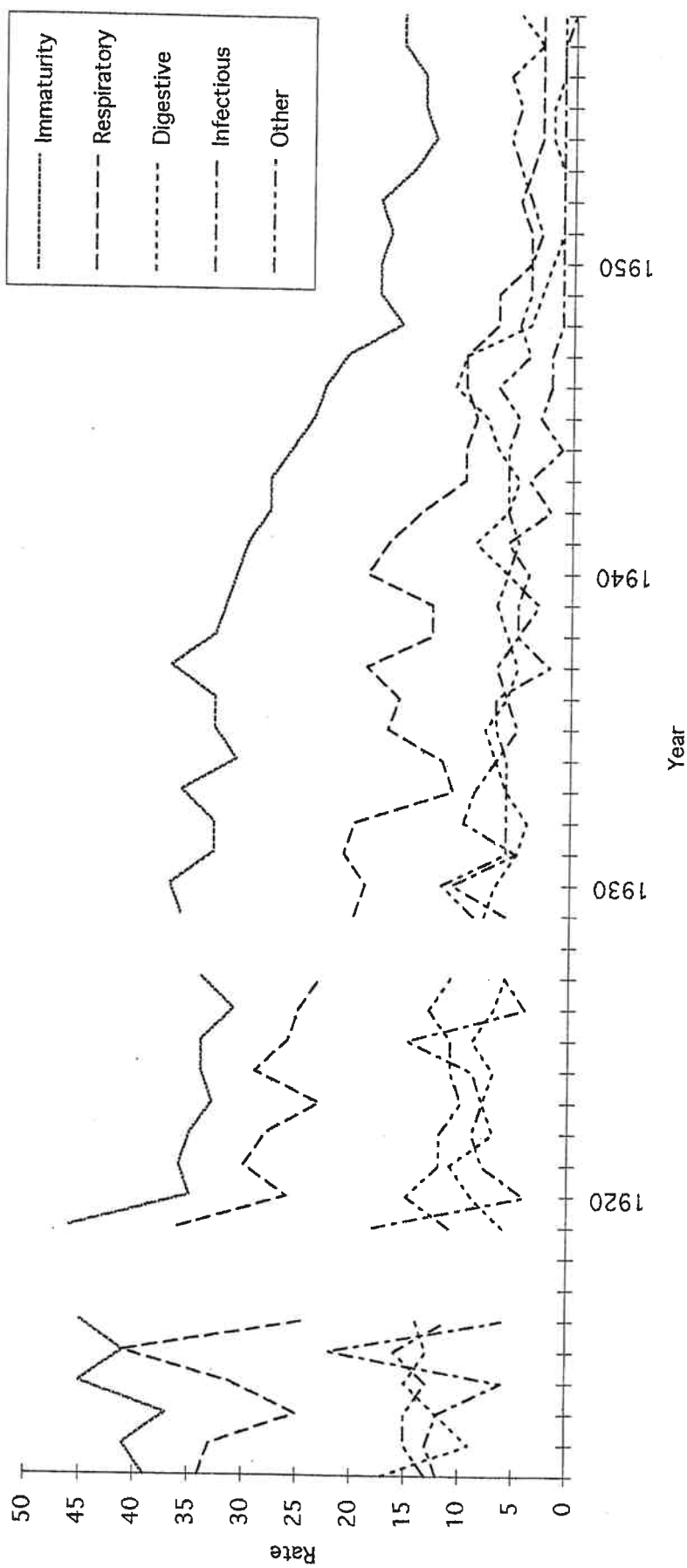
IV. THE MULTI-CROSS SECTIONAL EVIDENCE

We have established a *prima facie* case for arguing that overcrowding, particularly when it is concentrated in small houses, is a significant cause of infant mortality. However, we do not have sets of annual data on the occupancy of different types of houses that we can employ in a time series study of the hypothesised relationship. Furthermore, we know that such a study would be fraught with difficulty because the early twentieth century witnessed significant medical, healthcare and social welfare advances that would be difficult to be take into account in any such study. However, data concerning the occupancy of different types of house are available in some Census years at the city district level in both our chosen cities. Given that accurate vital statistics are also available annually by city district, it becomes possible to construct a cross section regression model that can address our hypothesis.

For Glasgow it was possible to use census data for 1911, 1931, 1951 and 1961 and, for Edinburgh, 1931, 1951 and 1961.[35] For both Glasgow and Edinburgh the sources of data were the Census and the Annual Reports of the Medical Officer of Health for each city. These sources provide a complete set of very reliable data, with no need to generate additional data from other sources or by other means. For each of the Census years listed, infant mortality rates, proportions of house sizes from 1 to 5+ rooms and the number of people living in each type of house are available by city district. Investigating cross-section correlations between city district level data at different points in history has some clear advantages over a historical study that employs city-wide time series data. Many of the causal factors in a time series study that are difficult to quantify, such as developments in healthcare and disease prevention, are relatively unimportant in a cross-section study at a point in time. Such developments, in the main, affect the city-wide average infant mortality rate, around which district rates are distributed, as we move from one Census year to another.

To test the hypothesis that overcrowding in small houses influences infant mortality rates, it was necessary to construct a model of infant mortality determination so that it is clear how the forces at work operate. Furthermore, in such a study, omitted variables must exist and the careful specification of a model helps us to assess their impact upon our findings. We have already sought to provide preliminary support for our general hypothesis that high levels of overcrowding in small houses raises infant mortality by drawing upon a range of authoritative sources and from observing the associated high incidence of respiratory and digestive diseases as causes of infant death. However, we know that infant mortality is affected by a range of other causal factors such as, for example, hereditary defects, malnutrition, lack of protection from the elements, accidents and acts of violence and neglect.

Some of these factors will be related to the economic circumstances of families in a district. For example, poverty affects dietary choice, and it is known that Glasgow's overall diet was more deficient in fruit and vegetables than Edinburgh's and that, during the interwar years, Glasgow's workers and unemployed people had amongst the lowest caloric intake of any location within Scotland.[36] Quality of diet is likely to have had an impact on infant death, particularly in the deaths classified as due to immaturity and digestive causes. Thus, if poverty is taken into account, many of the factors listed become poverty *transmitters* to infant mortality. After allowing for the impacts of poverty, it is unlikely that such factors would have any *systematic* impact upon the average infant mortality rate of a city district in any given year.

We know that higher levels of poverty existed in all Census years in Glasgow compared to Edinburgh. Differential poverty rates between the two cities were mainly a function of their

major economic activities. Glasgow was an industrial city, whereas Edinburgh was, in the main, a service centre. As an industrial city, Glasgow attracted greater numbers of unskilled and semi-skilled workers, especially during the interwar years, when the level of overall economic activity for Britain was low. Thus, Glasgow tended to have a higher unemployment rate than Edinburgh throughout the twentieth century. The general level of incomes was also lower, resulting in lower expenditure on all goods and services, including housing. Just as there was a demand for cheaper, lower quality food in Glasgow, there was a parallel demand for cheaper, lower quality housing.

Adopting such a 'demand-side' perspective leads to two conclusions. First, overcrowding can be a 'voluntary' phenomenon in the sense that it is actively chosen given the economic circumstances of a family. Second, overcrowding will be paralleled by a range of other manifestations of poverty, so evidence that overcrowding is related to infant mortality must be interpreted more broadly, as an indicator of the impact of poverty. Furthermore, it has to be acknowledged that household income is not the only argument that enters the housing demand equation – rental prices will also be determinants of demand. Quite straightforwardly, poor families will not choose large houses because they are too expensive.

When we introduce rental prices, we must address supply-side considerations as well. If supply is responsive to rental prices, increases in demand will cause them to rise and property developers should respond by constructing houses of an appropriate size and quality to fit the budgets of families on a range of incomes. In a city such as Glasgow, with lower average incomes than Edinburgh, we would expect to see families, on average, choosing inferior accommodation with fewer rooms. Inasmuch as the generalised effect of poverty causes infant mortality to be higher, we should discern an association with overcrowding, measured by average room density, which is more pronounced in Glasgow. However, the infant mortality experience of Glasgow, we hypothesise, reflects much more than the housing choices of poor families.

On the supply side, property developers faced serious difficulties in meeting the needs of families prior to the Second World War. As we have noted, high land prices and related restrictions on the release of land for building worked to limit the construction of new housing by private developers. Furthermore, rent controls provided a disincentive to both the construction of new and the maintenance of existing properties. In the face of both inward migration and increases in the demand for larger houses because of rises in real income, these restrictions resulted in an excess demand for housing. This market failure resulted in 'involuntary' overcrowding because the Glasgow City Council was unable to accept its responsibility to increase the availability public housing supply to compensate for the fact that regulatory intervention had removed the private sector's incentive to provide new and maintain old housing. The severe shortage of housing that arose was only reduced after World War II when the Council began to commandeer land for public housing on a large scale.

How did 'involuntary' overcrowding manifest itself? Many Glasgow families were confronted with the reality that they must occupy less space than they would wish to, given their income and the prevailing rental price. However, such overcrowding was not spread evenly across the housing stock - planning regulations discouraged the sub-division of larger houses, so involuntary overcrowding was concentrated in smaller houses that were inappropriate for families. Some better off families were able to obtain larger houses in the interwar years outside the City boundaries, in suburban areas such as Bearsden, Milngavie, Clarkston and Newton Mearns, where property development for owner occupancy was

permitted. However, this was not an option for the bulk of the population. Severe shortages of adequate family housing in the City forced families to remain in the old stock of one and two room houses which were also in demand by inward migrants. Of course, it was the poorest families that suffered the most severe involuntary overcrowding in small houses, providing 'death trap' conditions for infants. However, it was not poverty *per se* that was the main problem, but the tendency for the housing stock to remain as it was in the late nineteenth century, while other cities in Britain, such as Edinburgh, provided the conditions for modernisation and property development to meet emerging needs.

How do we test the hypothesis that involuntary overcrowding caused infant deaths in Glasgow? Of course, involuntary overcrowding raises average room density in a district, over and above that associated with voluntary overcrowding. However, we cannot distinguish them using only average room density statistics. The distinctive feature of involuntary overcrowding is that it exists when there is lack of improvements and new constructions that provide families with the larger houses that they desire. Thus, a key indicator of such a situation will be the observation of a relatively high proportion of families living in small houses. If involuntary overcrowding has a strong effect on infant mortality, we should observe a strong positive correlation of the latter with the proportion of small houses occupied, over and above any correlation with average room density, which is a measure that takes no account of the size of house in which rooms are located.

FIGURE 1

Stylised Demand and Supply of Housing
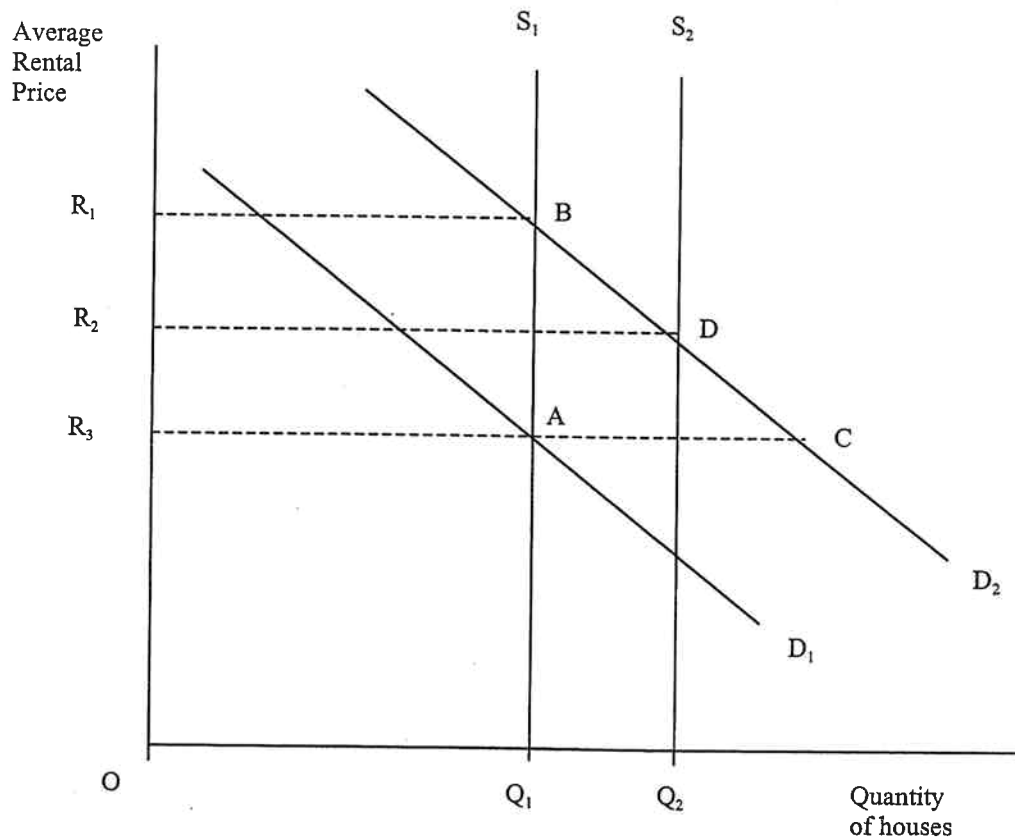
In order to test our hypothesis concerning the impact of involuntary overcrowding, it is necessary to embed it in a model that can address the cross district data that we have. In Figure 1 we have a stylised representation of the demand and supply of housing in Glasgow. Point **A** represents a market equilibrium relevant to the first decade of the twentieth century, following a significant phase of urban expansion in Glasgow. Then, in the ensuing three decades, expansion in housing supply became severely restricted by planning controls. If the demand curve moved from D_1 to D_2 and rents were controlled at R_3, then excess demand, **AC**, would emerge. Given that people have to live somewhere, room density would have risen within the old housing stock. The relatively static nature of the stock implies a high 'black market' rental price, R_1, and it is this that made the construction of 'white collar' housing by private property developers outside the city limits so profitable. Had a higher level of property development been possible in the city, coupled with market determined rents, then the supply curve would have shifted to S_2 and a point like **D** would have been attained. The average rental price (actual or imputed) in equilibrium, R_2, would have been higher than R_3 because of the greater provision of larger, more expensive, houses. Quantity demanded would be less at **D** than **C** because higher rents would have slowed inward migration into the city.

In order to test whether involuntary overcrowding resulted in significant numbers of infant deaths, we begin with the following general model:

$$IMR = f(Y, AF, P, Z) \qquad (1)$$

where IMR is a city district's infant mortality rate
  Y is average income per capita in a district
  AF is average family (=household) size in a district
  P is the proportion of houses with less than three rooms in a district
  Z is a vector of other factors

As we have discussed, poverty, in itself, does not cause high infant mortality. It must be associated with other mediating conditions, such as: malnutrition (M), lack of clothing (C) or poor housing conditions (H). Thus:

$$IMR = f[(M, C, H), AF, P, Z] \qquad (2)$$

It is not automatic that poverty impacts on infant mortality in the same way in different locations. We have already noted that, in some studies, infant mortality rates have been found to be lower in rural, compared to urban, areas where poverty is much higher. However, in urban areas, income is, to a much greater extent, quantified in terms of the purchasing power of money. If supply shortages of food, clothing or housing develop, then prices rise to equate demand with reduced supply or, if price controls are in operation, rationing occurs. In both cases, reduced consumption of one or more of these necessities of life can occur from any initial level of average income. Thus, shortages caused by supply side factors can result in drops in consumption that are independent of income. They do, however, always impact most severely on the poor who bear the brunt of such market adjustments. How do housing characteristics in H tend to affect the infant mortality rate?

Overcrowding, poor sanitation, poor ventilation, inadequate heating and dampness are all factors that can aggravate ill-health and raise the infant mortality rate. Of these, our data only permit us to measure the degree of overcrowding: average room density (ARD) in a district, which is average household size (AF) divided by average number of rooms (AR). Thus, we

must associate all other internal housing characteristics with *ARD*, otherwise they become part of the *Z* vector in eq.(2)

We have chosen the proportion of one and two roomed houses as 'small' houses to capture involuntary overcrowding effects. It should be stressed that although we would expect the poor to tend to choose small houses in which to voluntarily overcrowd, it is not automatic that a high value of *P* will be associated with a high *ARD*. Indeed, today, in a city such as Glasgow, small houses tend to have low room densities because they contain few children. The proportion of small houses in a district should have no impact on *IMR*, over and above any *ARD* effects associated with poverty, unless involuntary overcrowding of small houses by families is a severe problem.

We have included average family (approximated by household) size (*AF*) in eq. (3) in addition to *ARD*, because there is some evidence that it can impact upon infant mortality, irrespective of income level or room density. From our perspective, it is a demand side effect - as *AF* rises, it lowers family per capita income, inducing poverty effects that may not be closely related to overcrowding. For example, a large family living in a large house may not be able to afford to heat it. However, inasmuch as larger families do tend to choose to overcrowd, then we should see a collinear association with *ARD* in the data. However, *AF* is, explicitly, a voluntary demand-side variable, so strong collinearity may not be present when average room density is affected by involuntary supply side overcrowding.

Our modified model is as follows:

$$IMR = f(ARD, AF, P, \mathbf{Z}) \tag{3}$$

Eq (3) is specified as a simple nonlinear relationship, linearised in natural logarithms:

$$IMR = k(ARD)^a (AF)^b (P)^c \mathbf{Z} \tag{4}$$

Therefore:

$$lnIMR = lnk + a\, ln(ARD) + b\, ln(AF) + c\, ln(P) + ln\mathbf{Z} \tag{5}$$

Average room density and average family (household) size for each district were obtained for all Census years as follows:

$$ARD = P1.RD1 + P2.RD2 + P3.RD3 + P4.RD4 + P5.RD5 \tag{6}$$

where *ARD* is a weighted average room density, *P1* is the proportion of 1 room houses in a district, etc., and *RD1* is the room density of 1 room houses in a district, etc. Similarly:

$$AF = P1.HD1 + P2.HD2 + P3.HD3 + P4.HD4 + P5.HD5 \tag{7}$$

where *AF* is the weighted average house density, *P1* is the proportion of 1 room houses in a district, etc., and *HD1* is the house density of 1 room houses in a district, etc.

Considerable care must be exercised in estimating a specification such as eq.(5) since all factors in the *Z* vector are contained in the regression error term (*u*). Despite the fact that the aggregation of households in districts will cancel out many omitted factors, any tendency for the *u* distribution to be non-normal results in omitted variable bias. Fortunately, diagnostic tests can be applied to check if this is the case. Our research strategy was to begin with a

simple univariate version of eq. (5) restricting $b = c = 0$. This is a room density model with no distinction made between voluntary and involuntary overcrowding. We examine whether there is any association with infant mortality. The results of this regression exercise for Glasgow are reported in Table 3. Where recursive least squares estimation indicated the presence of outliers, which can bias estimated coefficients, their impact was removed by the inclusion of impulse dummy variables. Where relevant, these results are also reported.

In all four Census years the cross-district regression results for Glasgow *ARD* is highly significant. Generally, these results offer strong support to the hypothesis that overcrowding and other associated aspects of poverty were important determinants of infant mortality up to as late as the 1960s in Glasgow. Looking at the individual results, those for 1911 and 1931 are very similar, confirming that little had changed in twenty years. After 1931 we observe a steady decline in the estimated constant term, indicating, as might be expected, that the city-wide average infant mortality rate experienced a secular decline as various health-care and social welfare improvements occurred. The adjusted R^2 statistics reported have the impact of the powerful constant term removed and indicate that the correlation is strongest in 1931, after which it weakens as the aforementioned improvements begin to blur the relationship across districts. Interestingly, the estimated coefficient on *lnARD* falls noticeably, from about unity to about 0.6, between 1931 and 1951, after which it rises to about 0.8 in 1961.

In Table 4 we go on to report the regression results for our restricted univariate version of eq.(5) for Edinburgh. Generally, a much weaker association is observed, both in terms of estimated coefficient size and explanatory power. The most striking contrast is in 1931, when a marginally significant estimated coefficient of only 0.33 is reported. However, the estimated coefficients for 1951 and 1961 are quite similar to their Glasgow counterparts. By 1961 the only substantive difference between the results is in the constant terms which reflect the difference in city-wide infant mortality (23/1000 in Edinburgh and 31/1000 in Glasgow). This difference can be associated with the difference in city-wide average room density (0.94 in Edinburgh and 1.213 in Glasgow), applying a unitary elasticity. Thus, we can attribute both the Glasgow/Edinburgh difference and district variations within each city in 1961 to variations in poverty. What is striking about our results is that we can still observe robust room density effects even when infant mortality rates are extremely low by historical standards. The absolute effects of poverty variation are small, but they offer a warning concerning the outcome of rising poverty levels.

Interesting as these results are, we must proceed with great care in interpreting them, given that omitted variables can lead to coefficient bias. Although none of the specifications estimated exhibit any residual normality or heteroskedasticity problems, high Ramsey RESET statistics in 1911 and 1931 for Glasgow indicate that misspecification problems may be present. Thus, we went on to estimate the full model specified in eq.(5). The results for Glasgow are contained in Table 5.

TABLE 3

OLS Regression Results for a Restricted Version of Eq.(5) for Glasgow in 1911, 1931, 1951 and 1961

1911 (26 Districts):

$$lnIMR1911 \quad = \quad 4.063 \quad + \quad 1.032 \; lnARD$$
$$(t \; value) \qquad\qquad (22.698) \qquad (4.587)$$

R^2 adj. = 0.4678, F(1, 24) = 21.09, Normality Chi^2 (2) = 0.25, Heteroscedasticity Xi^2 F[2, 21] = 3.68, RESET F[1, 23] = 6.06.

Corrected for Exchange, Blytheswood and Broomielaw outliers:

$$lnIMR1911 \quad = \quad 3.890 \quad + \quad 1.165 \; lnARD$$
$$(t \; value.) \qquad\qquad (29.923) \qquad (7.103)$$

R^2 adj. = 0.6770, F(1, 24) = 50.2, Normality Chi^2 (2) = 2.98, Heteroscedasticity Xi^2 F[2, 21] = 5.19, RESET F[1, 23] = 2.89.

1931 (33 Districts):

$$lnIMR1931 \quad = \quad 3.988 \quad + \quad 0.930 \; lnARD$$
$$(t \; value) \qquad\qquad (40.283) \qquad (6.370)$$

R^2 adj. = 0.5673, F(1, 31) = 40.65, Normality Chi^2 (2) = 1.10, Heteroscedasticity Xi^2 F[2, 28] = 4.38, RESET F[1, 30] = 7.55.

1951 (30 Districts):

$$lnIMR1951 \quad = \quad 3.514 \quad + \quad 0.651 \; lnARD$$
$$(t \; value) \qquad\qquad (45.051) \qquad (3.869)$$

R^2 adj. = 0.3512, F(1, 28) = 15.15, Normality Chi^2 (2) = 0.13, Hereroscedasticity Xi^2 F[2, 25] = 1.40, RESET F[1, 27] = 0.07.

Corrected for North Kelvinside and Partick East outliers:

$$lnIMR1951 \quad = \quad 3.578 \qquad + \quad 0.570 \; lnARD$$
$$(t \; value.) \qquad\qquad (50.074) \qquad (3.776)$$

R^2 adj. = 0.5476, F(1, 28) = 10.491, Normality Chi^2 (2) = 3.36, Heteroscedasticity Xi^2 F[2, 25] = 0.52, RESET F[1, 27] = 0.86.

1961 (30 Districts):

$$lnIMR1961 \quad = \quad 3.206 \quad + \quad 0.754 \; lnARD$$
$$(t \; value.) \qquad\qquad (45.155) \qquad (3.142)$$

R^2 adj. = 0.2616, F(1, 28) = 9.92, Normality Chi^2 (2) = 0.31, Xi^2 Heteroscedasticity F[2, 25] = 2.85, RESET F[1, 27] = 0.00

Corrected for North Kelvinside outlier:

$$lnIMR1961 \quad = \quad 3.164 \quad + \quad 0.871 \; lnARD$$
$$(t \; value.) \qquad\qquad (48.677) \qquad (3.923)$$

R^2 adj. = 0.3544, F(1, 28) = 15.37, Normality Chi^2 (2) = 0.18, Heteroscedasticity Xi^2 F[2, 25] = 1.59, RESET F[1, 27] = 0.00

TABLE 4

OLS Results for a Restricted Version of Eq.(5) for Edinburgh in 1931, 1951 and 1961

1931 (22 Districts):

$$lnIMR1931 \quad = \quad 4.040 \quad + \quad 0.333 \; lnARD$$
$$(t \; value) \qquad (46.515) \qquad (1.712)$$

R^2 adj. = 0.1225, $F(1, 20)$ = 2.9308, Normality Chi^2 (2) = 0.71, Heteroscedasticity Xi^2 $F[2, 17]$ = 0.871, RESET $F[1, 19]$ =0.90.

1951 (22 Districts):

$$lnIMR1951 \quad = \quad 3.314 \quad + \quad 0.475 \; lnARD$$
$$(t \; value.) \qquad (33.191) \qquad (1.165)$$

R^2 adj. = 0.0635, $F(1, 20)$ = 1.3578, Normality Chi^2 (2) = 0.26 Hereroscedasticity Xi^2 $F[2, 17]$ = 1.21, RESET $F[1, 19]$ = 0.01.

Corrected for South Leith and Colinton outliers:

$$lnIMR1951 \quad = \quad 3.077 \quad + \quad 0.801 \; lnARD$$
$$(t \; value.) \qquad (36.978) \qquad (2.257)$$

R^2 adj. = 0.4120, $F(3, 18)$ = 4.2176, Normality Chi^2 (2) = 0.02 Hereroscedasticity Xi^2 $F[2, 17]$ = 0.97, RESET $F[1, 19]$ = 0.11.

1961 (23 Districts):

$$lnIMR1961 \quad = \quad 3.071 \quad + \quad 1.2095 \; lnARD$$
$$(t \; value.) \qquad (34.491) \qquad (2.318)$$

R^2 adj. = 0.1964, $F(1, 21)$ = 5.375, Normality Chi^2 (2) = 0.12, Xi^2 Heteroscedasticity $F[2, 18]$ = 0.52, RESET $F[1, 20]$ =1.97

Corrected for Corstorphine District outliers:

$$lnIMR1961 \quad = \quad 3.0876 \quad + \quad 0.992 \; lnARD$$
$$(t \; value.) \qquad (36.804) \qquad (1.979)$$

R^2 adj. = 0.3255, $F(2, 20)$ = 5.0691, Normality Chi^2 (2) = 0.15, Xi^2 Heteroscedasticity $F[3, 16]$ = 0.46, RESET $F[1, 19]$ =2.33

TABLE 5

OLS Results for an Unrestricted Version of Eq.(5) for Glasgow in 1911, 1931, 1951 and 1961

1911 (26 Districts):

$$lnIMR1911 = -3.398 + 4.3991 \, lnAF + 0.354 \, P$$
$$(t \, value) \quad (-3.292) \quad (6.634) \quad (7.512)$$

R^2adj. = 0.827, F(2, 23) = 55.05, Normality Chi^2 (2) = 5.05, Heteroscedasticity Xi^2 F[2, 21] = 0.78, RESET F[1, 23] = 1.67.

1931 (33 Districts):

$$lnIMR1931 = -2.450 + 0.958 \, lnAF + 0.321 \, P$$
$$(t \, value) \quad (-0.769) \quad (1.772) \quad (7.532)$$

R^2adj. = 0.707, F(2, 30) = 36.027, Normality Chi^2 (2) = 0.942, Heteroscedasticity Xi^2 F[2, 21] = 0.725, RESET F[1, 29] = 0.001

1951 (30 Districts):

As in Table 3

1961 (30 Districts):

As in Table 3

The regression results reported in Table 5 are 'parsimonious', in the sense that statistically insignificant variables were removed. The first thing to notice is that in both 1911 and 1931 we obtain a much higher level of explanatory power using our full model specification. For 1911, we find that average room density ceases to be a significant variable in the presence of both family size and the proportion of small houses. Nearly double the variation in infant mortality is explained. These results support the contention that overcrowding has both a voluntary demand-side impact, related to family size, and an involuntary supply-side component related to the density of small house occupancy in a district. The former is much more powerful in 1911 compared to 1931, indicating an easing of demand side pressure, as average family size fell. The small house effect is very strong in both 1911 and 1931, with a 1% higher proportion of small houses inducing about a 0.33% higher infant mortality rate.

By 1951 the small house supply-side effect was found to be no longer important, even though a sizeable proportion of the population still resided in small houses. The advent of healthcare, medical advances and child welfare programs seem to have counteracted the small house effect leaving only demand-side, voluntary overcrowding effects related to poverty. However, the intensity of the poverty effects is reduced because of smaller family size. Our parsimonious result is the same as that in Table 5 but it should be noted that family size and average room density are highly collinear when entered together and record very similar levels of explanatory power when entered separately. Thus, the impact of overcrowding appears to have become mainly a demand side outcome associated with poverty in the postwar era. This general picture is further confirmed in 1961.

Turning to the Edinburgh results, we have not presented them in tabular form because no significant small house effects were discovered, suggesting that, even in 1931, housing supply was generally adequate for the needs of families across a range of incomes.[37] Overcrowding did occur in some districts but it was not involuntary. As in the 1951 and 1961 results for Glasgow, family size and room density are collinear when entered together in all three Census years. It is also worth noting that the 1931 Census for Edinburgh provides district information concerning rents paid and there is clear evidence that the lowest rents are paid in districts with the highest average room densities, confirming that poor families tended to live more densely together. However, the effects of overcrowding are in sharp contrast to those prevailing in Glasgow in that same year.

The relatively poor infant mortality record of Glasgow up to the early 1960s (when it was 33% above the rate of, for example, Birmingham after being 10% below back in 1911) can be attributed to both the slowness in improving housing conditions and to poverty. There is little reason to suppose that healthcare improvements were introduced in Glasgow at a significantly slower rate than other cities. On the contrary, pioneering advances in healthcare and medicine occurred in Glasgow throughout the half-century that we have considered. It is clear that household income was low in Glasgow (about 20% lower than in Edinburgh in the interwar period) but we do not feel that poverty was the decisive factor in determining the very high infant mortality rates that were recorded in the interwar period. In many cases families with respectable income levels had to endure cramped and inadequate accommodation because there was nothing else available. The nature of Glasgow's housing stock was the problem.

From the early 20th century the private sector failed to meet housing demand (between 1919 and 1955 only 9.6 per cent of the 108,941 permanent houses constructed in Glasgow were built by the private sector).[38] Public housing construction, in the face of such a massive problem was also very inadequate. No less than 59.5 per cent were houses of 3 or less

rooms.[39] It has been calculated by others that in 1931, 59.9 per cent of Glasgow's population lived in overcrowded housing. The corresponding figure for 1951 was still as high as 44.2 per cent,[40] yet the impact of involuntary overcrowding was mitigated by timely interventions by the State on a number of fronts to alleviate disease, dietary deficiencies and other disadvantages suffered by infants in small, overcrowded housing. Also, by that time, the State was providing funds to cities throughout the UK to build council housing, and by 1951 the families in some of the worst housing had been relocated to more expansive accommodation in council estates on Glasgow's city boundaries. Although such moves did not necessarily remove the impact of poverty on infant mortality, the provision of houses with more rooms altered fundamentally the sleeping arrangements of families and provided opportunities to isolate family members with infectious diseases. The one and two-roomed house ceased to be a family death trap, but instead became accommodation for childless people.

4.   CONCLUSIONS

The conclusions that can be drawn from the above research are clear and can be summarised. We have discovered that infant mortality is strongly related to overcrowding in certain conditions, typified by those prevailing in Glasgow in the first half of the twentieth century. Generally, we shall always observe some kind of overcrowding effect simply because of poverty. For this reason, room density is a useful indication of the quality of life. However, in this paper we have sought to identify something much more serious by making a crucial distinction between voluntary and involuntary overcrowding. We have shown that, because of serious failures in housing policy in the first half of the twentieth century, Glasgow City Council precipitated conditions of involuntary overcrowding in very small houses which induced very high infant mortality rates. In contrast, a more enlightened Edinburgh City Council took steps much earlier to allow the housing stock to expand and improve, in line with a number of other British cities. Thus, the absence of significant involuntary overcrowding in Edinburgh resulted in only marginally significant links between measures of overcrowding and infant mortality.

Why the councillors of Glasgow chose to restrict the availability of land for housing development, to control rents at low levels and, having done both, to fail to provide public housing to alleviate the situation, is beyond the scope of this paper. However, our lack of analysis of the politics of the situation does not prevent us from drawing important lessons from history that can inform our understanding of why infant mortality rates are so high in cities where inward migration is strong but the provision of appropriate housing, private or public, is low. One- and two-roomed houses do not present a problem unless they are occupied by families with young children and infants, rather than single people and couples. In this regard, our findings offer a warning to public authorities to be vigilant and forward-looking concerning the size and composition of the housing stock as inward migration and economic conditions vary over time. Even in modern industrial cities, public authorities must be wary of population influxes occurring at rates faster than the expansion rate of the housing stock, leading to involuntary overcrowding and the emergence of 'death trap' conditions. In point of fact we cannot say that infants are dying because of small house overcrowding, in addition to the generalised effects of urban poverty, in cities currently experiencing rapid influxes of population because little in-depth research seems to have been done on the question. However, we believe that the strong evidence that we have presented for the 'Second City' of the British Empire, in its twilight years as an industrial centre, should provide a stimulus for further historical and contemporary research in a range of urban settings .

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[3]   Royal Commission, *Housing of the Industrial Population*, 1917, p. 95.

[4]   See for example, Supplements to the Local Government Board Annual Reports for 1909-10 [Cd. 5263], 1912-13 [Cd. 6909], 1915-16 [Cd. 8496], and 1917-18 [Cd. 9169].

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[22] Department of Health for Scotland, *Housing Overcrowding Survey*, 1936, Cmd. No. 5171, pp. 10-13.

[23] Butt, "Working Class Housing", p. 261.

[24] Butt, "Working Class Housing", p. 256.

[25] Butt, "Working Class Housing", p. 259.

[26] Butt, "Working Class Housing", p. 261.

[27] Royal Commission, *Housing of the Industrial Population*, 1917, p. 90.

[28] Royal Commission, *Housing of the Industrial Population*, 1917, p. 93.

[29] Royal Commission, *Housing of the Industrial Population*, 1917, p. 94.

[30] Royal Commission, *Housing of the Industrial Population*, 1917, p. 94.

[31] Royal Commission, *Housing of the Industrial Population*, 1917, p. 94.

[32] Royal Commission, *Housing of the Industrial Population*, 1917, p. 94.

[33] Royal Commission, *Housing of the Industrial Population*, 1917, p. 111.

[34] Royal Commission, *Housing of the Industrial Population*, 1917, p. 112.

[35] It must be noted that the 1921 and 1941 census did not contain detailed information on housing, therefore, these years have been excluded from this study.

[36] Rory Williams, "Medical, economic and population factors in areas of high mortality: the case of Glasgow", *Sociology of Health & Illness: A Journal of Medical Sociology*, Vol. 16, No. 2, March 1994, pp. 165-66. Interestingly, Williams also indicates that the "heights of children diminished as dwelling space dimished and crowding increased", and that Glasgow children in the interwar period were 5 cm shorter than London children (p. 166).

[37] These results are available on request from the authors.

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