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Housing in early life and later mortality

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Abstract

***Study objectives*—The aim was to examine the influence of domestic crowding and household amenities in early life on later mortality from all causes and specifically from stomach cancer, chronic obstructive pulmonary disease, and rheumatic heart disease.**

***Design*—This was a retrospective cohort study of people whose houses had been surveyed in 1936 and whose household size was known from the 1939 census. Subjects were followed through the National Health Service Central Register from 1951 to 1989.**

***Setting*—The housing survey had been carried out in the midland town of Chesterfield.**

***Subjects*—Subjects comprised 8138 men and women born after 1900.**

***Results*—A total of 2929 deaths were observed during the follow up period. All causes mortality in the full cohort was not consistently related to any of the housing variables examined, but among subjects who were still children at the time of the housing survey, death rates were higher in those whose houses were crowded or lacked a hot water tap. No associations could be shown between stomach cancer and domestic crowding or food storage facilities; chronic obstructive pulmonary disease and domestic crowding or use of gas for cooking; or rheumatic heart disease and domestic crowding. There were few deaths from these causes, however, in subjects who were children at the time of housing survey.**

***Conclusions*—The findings suggest that the housing of young adults in Chesterfield during the 1930s had little effect on their later mortality. Further follow up of the cohort is needed before firm conclusions can be drawn about the influence of housing at younger ages.**

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Improvements in housing have had a major impact on public health in Britain over the past century. In particular, the clearance of damp, overcrowded slums and the provision of clean, piped water and mains drainage have contributed substantially to the decline of many acute infections. Attention has now turned to the influence of housing on chronic disease. For example, respiratory illness in children has been linked with damp accommodation[1 2] and with combustion products from the use of gas for cooking.[3–5] Stomach cancer has been associated with domestic crowding in early life (suggesting a possible infectious aetiology),[6] and with poor facilities for food storage in childhood (perhaps because of fungal contamination of food).[7] And the epidemic of acute appendicitis over the past 100 years has been related to the growth in the number of houses with hot water systems and bathrooms, suggesting that the disease is triggered by delayed enteric infection in people who have not acquired immunity in infancy.[8]

Investigation of these associations can be difficult if the disease outcome is manifest only after a long latent interval, since personal recall of domestic conditions many years earlier may not be accurate. The discovery of archived records from a detailed survey of housing carried out in the midland town of Chesterfield in 1936 has enabled us to examine the influence of housing in early life on later mortality with more reliable measures of crowding and household amenities. We have looked at associations with overall mortality and with diseases that might be expected to relate to specific aspects of housing—stomach cancer with crowding and food storage facilities, chronic obstructive pulmonary disease (COPD) with crowding and use of gas for cooking, and rheumatic heart disease with crowding.

Method

The survey had been carried out in response to the 1935 Housing Act which required local authorities in England and Wales to assess all working class housing in their areas as a prelude to slum clearance. It came to attention only recently when a report of the findings nationally was released under the 50 year rule.[9] (In Britain, potentially sensitive government documents are sometimes only made public after an interval of 30, 50, or 100 years). Data were held in a card index and covered most of the houses in Chesterfield in 1936. Recorded for each address were the surname of the tenant or subtenant; the total number of rooms and number of bedrooms; whether the house had a hot water tap and/or fixed bath; the type of foodstore, cooking facilities, and lavatory; and the number of residents allowed according to criteria defined in the 1935 Act. The latter depended on the number and size of rooms. From these records we selected a sample of 4370 addresses to cover a range of housing quality.

The list of addresses together with the names of tenants and subtenants was sent to the Office of Population Censuses and Surveys (OPCS) who identified each address in the census of September 1939. From the census, the OPCS abstracted the sex and dates of birth of the residents and forwarded this anonymised information to us with a note of whether persons of the same surname as

the tenant or subtenant at the time of the survey were still living at the address in 1939. In addition, residents born after 1 January 1900 were traced through the National Health Service Central Register (NHSCR) and followed up to 31 December 1989. For subjects who died during the period of follow up, the OPCS provided us with the date and underlying cause of death (coded to the 9th revision of the *International Classification of Diseases*). Where subjects were lost to follow up for other reasons (mostly emigration), we were given the date when they were last known to be alive.

Of the 4370 addresses selected for study, 3563 (82%) were occupied in 1939 by a person of the same name as the tenant or subtenant in 1936. The mortality of residents at these addresses during follow up from 1951 to 1989 was compared with that of the national population by the person-years method, with allowance for age and calendar period in five year intervals. Deaths before 1951 were excluded from the analysis because of uncertainties about the completeness of ascertainment during this period.

Within the cohort, associations between housing characteristics and mortality during 1951–89 were examined by a Cox's proportional hazards model. In addition to variables derived directly from the housing survey, we estimated risk in relation to a crowding index, defined as:

$$\frac{\text{total persons resident in 1939}}{\text{persons allowed in 1936}}$$

and according to "persons per bedroom" defined as:

$$\frac{\text{total persons resident in 1939}}{\text{number of bedrooms in 1936}}$$

Risk estimates were adjusted for sex and for year of birth in five year strata (because of small numbers of deaths a 10 year stratum was used for 1930–39).

Results

The 3563 households analysed included 8812 persons born after 1900 of whom 8138 were alive and under follow up at the beginning of 1951. During 1951–89, 2929 died, 118 emigrated, and 50 were lost to follow up for other reasons. All causes mortality was similar to that in the national population (Standardised mortality rate (SMR)=1·03) as were death rates from circulatory disease (SMR=1·07), COPD (SMR=0·96), and stomach cancer (SMR=1·07).

Table I shows the distribution of deaths from all causes within the cohort according to housing in the 1930s. Almost all houses had water closets, and the numbers with other types of lavatory were too small for meaningful analysis. Provision of a fixed bath correlated almost exactly with having a hot water tap, so results are presented for the latter only. None of the housing variables examined showed a clear relation to mortality in the full cohort. When analysis was restricted to those born after 1915, however, and especially to those born after 1925 (that is, those who were still children at the time of the housing survey), there was a trend to higher death rates in those with crowded dwellings and with no hot water tap. The strongest association was with the number of persons per bedroom in subjects born after 1925. This relation could not be attributed to any single cause of death, but the major contributions came from ischaemic heart disease, cerebrovascular disease, rheumatic heart disease, and COPD.

Table II shows mortality from stomach cancer according to crowding and food storage facilities. There was no association with any of the variables examined, but the number of deaths among subjects born after 1915 was too small to rule out a relation to crowding or food storage in this subgroup. Only six deaths from stomach cancer were recorded in people born after 1925.

Table I Overall mortality according to housing in the 1930s

	All subjects		*Subjects born 1915–39*		*Subjects born 1925–39*	
Exposure	*Deaths*	*RR (95% CI)*	*Deaths*	*RR (95% CI)*	*Deaths*	*RR (95% CI)*
Crowding index:						
<0·50	954	1	171	1	49	1
0·50–0·99	1489	0·99 (0·91,1·07)	559	1·03 (0·87,1·23)	196	1·03 (0·75,1·41)
1·00+	478	1·03 (0·92,1·15)	230	1·11 (0.91,1·36)	119	1·21 (0·86,1·68)
Persons per bedroom:						
<1·50	854	1	156	1	35	1
1·50–2·49	1393	0·97 (0·89,1·09)	493	1·14 (0·96,1·37)	171	1·34 (0·93,1·93)
2·50+	674	1·05 (0·94,1·16)	311	1·18 (0·97,1·43)	158	1·61 (1·12,2·32)
Hot water tap:						
Yes	1240	1	378	1	150	1
No	1672	1·06 (0·98,1·14)	578	1·13 (1.00,1·29)	211	1·15 (0·93,1·41)
Purpose built larder:						
Yes	1344	1	444	1	167	1
No	1569	1·01 (0·94,1·09)	512	1·03 (0·91,1·17)	194	1·09 (0·88,1·33)
Gas cooker:						
No	1821	1	579	1	216	1
Yes	1090	0·96 (0·89,1·03)	376	0·96 (0·84,1·09)	145	1·01 (0.82,1·24)

Risk estimates were derived from a Cox's proportional hazards model. Information on specific exposures was missing for small numbers of subjects
RR=relative risk; CI=confidence interval

Table II Mortality from stomach cancer according to crowding and food storage facilities

	All subjects		*Subjects born 1915–39*	
Exposure	*Deaths*	*RR(95% CI)*	*Deaths*	*RR (95% CI)*
Crowding index:				
<0·50	33	1	5	1
0·50–0·99	28	0·5 (0·3,0·9)	12	0·8 (0·3,2·2)
1·00+	12	0·8 (0·4,1·6)	4	0·7 (0·2,2·7)
Persons per bedroom:				
<1·50	32	1	7	1
1·50–2·49	26	0·5 (0·3,0·8)	8	0·4 (0·1,1·2)
2·50+	15	0·7 (0·4,1·2)	6	0·5 (0·2,1·6)
Purpose built larder:				
Yes	35	1	11	1
No	36	0·9 (0·5,1·4)	10	0·8 (0·3,1·9)

In subjects born after 1915, the risk of COPD showed a weak association with measures of crowding (table III), but there was no parallel association in the full cohort, nor any evidence of a relation to use of gas for cooking. Mortality from rheumatic heart disease was unrelated to crowding in the full cohort (table IV).

Discussion

This study had the advantage that it used archived records of housing rather than relying on people's recall of their past accommodation. Moreover, the 1936 survey was designed specifically to identify crowded dwellings which lacked adequate amenities, and thus could be expected to provide accurate information on the size and design of houses.

Against this, however, must be weighed several weaknesses. The housing data related to one point in time only. We do not know whether dwellings

were improved or otherwise altered in the years before or after the survey. Nor do we know how long subjects lived at the addresses studied, beyond the fact that their families were resident both in 1936 and 1939. It is likely that for most of the subjects born before 1915 the house will have been the marital home and not where they spent their childhood. Information about the number of residents at each address was also limited to one date, although the size of most households will have varied over time.

Such imprecision in the classification of exposure would normally tend to obscure associations. Nevertheless, clear correlations have been shown in ecological studies using similar cross sectional data on housing. For example, an analysis of mortality in 212 areas of England and Wales during 1968–78 in relation to an index of crowding derived from the 1936 survey showed correlation coefficients of 0·64 for stomach cancer, 0·60 for bronchitis, and 0·53 for rheumatic heart disease.[6]

Table III Mortality from chronic obstructive pulmonary disease according to crowding and use of gas cooker

	All subjects		*Subjects born 1915–39*	
Exposure	*Deaths*	*RR(95% CI)*	*Deaths*	*RR (95% CI)*
Crowding index:				
<0·50	50	1	3	1
0·50–0·99	72	1·0 (0·7,1·5)	15	1·6 (0·4,5·4)
1·00+	17	0·8 (0·5,1·5)	5	1·4 (0·3,6·1)
Persons per bedroom:				
<1·50	39	1	2	1
1·50–2·49	74	1·2 (0·8,1·8)	15	2·8 (0·6,12·2)
2·50+	26	1·1 (0·7,1·8)	6	1·8 (0·4,9·2)
Gas cooker:				
No	92	1	18	1
Yes	47	0·8 (0·6,1·2)	6	0·5 (0·2,1·2)

Table IV Mortality from rheumatic heart disease according to crowding

	All subjects		*Subjects born 1915–39*	
Exposure	*Deaths*	*RR(95% CI)*	*Deaths*	*RR (95% CI)*
Crowding index:				
<0·50	17	1	6	1
0·50–0·99	24	0·8 (0·4,1·5)	13	0·7 (0·3,1·9)
1·00+	10	1·0 (0·4,2·3)	6	0·9 (0·3,2·8)
Persons per bedroom:				
<1·50	16	1	8	1
1·50–2·49	25	0·9 (0·5,1·6)	10	0·5 (0·2,1·2)
2·50+	10	0·7 (0·3,1·6)	7	0·6 (0·2,1·6)

The strongest association in our cohort study was between all causes of mortality and the number of persons per bedroom in subjects born after 1925 (table I). This measure reflects the need to share bedrooms, possibly a more potent influence on the transmission of respiratory and enteric infections than other aspects of crowding. Unfortunately, the number of deaths in the subgroup born after 1925 was too small to allow detailed analysis by specific cause of death. The fact that no similar association was apparent in the full cohort, however, suggests that the long term influence of housing on health is greatest in younger children.

This may explain our failure to show associations between housing and diseases such as stomach cancer, COPD, and rheumatic heart disease. Stomach cancer has been linked both with poor food storage facilities[7] and with crowding in childhood.[6] The latter association emerged from a geographical analysis of mortality in relation to housing data similar to those on which the current study was based, and prompted the suggestion that the disease could result from an infection in early life. In support of this hypothesis, several studies have now shown an increased risk of stomach cancer in people infected by *Helicobacter pylori*,[10–12] while others indicate that crowding in childhood makes infection by *H pylori* more likely.[13 14] Our data suggest that crowding in early adult life is not an important determinant of risk, but because there were few deaths from stomach cancer in subjects who were below 10 years of age at the time of the housing survey, they cannot be regarded as strong evidence against an effect of crowding in childhood.

Suspicion that housing in early life might influence later risk of COPD arises from the observation that crowding[15] and use of gas for cooking[3–5] are both associated with an increased risk of respiratory disease in childhood, and that childhood respiratory illness in turn predisposes to obstructive lung disease in adults.[15–17] In addition, at least one study has linked adult COPD directly with domestic crowding at age two.[15 18] Again, our observations suggest that any effect of crowding or use of gas for cooking on the development of COPD is likely to be small in early adult life but might be greater during childhood. Similarly, we found no evidence that crowding in early adulthood is an important determinant of mortality from rheumatic heart disease, although it is a well established risk factor for rheumatic fever.[19] We think it unlikely that these negative findings can be explained by confounding effects. For example, it seems improbable that young adults from crowded dwellings would smoke considerably less or be better nourished than those with superior housing. Nor is it likely that they would have experienced substantially less air pollution. All of the houses studied lay within a 7 km radius of the town centre with the most crowded dwellings situated centrally. Almost all houses had coal ovens whether or not gas was used for cooking.

Overall, this study suggests that the housing of young adults in Chesterfield during the 1930s had little effect on their later mortality. Important questions remain, however, concerning the influence of housing at younger ages. Continued follow up of the cohort should help to provide answers.

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1 Burr ML, St Leger AS, Yarnell JWG. Wheezing, dampness and coal fires. *Community Medicine* 1981; **3**: 205–9.
2 Burr ML, Miskelly FG, Butland BK, Merrett TG, Vaughan-Williams E. Environmental factors and symptoms in infants at high risk of allergy. *J Epidemiol Community Health* 1989; **43**: 125–32.
3 Melia RJW, Florey C du V, Altman DG, Swan AV. Association between gas cooking and respiratory disease in children. *BMJ* 1977; **2**: 149–52.
4 Melia RJ, Florey C du V, Chinn S. The relation between respiratory illness in primary school children and the use of gas for cooking. 1. Results from a national survey. *Int J Epidemiol* 1979; **8**: 333–8.
5 Ogston SA, Florey C du V, Walker CHM. The Tayside infant morbidity and mortality study: effect on health of using gas for cooking. *BMJ* 1985; **290**: 957–60.
6 Barker DJP, Coggon D, Osmond C, Wickham C. Poor housing in childhood and high rates of stomach cancer in England and Wales. *Br J Cancer* 1990; **61**: 575–8.
7 Coggon D, Barker DJP, Cole RB, Nelson M. Stomach cancer and food storage. *J Natl Cancer Inst* 1989; **81**: 1178–82.
8 Barker DJP. Acute appendicitis and dietary fibre: an alternative hypothesis. *BMJ* 1985; **290**: 1125–7.

9 Ministry of Health. *Housing Act 1935: Report on the overcrowding survey in England and Wales 1936*. London: HMSO, 1936.
10 Forman D, Newell DG, Fullerton F et al. Association between infection with Helicobacter pylori and risk of gastric cancer: evidence from a prospective investigation. *BMJ* 1991; **302**: 1302–5.
11 Parsonnet J, Friedman GD, Vandersteen DP *et al.* Helicobacter pylori infection and risk of gastric carcinoma. *N Eng J Med* 1991; **325**: 1127–31.
12 Nomura A, Stemmermann GN, Chyou PH, Kato I, Perez-Perez GI, Blaser MJ. Helicobacter pylori infection and gastric carcinoma among Japanese Americans in Hawaii. *N Eng J Med* 1991; **325**: 1170–1.
13 Galpin OP, Whitaker CJ, Dubiel AJ. Helicobacter pylori infection and overcrowding in childhood. *Lancet* 1992; **339**: 619.
14 Mendall MA, Goggin PM, Molineaux N *et al.* Childhood living conditions and Helicobacter pylori seropositivity in adult life. *Lancet* 1992; **339**: 896–7.
15 Mann SL, Wadsworth MEJ, Colley JRT. Accumulation of factors influencing respiratory illness in members of a national birth cohort and their offspring. *J Epidemiol Community Health* 1992; **46**: 286–92.
16 Burrows B, Knudson RJ, Lebowitz MD. The relationship of childhood respiratory illness to adult obstructive airway disease. *Am Rev Resp Dis* 1977; **115**: 751–60.
17 Cooreman J, Redon S, Levallois M, Liard R, Perdrizet S. Respiratory history during infancy and childhood, and respiratory conditions in adulthood. *Int J Epidemiol* 1990; **19**: 621–7.
18 Britten N, Davies JMC, Colley JRT. Early respiratory experience and subsequent cough and peak expiratory flow rate in 36 year old men and women. *BMJ* 1987; **294**: 1317–20.
19 Morris JN, Titmuss RM. Health and social change. I—The recent history of rheumatic heart disease. *The Medical Officer* 1944; **72**: 69–71, 77–79, 85–87.