

INFANT DEATH IN METROPOLITAN AUSTRALIA 1970-1973

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Résumé — Des variations locales dans les taux moyens de mortalité juvénile pour une période de quatre ans (1970-1973) à l'intérieur de cinq régions métropolitaines australiennes (Sydney, Melbourne, Brisbane, Adelaide, Perth) sont ici examinées dans le contexte de leurs rapports avec un nombre de variables descriptives de composition socio-économique, concernant le logement et démographique au moyen d'une analyse de corrélation et de régression prolongées. Alors que les techniques de multiple régression avaient produit des niveaux relativement élevés d'explication statistique, le sens des résultats n'était pas entièrement clair. La substitution d'une série de variables statistiquement indépendante tirées d'une analyse de composant principal de la matrice des données de la variable originale indépendante et la reconstitution des coefficients de régression qui en découlent, suggère, cependant, que dans chaque région métropolitaine des variations dans les niveaux de mortalité ont des rapports significatifs avec les variations du contexte écologique; en particulier, avec les variations dans la nature et la composition de la famille ou l'unité vivante et l'environnement de la demeure ou du voisinage. Alors qu'on peut apporter peu d'éclaircissement sur les mécanismes causaux opératifs, de tels résultats suggèrent qu'un cas existe pour diriger une recherche additionnelle au niveau de l'individu.

Abstract — Local variations in four-year average (1970-1973) infant death rates within five Australian metropolitan areas (Sydney, Melbourne, Brisbane, Adelaide, Perth) are examined in the context of their relationships with a number of variables descriptive of socio-economic, housing and demographic composition by means of an extended correlation and regression analysis. While stepwise multiple regression techniques were productive of relatively high levels of statistical explanation the meaning of the results was not entirely clear. The substitution of a set of statistically independent variables derived from a principal component analysis of the original independent variable data matrix and the reconstitution of the resulting regression coefficients suggest, however, that in each metropolitan area variations in mortality levels are significantly associated with variations in the ecological context, in particular with variations in the nature and composition of the family or living unit and the housing or neighbourhood environment. While little elucidation of the causal mechanisms operative can be achieved such results suggest that a case exists for conducting further research at the level of the individual.

Key Words — infant mortality, regression analysis, principal component analysis, ecological variation, socio-economic deferment

In most, if not all, western societies significant sectoral and/or areal mortality differentials can still be seen despite their substantial diminution during the last phases of demographic modernization. While any differential having its origins, for example, in the stratification of society deserves ameliorative action, significant disparities in the likelihood of surviving the first day, week, month, or year of infancy demand even more urgent attention, for those at risk are at once totally devoid of responsibility for their plight and quite powerless to alter their situation. To monitor such differentials and, where possible, to isolate causal mechanisms, therefore, should be basic challenges in the

health and welfare programme of any society paying even lip service to the ideal of "equal opportunity."

While it is clear that fullest comprehension of the origins of such differentials requires analyses based on data relating to the social, medical and familistic circumstances of individual deaths (see, for example, Chase, 1973; Morris, Heady, *et al.*, 1955) a consideration of broader "environmental/ecological" and locational situations would also seem to be desirable for while high-risk categories may be described in terms of birth weight, maternal age, parity, or ethnicity, for example, a successful attack upon what has been described as "one of the major public health problems today" (Shah and Abbey, 1971; 33) may also depend upon areal analysis which takes cognisance of locality specific social or environmental problems such as atmospheric pollution (see, for example, Hunt, 1967; Hunt and Cross, 1975). Where individual data are not available, of course, areal-aggregate studies, despite their manifest limitations, may also be productive of useful insight into the origins of differentials and suggestive of remedial strategies.

This paper attempts to define the ecological conditions associated with spatial variations in the incidence of infant death — total and component — in each of Australia's five mainland state capital cities (Sydney, Melbourne, Brisbane, Adelaide, Perth) during the early 1970s. From the application of extended correlation and regression techniques emerges a small number of relationships apparently worthy of more detailed investigation of some potential interest to health care planners.

Mortality Data: Sources and Reliability

Mortality patterns analysed in this paper are derived from registered infant deaths, still-births and live births, allocated back to the Local Government Area (LGA) of mother's usual residence, occurring in the period 1970-1973. As such they constitute a virtually complete data set that is said to be fairly accurate in its attribution of events to localities, is readily comparable with other census-derived data and lends itself to the computation of conventional infant mortality rates (in the form of deaths under one year, <1 month, <1 week/1,000 live births) for each statistical area.

Such an approach has been adopted in this paper, modified only by the need to compute four-year mortality rates, in some cases for amalgamated LGAs, in order to minimize the effects of random fluctuations in the incidence of deaths where only a small number of events occurred, for example in the smallest, least populated LGAs. In only a few instances are the resultant rates based upon less than 20 infant deaths. While it must be accepted that this has probably injected some indeterminate amount of measurement error into the analysis, Jiobu (1972) has indicated that errors of this type will tend to decrease rather than increase the observed relationship between mortality and indicator variables. Their presence, therefore, should not greatly weaken the analysis.

Other sources of potential error should also be noted. First, and possibly most important, but at this stage unmeasurable in its impact, is the common enough problem of the hybrid nature of the mortality rate itself which relates deaths in a population to a different base population. This is further compounded by the equally unmeasured and unmeasurable effects of maternal residential relocation in the period between the birth and subsequent death of an infant. While the former may be seen as probably spatially invariant, the same cannot be said for the latter. Until matched birth and death records can be obtained, however, all that can be done is to recognize the problem's likely existence.

Error is also likely to arise from the system of areal units employed for these depart significantly from the ideal in several respects. In particular, major variations in size and

shape occur both within and between cities. LGAs or amalgamated LGAs will therefore differ considerably in levels of socio-economic homogeneity; perhaps, in the most flagrant cases, to a point at which the utility of the ecological indices calculated is seriously if not totally impaired. In the smaller cities, too, the total number of observational units is less than might be desired for use in regression studies.

The problem of temporal discrepancy within the data set must also be mentioned but, because the bulk of the independent variables employed in this study relate to a point which is almost central to the period over which mortality data have been aggregated, it seems unlikely that such effects would be of sufficient magnitude to influence the analysis.

The Pattern of Mortality

As Table 1 indicates, the level of variation in each of the measures of infant death within the metropolitan areas is considerable — TIDRs, for example, range between 7 and 60 — while the spatial patterns of mortality exhibit a high degree of non-randomness (see Figure 1), a good deal of similarity (Table 2) and, in the majority of cases, depart sufficiently from the accepted bounds of variation about the average level of mortality for it to seem likely that factors other than chance are at work (Table 3). In each metropolis, the highest rates are most characteristic of the inner city areas: in each there is a general tendency for rates to decrease outwards; in several instances there are what might be described as quasi-sectoral effects; yet on occasion there is an apparent reversal of the centre-periphery gradient on the outer margins of the city. Despite the often extreme coarseness of the areal mesh, such a situation suggests almost inevitably that the key to the understanding of spatial variations in infant death rates in metropolitan Australia might well lie in their socio-spatial structure; that is, in the spatial disposition within cities of communities or sub-populations differentiated, in essence, on those basic urban factorial-ecologic dimensions of socio-economic status, familism and ethnicity (see, for example, Logan *et al.*, 1975). In effect, therefore, this study will also provide commentary upon the relevance of urban ecological theory to the understanding of infant death patterns in Australian cities.

The Origin of Variations in Infant Death Levels: The Selection of Independent Variables

In the now considerable literature on infant mortality many "explanatory" variables have been identified or proposed. At the broadest level a distinction is drawn between *exogenous* or environmental variables and *endogenous*, more specifically biological, medical, or genetic variables. Such a distinction, though convenient, is not especially useful, however, for it is clear that the latter are not entirely unaffected by the former; that is, that fetal health can be influenced significantly by the maternal environment. More useful in this context is an approach which recognizes that death rates for areally defined population aggregates are the outcome of a multiplicity of complexly interrelated factors impinging directly or indirectly upon fetal survival chances and operating at a variety of spatial scales.

At the centre of this nest of potentially causal influences may be placed factors relating to fetal development, e.g., duration of gestation, birth weight, congenital defects, multiple birth (see Erhardt *et al.*, 1970; Hunt, 1967; Morris and Heady, 1955; Shah and Abbey, 1971; Thompson, 1968). At one stage removed from this group may be placed effects related to maternal physiology, influenced, for example, by maternal age and birth parity (Bonham, 1969; Daly *et al.*, 1955; Erhardt *et al.*, 1970; Heady *et al.*, 1955a, 1955b;

TABLE 1 SUMMARY STATISTICS COMPONENT AND TOTAL INFANT MORTALITY RATES, SELECTED AUSTRALIAN METROPOLITAN AREAS, 1970-1973

	TIDR ^b	NNDR ^c	ENNDR ^d	PNDR ^e
<u>SYDNEY</u>				
weighted mean ^a	17.84	13.14	10.46	25.08
unweighted mean ^f	18.18	13.43	12.20	25.78
standard deviation	3.85	2.65	2.34	4.40
maximum value	32.36	21.92	19.31	34.93
minimum value	11.04	8.72	7.55	16.57
<u>MELBOURNE</u>				
weighted mean	14.52	10.51	9.29	22.76
unweighted mean	14.86	10.77	9.63	23.98
standard deviation	3.16	2.33	2.33	6.77
maximum value	28.54	20.21	19.02	41.67
minimum value	10.21	7.04	6.31	12.83
<u>BRISBANE</u>				
weighted mean	15.18	11.08	9.53	20.50
unweighted mean	15.66	11.36	9.69	20.65
standard deviation	4.44	3.54	3.01	5.72
maximum value	27.31	18.65	16.04	36.87
minimum value	7.51	4.29	3.22	11.20
<u>ADELAIDE</u>				
weighted mean	13.94	10.94	7.02	21.28
unweighted mean	17.04	11.44	10.09	21.85
standard deviation	10.25	3.23	2.56	4.82
maximum value	59.91	23.04	18.43	37.82
minimum value	11.15	6.83	6.83	15.63
<u>PERTH</u>				
weighted mean	15.87	11.48	9.74	23.28
unweighted mean	16.94	11.97	10.58	4.89
standard deviation	4.51	2.64	2.40	4.89
maximum value	30.25	16.68	14.69	33.06
minimum value	11.13	7.39	6.61	14.62

a) deaths/1,000 livebirths (or births) for defined study area

b) deaths under 1 year/1,000 live births

c) deaths within 28 days/1,000 live births

d) deaths within 7 days

e) still births and deaths within 28 days/1,000 births

TABLE 2 LGA DEATH RATE INTERCORRELATIONS, AUSTRALIAN METROPOLITAN AREAS 1970-1973

INTERCORRELATION	SYDNEY	MELBOURNE	BRISBANE	ADELAIDE	PERTH
NN - ENN*	0.968	0.967	0.953	0.949	0.957
NN - TID	0.945	0.890	0.928	0.900	0.759
TID - ENN	0.931	0.877	0.843	0.826	0.829
TID - PN	0.695	0.699	0.813	0.807	0.579
NN - PN	0.721	0.677	0.825	0.899	0.544
ENN - PN	0.696	0.620	0.779	0.806	0.592
n	41	49	26	21	19

* NN = neonatal death rate

ENN = early neonatal death rate

TID = total infant death rate

PN = peri-natal death rate

n = number of L.G.A.'s or Aggregated L.G.A.'s in Metropolitan area.

Struening *et al.*, 1970). Beyond these again lie influences deriving from maternal "behaviour" for example, health care, nuptial status (Chase, 1973; Jiobu, 1972; Steglich and Deardorff, 1968; Thompson, 1968), nutrition (Morris and Heady, 1955), lifestyle, e.g., work force involvement (Martin, 1967; Morris and Heady, 1955) which in turn may be negated, partially offset, or reinforced by the effects of conditions related to household, family or living unit, for example, family size (Heady *et al.*, 1955); residential crowding levels (Martin, 1967); type and quality of accommodation (Bruen and Hennessy, 1970; Martin, 1967; Morris *et al.*, 1955; Thompson, 1968); family stability (Erhardt, *et al.*, 1970; Jiobu, 1972; Struening *et al.*, 1969); and economic status (see, for example, Chase, 1973; Morris *et al.*, 1955; Pyle and Rees, 1971; Stockwell, 1962). Finally, the influences of the micro living environment may itself be affected by neighbourhood, locality, or more broadly location-specific effects including, for example, population density (Martin, 1967; Pyle and Rees, 1971; Schmitt, 1966); atmospheric pollution (Howe, 1972; Hunt and Cross, 1975; Koshal and Koshal, 1973); or perhaps even water hardness (Crawford *et al.*, 1972).

Unfortunately, not all these factors can be adequately operationalized for inclusion in such a study. Pollution level measurements, for example, are not available for each LGA in each city and even if they were, it is debatable whether single station readings would be sufficiently representative. Nor is information relating to maternal health care behaviour available in even roughly surrogate form. Nevertheless, an initial list of some 27 variables indicative in direct or proxy form of one or more of these factors was prepared for

TABLE 3 CHI-SQUARE VALUES, METROPOLITAN INFANT DEATHS, 1970-1973^a

MORTALITY RATE	SYDNEY	MELBOURNE	BRISBANE	ADELAIDE	PERTH
TIDR	137.39***	91.77***	68.40***	127.09***	37.98**
NNDR	87.36***	84.40***	44.10*	25.24 ⁿ	24.70 ⁿ
ENNDR	234.94***	88.07***	44.35**	20.06 ⁿ	21.25 ⁿ
PNDR	115.74***	96.26***	76.20***	36.22*	27.90 ^o
df	40	48	25	20	18

a Derived from comparison of actual numbers of deaths occurring in L.G.A. with those expected from application of Metropolitan Mean rate to birth totals in L.G.A.'s.

*** $P \leq 0.001$

** $P \leq 0.01$

* $P \leq 0.05$

o $0.10 > P > 0.05$

n $P > 0.10$

correlation against computed LGA mortality rates (Table 4). In general the position of a variable in the table is indicative of its hypothesized scale of operation in the nested scheme outlined above.

Analysis

As Table 5 indicates, infant death rates are not strongly correlated with many of the selected variables. In most cities, however, highest rates seem to be experienced in areas also characterized by higher concentrations of young mothers, small family units, ex-nuptial births, separated and divorced females of reproductive age, households headed by unmarried females, working reproductively aged females, and rented accommodation. In Sydney, high residential densities and ethnicity also assume some importance, as do occupational characteristics and female educational levels in Brisbane and crowding measures in Adelaide and Perth. In short, aspects of the social composition of areally defined populations seem substantially to underlie the patterning of infant death rates. In that Australian metropolitan society is less sharply differentiated on economic or ethnic grounds than its North American or British counterparts and lacks almost entirely their inheritance of sub-standard or seriously deteriorated 19th-century, high-density housing this is perhaps hardly surprising. Nevertheless, it is certainly suggestive that in this respect at least, "the egalitarian society" falls far short of its ideal.

Regression Analysis: To determine whether variability in mortality could more effectively be accounted for by linear combinations of selected variables the several sets of infant death rates were subjected to stepwise multiple regression analysis. In

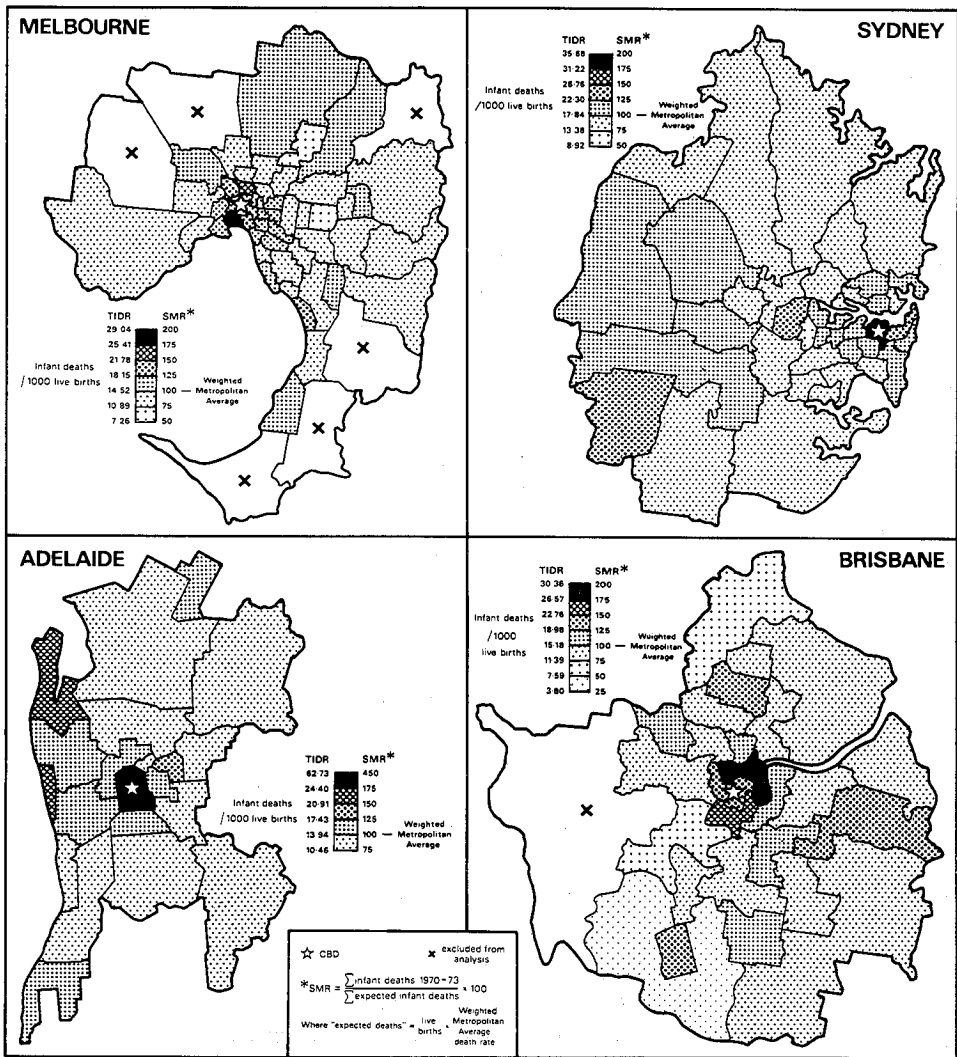


FIGURE 1 PATTERNS OF INFANT DEATH IN FOUR AUSTRALIAN METROPOLITAN AREAS; TOTAL INFANT DEATH RATES (TIDR) AND STANDARDISED MORTALITY RATIOS (SMR) 1970-73

three-quarters of the analyses a substantially enhanced level of explanation was achieved by this procedure and in more than half coefficients of multiple determination exceeding 0.5 were obtained, with R^2 levels ranging between 0.15 and 0.90 (Table 6).

While most variables entered at least one of the 20 solutions, three — ex-nuptial births; divorced or separated females; “crowding” — were especially heavily involved and four more appeared frequently. It would seem, therefore, that size, function, ethnic composition, housing, and other differences notwithstanding there is at least a reasonable degree of between-city comparability and some degree of generality in the ecological context of infant death in Australia’s largest cities, since, in general, intra-city variations in

TABLE 4 SOCIO-ECONOMIC INDICATORS SELECTED FOR INCLUSION IN ANALYSIS OF INFANT DEATH RATES

- 1) % births in LGA occurring to mothers under 21 years of age
- 2) % births in LGA occurring to mothers over 34 years of age
 - 1a) % females 15-40 under 20 (Brisbane only))
 - 2a) % females 15-40 over 34 (Brisbane only))(Surrogate for 1 & 2)
- 3) % families with less than 2 children
- 4) % families with more than three children
- 5) % births ex-muptial (not available for Melbourne)
- 6) % females completed school with less than 6th level (= completed primary school)
- 7) % females 15-39 in labour force
- 8) % families with more than five resident members
- 9) % families with less than 3 redicent members
- 10) Average population/occupied private dwelling
- 11) % families headed by a never married female
- 12) % females 20-39 separated or divorced
- 13) Bedrooms/100 inhabitants
- 14) % occupied private dwellings with less than 2 bedrooms
- 15) % total population born overseas
- 16) % total population born in Italy, Greece, Yugoslavia
- 17) % females in work force engaged in professional, administrative or clerical activities
- 18) % males in work force in professional, administrative/management activities
- 19) % males in work force engaged in processing/craftsmen activities
- 20) Overall population density (population/acre)
- 21) Residential population density (population/acre of residential land)
- 22) % occupied private dwellings "flats" (apartments)
- 23) % occupied private dwellings tenanted
- 24) % occupied private dwellings rented from State Housing Authority
- 25) Average family income, 1971 (Sydney only) (6)
- 26) % population resident in apartment blocks containing more than 8 units
- 27) Average weekly rental, unfurnished houses
- 28) Average weekly rental, unfurnished apartments

infant death rates are significantly, and in some cases closely, related to the spatial patterning of (a) non-familistic life styles (social disorganization); (b) residential environmental conditions; and, less commonly, (c) parity and socio-economic status.

While it would be useful at this stage to be able to argue that much of the variability in infant death rate within any or all of the capital cities can be attributed to variations in, say, the proportion of separated and divorced females aged 20-40 years, with the implication that somehow, perhaps through inadequate mothering in disrupted households, survival probabilities are thereby diminished, it is clear that, the ecological fallacy aside, major impediments to interpretation still exist in no small measure because of the high level of multi-collinearity in the data matrices. The inclusion of one variable rather

TABLE 5 ZERO ORDER CORRELATION COEFFICIENTS—COMPONENT AND TOTAL
INFANT DEATH RATES WITH INDEPENDENT VARIABLES

	SYDNEY				MELBOURNE				BRISBANE				ADELAIDE				PERTH			
VARIABLE	TIDR	NNDR	ENNDR	PNDR	TIDR	NNDR	ENNDR	PNDR	TIDR	NNDR	ENNDR	PNDR	TIDR	NNDR	ENNDR	PNDR	TIDR	NNDR	ENNDR	PNDR
% births to F<21	(+)	503	519	533	474	428	234	281	197	455	461	431	599	309	240	199	216	375	371	302
% births to F≥24	(+)	-127	-112	-173	-183	-005	-079	-029	-019	-144	-165	-078	-251	-104	018	156	029	-130	-026	-018
% families 4+ children	(+)	-385	-456	-455	-389	-391	-325	-422	-290	-293	-252	-182	-387	-436	-325	-279	-377	-288	-320	-418
% families 2+ children	(+)	401	489	504	464	380	325	405	324	041	050	082	093	362	341	325	339	405	303	396
% live births ex-nuptial	(+)	665	658	655	648	n.a.	n.a.	n.a.	n.a.	522	505	395	561	187	065	006	083	602	559	548
% females 20-40 sep/div.	(+)	607	655	641	639	441	347	382	299	679	684	631	674	621	559	586	420	457	378	405
% families n.m. female heads(+)	447	465	474	464	363	317	357	419	352	287	204	330	338	297	300	260	546	506	620	476
% private dwells. 3 inhabs.(+)	432	473	486	496	408	390	435	381	341	299	219	431	468	398	369	369	554	532	644	569
% private dwells. 6+ inhabs.(+)	-371	-414	-432	-336	-303	-339	-413	-313	-296	-269	-213	-373	-410	-249	-203	-283	-406	-394	-490	-413
Pop/occ. private dwells.	(+)	-380	-466	-458	-366	-361	-385	-429	-292	-316	-301	-275	-391	230	198	158	177	-442	-436	-507
Bedrooms/100 inhabs.	(-)	-188	-133	-109	-212	-125	098	111	-038	-279	-245	-169	-196	-834	-685	-619	-605	-476	-583	-563
% occ. priv. dwell. 2 bedrooms	(+)	564	595	583	657	525	367	392	376	274	259	142	373	463	390	408	309	513	343	435
% occ. priv. dwells.'flats'(+)	301	364	377	402	514	365	428	351	241	238	175	360	495	383	384	318	378	285	327	235
% occ. priv. dwells. rented	(+)	531	534	511	606	494	313	357	343	577	566	537	533	535	501	548	317	515	310	341
% occ. priv. dwells SHH tenanted	(+)	-092	-106	-119	013	160	-045	015	-014	-137	-007	111	-275	288	-267	-276	-237	141	066	001
% pop. in blocks 9+ apartments	(+)	298	369	371	374	497	328	389	290	280	212	155	597	311	166	175	162	199	333	270
Pop./acre	(+)	390	478	475	421	193	106	228	018	517	459	485	465	017	-050	-049	-098	146	267	222
Av. weekly rental; houses	(-)	-110	-126	-083	-154	-150	015	089	028	-135	-193	-215	-004	-014	-190	-200	-156	-082	022	092
Av. weekly rental; flats	(-)	038	042	033	-097	-109	033	107	027	-156	-111	-126	-099	303	275	265	334	-073	166	095
% females 15-39 in work force	(+)	374	428	455	415	453	274	358	276	395	374	346	440	702	588	515	575	448	511	535
% females in workforce prof/admin/cler.	(-)	-191	-212	-175	-317	-296	-063	-047	-030	-102	-224	-209	-141	135	039	083	025	024	058	196
% males in workforce prof/admin/man.	(-)	-208	-230	-195	-306	-299	-048	-033	-026	-394	-493	-513	-450	-150	-215	-142	-228	-006	040	187
% males in workforce processing etc.	(+)	017	129	092	147	187	-018	-029	-097	341	431	418	328	054	146	064	167	-057	-059	-223
% females ed. 6th level	(+)	218	322	281	419	387	139	186	137	486	579	575	485	111	265	233	309	335	333	219
% pop. born overseas	(+)	508	541	494	609	377	183	214	133	343	390	197	422	155	091	-021	069	045	047	-053
% pop. born S. Europe	(+)	227	308	271	373	310	098	167	127	407	397	314	493	282	394	246	445	049	-062	018
Av. family income (\$/yr.)	(-)	-180	-221	-192	-259	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.
Resid. pop. density	(+)	681	699	656	685	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.

TABLE 6 STANDARDIZED PARTIAL REGRESSION COEFFICIENTS FROM STEPWISE
MULTIPLE REGRESSION OF INFANT DEATH RATES ON SOCIAL ENVIRONMENTAL
VARIABLES, AUSTRALIAN METROPOLITAN CENTRES

VARIABLE ¹	SYDNEY				MELBOURNE				BRISBANE				ADELAIDE				PERTH			
	TID	NH	ENH	PN	TID	NH	ENH	PN	TID	NH	ENH	PN	TID	NH ²	ENH ²	PN	TID	NH ²	ENH ²	PN ²
1																				
2	.25 ^a		.66 ^a														-1.66 ^a			
4														.32 ^c		.32 ^d				.50 ^d
5	.98 ^a	.46 ^c	.50 ^a	.34 ^c	n.a.	n.a.	n.a.	n.a.	.68 ^a	.52 ^d	.45 ^b	.53 ^a			-35 ^d	2.64 ^a				
6		.48 ^c										-1.04 ^a			1.12 ^a					
7																				
8						.39 ^a	.43 ^a												.64 ^a	
9																				
10	.40 ^a		.57 ^a					.63 ^c												
11																				
12						.49 ^a														
13	.32 ^b																			
14	-.49 ^c	-.92 ^a	-.86 ^a																	
16								.68 ^a												
17								-.29 ^c												
21																				
24																				
25										.30 ^d	.33 ^d									
28	.69 ^a	.61 ^a	.69 ^a	.45 ^a	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.
R ² x 100	78.4	69.29	76.49	53.05	32.24	15.20	18.91	23.59	46.16	53.03	47.68	66.30	90.55	56.88	68.32	46.69	94.68	33.96	41.53	58.89
F Ratio	20.65 ^a	20.31 ^a	18.44 ^a	21.47 ^a	10.95 ^a	8.42 ^a	10.96 ^a	7.10 ^a	20.58 ^a	12.98 ^a	10.48 ^a	10.33 ^a	38.35 ^a	11.87 ^a	12.22 ^a	7.88 ^a	27.96 ^c	8.74 ^a	12.07 ^a	7.16 ^a

a = P < 0.01
b = 0.02 > P < 0.01
c = 0.05 > P < 0.02
d = 0.10 > P < 0.05
* = Pattern of mortality rates not significantly different (P < 0.01) overall from metropolitan average (see Table 3)
+ = For key to variables see Table 5

TABLE 7 STANDARDIZED PARTIAL REGRESSION COEFFICIENTS, MORTALITY RATES ON COMPONENT SCORES

	COMPONENT				
	1	2	3	4	R ² x 100
SYDNEY					
TIDR	0.549***	0.208	-0.198		39.28 ^a
NNDR	0.604***	0.227*	-0.094		43.61 ^d
ENNDR	0.601***	0.195	-0.133		42.70 ^a
PNDR	0.613***	0.282**	-0.107		47.76 ^a
MELBOURNE					
TIDR	0.469**	0.105	-0.089		24.73 ^a
NNDR	0.333***	-0.066	(-0.096)		12.42 ^b
ENNDR	0.394***	-0.063	-0.025		16.58 ^b
PNDR	0.331***	-0.107	-0.137		14.35 ^b
BRISBANE					
TIDR	0.463***	-0.462***	0.257**	0.200	54.80 ^a
NNDR	0.438***	-0.483***	0.347**	0.087	56.69 ^d
ENNDR	0.367***	-0.424***	0.448***	0.106	53.61 ^a
PNDR	0.531***	-0.432***	0.175	0.081	52.44 ^a
ADELAIDE					
TIDR	0.339***	0.292***	-0.563***	-0.463***	83.50 ^a
NNDR* ⁿ	0.368**	0.375**	-0.331**	-0.481***	61.93 ^a
ENNDR*	0.359*	0.309	-0.302	-0.406	48.38 ^a
PNDR	0.331*	0.321*	-0.305	-0.516***	53.90 ^a
PERTH					
TIDR	0.519**	0.129	-0.193	0.181	37.67 ^b
NNDR	0.453*	(0.081)	(-0.034)	(0.138)	31.90 ^b
ENNDR	0.547**	-0.043	(-0.056)	(0.106)	31.06 ^b
PNDR*	0.542***	0.155	-0.253	0.337*	49.94 ^b

*** = P < 0.01

** = 0.025 > P > 0.01

* = 0.05 > P > 0.025

a = P > 0.01

b = P > 0.05

n = non significant Chi-square

() = variables not included in regression for determination of significant R²

than another in a regression would often seem more fortuitous than meaningful, therefore, while relationships which are directionally consistent at the correlation stage now exhibit directional instability as, for example, in the case of "rented dwellings" (X14).

Under such circumstances it is clearly undesirable to attach specific intrinsic meaning

to the combinations of "explanatory" variables thrown up by this phase of the analysis. Instead some attempt must be made to extract from the independent variable matrix underlying dimensions of variation which are themselves more truly independent.

Principal Component Analysis: To this end three or four principal components, accounting for just on 85 per cent of the original total variance, were extracted from reduced data matrices for each city, and area scores on each component were calculated to produce new sets of statistically independent variables for use in a further series of regression analyses. In each city the first component defined an inner city, "low-familism" syndrome (high loadings on working, divorced or separated, unmarried, head of household females, ex-nuptial births, births to young mothers, apartments, rental accommodation, etc.) and the second, apparently, low socio-economic status (highest loadings blue collar workers, low female educational attainment, low family income [Sydney only], crowding). Other components resist simple interpretation but in each case the variable "percentage births to mothers > 34 years" loads heavily, though there is considerable between-city variation in the social context.

The outcome of regressing infant death rates on component scores is summarized in Table 7. As might be expected the use of these "composite" variables reduces the explanatory power of the models considerably. Even so, multiple R^2 values in excess of 0.45 are still common. In all cases "low familism" dominates the regressions, with low socio-economic status apparently also contributory in Brisbane, Sydney, and Adelaide. "Late maternity" also contributes slightly to the explanation in Brisbane and Adelaide.

At this point it seems possible to conclude that spatial variations in the level of infant mortality within Australian metropolitan areas reflect, at least in part, the basic ecological structuring of the cities concerned, though clearly the associations are weaker than might be wished for. From an interventionist viewpoint, however, such conclusions are less than totally helpful for, given the range of area and resident attributes covered by these classical ecological umbrella concepts, they provide no clear suggestion of where remedial action might most usefully be undertaken. Some progress toward this goal might be achieved if the true significance of individual independent variables, stripped of their interactions, can be indicated.

Such a result can be achieved by reconstitution of the component score regression coefficients to generate sets of standardized weightings (Table 8) which indicate the relative importance of each variable included in the component analysis (Riddell, 1970). In this study, however, the product of this somewhat tortuous procedure seems really to advance the analysis very little, if at all, beyond the zero-order correlation stage for as Table 8 demonstrates many of the reconstituted coefficients have approximately similar weight. In sum, this final piece of data manipulation seems merely to confirm that in the five metropolises under consideration intra-city variation in infant mortality rates is clearly associable with specific ecological contexts, highest rates occurring in LGAs characterized by:

- (a) a housing stock containing high proportions of apartments — particularly in blocks of nine or more units — or other forms of rented accommodation;
- (b) a resident population in which singles or pairs, broken marriages, and female-headed living units are common, in which high proportions of reproductively "at risk" females are employed outside the home and where the incidence of births to women under 21 and to unmarried mothers is highest; and, in Brisbane and Sydney by
- (c) high proportions of overseas-born, less educated females and male, blue-collar workers.

TABLE 8 MAJOR RECONSTITUTED STANDARDIZED REGRESSION COEFFICIENTS —
INFANT DEATH RATES ON SOCIO-ECONOMIC VARIABLES

VARIABLE†	SYDNEY				MELBOURNE				BRISBANE				ADELAIDE				PERTH			
	TIOR	NN	ENN	PN	TIOR	NN	ENN	PN	TIOR	NN	ENN	PN	TIOR	NN	ENN	PN	TIOR	NN	ENN	PN
1	10.08			11.92	10.39				15.41	13.98	12.48	10.38								11.54
2																				-11.63
4	10.93	11.83	11.83															10.20	10.32	14.56
5	14.11	14.77	14.71	15.41	n.a.	n.a.	n.a.	n.a.	14.21	13.29	11.87	8.25					11.53	10.26	10.59	12.32
6	13.62	14.70	14.65	14.95	12.86	7.91	9.09		16.99	16.73	20.82	10.33	12.72	11.00	9.52	8.87	11.07	10.26	11.19	11.71
7																	10.56		11.64	
8	10.04		12.66			7.97							8.83				11.20		12.39	11.31
9									11.65				-19.91	-15.84	-9.47	-14.84	-10.30		-11.69	-11.13
11																				
12	13.82	14.34	14.47	14.64	11.25	8.40	9.35	8.54									13.08		12.11	
13	10.82	12.18	12.19	12.05	11.19	8.47	9.80	8.32									12.83	11.12	12.62	
14	14.29	14.71	14.75	15.34	10.85	7.83		8.05	13.97	13.19	11.65		12.01	9.42		7.04	12.81	10.83	11.48	10.69
16	10.61	12.04	12.03	11.09	11.08		9.14	7.76									10.62		10.46	
17							17		10.97											
20	11.23	12.73		12.69	11.10		9.25	7.66					15.01	13.28	10.94	11.87	11.42		11.85	14.50
23										10.05		8.57								
24										13.98	12.52	11.50								-11.09
25	10.40	12.40	11.71	13.00						10.58										
26									11.16	16.34										
28	13.06	13.39	12.24	14.20	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	9.58			n.a.	n.a.	n.a.	n.a.

† For key to variables see Table 5

Discussion

Three points seem indisputable. First, there is in each metropolitan centre a measurable, statistically significant, and on occasion surprisingly high level of association between local levels of infant death and variations in aspects of urban socio-economic and demographic structure. Second, despite inter-city differences in the way individual variables combine or weigh, substantially the same sorts of factors seem to be involved, whether for early or overall infant death rates. Finally, despite these ecological associations it is manifestly clear that little, if any, progress has been made in identifying the mechanisms giving rise to these associations for few of the original variables lend themselves to a single, precise, unambiguous interpretation. Yet, if conclusions of any practical utility are to emerge some attempt must now be made to explore — albeit subjectively — their meaning.

The reconstituted variable weightings break up into some half-dozen subjectively defined clusters. Variables descriptive of *family status* are at once most frequent in appearance and most heavily weighted. Three possibilities present themselves. First, and notwithstanding the ecological fallacy, is the suggestion of some direct, causal link between “non-standard families” and infant death, perhaps through inadequate ante-natal care, poorer maternal condition, less effective “mothering,” or even deliberate maltreatment (*vide* the rising incidence of “battered babies”). Were this to be so the implications would, of course, be serious. Alternatively, it may be argued that the association results from higher mortality among infants born to women living in such areas but who themselves possess other attributes. If so, the problems remain: which categories of birth are at greatest risk, and why? The answer to such questions can be determined only by micro-level research outside the scope of this paper. Finally, it is possible that these associations merely reflect the higher risk associated with first births but the magnitude of the differentials would seem to weaken this argument.

The several variables relating to *neighbourhood environment* seem also suggestive of a “first birth” effect for it is less common for apartment dwellers to be well advanced in family formation. Nonetheless, high density areas are characterized by higher incidences of other social pathologies (see, for example, Burnley, 1977) and may well be less than adequately supplied with readily accessible health services, though this was not apparent in a comparison of residuals from the regressions against the per capita availability of GPs in Sydney (see Freestone, 1975).

Variables descriptive of the *household* (e.g., crowding levels) point in two different directions. On the one hand they reinforce the possible effects of first births in small family areas; but there is also a suggestion that areas characterized by larger families tend to experience higher mortality. Whether this relates to diminished “mothering” under conditions of stress or to more direct, class-based effects (related, for example, to differentials in disposable income, attitudes toward or diminished access to health care), however, remains to be seen, though there is a suggestion that in Brisbane at least, this may well be the case.

Maternal characteristic (percentage births to mothers < 21 years; percentage of births to unmarried mothers) and *ethnic status* variables temptingly offer themselves for interpretation in a more directly causal sense. Because the former variables are birth-based rather than merely ecological this may well be correct, in which case we have a possible combination of parity and social disadvantage suggesting the need for additional research and possible remedial treatment. Equally, the apparently common relationship between higher mortality and “ethnicity” — one of the major lines of cleavage in Australian society — is also suggestive of social disadvantage and/or, more

specifically, culture-based differences in access to information about and use of pre-, post-, and at-birth medical attention. But whatever the process, further investigation of the risk level among births to overseas-born parents is clearly called for.

Conclusion

This study demonstrates that within Australia's major metropolitan centres significant — though variable — proportions of intra-city variation in infant mortality (total and component) can be statistically accounted for in urban ecological structural terms, in particular socio-demographic rather than economic structure, as epitomized in the primacy of a family status and related housing dimension; that is, in terms of conditions tending to vary zonally rather than sectorally.

Whether these associations have their origins in areally varying "environmental" attributes (e.g., the quality of housing, crowding levels) or in the behaviour of individuals resident in areas so characterised is not possible to determine with the data available, however, for the effect of variations in the incidence of mothers at higher risk on biological grounds cannot effectively be separated out. Nor, so long as it remains necessary to work in the aggregate ecological mode with large and often heterogeneous populations in a society in which infant death is a comparatively rare event, does it seem likely that more strictly causal influences can more precisely be isolated. Conceivably, the inclusion in the analysis of additional, more immediately and unambiguously interpretable variables relating, for example, to the frequency of health centre (see Wilson, 1978) or medical practitioner use or to levels of atmospheric pollution may improve matters; but, in the end, it seems likely that the critical findings will come only from detailed analysis of individual infant deaths — by locality as well as by maternal, residential, and other characteristics. Until such time as studies of this nature are possible, therefore, it would seem reasonable to conclude, with Schwirian (1971) that mortality rates is certainly sensitive to the total social and ecological system; but necessary, too, to suggest that the process links between social and ecological structure and mortality are less than unequivocally revealed. Thus, while those responsible for, or concerned with, social policy formulation and implementation, including those relating to the delivery of health care, may find these analyses of some use, this is more likely to derive from their stimulus function than as a basis for ameliorative action.

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