Infection and Malnutrition Interaction Effects On Child Mortality in Scania, Sweden 1766-1894

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Background

In the 18th and 19th centuries, food production was at such low levels that most parts of the European population was chronically malnutritioned, and any short-term food scarcity caused by normal harvest fluctuations or famine would have affected mortality, especially for the most vulnerable groups as children or elderly.[1] Further, any food scarcity should have affected mortality of the ones suffering from any illness most, since low nutritional status makes an individual more susceptible to infectious disease, and when illness does strike, it is more severe, prolonged, and carries increased risk of permanent damage or death.[2] Infections on the other hand worsen the nutritional status and body claims are much higher during sickness.[3] But the body is also easier to infect in the first place if malnourished, since immunity is damaged by malnutrition and infection.[4] Thus, not only disease and malnutrition are important for mortality but also the interaction of disease and malnutrition should be an important determinant for mortality. Hence, it is of vital interest to include the interaction of infection and malnutrition in the theoretical mortality model, and in this paper it will be tested empirically. The investigation is of interest since: 1) it contributes to explaining the general mortality decline, which still is only partly understood[5] and 2) the developing countries today face about the same situation as in 18th and 19th century Sweden, and any investigation that can increase the knowledge of historical child mortality can improve the understanding of child mortality in the developing countries today.

Age-group and period sub-division

The age-group used in the empirical analysis is children in age 1 to 14 since the consequences of malnutrition and disease are considered more serious for infants and children than for adults,[6] and the setting is rural Sweden during 18th and 19th century. A period subdivision accounts for enclosures, land partitionings, and industrialisation, making it possible to investigate child mortality during the transition from one type of society to another.

Data

The empirical analysis uses data from SDD on five parish populations in Scania, Southern Sweden.[7] This database contains data on demographic events and economic conditions for several thousands of individuals from 1650-1894 collected from parish registers and church records, and supplemented with socio-economic information from poll tax register. A recent 50 % increase of this base due to the addition of a fifth parish, Kågeröd, makes it possible to use it for analysing interactions of malnutrition and disease in a pre-industrial population. The area is an rural area, where a selection of farmers, smallholders, semilandless, and landless has been made for this study, which means that there are no priest, teachers, estate-owners, and similar in the study due to small numbers for these categories. Since most of the supplement information on socio-economic status is only available for all parishes after 1766, the study is limited to 1766-1894.

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[1] Fogel (2000).
[2] Scrimshaw, Taylor & Gordon (1968), Pollard (1982), Scrimshaw & SanGiovanni (1997), Mata, Urrita & Garcia (1967), Scrimshaw (2003), Gopolan (2000).
[3] Lunn (1992), Fogel (2000), Scrimshaw, Taylor & Gordon (1968), Osmani (2000).
[4] Gershwin, Beach & Harley (1985), Fogel (1986, 1994), Pollard (1982), Chandra (1989).
[5] Johansson (2004: Chapter 2).
[6] Scrimshaw, Taylor & Gordon (1968).
[7] Scanian Demographic Database.

Theoretical and empirical model

Child mortality is directly dependent on nutrition, disease, and the combination of these, which in turn depends on family preferences and socio-economic conditions, as well as biological endowments.[8] The theoretical child mortality model in the paper draws on the model in Johansson (2004) and accounts for nutrition and disease but also the interaction of nutrition and disease. It also considers variables as socio-economic conditions, individual

Theoretical model	**Operationalisation**	**Variable in empirical model**
Community level		
Environment	Parish	Hög (reference group) Kävlinge Halmstad Kågeröd
		Sireköpinge
Nutrition	Food price	Current local rye price in time *t*
		Current local rye price in time *t*-1
Disease load	Mortality rate	Local current infant mortalty rate in time *t*
		Local current infant mortalty rate in time *t* -1 Local infant mortality rate during infancy
Nutrition & disase load interaction	Food price * mortality rate	Current local rye price in time *t* * local current infant mortality rate in time *t* Current local rye price in time *t*-1 * local current infant mortality rate in time *t*-1
Family level		
Family wealth	Socio-economic status	Landless (reference group) Smallholder with LT 1/16 *mantal* Freeholder/crown tenant GE 1/16 *mantal* Noble tenant with GE 1/16 *mantal*
Family knowledge	Shared components	Family-based frailty
Family values/preferences	Shared components	Family-based frailty
Genetics	Shared components	Family-based frailty
Individual level		
	Birth cohort	Year of birth
Maternal factors	Low maternal age Normal maternal age High maternal age	Maternal age at childbirth under 22 Maternal age at childbirth 22 to 34 (ref grp) Maternal age at childbirth 35 and over
Nutrition & disease load	Birth quarter	Winter quarter (reference group) Spring quarter Summer quarter Autumn quarter
Sex		Male (reference group) Female

Note: Abbreviations in table: GE = Greater or Equal to. LT = Less Than.

[8] Mosley & Chen (1984).

characteristics, family belonging, and conditions in very early life (in utero and during infancy).[9] It is then operationalised as given below to work within a multilevel survival regression framework with frailty at the family level.

Main hypotheses

Four main hypotheses will be tested according to the expectations below:

- a positive effect of current nutrition on relative mortality risk (= increased risk of dying when food prices are going up) is expected,

- a positive effect of current disease load on relative mortality risk is expected,

- a positive interaction effect of current nutrition and current disease load on relative mortality risk is expected, and

- a delayed positive effect of current nutrition lagged one year, disease load lagged one year, as well as the interaction of them lagged on year; at least in last sub-period, is expected.

Results

Pre-transformation period: most results were in line with what was expected and nutrition intake and disease load during childhood had a significant positive effect on the mortality risk of children, and also the interaction between nutrition intake and disease load:

variable	exp(coef)	p
as.integer(bthdate)	0.992	-6.5e-03
Ref: parish Hög	1.000	
as.factor(parish)Kävlinge	1.313	1.4e-01
as.factor(parish)Halmstad	1.128	5.4e-01
as.factor(parish)Sireköpinge	1.385	8.9e-02
as.factor(parish)Kågeröd	1.185	3.4e-01
imrd	1.002	6.2e-01
lryedtv	1.852	2.3e-03
imrdtv	1.027	2.3e-06
Interaction lryedtv:imrdtv	1.097	1.8e-04
Ref: socc Landless	1.000	
as.factor(socc)Small	1.066	5.8e-01
as.factor(socc)Fr+Cr	1.210	2.7e-01
as.factor(socc)Noble	0.879	-2.8e-01
mthagbc < 22TRUE	1.006	9.6e-01
Ref: mthagbc 22-34	1.000	
mthagbc > 34.999TRUE	1.155	1.2e-01
Ref: season Winter	1.000	
seasonSpring	0.812	-7.3e-02
seasonSummer	0.915	-4.4e-01
seasonAutumn	0.897	-3.2e-01
Ref: sex Female	1.000	
sex male	1.184	3.7e-02
frailty.gaussian(fam)		2.4e-01

LR-test p=3.21e-06 Variance of random effect= 0.167

Transformation period: most results were in line with what was expected, and nutrition and disease had the expected sign, but only nutrition was significant. The interaction between nutrition and disease was highly significant and also positive. Lagged interaction effect had the expected sign and was not that far from being significant (p=0.14):

var	exp(coef)	p
as.integer(bthdate)	0.999	0.6700
as.factor(parish)Kävlinge	1.287	0.0680
as.factor(parish)Halmstad	1.046	0.7500

[9] Based on a theoretical child mortalty model derived in Johansson (2004); Chapter 5.

as.factor(parish)Sireköpinge	0.950	0.7200
as.factor(parish)Kågeröd	0.714	0.0130
imrd	1.000	0.9300
lryedtv	1.457	0.0600
imrdtv	1.003	0.5900
lryedtv:imrdtv	1.070	0.0065
as.factor(socc)Small	0.951	0.5600
as.factor(socc)Fr+Cr	0.802	0.1100
as.factor(socc)Noble	1.138	0.2500
mthagbc < 22TRUE	0.923	0.4000
mthagbc > 34.999TRUE	1.170	0.0650
seasonSpring	0.985	0.8700
seasonSummer	0.832	0.0800
seasonAutumn	0.970	0.7600
sex	0.834	0.0120
frailty.gaussian(fam)		0.0820

LR-test p=1.31e-07 Variance of random effect= 0.254

Industrialisation period: the effect of food prices are as expected but not significant and the mortality rate has an unexpected sign. However, the interaction is strong, positive and significant (see table below). There are also strong and significant effects from food prices the year before and mortality the year before (see next table).

var	exp(coef)	p
as.integer(bthdate)	0.990	5.3e-02
as.factor(parish)Kävlinge	1.491	3.0e-02
as.factor(parish)Halmstad	0.774	1.6e-01
as.factor(parish)Sireköpinge	0.975	8.8e-01
as.factor(parish)Kågeröd	0.789	1.8e-01
imrd	0.992	5.1e-01
lryedtv	1.471	1.7e-01
imrdtv	0.950	6.2e-04
lryedtv:imrdtv	1.229	8.8e-03
as.factor(socc)Small	0.942	6.0e-01
as.factor(socc)Fr+Cr	0.570	1.1e-03
as.factor(socc)Noble	1.049	7.8e-01
mthagbc < 22TRUE	1.008	9.4e-01
mthagbc > 34.999TRUE	1.494	7.9e-05
seasonSpring	0.958	7.3e-01
seasonSummer	0.974	8.3e-01
seasonAutumn	1.136	2.8e-01
sex	1.010	9.1e-01
frailty.gaussian(fam)		0.01500

LR-test p=3.33e-08 Variance of random effect= 0.397

var	exp(coef)	p
lryedtv	1.482	1.6e-01
lryedtv1	3.597	4.6e-05
imrdtv	0.955	2.2e-03
imrdtv1	1.038	1.7e-02
lryedtv:imrdtv	1.230	6.4e-03
lryedtv1:imrdtv1	0.860	7.3e-02

Conclusion

The estimates show that there was indeed a significant effect of low nutrition and also of high disease load on child mortality, and that the interaction of low nutrition and high disease load had a large and significant increase of mortality risk for children in all three sub-periods. Hence, current nutrition but also current disease load had an influence on child mortality during the Swedish mortality decline, and additionally there is a relative risk increase of mortality for children by 7 to 22 % caused by the interaction between malnutrition and disease, depending on sub-period. The conclusion is therefore that this interaction is important for explaining child mortality in pre-industrial Sweden, and current nutrition and current disease load as well as the interaction between them should be included in theoretical models of child mortality.