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PATHWAYS OF INEQUALITIES IN PAEDIATRIC INFECTIOUS DISEASES

Evidence from register-based cohort studies in Sweden

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To my family

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Abstract

Background:

In Sweden, the risk of paediatric infectious diseases is associated with low parental education and low household income. Health-related behaviours that increase the risk of paediatric infectious diseases include high pregnancy body mass index (i.e., body mass index ≥25), smoking during pregnancy, and low exclusive breastfeeding (i.e., exclusive breastfeeding for <4 months). These behaviours may serve as pathways linking socioeconomic circumstances to paediatric infectious diseases; however, this has rarely been studied. The overarching aim of this thesis was to investigate whether and how health-related behaviours link socioeconomic circumstances to infectious disease hospitalisations in the first 5 years of life.

Methods:

We created four register-based cohorts by combining data from the Medical Birth Register, the National Inpatient Register, the Cause of Death Register, the Longitudinal Integrated Database for Health Insurance and Labour Market Studies, the Total Population Register, and the Child Healthcare Quality Database in Uppsala. In the first study, we examined intersectional inequalities in paediatric infectious diseases in relation to maternal education, household income, sex/gender, and maternal migration status, as well as their combinations, using an analysis of individual heterogeneity and discriminatory accuracy approach. In the second study, we examined associations between high pregnancy body mass index and smoking during pregnancy—and paediatric infectious disease hospitalisations using regression models. In the third study, we examined associations between breastfeeding practice and paediatric infectious diseases using regression models. In the fourth study, we explored whether the effect of disadvantaged socioeconomic circumstances on the risk of paediatric infectious diseases was mediated by high pregnancy body mass index, smoking during pregnancy, and low exclusive breastfeeding, using causal mediation analysis.

Results:

In the first study, we identified complex inequalities in paediatric infectious diseases related to maternal education, household income,

sex/gender, and maternal migration status, as well as their combinations. Children from lower socioeconomic groups were more likely to be hospitalised for overall, upper respiratory, lower respiratory, and enteric infections, with maternal education being the primary driver of these socioeconomic inequalities. Additionally, we found that the overall risk of paediatric infectious diseases was unrelated to household income in children born to highly educated mothers, suggesting that maternal education may protect children from the adverse consequences of poverty.

In the second study, we found that high pregnancy body mass index and smoking during pregnancy were associated with an increased risk of paediatric infectious disease hospitalisations. Pregnancy overweight and obesity increased the risk of overall paediatric infectious diseases by 5% and 18%, respectively. Smoking during pregnancy increased the risk of respiratory infections by 29%.

In the third study, we found that the overall risk of paediatric infectious disease hospitalisation decreased with the duration of exclusive breastfeeding up to 4 months of age. Additionally, we found that breastfeeding reduced the risk of both respiratory and enteric infections in children under 2 years and the risk of respiratory infections in children aged 2–4 years.

In the fourth study, we found that higher exposure to health-related behaviours—namely, high pregnancy body mass index, smoking during pregnancy, and low exclusive breastfeeding—mediated approximately half of the association between maternal education level and paediatric infectious diseases. Most of this mediation effect was attributed to low exclusive breastfeeding. However, high pregnancy body mass index and smoking during pregnancy accounted for approximately 20% of the increased risk of infectious diseases observed in children born to mothers with less than 13 years of education.

Conclusion:

Our findings suggest that higher exposure to health-related behaviours is an important pathway linking socioeconomic circumstances to paediatric infectious diseases. Consequently, interventions targeting these behaviours may help reduce inequalities in paediatric infectious diseases.

Abbreviations

AIHDA	analysis of individual heterogeneity and discriminatory accuracy
aIRR	adjusted incidence rate ratio
AUC	area under the receiver operating characteristic curve
BF	breastfeeding
BMI	body mass index
CI	confidence interval
COVID-19	coronavirus disease 2019
EBF	exclusive breastfeeding
HR	hazard ratio
ICD	International Classification of Diseases
IR	incidence rate
IRR	incidence rate ratio
MIE	mediated interactive effect
OR	odds ratio
PNDE	pure natural direct effect
PNIE	pure natural indirect effect
PY	person-year
Q	quintile
RERI	relative excess risk due to interaction
RSV	respiratory syncytial virus

SECs	socioeconomic circumstances
TE	total effect

Enkel sammanfattning på svenska

Risken att drabbas av infektionssjukdomar under barndomen skiljer sig mellan sociala grupper. Vi vet att barn i Sverige till föräldrar med låg utbildningsnivå och låg inkomst har en ökad risk att drabbas av både vanliga och allvarliga infektioner. Vi vet även att vissa hälsorelaterade beteenden, som övervikt/obesitas under graviditeten, rökning under graviditeten och ingen/kortvarig helamning, ökar risken för infektionssjukdomar hos barn. Det är möjligt att dessa hälsorelaterade beteenden förmedlar sambandet mellan sociala faktorer och infektionssjukdomar hos barn. Detta har dock inte studerats i någon större omfattning. Syftet med denna avhandling var därför att studera sambandet mellan sociala faktorer och risken att barn drabbas av infektionssjukdomar, samt att studera hur ett eventuellt samband förmedlas av hälsorelaterade beteenden.

Denna avhandling består av fyra registerbaserade observationsstudier. Studierna använder information från medicinska födelseregistret, dödsorsaksregistret, patientregistret, longitudinell integrationsdatabas för sjukförsäkrings- och arbetsmarknadsstudier, registret över totalbefolkningen och barnhälsovårdsregistret i Uppsala.

I den första studien undersökte vi ojämlikheter i infektionssjukdomar relaterade till mors utbildningsnivå, mors födelseland, familjens disponibla inkomst, kön/genus samt deras interaktioner. I studien ingick barn 0–4 år födda i Sverige mellan 1998 och 2015 (n=1 785 588). Vi fann att låg utbildningsnivå hos modern och låg disponibel familjeinkomst ökade risken för infektionssjukdomar. Vi fann även att dessa två faktorer interagerade med varandra; det fanns inget samband mellan disponibel familjeinkomst och infektionssjukdomar hos barn till högutbildade mammor.

I den andra studien undersökte vi sambandet mellan övervikt/obesitas samt rökning under graviditeten och infektionssjukdomar. I studien ingick barn 0–4 år födda i Sverige mellan 1998 och 2006 (n=693 007). Vi fann att övervikt under graviditeten ökade risken för infektionssjukdomar hos barn med 5% och att obesitas under graviditeten ökade risken för infektionssjukdomar hos barn med 18%. Vi fann även att rökning under graviditeten ökade risken för nedre luftvägsinfektioner med 29%.

I den tredje studien undersökte vi sambandet mellan amning och infektionssjukdomar. I studien ingick barn 0–4 år födda i Uppsala län mellan 1998 och 2010 (n=37 825). Vi fann att helamning upp till fyra månaders ålder minskade risken för infektioner under de första två åren samt att amning minskade risken för luftvägsinfektioner mellan två och fem års ålder.

I den fjärde studien undersökte vi om sambandet mellan mors utbildningsnivå och infektionssjukdomar förmedlades av hälsorelaterade beteenden. I studien ingick barn 0–4 år födda i Uppsala län mellan 1997 och 2015 (n=63 216). Vi fann att övervikt/obesitas under graviditeten, rökning under graviditeten samt ingen/kortvarig helamning förmedlade ungefär halva sambandet mellan mors utbildningsnivå och infektionssjukdomar hos barnen. Detta berodde till stor del på skillnader i amning mellan barn till låg och högutbildade mammor.

Sammanfattningsvis fann vi att ojämlikheter i infektionssjukdomar under barndomen till stora delar förmedlades av hälsorelaterade beteenden. Dessa ojämlikheter bör därför kunna minskas genom insatser som stöttar amning och motverkar övervikt/obesitas samt rökning.

Original papers

This thesis is based on the following papers, referred to as Study I, II, III and IV.

I Videholm S, Silfverdal SA, Gustafsson PE. Socioeconomic circumstances, health-related behaviours and paediatric infections: a mediation analysis. *Archives of Disease in Childhood* 2024;109:195-200.

II Videholm S, Silfverdal S, Reniers G. Maternal weight and infections in early childhood: a cohort study. *Archives of Disease in Childhood* 2019;104:58-63.

III Videholm S, Wallby T, Silfverdal S. Breastfeeding practice, breastfeeding policy and hospitalisations for infectious diseases in early and later childhood: a register-based study in Uppsala County, Sweden. *BMJ Open* 2021;11:e046583.

IV Videholm S, Silfverdal SA, Gustafsson PE. Intersectional inequalities in paediatric infectious diseases: a national cohort study in Sweden. *J Epidemiol Community Health* 2024;78:417-423.

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Introduction

In 2019, the global burden of infectious diseases in children under 5 years of age was estimated at 2.4 million deaths and 6.9 million years of healthy life lost to disability, amounting to 217.2 million years of health lost due to death and disability. Infectious diseases accounted for approximately 45% of the total disease burden in this age group.[1]

Inequalities in paediatric infectious diseases

There are substantial inequalities in paediatric infectious diseases between countries. The majority of deaths caused by infectious diseases occur in countries with a low socio-demographic index, a composite measure of fertility, education, and income.[1 2] Mortality rates for lower respiratory infections and diarrhoea, in particular, are strongly associated with the socio-demographic index.[3 4] However, paediatric infectious diseases remain a significant cause of hospitalisations even in high-income countries.[5-7]

Within high-income countries, paediatric infectious diseases disproportionately affect children from lower socioeconomic groups. The overall risk of paediatric infectious diseases is associated with low parental education levels, low household income, and ethnicity.[6 8] Children born to immigrant parents have an increased risk of certain infections, such as parasitic infections and tuberculosis.[9] Additionally, children facing multiple social disadvantages are more likely to acquire specific infections. For example, ethnic minority children born to low-educated parents are more susceptible to *Helicobacter pylori*, hepatitis A, and cytomegalovirus infections.[10]

In Sweden, children of low-educated mothers are at higher risk of hospitalisation for lower respiratory infections, gastroenteritis, sepsis, and meningitis.[11-14] Children living in low-income families are at higher risk of gastroenteritis and experience higher mortality rates from respiratory infections.[13 15] Children of immigrants are more susceptible to gastroenteritis and are more likely to be prescribed antibiotics.[13 16 17] Compared with Swedish-born children, tuberculosis is much more common among children born in certain high-endemic countries, particularly Somalia.[18] Compared with resident children, asylum-seeking children are more susceptible to infectious diseases, including respiratory infections, gastroenteritis, and skin infections.[19] A summary

of Swedish studies examining socioeconomic circumstances (SECs) and paediatric infectious diseases is provided in Appendix Table A1.

Intersectional inequalities

Studies examining inequalities in paediatric infectious diseases in Sweden have typically focused on a single social disadvantage, such as low parental education, low household income, and parental migration status, or they have examined multiple disadvantages separately (Appendix Table A1). However, this approach does not capture the complex inequalities shaped by the interplay of multiple social disadvantages.

Intersectionality is a theoretical framework for analysing multiple social disadvantages. The term intersectionality was first introduced by Kimberlé Crenshaw (1989) to describe how the lives of Black women are shaped by racism and sexism in ways that separate analyses of race and gender cannot capture.[20] Since then, intersectionality has influenced various academic disciplines, including feminist legal studies, gender studies, psychology, and public health.[21 22] An intersectional approach assumes that multiple social identities—such as socioeconomic position, sex/gender, and ethnicity—and their interactions shape individuals' health. These identities reflect interlocking systems of privilege and oppression, including poverty, racism, and sexism. Such an approach can (i) uncover differences within population groups often considered homogeneous, such as men, women, ethnic minorities, and migrants, and (ii) reveal how complex power structures shape inequalities.[21 23]

Pathways of inequalities

SECs may influence child health through material, psychosocial, behavioural, and structural pathways.[24]

A framework developed by Diderichsen et al.[24 25] illustrates these pathways and the corresponding policy entry points (Figure 1). In this framework, the primary driver of health inequalities is social stratification shaped by structural forces. These structural forces—political, economic, cultural, and commercial—affect child health by determining control over power and resources. The second driver is higher exposure to material, psychosocial, and behavioural risks among lower socioeconomic groups. Material risks include unhealthy food, inadequate clothing, and poor housing. Psychosocial risks involve negative feelings and stress. Behavioural risks include alcohol consumption, unhealthy diets, and physical inactivity. The third driver is increased vulnerability to health risks in lower socioeconomic groups, who are often exposed to multiple interacting risks. The fourth driver involves the differential consequences of childhood illness, which may perpetuate health inequalities across the life course and generate further social stratification (the fifth driver).[24 25]

This thesis focuses on how the behavioural pathway, in the form of high pregnancy body mass index (BMI), smoking during pregnancy, and breastfeeding (BF) may link SECs to paediatric infectious diseases. More generally, health-related behaviours may link SECs to paediatric infectious diseases due to differences in health literacy, defined as the 'capacity to obtain, process and understand basic health information and services needed to make appropriate health decisions'.[26] Health literacy is associated with both education level and child health.[27-29] Additionally, material deprivation, psychosocial stress, and structural forces may influence the risk of paediatric infectious diseases through health-related behaviours (Figure 1). In Sweden, one study has shown that health-related behaviours partly mediate the effect of low maternal education on the risk of respiratory infections (Appendix Table A1).[11]

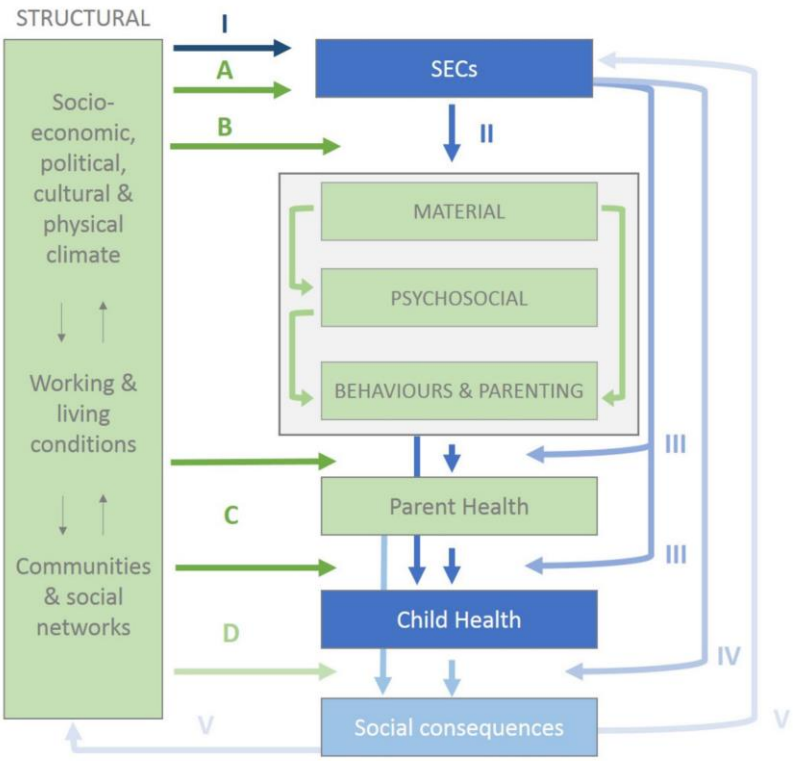

Figure 1: Conceptual framework illustrating pathways of health inequalities and corresponding policy entry points. The drivers of health inequalities include (I) social stratification, (II) differential exposure, (III) differential vulnerability, (IV) differential consequences, and (V) further social stratification. Aligned with these drivers are four key policy entry points: (A) social stratification, (B) exposure to health-related factors, (C) vulnerability to health-related factors, and (D) consequences of ill health. From Pearce et al, Figure 3.[24] The figure is re-published with permission from the publisher.
SECs, socioeconomic circumstances.

Health-related behaviours and paediatric infectious disease

This thesis examines three health-related behaviours: high pregnancy BMI, smoking during pregnancy, and BF. These health-related behaviours are important determinants of child health that interventions can target. Additionally, smoking and low BF rates are well-established risk factors for paediatric infectious diseases,[3 4] while emerging evidence also suggests that high pregnancy BMI may increase the risk of such infections.[30 31]

High pregnancy BMI

High pregnancy BMI is associated with numerous adverse outcomes for both mother and child, including miscarriage and stillbirth, gestational diabetes, obstetric complications, maternal death, neonatal deaths, and childhood obesity.[32-34] Increasing evidence indicates that children of mothers with obesity are at higher risk of paediatric infectious diseases, particularly respiratory infections.[30 31 35-38] Additionally, Swedish studies shown an increased susceptibility to neonatal sepsis among children of mothers with high pregnancy BMI (Table 1).[39 40] During pregnancy, dietary and lifestyle interventions may reduce maternal weight gain and lower the risk of pre-eclampsia, while dietary interventions alone may help prevent preterm birth.[41] Before pregnancy, taxing unhealthy foods, subsidising healthy options, restricting the marketing of unhealthy foods and mandatory understandable nutrition labelling can help reduce rates of overweight and obesity.[42 43] Additionally, anti-obesity medications and bariatric surgery can effectively treat obesity and reduce its complications.[44] For example, treatment with bariatric surgery has been shown to reduce the risk of adverse delivery outcomes in later pregnancies.[45]

Table 1: Swedish studies examining pregnancy overweight and obesity in relation to paediatric infectious diseases

Study	Study design	Population	Outcome	Findings
Håkansson and Källen[39]	Cohort study	Sweden 1997–2001 n = 344 127	Early-onset GBS	BMI 18.5–24.9 = OR 1.0 BMI 25.0–29.9 = OR 1.5 (1.1–2.0) BMI ≥30.0 = OR 1.7 (1.2–2.6)
Videholm et al.[14]	Cohort study	Sweden 1997–2013 n = 1 406 547	Sepsis and bacterial meningitis 28 days to <2 years	Sepsis: BMI 18.5–24.9 = IRR 1.0 BMI 25.0–29.9 = IRR 1.10 (0.92–1.30) BMI ≥30.0 = IRR 1.11 (0.88–1.39) Bacterial meningitis: BMI 18.5–24.9 = IRR 1.0 BMI 25.0–29.9 = IRR 1.06 (0.82–1.36) BMI ≥30.0 = IRR 0.99 (0.72–1.37)
Villamor et al.[40]	Cohort study	Sweden 1997–2016 n = 1 971 346	Early-onset neonatal sepsis (≤72h)	BMI 18.5–24.9 = OR 1.0 BMI 25.0–29.9 = OR 1.19 (1.08–1.32) BMI 30.0–34.9 = OR 1.70 (1.49–1.94) BMI 35.0–39.9 = OR 2.11 (1.73–2.58) BMI ≥40.0 = OR 2.50 (1.86–3.38)

Studies were identified through a PubMed search restricted to children (0–18 years of age) using the following terms: infect* AND (pregnancy overweight OR pregnancy obesity OR pregnancy BMI) AND Sweden. The search yielded 36 articles; 6 were reviewed in full text, 1 additional article was added manually, and 3 articles were included in the final selection.
BMI, body mass index; GBS, group B streptococcal disease; IRR, incidence rate ratio; OR, odds ratio.

Pregnancy smoking

Smoking during pregnancy has detrimental effects on child health. Children born to mothers who smoke during pregnancy are at higher risk of preterm birth, congenital malformations, sudden infant death syndrome, and experience higher perinatal mortality rates.[46 47] Later in life, they are more likely to experience complications such as obesity, type 2 diabetes, attention deficit hyperactivity disorder, and substance abuse.[48] It is also well established that pregnancy smoking increases the risk of paediatric infectious diseases, particularly respiratory infections.[46 49 50] Swedish studies have shown that children born to mothers who smoke during early pregnancy are more likely to be hospitalised for respiratory infections and bacterial meningitis (Table 2).[11 14] There is strong evidence that smoking cessation counselling in early pregnancy increases the likelihood of quitting smoking.[48] Additionally, the use of nicotine replacement therapy during pregnancy is considered safe and may improve cessation rates.[48 51]

Table 2: Swedish studies examining pregnancy smoking in relation to paediatric infectious diseases

Study	Study design	Population	Outcome	Findings
Håkansson and Carlsson[52]	Cohort study	Teleborg 1983–1984 n = 192	Antibiotic-treated respiratory tract infections <1 year	Children of smoking mothers: 60% vs. children of non-smoking mothers: 40%
Hjern et al.[11]	Cohort study	Stockholm 1990–1994 PY = 820 871	Lower respiratory infections 0–4 years	Pregnancy smoking No = OR 1.0 Yes: Year 1 = OR 1.2 (1.1–1.4) Year 2 = OR 1.3 (1.2–1.5) Year 3 = OR 1.2 (1.03–1.4) Year 4 = OR 1.0 (0.8–1.2) Year 5 = OR 0.9 (0.7–1.2)
Mangrio et al.[17]	Cross-sectional study	Malmö 2003–2006 n = 7266	Antibiotic use 0–7 months	Pregnancy smoking No = OR 1.0 Yes: OR 0.93 (0.72–1.20)
Lind et al.[13]	Cohort study	Sweden during 2006–2012 n = 752 078	Gastroenteritis <5 years	Maternal smoking during early pregnancy: No = HR 1.0 Yes = HR 0.96 (0.91–1.01)
Rhedin et al.[53]	Case-control study	Stockholm 2011–2014 n = cases /controls 93/240	Pneumonia ≤5 years	≥1 parent smoking = cases 19% vs. controls 10%, p = 0.01
Videholm et al.[14]	Cohort study	Sweden 1997–2013 n = 1 406 547	Sepsis and bacterial meningitis 28 days to <2 years	No = IRR 1.0 Yes: Sepsis = IRR 0.96 (0.75–1.25) Bacterial meningitis = IRR 1.46 (1.05–2.04)

Studies were identified through a PubMed search restricted to children (0–18 years of age) using the following terms: infect* AND pregnancy AND (smoking OR smoke) AND Sweden. The search yielded 58 articles; 14 were reviewed in full text, 4 additional articles were added manually, and 6 articles were included in the final selection.

HR, hazard ratio; IRR, incidence rate ratio; OR, odds ratio; PY, person-years.

Breastfeeding

BF is a key determinant of child health. In high-income countries, it is associated with a reduced risk of childhood cancer, childhood obesity, inflammatory bowel disease, coeliac disease, type 1 diabetes, and type 2 diabetes as well as higher intelligence.[54-56] Randomised controlled trials and large observational studies have demonstrated that exclusive BF (EBF) reduces the risk of paediatric infectious diseases.[57-59] For example, the 'Promotion of Breastfeeding Intervention Trial' showed that an intervention based on the 'Baby-Friendly Hospital Initiative' increased the prevalence of EBF at 3 months from 6% to 43% and reduced the risk of gastrointestinal infections by approximately 40%.[60] Additionally, growing evidence suggests that BF provides protection against respiratory infections beyond infancy.[61 62] Swedish studies have shown that BF protects against invasive *Haemophilus influenzae*, urinary tract infections, and otitis media and that breastfed children are less likely to be treated with antibiotics (Table 3).[17 63-66] BF support from the antenatal to postnatal periods promotes EBF.[67-69] There is also evidence supporting skin-to-skin care, rooming-in (keeping the infant and mother together), and cup feeding.[67] Additionally, paid maternity leave has been shown to promote BF.[67 68]

Table 3: Swedish studies examining breastfeeding in relation to paediatric infectious diseases

Study	Study design	Population	Outcome	Findings
Harsten et al.[70]	Cohort study	Lund n = 113	Recurrent AOM 0–2 years	No association between duration of BF and infections
Håkansson and Carlsson[52]	Cohort study	Teleborg 1983–1984 n = 192	Antibiotic-treated respiratory infections <1 year	No association between duration of BF and infections
Aniansson et al.[66]	Cohort study	Göteborg n = 400	AOM URI <1 year	Age at first AOM was reversely associated with duration of BF Lower risk of URI between 4–12 months in breastfed children

Silfverdal et al.[63]	Case-control study	Örebro County 1987–1992 n = cases/controls 54/139	Invasive *Haemophilus influenza* <6 years	EBF: ≥13 wk = OR 1.0 0–12 wk = OR 3.79 (1.6–8.8) Any BF: ≥21 wk = OR 1.0 0–20 wk = OR 2.92 (1.3–6.5)
Silfverdal et al.[64]	Ecological study	Örebro County 1956–1992	*Haemophilus influenzae* meningitis 1 mo to 16 years	Strong (negative) correlation between BF and infection with time lag of 5–10 years
Mårild et al.[65]	Case-control study	Göteborg 1993–1995 n = cases/controls 200/336	Urinary tract infections 0–6 years	Non-BF vs. BF HR 2.30 (1.56–3.39) Larger effect for girls than for boys (HR 3.78 vs. HR 1.63)
Mangrio et al.[17]	Cross-sectional study	Malmö 2003–2006 n = 7266	Antibiotic use ≤8 months	Any BF: 0 mo = OR 1.92 (1.31–2.80) 0.1–3 mo = OR 0.97 (0.77–1.24) 3.1–6 mo = OR 1.00 6.1–8 mo = OR 0.92 (0.65–1.32)

Studies were identified through a PubMed search restricted to children (0–18 years of age) using the following terms: infect* AND (breast feeding OR breastfeeding OR breastfed) AND Sweden. The search yielded 190 articles; 12 were reviewed in full text, and 7 were included in the final selection.
AOM, acute otitis media; BF, breastfeeding; EBF, exclusive breastfeeding; HR, hazard ratio; mo, months; OR, odds ratio; URI, upper respiratory infections; wk, weeks.

The Swedish setting

Sweden is a wealthy egalitarian society with comparatively low economic inequalities and high social mobility.[71] However, economic inequality has increased over the past few decades. For example, Sweden's Gini coefficient rose from 0.22 in 1990 to 0.31 in 2023.[72] At the same time, the Swedish government's commitment to reducing inequalities has declined; Sweden dropped from 1st place in 2017 to 24th place in 2024 in the Commitment to Reducing Inequality Index.[73] [74]

Public health

Sweden has a cross-sectoral public health policy framework. The overarching goal of this framework is 'to create societal prerequisites for good health on equal terms for the entire population'.[75] The policy is structured around eight target areas:

- Conditions in early life
- Knowledge, skills, and education/training
- Work, working conditions, and work environment
- Income and economic resources
- Housing and neighbourhood conditions
- Health behaviours
- Control, influence, and participation
- Equitable and health-promoting health and medical services

Additionally, Sweden has specific public health strategies that complement its overarching policy framework. For example, Sweden has a comprehensive national strategy for the prevention of alcohol, narcotics, doping, and tobacco. Tobacco prevention measures include taxation of tobacco products, smoke-free legislation, and smoking cessation programmes. This strategy aims to achieve a 'non-smoking Sweden' (smoking prevalence of <5%).[76] Sweden also has a national strategy for BF based on the Baby-Friendly Hospital Initiative, which recommends that all women receive tailored BF support during pregnancy and after birth.[77] By contrast, Sweden does not have a national strategy for the prevention of overweight and obesity.[78]

Preventive care

The Swedish healthcare system is publicly financed and explicitly committed to ensuring the health of all citizens.[79] Preventive maternal and child healthcare is provided through antenatal and well-baby clinics.

Antenatal care in Sweden is free of charge and has an almost universal uptake. All women are offered approximately 10–12 visits, which include health examinations, guidance on alcohol consumption, a healthy diet, physical activity, parenting, and information about labour and birth. BF recommendations follow the 'Ten Steps to Successful Breastfeeding'.[80 81] Women at increased risk of complications—such as those with overweight, smoking or snuff use, cardiovascular disease, diabetes mellitus, or mental health conditions—are offered additional follow-up.[82] This includes lifestyle interventions in line with national guidelines for obesity care and smoking cessation counselling. Women with complications are referred to an obstetrician if necessary.[80 81]

A preventive child health programme is provided free of charge by well-baby clinics, with an uptake that is virtually complete across all socioeconomic groups.[83] The programme is designed based on the concept of 'proportionate universalism',[84 85] which refers to actions that are 'universal, but with a scale and intensity that is proportionate to the level of disadvantage'.[86] It has three levels, depending on the needs of the family. The first level is offered to all children and includes health examinations, developmental and growth monitoring, vaccinations, and health advice, provided by public health nurses and general practitioners. This level consists of approximately 12 visits. The second level is targeted at families requiring additional support due to medical conditions or social circumstances and includes extra visits, parental support, and additional vaccinations for at-risk groups. The third level is designed for families with the greatest needs and provides enhanced support in collaboration with social services and referrals to other healthcare providers if needed.[84 85]

The Swedish national vaccination programme for children includes vaccines against diphtheria, tetanus, whooping cough, polio, measles, mumps, rubella, *Haemophilus influenzae* type b, *Streptococcus pneumoniae* (since 2009), human papillomavirus (girls since 2010 and boys since 2020), and rotavirus (since 2019). Additionally, all regions offer vaccinations against hepatitis B (since 2016).[87] In 2024, the Public Health Agency of Sweden recommended that the government introduce varicella vaccination into the national programme.[88] However, this recommendation has not yet been implemented. The vaccination coverage rate is approximately 95% for diphtheria, tetanus, whooping cough, polio, *Haemophilus influenzae* type b, and *Streptococcus pneumoniae*; slightly lower for measles, mumps, and rubella; and around 90% for rotavirus.[87] Coverage is equitable across socioeconomic groups.[83]

Health-related behaviours

The prevalence of pregnancy overweight and obesity has increased over the past three decades (Figure 2). This rise in pregnancy BMI is observed across all educational levels; however, the rise has been slower for women with higher education.[89]

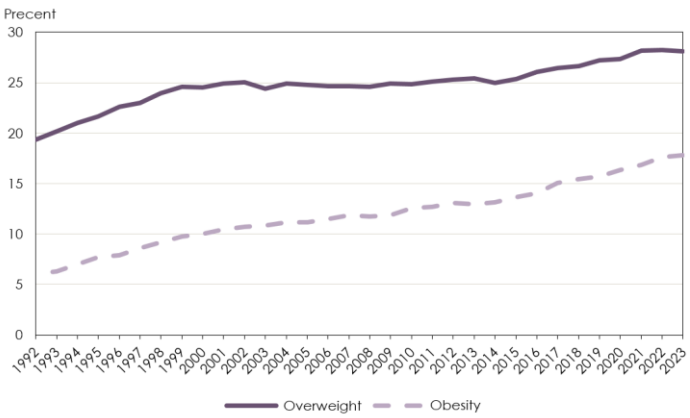

Figure 2: Proportion of pregnant women with overweight (BMI 25.0–29.9) and obesity (BMI 30.0 or more) at registration to antenatal care, 1992–2023.
Source: Medical Birth Register, National Board of Health and Welfare.
BMI, body mass index.

The prevalence of pregnancy smoking has decreased markedly over the past four decades (Figure 3). Smoking during pregnancy is strongly associated with low maternal education.[90]

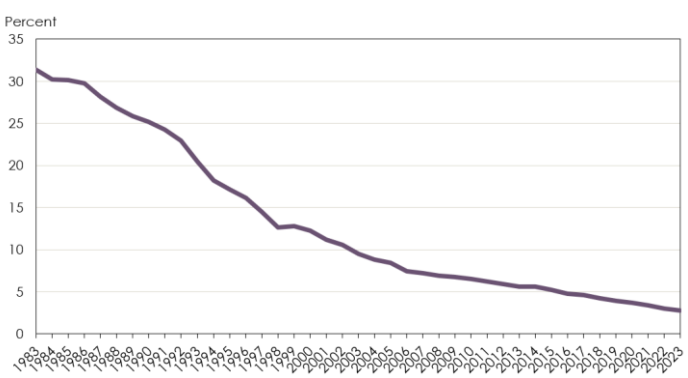

Figure 3: Maternal smoking at registration to antenatal care, 1983–2023.
Source: Medical Birth Register, National Board of Health and Welfare.

In 2021, approximately 50% of Swedish children were exclusively breastfed until 4 months of age, and approximately 65% were breastfed until 6 months (Figure 4). Over time, BF rates gradually declined (Figure 5), with decreases observed across all educational groups.[91]

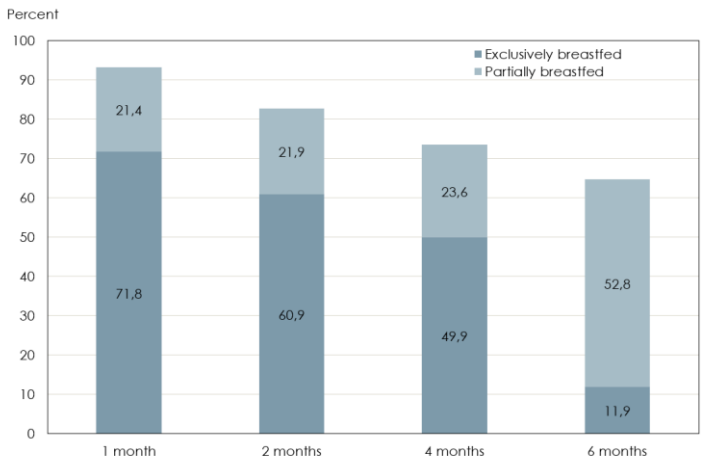

Figure 4: Exclusively and partially breastfed infants by age. Infants born 2021. Source: Breastfeeding Statistics, National Board of Health and Welfare.

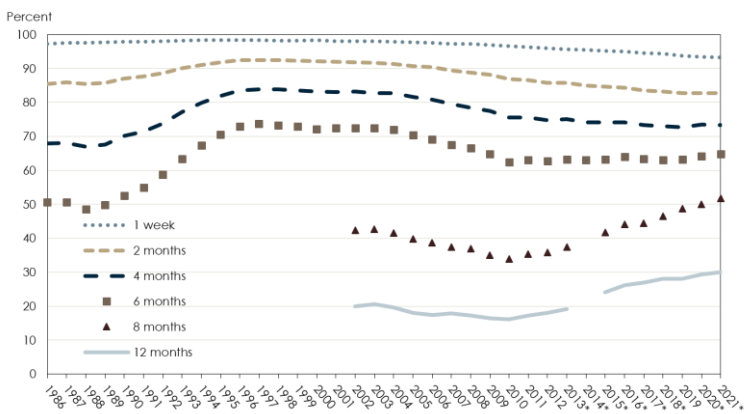

Figure 5: Exclusively or partially breastfed infants born 1986–2021. Information on breastfeeding at 8 and 12 months started in 2002. Source: Breastfeeding Statistics, National Board of Health and Welfare.
*National prevalence in 2013–2018 and 2020–2021 were estimated due to missing data for some regions. In 2014, no national prevalence is shown due to low data quality.

Paediatric infectious diseases

Among children under 5 years of age, the incidence of infectious disease hospitalisations is 3521 per 100 000 person-years (PY) for boys and 2798 per 100 000 PY for girls (Figure 6). This represents 28% of all hospitalisations in this age group. Upper and lower respiratory infections account for nearly half of these admissions. The leading aetiologies of lower respiratory infections—such as community-acquired pneumonia—are influenza viruses, metapneumovirus, and respiratory syncytial virus (RSV).[53] Bacterial causes include *Streptococcus pneumonia* and *Haemophilus influenzae* type b.[92] Enteric infections account for approximately one-fifth of all hospitalisations, with the leading causes being rotavirus, norovirus, adenoviruses, and *Campylobacter jejuni*.[92 93]

The incidence of paediatric infectious diseases in Sweden has changed over the past two decades (Figure 6). Prior to the coronavirus disease 2019 (COVID-19) pandemic, the incidence of all major infectious disease categories declined steadily, with the exception of genitourinary infections. The declining rates of respiratory and enteric infections may partly be attributed to the introduction of pneumococcal and rotavirus vaccination.[94 95] Following the COVID-19 pandemic, the incidence of lower respiratory infections markedly increased. The most likely explanation is that the usual circulation patterns of paediatric infectious diseases were disrupted during the pandemic, resulting in a population immunity debt to certain pathogens, including RSV, Bordetella pertussis (whooping cough), and Mycoplasma *pneumoniae*.[96]

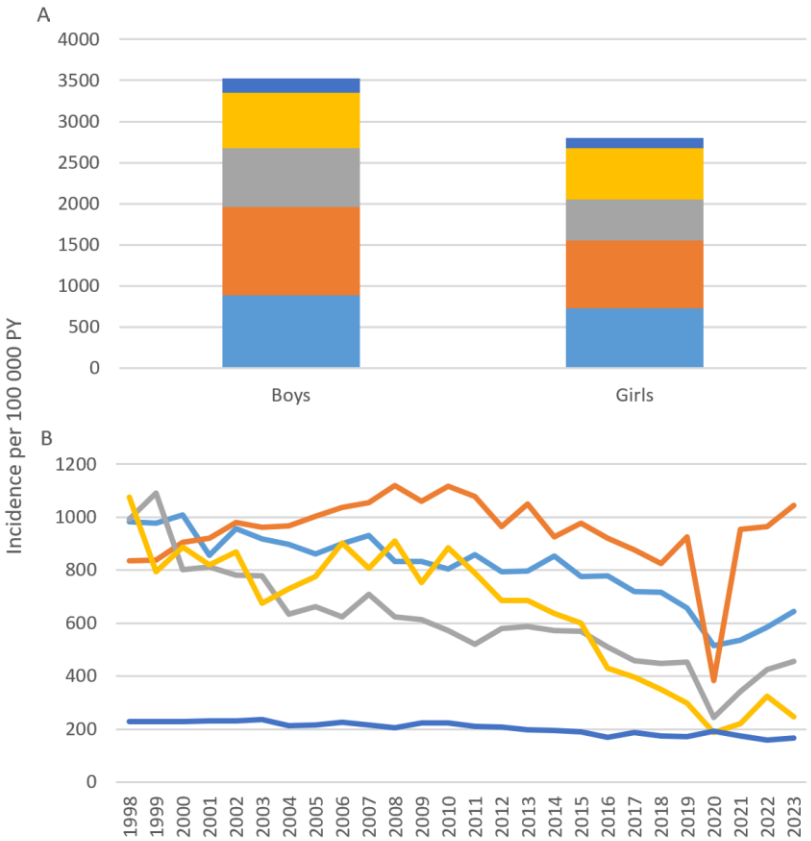

Figure 6: Incidence of genitourinary (dark blue), enteric (yellow), upper respiratory (grey), lower respiratory (orange), and other (light blue) infections by (A) sex/gender and (B) calendar year. Infectious disease categories were defined according to ICD-10 codes presented in Appendix Table A2 (using 3 characters). The total incidence of hospitalisations between 1998 and 2023 was 12 399 per 100 000 PY for boys 0–4 years and 9864 per 100 000 PY for girls 0–4 years. Source: Inpatient Register, National Board of Health and Welfare. ICD-10, International Classification of Diseases, 10th Revision; PY, person-years.

Rationale for this thesis

Several previous studies have shown that SECs influence the risk of paediatric infectious diseases in Sweden. However, no prior study has examined intersectional inequalities in paediatric infectious diseases.

It is known that high pregnancy BMI, pregnancy smoking, and low EBF increase the risk of paediatric infectious diseases in Sweden. Nevertheless, further research is needed because no previous studies have systematically examined associations between high pregnancy BMI, pregnancy smoking, BF practices, and hospitalisations for overall and major categories of paediatric infectious diseases in Sweden. Only a few studies have explored the association between BF and infectious diseases beyond infancy.

Additionally, no previous study has examined the extent to which the effect of SECs on the risk of overall paediatric infectious diseases is mediated by health-related behaviours in Sweden.

Objectives

The overarching aims of this thesis were to (i) describe inequalities in paediatric infectious diseases, (ii) explore how health-related behaviours may influence the risk of paediatric infectious diseases, and (iii) investigate whether, and in what ways, health-related behaviours may link SECs to paediatric infectious diseases.

The specific objectives of this thesis were as follows.

Study I: To examine inequalities in paediatric infectious diseases in relation to maternal education, household income, sex/gender, maternal migration status, and their combinations.

Study IIa: To examine the association between high pregnancy BMI and hospitalisations for paediatric infectious diseases.

Study IIb: To examine the association between pregnancy smoking and hospitalisations for paediatric infectious diseases.

Study III: To examine the association between BF practices and hospitalisations for paediatric infectious diseases.

Study IV: To examine whether the effect of disadvantaged SECs on the risk of paediatric infectious diseases is mediated by high pregnancy BMI, pregnancy smoking, and low EBF.

Materials and Method

This project consists of four register-based cohort studies (see Table 4 for an overview). The first and second studies were national register-based cohort studies, including children born in Sweden between 1998–2015 and 1998–2006, respectively. The third and fourth studies were regional register-based cohort studies of children born to mothers residing in the Uppsala Region between 1998–2010 and 1997–2015, respectively.

Study design

A cohort study is an observational study that follows 'exposed' and 'unexposed' individuals over time, during which the outcome of interest is recorded. The occurrence of the outcome in the exposed group is then compared with that in the unexposed group. Cohort studies can include multiple exposures and multiple outcomes. Compared with case-control studies, cohort studies are less prone to recall bias, a form of differential misclassification that occurs when individuals with the outcome are more likely to recall the exposure.[97] This also applies to retrospective cohort studies if data have been collected prospectively.[98] However, cohort studies are susceptible to selection bias; i.e., systematic differences between individuals who participate and those who are eligible but do not participate. Like all observational studies, cohort studies are also prone to confounding, which occurs when a third factor distorts the apparent association between an exposure and an outcome. Confounding can be addressed through restriction, matching, stratification, or statistical modelling (e.g., regression analysis).[97]

A register-based study uses data obtained from health and administrative registers.[99] This approach offers several strengths for medical research. Health and administrative registers are typically population-based, which reduces the risk of selection bias due to non-response or loss to follow-up.[99 100] Register data are usually collected prospectively, thereby minimising the risk of recall bias. Additionally, compared with studies relying on primary data collection, register-based research is often more time- and cost-efficient. Limitations are mainly related to data quality and data availability.[100]

Table 4: Overview of the studies

	Study I	**Study II**	**Study III**	**Study IV**
Study design:	- Register-based cohort study			
Study population:	- Children born in Sweden 1998–2015	- Children born in Sweden 1998–2006	- Children born to mothers who resided in Uppsala Region between 1998–2010	- Children born to mothers who resided in Uppsala Region between 1997–2015
Data sources:	- Medical Birth Register - National Inpatient Register - Cause of Death Register - Longitudinal Integrated Database for Health Insurance and Labour Market Studies - Total Population Register	- Medical Birth Register - National Inpatient Register - Cause of Death Register - Longitudinal Integrated Database for Health Insurance and Labour Market Studies - Total Population Register	- Medical Birth Register - National Inpatient Register - Cause of Death Register - Longitudinal Integrated Database for Health Insurance and Labour Market Studies - Total Population Register - Child Healthcare Quality Database in Uppsala	- Medical Birth Register - National Inpatient Register - Cause of Death Register - Longitudinal Integrated Database for Health Insurance and Labour Market Studies - Total Population Register - Child Healthcare Quality Database in Uppsala
Exposures and mediators:	Exposures: - Intersectional variable (maternal education, disposable household income, sex/gender, and maternal migration status)	Exposures: - Pregnancy BMI - Pregnancy smoking*	Exposure: - Breastfeeding	Exposure: - Maternal education Mediators: - Pregnancy smoking - Pregnancy BMI - Breastfeeding

Main outcome:		- Paediatric infectious diseases in the first 5 years of life		
Covariates:	- Year of birth	- Maternal age - Maternal education level - Pregnancy smoking - Pregnancy BMI* - Parity - Geographic region - Year of birth	- Small for gestational age - Large for gestational age - Congenital malformation - Sex - Maternal age - Maternal education level - Pregnancy smoking - Parity - Maternal country of birth - Year of birth - Previous admissions	- Maternal age - Maternal country of birth - Year of birth
Statistical methods:	- Logistic regression models - Area under the receiver operating characteristic curve	- Negative binomial regression models - Multiple imputation†	- Negative binomial regression models - Generalised estimating equations	- Poisson regression models - Natural effect models - Multiple imputation

*In analysis of pregnancy smoking.

†In sensitivity analysis, results presented in the original paper.

BMI, body mass index.

Data sources

We combined data from the Medical Birth Register, the National Inpatient Register, the Longitudinal Integrated Database for Health Insurance and Labour Market Studies, the Cause of Death Register, the Total Population Register, and the Child Healthcare Quality Database in Uppsala. These registers were linked by the Centre for Epidemiology at the Swedish National Board of Health and Welfare, using the unique personal identification number assigned to all Swedish residents at birth or upon immigration.[101] All data were pseudo-anonymised, with the key retained by the Swedish National Board of Health and Welfare.

Information on pregnancies, deliveries, and new-born children was obtained from the Medical Birth Register. The register was established in 1973 by the National Board of Health and Welfare.[102] All healthcare providers in Sweden are legally required to report pregnancy, delivery, and neonatal data to the Medical Birth Register. Both coverage and overall data quality are very high. The register includes approximately 98% of all births in Sweden since 2000 and more than 99% since 2015. Key variables such as birthweight, gestational age, and maternal age have less than 1% missing data.[103] However, other variables—such as those on congenital malformations—have a higher degree of missingness.[102]

Data on hospital admissions were retrieved from the National Inpatient Register. The register was established in 1964 when the National Board of Health and Welfare began collecting data on somatic inpatient care in six Swedish counties.[104] It has had national coverage of inpatient hospital care since 1987 and of hospital-based outpatient care since 2001. Diagnoses in the National Inpatient Register are coded using the Swedish version of the International Classification of Diseases (ICD). In Sweden, the 10th Revision (ICD-10) has been used since 1997, except in Region Skåne, where the ICD-10 was introduced in 1998. The coverage is very high for inpatient care and approximately 80% for hospital-based outpatient care. The positive predictive value of most diagnoses in the register exceeds 85%.[104]

Socioeconomic data were retrieved from the Longitudinal Integration Database for Health Insurance and Labour Market Studies. This register is managed by Statistics Sweden and it covers the adult (≥16 years) Swedish population since 1990.[105] It compiles data from other

government-administered registers and includes information on income, occupation, education, and demographic characteristics. In Sweden, participation in government-administered registers is compulsory, resulting in very high coverage. For example, information on education is available for more than 98% of individuals aged 25–64 years. However, the proportion of individuals with low education is likely overestimated, and missing data on education is more common among immigrants. The overall accuracy for the highest attained level of education is estimated to be around 85%.[105]

Information on date of death was obtained from the Cause of Death Register. Managed by the National Board of Health and Welfare, this register contains electronic data on deaths in Sweden since 1952.[106] The underlying cause of death is classified using ICD codes. Coverage is virtually complete, with information on the underlying cause of death missing in less than 5% of cases. However, the accuracy of the register may be influenced by physicians' ability to certify deaths, the autopsy rate (around 7% for women and 15% for men in 2014), and the processing of death certificates by the National Board of Health and Welfare.[106]

Information on date of emigration and maternal country of birth was obtained from the Total Population Register. This register was established in 1968 following the digitalisation of Swedish population data in 1967.[107] Sweden maintains two registers for population data: the Population Register, managed by the Swedish Tax Agency, and the Total Population Register, managed by Statistics Sweden. The Swedish Tax Agency compiles population data in the Population Register, which is then transferred to the Total Population Register. The quality of population data in Sweden is high—virtually all births and deaths, 95% of immigrations, and 91% of emigrations are reported to the Population Registers within 30 days. However, over-coverage in the registers is estimated at 0.25%–0.50% of the population, mainly due to unreported emigration and deaths occurring abroad.[107]

Information on BF was obtained from the Child Healthcare Quality Database in Uppsala. The agreement between BF data recorded in the Child Healthcare Quality Database and that recorded in medical journals is approximately 95%.[108]

Exposures and mediators

Intersectional inequalities

The first study examined inequalities in paediatric infectious diseases in relation to maternal education, household income, sex/gender, maternal migration status, and their combinations. Information on maternal education and disposable household income was obtained from the Longitudinal Integrated Database for Health Insurance and Labour Market Studies. Maternal education was categorised as follows: secondary school or less (≤9 years), upper secondary school (10–12 years), and post-secondary education (≥13 years). Disposable household income at birth was divided into annual quintiles (Q1–Q5), with the fifth quintile (Q5) representing the highest income group. Information on sex/gender was obtained from the Medical Birth Register and categorised as girl or boy. Maternal migration status was obtained from the Total Population Register and categorised as non-immigrant (Swedish-born) or immigrant (foreign-born).

We created an intersectional strata variable comprising 60 strata by combining the 5 categories of household income, the 3 categories of maternal education, the 2 categories of sex/gender, and the 2 categories of maternal migration status. This variable was used to capture the joint effect of all sociodemographic variables and their interactions.

Health-related behaviours

The second study examined associations between high pregnancy BMI, pregnancy smoking, and hospitalisations for paediatric infectious diseases. Information on maternal height, weight, and smoking status at the first antenatal care visit was obtained from the Medical Birth Register.[102] Pregnancy BMI was calculated as weight (kg) divided by height $(m)^2$ and categorised as underweight (<18.5), normal weight (18.5–24.9), overweight (25.0–29.9), and obese (≥30.0). High pregnancy BMI was defined as pregnancy BMI ≥25 kg/m^2. Pregnancy smoking was categorised as yes or no.

The third study examined the association between BF practices and paediatric infectious diseases. Information on exclusive, partial, or no BF at 1 week, and at 2, 4, and 6 months was obtained from the Child Healthcare Quality Database in Uppsala. BF was categorised as no BF if 'no breastfeeding' was reported at 1 week. EBF was considered to be present until the first report of either 'partial breastfeeding' or 'no

breastfeeding', and any BF was considered present until 'no breastfeeding' was reported. BF was then categorised into six groups: no BF, EBF <4 months with BF <6 months, EBF <4 months with BF ≥6 months, EBF 4–5 months with BF <6 months, EBF 4–5 months with BF ≥6 months, and EBF ≥6 months.

Socioeconomic circumstances, mediation by health-related behaviours

The fourth study examined how the effect of disadvantaged SECs on the risk of paediatric infectious diseases was mediated by health-related behaviours.

In Sweden, as in other European countries, education is the preferred indicator of socioeconomic position.[109] Accordingly, children's SECs were captured through maternal education level. Maternal education was categorised as follows: secondary school or less (≤9 years), upper secondary school (10–12 years), short postsecondary education (13–14 years), and long postsecondary education (≥15 years). Mediators included high pregnancy BMI (i.e., pregnancy BMI of ≥25 kg/m^2), pregnancy smoking, and low EBF (i.e., EBF of <4 months).

Outcomes

The primary outcome in all studies was the overall number of paediatric infectious disease hospitalisations recorded in the National Inpatient Register. The secondary outcomes were the number of hospitalisations for specific categories of infectious diseases (Table 5). A previously developed coding scheme was used to identify hospital admissions with infection-related ICD codes (Appendix Table A2). This coding scheme has been evaluated by a panel of infectious disease clinicians, microbiologists, and disease coding experts.[6]

Table 5: Secondary outcomes

Study I	Study II	Studies III and IV
Upper respiratory infections	Upper respiratory infections	Respiratory infections
Lower respiratory infections	Lower respiratory infections	
Enteric infections	Enteric infections	Enteric infections
Genitourinary infections	Genitourinary infections	
	Perinatal infections	
	Skin and soft tissue infections	
	Neurological and eye infections	
	Bloodstream infections	
	Digestive infections	
	Other infections	

Statistical analyses

Intersectional inequalities

In the first study, we examined intersectional inequalities in paediatric infectious diseases using an analysis of individual heterogeneity and discriminatory accuracy (AIHDA) approach. In AIHDA, each group is treated as representing a specific social context.[110] In the first step, differences between groups are visualised using plots. In the second step, these differences are quantified by estimating the proportion of total individual variance that exists at the group level—that is, the share explained by social context.[110 111]

Analyses were performed in three steps for each outcome: overall, upper respiratory, lower respiratory, enteric, and genitourinary infections. First, we calculated incidence rates (IRs) with 95% confidence intervals (CIs) for each intersectional stratum—that is, for each unique combination of maternal education, disposable household income, sex/gender, and maternal migration status. Second, we fitted three consecutive logistic regression models for each outcome; the results were presented as odds ratios (ORs) with 95% CIs. The first model included year of birth only. The second model included sociodemographic variables and year of birth, estimating the joint effect of sociodemographic variables without interaction effects. The third model included the intersectional strata variable and year of birth, allowing for interaction effects between the sociodemographic variables. Third, we quantified the ability of each logistic model to discriminate between individuals with and without the outcome. This measure of discriminatory accuracy reflects how much of the total individual variance in an outcome that is explained by the predictor variable(s).[110] Discriminatory accuracy was assessed using the area under the receiver operating characteristic curve (AUC), which ranges from 0.5 (no discrimination) to 1.0 (perfect discrimination).[112] AUC values were interpreted as follows: 0.5–0.6 (absent or very small), >0.6–≤0.7 (moderate), >0.7–≤0.8 (large), and >0.8 (very large).[112] Finally, we calculated the incremental change in AUC (ΔAUC) between models. The inclusion of the intersectional variable in the third model allows for interaction effects; a positive ΔAUC indicates that interaction is present.[111]

Health-related behaviours

In the second study, we examined associations between high pregnancy BMI and paediatric infectious diseases using negative binomial regression models. A separate model was fitted for each outcome: overall, upper respiratory, lower respiratory, enteric, genitourinary, perinatal, skin and soft tissue, neurological and eye, bloodstream, digestive, and other infections. Results were presented as crude incidence rate ratios (IRRs) and adjusted IRRs (aIRRs) with 95% CIs. The adjusted analyses controlled for the potentially confounding effects of maternal age, maternal education level, pregnancy smoking, parity, geographic region, and year of birth. We used the same modelling approach to examine associations between pregnancy smoking and paediatric infectious diseases, with the adjusted analyses controlled for pregnancy BMI in place of pregnancy smoking.

In the third study, we examined associations between BF practices and hospitalisations for paediatric infectious diseases using negative binomial regression models. A separate model was fitted for each outcome (overall, respiratory, and enteric infections) and for each follow-up period (<2 years and 2–4 years). Results were presented as aIRRs with 95% CIs. All analyses were adjusted for year of birth. The adjusted analyses also controlled for the potentially confounding effects of small for gestational age, large for gestational age, congenital malformation, sex, maternal age, parity, pregnancy smoking, maternal education level, and maternal country of birth. In the later follow-up period, the adjusted analyses also included a control for previous admissions, using a binary variable indicating hospitalisation in the first follow-up period. In all analyses, we accounted for correlations between siblings using generalised estimating equations with robust standard errors.[113]

Socioeconomic circumstances, mediation by health-related behaviours and paediatric infections

In the fourth study, we examined how the effect of maternal education on the risk of paediatric infectious diseases was mediated by health-related behaviours using causal mediation analysis. For each outcome (overall, respiratory, and enteric infections), we fitted three natural effect models. The first model estimated the mediation effect of pregnancy smoking, the second model estimated the mediation effect of pregnancy smoking and high pregnancy BMI, and the third model estimated the mediation effect of pregnancy smoking, high pregnancy

BMI and low EBF. The first and second models were additional analyses not included in the original paper. Analysing multiple mediators *en bloc* is preferable to fitting separate models for each mediator because single-mediator models require independence between mediators.[114]

In these models, the total effect (TE) of maternal education on paediatric infectious diseases was decomposed into three components: the pure natural direct effect (PNDE), the pure natural indirect effect (PNIE), and the mediated interactive effect (MIE). The PNDE represents the effect of maternal education on childhood infections that is attributable to neither mediation nor interaction. The PNIE captures the portion of the effect that is due solely to mediation—i.e., differences in exposure to health-related behaviours. The MIE reflects the part of the effect due to an additive interaction between maternal education and the mediators—i.e., differences in susceptibility to health-related behaviours.[115] All analyses were adjusted for the potential confounding effects of maternal age, maternal country of birth, and year of birth.

Each mediation analysis was conducted in two steps. First, we imputed counterfactual outcomes for each potential level of maternal education. The dataset was expanded to include four observations per child—one for each potential level of maternal education. Mean values for the counterfactual outcomes were then imputed using a Poisson regression model that included maternal education, mediators, confounders, and an interaction term between maternal education and the mediators. Second, we estimated the PNDE, PNIE, TE, and MIE. The PNDE, PNIE, and a multiplicative interaction term were estimated by fitting a natural effect Poisson regression model to the expanded dataset. The TE was estimated as the linear combination of the PNDE, PNIE, and the multiplicative interaction term. The MIE was estimated as the relative excess risk due to interaction (RERI) = $IRR^{TE} - IRR^{PNDE} - IRR^{PINE} + 1$. In models with a TE > 1.05, we calculated the overall percentage mediated using the following formula: $[(IRR^{PNIE} - 1)/(IRR^{TE} - 1) + RERI^{MIE}/(IRR^{TE} - 1)] \times 100$.[115]

In all analyses, missing data were imputed using multiple imputation. Compared with single imputation, multiple imputation accounts for imputation uncertainty by introducing variability into the imputed values.[116] We generated 10 imputed datasets using a predictive model that included maternal education, pregnancy BMI, pregnancy smoking, EBF, maternal age, maternal country of birth, year of birth, parity, household income, follow-up time, overall infections, respiratory infections, and enteric infections. Regression results were pooled across

all imputed datasets using Rubin's rules, and percentages were calculated as averages across the imputed datasets.[117]

Methodological considerations

In the first study, intersectional inequalities were analysed using an AIHDA approach. Methods for intersectional analysis include cross-classification, regression with interaction terms, decision trees, fixed-effect AIHDA, and random-effect or multilevel AIHDA.[110 118] Compared to fixed-effect AIHDA, multilevel AIHDA is more closely aligned with intersectional theory because it explicitly models social context as a second level.[110] However, the fixed-effect AIHDA approach remains a valid method for intersectional analysis and has the advantage of being relatively straightforward to conduct and interpret.[110]

In the second and third studies, we examined associations between exposures and outcomes while adjusting for potential confounders. The causal inference framework offers an alternative to this traditional approach. According to this framework, analyses of observational data should emulate a hypothetical randomised trial with a clearly defined intervention. The effect of the hypothetical intervention is then estimated using methods such as propensity score matching, inverse probability weighting, or g-estimation.[119] Findings from well-conducted observational studies using causal inference methods may, in some cases, be interpreted as causal effects.[120 121] However, not all research questions can easily be framed as causal questions—particularly when no clear hypothetical intervention exists (e.g., SECs) or when numerous potential interventions are possible (e.g., high BMI).[119 122]

The fourth study examined how the effect of disadvantaged SECs on the risk of paediatric infectious diseases was mediated by health-related behaviours using causal mediation analysis. Compared with traditional mediation methods—such as the difference method, the product method, or path analysis using structural equation modelling—causal mediation analysis makes the assumptions required for causal interpretation explicit. These include (i) no confounding of the exposure–outcome relationship, (ii) no confounding of the mediator–outcome relationship, (iii) no confounding of the exposure–mediator relationship, and (iv) no mediator–outcome confounder that is itself affected by the exposure.[123] Additionally, causal mediation analysis allows for interactions between the exposure and mediator(s) and can therefore decompose the mediating effect into differential exposure and differential susceptibility.[115 124]

Ethical considerations

The fundamental ethical principles for research involving human subjects are respect for persons, beneficence, and justice.[125]

The principle of respect for persons requires that individuals be treated as autonomous agents and that participation in research be voluntary.[125] Register-based research is an exception to this principle because informed consent from individuals is not required. Instead, it is assumed that individuals do not object to their data being used for research purposes.[126] Surveys show that most people have a positive attitude toward the use of health data for medical research and are generally willing to forgo the requirement for informed consent.[127 128] However, concerns remain regarding data security and the potential for inappropriate access.[127 128] In this project, all data were pseudo-anonymised, and the risk of data leakage was minimal.

The ethical principle of beneficence requires that research maximises potential benefits while minimising possible harms. Researchers have an obligation to safeguard the well-being of participants and to consider what is beneficial for society as a whole.[125] For example, advancing child health through paediatric research is ethically justified, even when the participating children do not receive direct benefits from the research.[125] In this context, our research was ethically justified because it has the potential to improve children's overall health, despite study participants not receiving any direct individual benefit.

The ethical principle of justice states that the burdens of participating in research, as well as the benefits derived from it, should be distributed fairly across society.[125] Furthermore, this principle requires that the advances generated by research funded with public resources be made accessible to all.[125] Our project included all children, regardless of background; therefore, both the minimal burdens and the potential benefits of the research were equally distributed between privileged and underprivileged children.

All medical research involving human subjects requires approval from a research ethics committee.[129] This project was reviewed and approved by the Regional Ethical Review Board in Umeå (Ref. 2012-265-31M, with amendments 2013-320-32M, 2016-353-32M, and 2017-399-32M).

Results

Intersectional inequalities

The first study in this thesis examined intersectional inequalities in paediatric infectious diseases. It included 1 785 588 children born alive in Sweden between 1998 and 2015. Children were followed until 5 years of age or censored at death or emigration. The study comprised 7 974 833 PY of follow-up time and recorded 318 080 infectious disease hospitalisations. Most children were born to mothers with upper secondary (47%) or postsecondary (42%) education, and a substantial proportion (20%) were born to immigrant mothers.

Figure 7 shows the IR of overall infections for each intersectional stratum—that is, each unique combination of maternal migration status, sex/gender, maternal education, and household income. Figure 8 presents the IRs for upper respiratory, lower respiratory, enteric, and genitourinary infections across the same intersectional strata.

The incidence of overall infections decreased with increasing duration of maternal education and, to a lesser extent, with household income, among children born to both migrant and non-migrant mothers. However, among children born to highly educated mothers, the incidence of overall infections was unrelated to household income, suggesting an interaction between maternal education and household income (Figure 7). A similar pattern was observed for lower respiratory infections, where the incidence did not vary with household income among children of highly educated mothers. By contrast, the incidence of enteric infections was associated with household income regardless of maternal education level (Figure 8). Similar patterns were observed in the regression analyses (Appendix Table A3).

The ability of sociodemographic variables to discriminate between children with and without infections was low. The discriminatory accuracy ranged from an AUC of 0.55 (95% CI: 0.55–0.55) in the model for lower respiratory infections to an AUC of 0.59 (95% CI: 0.59–0.60) in the model for upper respiratory infections. Allowing for interactions did not increase the proportion of total individual variance explained by sociodemographic variables; the ΔAUC between models with an intersectional strata variable and those with sociodemographic variables was <0.01 in all cases (Appendix Table A3).

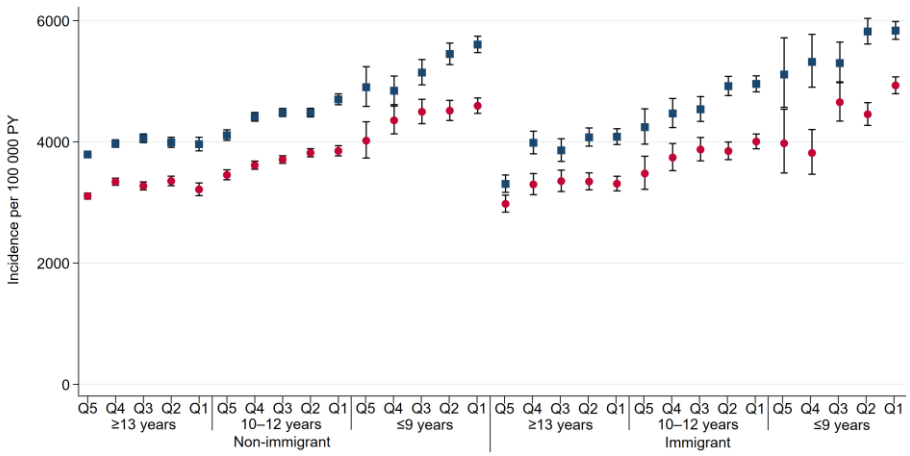

Figure 7: Incidence rates of hospitalisations for overall infections by sociodemographic factors. The figure shows boys (blue squares), girls (red circles), household income (Q5–Q1), maternal education (≥13 years, 10–12 years, and ≤9 years), and maternal migration status (non-immigrant and immigrant). Vertical lines represent 95% confidence intervals. From Videholm et al, Figure 1.[130]

PY, person-year.

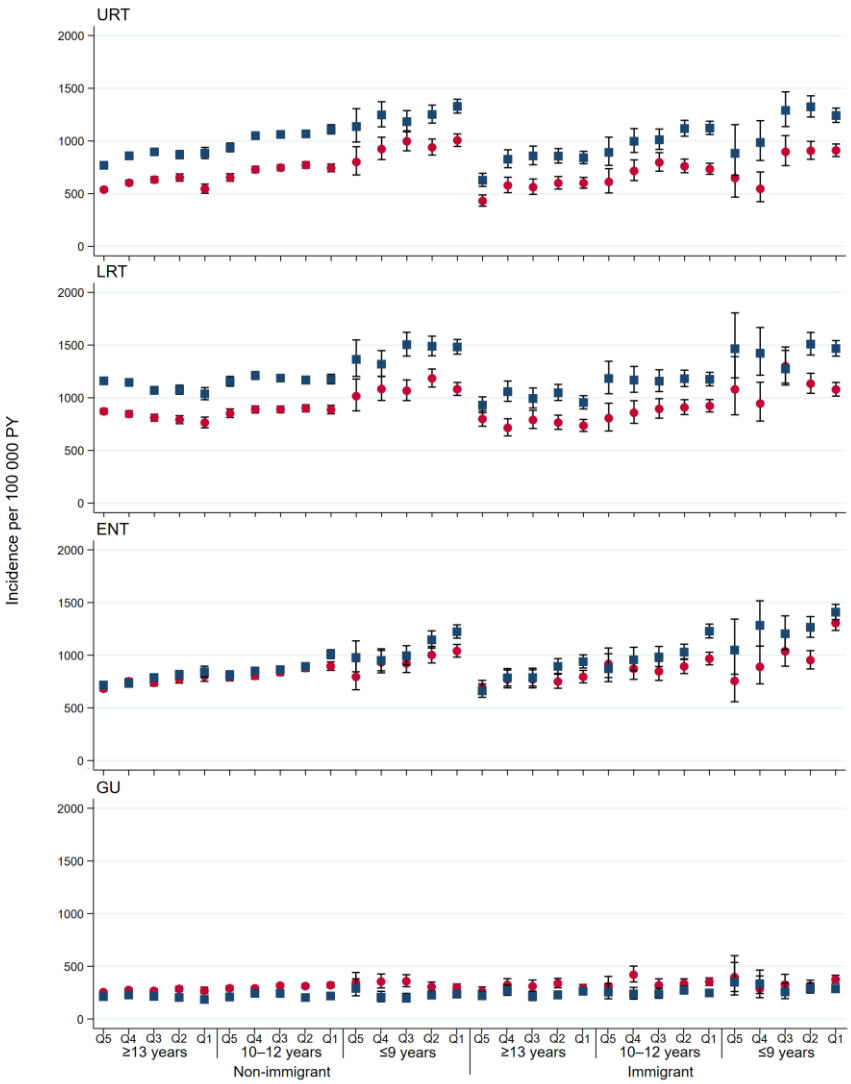

Figure 8: Incidence rates of hospitalisations for URT, LRT, ENT, and GU infections by sociodemographic factors. The figure shows boys (blue squares), girls (red circles), household income (Q5–Q1), maternal education (≥13 years, 10–12 years, and ≤9 years), and maternal migration status (non-immigrant and immigrant). Vertical lines represent 95% confidence intervals. From Videholm et al. Figure 2.[130]
ENT, enteric infections; GU, genitourinary infections; LRT, lower respiratory infections; PY, person-year; URT, upper respiratory infections.

High pregnancy BMI

The second study in this thesis examined the association between high pregnancy BMI and hospitalisations for paediatric infectious diseases. The study included 693 007 children born alive in Sweden between 1998 and 2006. Children were followed until 5 years of age or until censoring due to death or emigration. The study comprised 3 432 561 PY of follow-up time and recorded 125 010 infectious disease hospitalisations. A significant proportion of children were born to mothers with overweight (25%) or obesity (11%) during pregnancy.

Table 6 presents the associations between pregnancy BMI and hospitalisations for paediatric infectious diseases. Compared with children born to mothers with a normal pregnancy BMI, the risk of hospitalisation for paediatric infectious diseases was 5% higher (aIRR, 1.05; 95% CI, 1.03–1.06) in children born to mothers with pregnancy overweight and 18% higher (aIRR, 1.18; 95% CI, 1.16–1.21) in children born to mothers with pregnancy obesity. Children born to mothers with overweight or obesity during pregnancy were more likely to be hospitalised for certain infectious diseases, including respiratory, genitourinary, perinatal, and other infections. Additionally, children born to mothers with obesity during pregnancy had a higher likelihood of hospitalisation for enteric infections. Notably, the risk of hospitalisation for perinatal infections was 34% higher in children born to mothers with overweight (aIRR, 1.34; 95% CI, 1.25–1.44) and 72% higher in children born to mothers with obesity (aIRR, 1.72; 95% CI, 1.57–1.88).

Table 6: Associations between pregnancy BMI and hospitalisations for overall and specific categories of infectious diseases

	Crude IRR	**Adjusted IRR***
	(95% CI)	(95% CI)
All infections:		
<18.5	1.09 (1.04–1.14)	1.04 (1.00–1.09)
18.5–24.9	1 ref	1 ref
25.0–29.9	1.07 (1.06–1.09)	1.05 (1.03–1.06)
≥30.0	1.25 (1.22–1.28)	1.18 (1.16–1.21)
Upper respiratory infections:		
<18.5	1.02 (0.93–1.12)	0.97 (0.88–1.06)
18.5–24.9	1 ref	1 ref
25.0–29.9	1.12 (1.09–1.16)	1.08 (1.05–1.11)
≥30.0	1.28 (1.23–1.34)	1.19 (1.14–1.24)
Lower respiratory infections:		
<18.5	1.03 (0.94–1.12)	0.99 (0.91–1.08)
18.5–24.9	1 ref	1 ref
25.0–29.9	1.08 (1.05–1.11)	1.03 (1.00–1.06)
≥30.0	1.30 (1.25–1.35)	1.19 (1.15–1.24)
Enteric infections:		
<18.5	1.28 (1.18–1.38)	1.20 (1.11–1.29)
18.5–24.9	1 ref	1 ref
25.0–29.9	1.00 (0.98–1.04)	1.00 (0.97–1.03)
≥30.0	1.16 (1.12–1.21)	1.12 (1.08–1.17)
Genitourinary infections:		
<18.5	1.14 (0.98–1.33)	1.13 (0.97–1.32)
18.5–24.9	1 ref	1 ref
25.0–29.9	1.12 (1.06–1.18)	1.12 (1.05–1.18)
≥30.0	1.28 (1.18–1.37)	1.27 (1.17–1.37)
Perinatal infections:		
<18.5	0.84 (0.66–1.06)	0.84 (0.67–1.06)
18.5–24.9	1 ref	1 ref
25.0–29.9	1.31 (1.22–1.40)	1.34 (1.25–1.44)
≥30.0	1.63 (1.49–1.78)	1.72 (1.57–1.88)
Skin and soft tissue infections:		

<18.5	0.90 (0.70–1.15)	0.90 (0.70–1.15)
18.5–24.9	1 ref	1 ref
25.0–29.9	0.97 (0.89–1.06)	0.94 (0.86–1.02)
≥30.0	1.09 (0.97–1.23)	1.02 (0.91–1.15)
Neurological and eye infections:		
<18.5	0.90 (0.62–1.31)	0.87 (0.59–1.27)
18.5–24.9	1 ref	1 ref
25.0–29.9	0.91 (0.79–1.04)	0.93 (0.81–1.06)
≥30.0	0.96 (0.80–1.15)	1.02 (0.85–1.23)
Bloodstream infections:		
<18.5	0.94 (0.50–1.79)	1.04 (0.55–1.97)
18.5–24.9	1 ref	1 ref
25.0–29.9	1.12 (0.90–1.39)	1.08 (0.87–1.34)
≥30.0	1.02 (0.74–1.39)	1.05 (0.76–1.44)
Digestive infections:		
<18.5	1.32 (0.93–1.89)	1.32 (0.92–1.88)
18.5–24.9	1 ref	1
25.0–29.9	0.98 (0.86–1.13)	0.97 (0.84–1.11)
≥30.0	1.04 (0.85–1.26)	0.99 (0.82–1.21)
Other infections:		
<18.5	1.08 (0.97–1.20)	1.03 (0.92–1.14)
18.5–24.9	1 ref	1 ref
25.0–29.9	1.09 (1.05–1.13)	1.05 (1.01–1.10)
≥30.0	1.25 (1.19–1.32)	1.17 (1.11–1.23)

Analyses included 693 007 children, with 125 010 overall hospitalisations and the following category-specific hospitalisations: 25 609 upper respiratory, 32 770 lower respiratory, 29 028 enteric, 8676 genitourinary, 4406 perinatal, 3552 skin and soft tissue, 1744 neurological and eye, 1039 bloodstream, 1492 digestive, and 16 694 other infectious disease hospitalisations.
*Adjusted for maternal age, maternal education level, pregnancy smoking, parity, geographic region, and year of birth.
BMI, body mass index; CI, confidence interval; IRR, incidence rate ratio.

Pregnancy smoking

The second study in this thesis also examined the associations between pregnancy smoking and hospitalisations for paediatric infectious diseases in the previously described population. A minority of children (10%) were born to mothers who smoked during early pregnancy.

Table 7 presents the associations between pregnancy smoking and hospitalisations for paediatric infectious diseases. Compared with children born to mothers who did not smoke during pregnancy, the risk of overall infectious disease hospitalisation was 9% higher (aIRR, 1.09; 95% CI, 1.06–1.11) in children born to mothers who smoked during pregnancy. The risk of lower respiratory infections was 29% higher (aIRR, 1.29; 95% CI, 1.24–1.34) in this group. By contrast, associations between pregnancy smoking and other infectious disease categories were weaker or absent. The adjusted analyses are presented in the appendix of the original paper; the crude analyses are additional analyses not presented in the original paper.

Table 7: Associations between pregnancy smoking and hospitalisations for overall and specific categories of infectious diseases

	Crude IRR	**Adjusted IRR***
	(95% CI)	(95% CI)
All infections:		
No	1 ref	1 ref
Yes	1.21 (1.18–1.23)	1.09 (1.06–1.11)
Upper respiratory infections:		
No	1 ref	1 ref
Yes	1.22 (1.17–1.27)	1.05 (1.01–1.10)
Lower respiratory infections:		
No	1 ref	1 ref
Yes	1.40 (1.35–1.45)	1.29 (1.24–1.34)
Enteric infections:		
No	1 ref	1 ref
Yes	1.07 (1.03–1.11)	0.95 (0.91–0.99)
Genitourinary infections:		
No	1 ref	1 ref
Yes	1.00 (0.92–1.08)	0.94 (0.86–1.02)

Perinatal infections:		
No	1 ref	1 ref
Yes	1.16 (1.05–1.28)	1.11 (1.00–1.22)
Skin and soft tissue infections:		
No	1 ref	1 ref
Yes	1.08 (0.96–1.22)	1.02 (0.91–1.16)
Neurological and eye infections:		
No	1 ref	1 ref
Yes	1.11 (0.93–1.33)	1.13 (0.94–1.36)
Bloodstream infections:		
No	1 ref	1 ref
Yes	0.75 (0.55–1.04)	0.75 (0.53–1.05)
Digestive infections:		
No	1 ref	1 ref
Yes	1.16 (0.96–1.40)	1.10 (0.91–1.34)
Other infections:		
No	1 ref	1 ref
Yes	1.25 (1.18–1.31)	1.10 (1.04–1.16)

Analyses included 693 007 children, with 125 010 overall hospitalisations and the following category-specific hospitalisations: 25 609 upper respiratory, 32 770 lower respiratory, 29 028 enteric, 8676 genitourinary, 4406 perinatal, 3552 skin and soft tissue, 1744 neurological and eye, 1039 bloodstream, 1492 digestive, and 16 694 other infectious disease hospitalisations.
*Adjusted for maternal age, maternal education level, pregnancy BMI, parity, geographic region, and year of birth.
BMI, body mass index; CI, confidence interval; IRR, incidence rate ratio.

Breastfeeding practice

The third study in this thesis examined associations between BF practice and hospitalisations for paediatric infectious diseases. The study included 37 825 term and post-term singletons born alive to women residing in Uppsala County between 1998 and 2010. All children were followed until 2 years of age (early childhood) or until censoring due to death or emigration (n=204). The remaining 37 621 children were then followed from 2 to 5 years of age (later childhood) or until censoring due to death or emigration. The study comprised 187 763 PY of follow-up time and recorded 4705 infectious disease hospitalisations. The majority of children were exclusively breastfed for at least 4 months (Table 8).

Table 8: Breastfeeding practices

Breastfeeding practice:	
No BF	2 (613)
EBF <4 mo with BF <6 mo	19 (7335)
EBF <4 mo with BF ≥6 mo	11 (3998)
EBF 4–5 mo with BF <6 mo	4 (1488)
EBF 4–5 mo with BF ≥6 mo	40 (15007)
EBF ≥6 mo	25 (9384)

Data are presented as % (n).
BF, breastfeeding; EBF, exclusive breastfeeding; mo, months.

The overall risk of hospitalisations for paediatric infectious diseases decreased with the duration of exclusive breastfeeding in both early childhood (<2 years) and later childhood (2–4 years) (Table 9). Compared with children exclusively breastfed for ≥6 months, the highest risk of overall infectious disease hospitalisations in early childhood was found in children who were not breastfed (aIRR, 1.89; 95% CI, 1.45–2.47). Similarly, the highest risk of overall infectious disease hospitalisations in later childhood was observed in children who were not breastfed (aIRR, 1.52; 95% CI, 1.00–2.33). In later childhood, children who were not breastfed were 2.5 times more likely to be hospitalised for respiratory infections (aIRR, 2.53; 95% CI, 1.51–4.24). In general, children who were exclusively breastfed for 4–6 months had a similar risk of infection to those who were exclusively breastfed for ≥6 months.

Table 9: Associations between breastfeeding practices and hospitalisations for paediatric infectious diseases

	Early childhood (<2 years)		**Later childhood (2–4 years)**	
	Crude IRR (95% CI)	Adjusted IRR* (95% CI)	Crude IRR (95% CI)	Adjusted IRR† (95% CI)
All infections:				
No BF	2.13 (1.61–2.81)	1.89 (1.45–2.47)	1.98 (1.27–3.09)	1.52 (1.00–2.33)
EBF <4 mo with BF <6 mo	1.46 (1.30–1.64)	1.41 (1.25–1.59)	1.60 (1.29–1.98)	1.43 (1.16–1.75)
EBF <4 mo with BF ≥6 mo	1.33 (1.16–1.52)	1.31 (1.14–1.50)	1.35 (1.07–1.71)	1.27 (1.00–1.61)
EBF 4–5 mo with BF <6 mo	1.19 (0.95–1.50)	1.16 (0.92–1.46)	0.98 (0.69–1.38)	0.97 (0.68–1.37)
EBF 4–5 mo with BF ≥6 mo	1.04 (0.94–1.15)	1.05 (0.94–1.16)	1.05 (0.88–1.24)	1.05 (0.88–1.24)
EBF ≥6 mo	1 ref	1 ref	1 ref	1 ref
Respiratory infections:				
No BF	1.93 (1.30–2.87)	1.70 (1.15–2.52)	3.03 (1.78–5.13)	2.53 (1.51–4.24)
EBF <4 mo with BF <6 mo	1.43 (1.20–1.69)	1.39 (1.17–1.65)	1.56 (1.15–2.11)	1.47 (1.08–2.01)
EBF <4 mo with BF ≥6 mo	1.28 (1.04–1.58)	1.29 (1.05–1.59)	1.35 (0.98–1.87)	1.28 (0.92–1.77)
EBF 4–5 mo with BF <6 mo	1.02 (0.72–1.44)	0.99 (0.70–1.40)	0.99 (0.59–1.67)	1.03 (0.61–1.73)
EBF 4–5 mo with BF ≥6 mo	1.08 (0.93–1.26)	1.09 (0.93–1.26)	1.07 (0.83–1.37)	1.07 (0.84–1.38)
EBF ≥6 mo	1 ref	1 ref	1 ref	1 ref
Enteric infections:				
No BF	3.56 (2.30–5.52)	3.32 (2.14–5.14)	1.34 (0.46–3.91)	1.20 (0.41–3.55)
EBF <4 mo with BF <6 mo	1.55 (1.24–1.94)	1.50 (1.19–1.89)	1.42 (0.99–2.04)	1.35 (0.92–1.97)
EBF <4 mo with BF ≥6 mo	1.44 (1.09–1.91)	1.37 (1.03–1.82)	1.50 (0.90–2.50)	1.45 (0.85–2.47)

EBF 4–5 mo with BF <6 mo	1.46 (0.99–2.15)	1.43 (0.97–2.10)	1.15 (0.62–2.13)	1.12 (0.60–2.09)
EBF 4–5 mo with BF ≥6 mo	1.03 (0.83–1.27)	1.03 (0.83–1.27)	1.15 (0.82–1.60)	1.14 (0.82–1.59)
EBF ≥6 mo	1 ref	1 ref	1 ref	1 ref

In early childhood (<2 years), analyses included 37 825 children with 3327 overall hospitalisations, 1485 respiratory infectious disease hospitalisations, and 762 enteric infectious disease hospitalisations. In later childhood (2–4 years), analyses included 37 621 children with 1378 overall hospitalisations, 630 respiratory infectious disease hospitalisations, and 300 enteric infectious disease hospitalisations.

*Adjusted for year of birth, small for gestational age, large for gestational age, congenital malformation, sex, maternal age, parity, pregnancy smoking, maternal education level, and maternal country of birth.

†Adjusted for year of birth, small for gestational age, large for gestational age, congenital malformation, sex, maternal age, parity, pregnancy smoking, maternal education level, maternal country of birth, and previous admissions.

BF, breastfeeding; CI, confidence interval; EBF, exclusive breastfeeding; IRR, incidence rate ratio; mo, months.

Socioeconomic circumstances, mediation by health-related behaviours

The fourth study examined how the effect of maternal education on the risk of paediatric infections was mediated by health-related behaviours in children under 5 years of age. The study included 63 216 term and post-term (gestational age of ≥37 weeks) singletons born alive to mothers who resided in the Uppsala Region between 1997 and 2015. A large proportion (26%) of children had missing data on one or more variables. Missing values were imputed using multiple imputation, and the study included all 63 216 children. The study comprised 283 119 PY of follow-up time and recorded 6977 infectious disease hospitalisations.

Compared with children born to mothers with ≥15 years of education, children born to mothers with shorter education were more likely to be exposed to pregnancy smoking, high pregnancy BMI (BMI of ≥25 kg/m^2), and low EBF (EBF for <4 months) (Table 10). The largest difference between children of mothers with ≥15 years of education and those with ≤9 years of education was observed for low EBF (23.0% vs. 50.6%), followed by pregnancy smoking (0.9% vs. 24.6%).

Table 10: Mediators and their association with maternal education level

	≤9 years	**10–12 years**	**13–14 years**	**≥15 years**	**All**
	9.7	39.5	12.9	37.8	100
Pregnancy smoking:					
Yes	24.6	9.3	2.7	0.9	6.8
No	75.4	90.7	97.3	99.1	93.2
High pregnancy BMI:					
Yes	46.5	43.0	36.0	28.9	37.1
No	53.5	57.0	64.0	71.1	62.9
Low EBF:					
Yes	50.6	38.5	27.6	23.0	32.4
No	49.4	61.5	72.4	77.0	67.6

All values are percentages calculated as averages over all imputations. Analyses included all 63 216 children.
BMI, body mass index; EBF, exclusive breastfeeding.

We examined how the effect of maternal education on the risk of paediatric infections was mediated by different exposures (PNIEs) and different susceptibilities (MIEs) to health-related behaviours. Maternal education of ≥15 years was used as the reference group.

Pregnancy smoking mediated approximately 10% of the additional risk of overall infections and respiratory infections observed in children born to mothers with ≤13 years of education (Table 11). By contrast, pregnancy smoking did not mediate the additional risk of enteric infections observed in these children. These are additional analyses not presented in the original paper.

Pregnancy smoking and high pregnancy BMI mediated approximately 20% of the additional risk of overall infections and respiratory infections observed in children born to mothers with ≤13 years of education (Table 12). These are additional analyses not presented in the original paper.

Pregnancy smoking, high pregnancy BMI, and low EBF mediated approximately 60% of the additional risk of overall infections and respiratory infections observed in children born to mothers with ≤13 years of education (Table 13). Additionally, pregnancy smoking, high pregnancy BMI, and low EBF mediated 33% of the additional risk of overall infections observed in children born to mothers with 10–12 years of education and 40% of the additional risk of enteric infections in children born to mothers with ≤9 years of education. These analyses are presented in the original paper.

In summary, pregnancy smoking, high pregnancy BMI, and low EBF mediated approximately half of the association between maternal education level and paediatric infectious diseases. These mediators are likely dependent on each other; the mediation effect of each mediator can, therefore, not be determined precisely.[114] However, the proportion mediated by health-related behaviours increased substantially when low EBF was included. This suggests that low EBF was the main mediator. Additionally, all mediation effects were due to higher exposure to health-related behaviours; we found no evidence of increased susceptibility to health-related behaviours in children born to less educated mothers.

Table 11: Total and decomposed effects on all respiratory and enteric paediatric infections due to counterfactual changes in maternal education level; mediation due to pregnancy smoking

	All infections	Respiratory infections	Enteric infections
Maternal education level: 13–14 vs ≥15 years			
PNDE*	1.06 (0.96–1.17)	1.01 (0.88–1.16)	0.94 (0.77–1.14)
PNIE*	1.00 (0.99–1.01)	1.00 (0.99–1.01)	1.02 (0.99–1.04)
MIE†	−0.01 (−0.10–0.09)	−0.01 (−0.14–0.13)	−0.01 (−0.20–0.18)
TE*	1.06 (0.96–1.15)	1.01 (0.88–1.15)	0.93 (0.74–1.12)
TPM‡	−17	-	-
Maternal education level: 10–12 vs ≥15 years			
PNDE*	1.09 (1.02–1.18)	1.09 (0.98–1.20)	1.01 (0.87–1.17)
PNIE*	1.02 (0.97–1.06)	1.01 (0.96–1.07)	1.09 (0.97–1.22)
MIE†	0.00 (−0.07–0.08)	0.00 (−0.10–0.10)	−0.06 (−0.20–0.08)
TE*	1.11 (1.04–1.18)	1.10 (1.01–1.20)	1.04 (0.90–1.18)
TPM‡	18	10	-
Maternal education level: ≤9 vs ≥15 years			
PNDE*	1.33 (1.18–1.51)	1.34 (1.13–1.58)	1.32 (1.06–1.63)
PNIE*	1.04 (0.93–1.17)	1.04 (0.89–1.22)	1.25 (0.95–1.63)
MIE†	0.00 (−0.12–0.12)	−0.01 (−0.16–0.13)	−0.26 (−0.46– −0.06)
TE*	1.38 (1.26–1.50)	1.37 (1.22–1.51)	1.30 (1.10–1.51)
TPM‡	11	8	−3

Analyses included all 63 216 children. All models were adjusted for maternal age, maternal country of birth, and year of birth.
*PNDE, PNIE, and TE are presented as IRR with 95% CI.
†MIE estimated as excess risk due to interaction = $IRR^{TE} - IRR^{PNDE} - IRR^{PINE} + 1$.
‡TPM estimated as $[(IRR^{PNIE} - 1)/(IRR^{TE} - 1) + RERI^{MIE}/(IRR^{TE} - 1)] \times 100$ in models with a TE of IRR > 1.05.
CI, confidence interval; IRR, incidence rate ratio; MIE, mediated interactive effect; PNDE, pure natural direct effect; PNIE, pure natural indirect effect; TE, total causal effect; TPM, total percentage mediated.

Table 12: Total and decomposed effects on all respiratory and enteric paediatric infections due to counterfactual changes in maternal education level; mediation due to pregnancy smoking and high pregnancy BMI

	All infections	Respiratory infections	Enteric infections
Maternal education level: 13–14 vs ≥15 years			
PNDE*	1.05 (0.95–1.16)	1.01 (0.88–1.16)	0.95 (0.78–1.13)
PNIE*	1.01 (1.00–1.03)	1.02 (1.00–1.04)	1.03 (0.99–1.08)
MIE†	−0.01 (−0.11–0.09)	−0.01 (−0.15–0.12)	−0.03 (−0.23–0.17)
TE*	1.06 (0.96–1.15)	1.01 (0.88–1.15)	0.93 (0.74–1.13)
TPM‡	0	-	-
Maternal education level: 10–12 vs ≥15 years			
PNDE*	1.09 (1.01–1.17)	1.09 (0.98–1.20)	1.03 (0.89–1.13)
PNIE*	1.04 (0.99–1.09)	1.05 (0.98–1.12)	1.11 (0.98–1.31)
MIE†	−0.02 (−0.09–0.06)	−0.03 (−0.12–0.07)	−0.10 (−0.24–0.04)
TE*	1.11 (1.04–1.18)	1.10 (1.00–1.20)	1.04 (0.90–1.18)
TPM‡	18	20	-
Maternal education level: ≤9 vs ≥15 years			
PNDE*	1.29 (1.14–1.47)	1.31 (1.10–1.57)	1.29 (1.03–1.49)
PNIE*	1.08 (0.96–1.21)	1.09 (0.92–1.28)	1.27 (0.97–1.71)
MIE†	0.01 (−0.11–0.13)	−0.02 (−0.17–0.12)	−0.25 (−0.46– −0.05)
TE*	1.38 (1.26–1.50)	1.37 (1.23–1.52)	1.30 (1.11–1.51)
TPM‡	24	19	7

Analyses included all 63 216 children. All models were adjusted for maternal age, maternal country of birth, and year of birth.
*PNDE, PNIE, and TE are presented as IRR with 95% CI.
†MIE estimated as excess risk due to interaction = $IRR^{TE} - IRR^{PNDE} - IRR^{PINE} + 1$.
‡TPM estimated as $[(IRR^{PNIE} - 1)/(IRR^{TE} - 1) + RERI^{MIE}/(IRR^{TE} - 1)] \times 100$ in models with a TE of IRR > 1.05.
CI, confidence interval; IRR, incidence rate ratio; MIE, mediated interactive effect; PNDE, pure natural direct effect; PNIE, pure natural indirect effect; TE, total causal effect; TPM, total percentage mediated.

Table 13: Total and decomposed effects on all respiratory and enteric paediatric infections due to counterfactual changes in maternal education level; mediation due to pregnancy smoking, high pregnancy BMI, and low EBF

	All infections	Respiratory infections	Enteric infections
Maternal education level: 13–14 vs ≥15 years			
PNDE*	1.04 (0.94–1.14)	1.00 (0.87–1.15)	0.93 (0.76–1.13)
PNIE*	1.03 (1.02–1.05)	1.03 (1.01–1.05)	1.05 (1.02–1.08)
MIE†	−0.01 (−0.11–0.09)	−0.01 (−0.15–0.12)	−0.03 (−0.23–0.16)
TE*	1.06 (0.96–0.15)	1.01(0.88–1.15)	0.93 (0.73–1.13)
TPM‡	33	-	-
Maternal education level: 10–12 vs ≥15 years			
PNDE*	1.04 (0.97–1.13)	1.04 (0.94–1.15)	0.97 (0.84–1.13)
PNIE*	1.08 (1.03–1.13)	1.07 (0.99–1.15)	1.17 (1.05–1.31)
MIE†	−0.01 (−0.08–0.06)	−0.01 (−0.11–0.09)	−0.10 (−0.25–0.04)
TE*	1.11 (1.04–1.19)	1.10 (1.01–1.20)	1.04 (0.90–1.18)
TPM‡	64	60	-
Maternal education level: ≤9 vs ≥15 years			
PNDE*	1.17 (1.03–1.32)	1.17 (0.98–1.4)	1.18 (0.93–1.49)
PNIE*	1.11 (0.99–1.25)	1.12 (0.93–1.34)	1.32 (1.02–1.71)
MIE†	0.10(−0.02–0.22)	0.09 (−0.06–0.23)	−0.20 (−0.40–0.01)
TE*	1.38 (1.26–1.50)	1.37 (1.23–1.52)	1.30 (1.10–1.51)
TPM‡	55	57	40

Analyses included all 63 216 children. All models were adjusted for maternal age, maternal country of birth, and year of birth.
*PNDE, PNIE, and TE are presented as IRR with 95% CI.
†MIE estimated as excess risk due to interaction = $IRR^{TE} - IRR^{PNDE} - IRR^{PINE} + 1$.
‡TPM estimated as $[(IRR^{PNIE} - 1)/(IRR^{TE} - 1) + RERI^{MIE}/(IRR^{TE} - 1)] \times 100$ in models with a TE of IRR > 1.05.
CI, confidence interval; EBF, exclusive breastfeeding; IRR, incidence rate ratio; MIE, mediated interactive effect; PNDE, pure natural direct effect; PNIE, pure natural indirect effect; TE, total causal effect; TPM, total percentage mediated.

Discussion

This thesis has three main findings as related to the three aims. First, we found that inequalities in paediatric infectious diseases were associated with maternal education, household income, sex/gender, maternal migration status, and their combinations, with maternal education being the primary driver of socioeconomic inequalities. Second, we found that high pregnancy BMI, pregnancy smoking, and low EBF increased the risk of paediatric infectious diseases. Third, we found that these health-related behaviours mediated approximately half of the association between maternal education level and paediatric infectious diseases, with low EBF being the main mediator.

Inequalities

In our first study, we found complex inequalities in paediatric infectious diseases related to maternal education, household income, sex/gender, maternal migration status and their combinations.

Specifically, our study has three main findings. First, children from lower socioeconomic groups were more likely to be hospitalised for overall, upper respiratory, lower respiratory, and enteric infections. For all outcomes, maternal education was the primary driver of socioeconomic inequalities. These findings align with previous studies examining socioeconomic inequalities in paediatric infections among Swedish children.[11-15 53] Second, the overall risk of paediatric infectious diseases was unrelated to household income in children born to highly educated mothers. This suggests that maternal education can protect children from the detrimental effects of poverty. Third, inequalities related to education and income were overall similar in children of immigrant and non-immigrant mothers, suggesting that immigration does not significantly impact the overall risk of paediatric infectious diseases in Swedish children. One explanation for this may be the high and equitable coverage of the Swedish national vaccination programme.[83]

Our intersectional analysis revealed substantial inequalities in the average risk of paediatric infectious diseases. For example, the incidence of overall infectious disease hospitalisations ranged from around 3000 per 100 000 PY in girls of highly educated mothers to around 6000 per 100 000 PY in boys of poor and low-educated mothers. Despite this, the ability of the sociodemographic variables to discriminate between children with and without infections was poor. This indicates that a small proportion of the individual risk of infection was attributable to the social context.[110] Consequently, our findings support broad public health interventions that reach all children, rather than targeted interventions focusing on high-risk groups. However, the scale and intensity of these interventions may vary depending on the needs of different social groups.[86]

Health-related behaviours

High pregnancy BMI

In our second study, we found a moderate association between pregnancy obesity and the overall number of hospitalisations for infectious diseases in the first 5 years of life. Additionally, we found that pregnancy overweight and obesity increased the risk of certain infectious disease categories, including respiratory and perinatal infections.

To our knowledge, only three other studies have examined the association between high pregnancy BMI and overall paediatric infectious disease hospitalisations.[30 31 35] Our results are generally consistent with these studies. For example, an Israeli study found that children of mothers with pregnancy obesity were 13% more likely to be hospitalised for infectious diseases in the first 18 years of life.[31]

In our study, high pregnancy BMI was associated with an increased risk of respiratory infections. Several other studies support this finding.[36-38] For example, a recent Danish population-based cohort study found an increased risk of lower respiratory infections in children born to mothers with maternal overweight (adjusted hazard ratio [HR], 1.07; 95% CI, 1.05–1.09), obesity (adjusted HR, 1.16; 95% CI, 1.14–1.19), and severe obesity (adjusted HR, 1.21; 95% CI, 1.13–1.28).[131] One explanation is that pregnancy obesity alters the offspring's immune and respiratory development, including the frequency and function of immune cells.[132 133] Another explanation is that women with obesity may be less able to initiate and maintain BF.[134]

We found that pregnancy overweight and obesity increased the risk of perinatal infections. This finding is consistent with two previous Swedish studies reporting an increased risk of neonatal sepsis in children of mothers with high pregnancy BMI.[39 40] An increased risk of perinatal infections was expected, as pregnancy overweight and obesity increase the risk of many severe conditions that may, in turn, elevate the risk of neonatal infections.[32 34]

Pregnancy smoking

In our second study, we also found that pregnancy smoking was associated with an increased risk of lower respiratory infections.

Our finding is consistent with numerous previous studies reporting that smoking increases the risk of lower respiratory infections in children.[11 49]

[135] For example, a systematic review and meta-analysis found that smoking by either parent or other household members increases the risk of lower respiratory infections by approximately 50% (OR, 1.54; 95% CI, 1.40–1.69).[49] Moreover, a previous Swedish study reported that pregnancy smoking increased the risk of lower respiratory infections in the first 3 years of life.[11] An increased risk of respiratory infections in children exposed to tobacco smoke is expected. In the respiratory tract, tobacco smoke causes inflammation, fibrosis, higher mucosal permeability, and impaired mucociliary clearance. Additionally, tobacco smoke causes immunologic changes, such as decreased white blood cell function and lower immunoglobulin levels.[136]

Breastfeeding practices

Our third study found that the overall risk of infectious diseases in the first 5 years of life decreased with the duration of EBF until 4 months. Additionally, BF reduced the risk of both respiratory and enteric infections in children under 2 years of age, as well as the risk of respiratory infections in children aged 2–4 years. Our findings are consistent with previous research: overwhelming evidence shows that BF protects young children from respiratory and enteric infections,[57-59] and growing evidence suggests that BF also protects older children from respiratory infections.[61 62]

Duration of exclusive breastfeeding

The optimal duration of EBF is under debate. The World Health Organization recommends EBF until 6 months of age, followed by continued BF alongside complementary feeding in all settings.[137] The current Swedish guideline recommends exclusively breastfeeding until 6 months of age, with 'tiny tastes' (≤1 mL) permitted from 4 months.[138 139] Before 2002, the Swedish recommendation was EBF until 4–6 months of age.[140] In contrast, the European Society for Paediatric Gastroenterology, Hepatology, and Nutrition guideline recommends EBF for at least 4 months, with either EBF or predominant BF continuing for approximately 6 months.[141]

In our study, children who were exclusively breastfed for 4–6 months had a similar risk of infection as those who were exclusively breastfed for ≥6 months. Our findings are consistent with a Dutch study reporting that EBF until 4 months, followed by partial BF, decreased the risk of upper respiratory, lower respiratory, and gastrointestinal infections.[142] Similarly, a UK study found that children who were exclusively breastfed

for 4–6 months followed by partial breastfeeding had a comparable risk of chest and diarrhoeal infections to those exclusively breastfed for ≥6 months.[143] In contrast, a US study showed that EBF for 4 months—compared with 6 months—was associated with an increased risk of respiratory tract infection.[144] Additionally, observational analysis from the Promotion of Breastfeeding Intervention Trial found that EBF for 3 months followed by partial BF, compared with EBF for 6 months, was associated with an increased risk of gastrointestinal episodes; however, no significant association was found for gastrointestinal hospitalisations.[57] Consequently, there is support for guidelines recommending EBF for either 4–6 months or for a full 6 months.

Breastfeeding and respiratory infections after infancy

We found that BF reduced the risk of respiratory infections in children aged 2–4 years. Our findings are consistent with a Japanese study reporting that EBF at 6–7 months of age decreased the risk of respiratory infection hospitalisations (adjusted OR, 0.76; 95% CI, 0.58–0.99) between 30 and 42 months of age, with no association found between BF and diarrhoea hospitalisations.[61] Similarly, a Dutch study found that BF for at least 6 months decreased the risk of lower respiratory tract infections (adjusted OR, 0.71; 95% CI, 0.51–0.98) up to 4 years of age.[62] By contrast, analyses of BF data from the Environmental Determinants of Diabetes in the Young study found that BF increased the risk of respiratory infections (OR, 1.17; 95% CI, 1.09–1.26) in children aged 6 to 18 months.[145] Altogether, BF appears to protect children from respiratory infections beyond infancy. One explanation is that BF may have persistent effects on the immune system. For example, breastfed children have larger thymus size and an increased number of $CD8^+$ T cells after infancy.[146] [147]

Pathways of inequalities

Our fourth study showed that higher exposure to health-related behaviours—namely, high pregnancy BMI, pregnancy smoking, and low EBF—mediated approximately half of the association between maternal education level and paediatric infectious diseases. The main mediator was low EBF. Nevertheless, pregnancy smoking and high pregnancy BMI mediated approximately 20% of the association between maternal education and paediatric infectious disease in children born to mothers with less than 13 years of education.

To our knowledge, only two previous studies have examined whether health-related behaviours mediate the effect of SECs on the risk of paediatric infections. A Swedish study revealed that maternal smoking and family size partly mediated the effect of low maternal education on the risk of respiratory infections; adding these variables to a regression model reduced the association between maternal education and lower respiratory tract infection from an OR of 1.4 to an OR of 1.2.[11] Similarly, an Australian study reported that pregnancy smoking and family size partly mediated the effect of maternal education on a combined measure of child health that included infectious diseases.[148]

Health-related behaviours may link SECs to paediatric infectious disease in several ways. First, health literacy is strongly associated with both child health and education level.[27-29] For example, health literacy is linked to the ability to maintain EBF.[149] Second, lower socioeconomic groups are more likely to experience psychosocial stress, which increases the risk of unhealthy behaviours, including obesity, smoking, and early cessation of BF.[150-152] Additionally, financial hardship increases the risk of paternal smoking.[153] Third, deprived groups are more vulnerable to structural determinants of health. For example, lower socioeconomic groups are specifically targeted by commercial companies marketing unhealthy products, including tobacco and ultra-processed foods.[154]

Strengths and limitations

The major types of systematic error that can occur in cohort studies, such as those presented in this thesis, are selection bias, information bias, and confounding.

Selection bias

Selection bias arises from the procedures used to select study subjects or from factors influencing study participation.[97] All our studies were population-based, which reduces the risk of selection bias because the 'sample' ideally includes the entire 'source population'. However, selection bias may still occur, for example, due to missing data or loss to follow-up.

In Study I, we examined the association between maternal country of birth and paediatric infectious diseases. It is known that immigrants often remain in the population registers even when they are no longer residing in Sweden.[155] Consequently, our study may underestimate the incidence of paediatric infectious diseases in immigrant children.

In Studies II and IV, a large number of children had missing data on pregnancy BMI. Missing data can lead to biased estimates if there are systematic differences between missing and observed values. We addressed this issue by imputing missing data using multiple imputation. However, this approach only yields unbiased estimates if the data are missing 'completely at random' (no systematic differences) or 'missing at random' (systematic differences that can be explained by the observed data). Estimates will still be biased if the data are 'missing not at random' (systematic differences that cannot be explained by the observed data).[116] Our imputation models included variables that may reasonably predict BMI, such as maternal education level and age. Therefore, we consider it reasonable to assume that our data were 'missing at random'.

Information bias

Information biases arise when study variables—i.e., exposures, outcomes, mediators, or confounders—are inaccurately measured or classified. Misclassification biases can be either differential or non-differential. Differential misclassification occurs when the probability of misclassifying the exposure or outcome is related to the other. This type of bias can distort estimates either toward or away from the null. Non-

differential misclassification occurs when the probability of misclassification is unrelated to other variables. In most cases, this type of bias will shift estimates toward the null.[97]

In Studies III and IV, child health nurses collected information on BF practices at 1 week and at 2, 4, and 6 months during well-baby clinic visits. Compared with daily recordings, BF data reported at specific time points tend to overestimate the proportion of exclusively breastfed infants.[156] We expect the misclassification of BF practice to be independent of infectious disease hospitalisations and, therefore, non-differential.

In Studies II, III, and IV, information on pregnancy smoking and height was self-reported at the first antenatal visit. Self-reported data on health-related behaviours are prone to misclassification. However, self-reported information on pregnancy smoking in the Swedish Medical Birth Register is strongly correlated with cotinine levels (a metabolite of nicotine) in umbilical cord serum, indicating that the data on pregnancy smoking are valid.[157] Additionally, self-reported heights from women of reproductive age are known to be accurate and suitable for use in epidemiological research.[158]

In all studies, infection episodes were identified using register data. As a result, some misclassification is expected because cases could not be confirmed through clinical, laboratory, or radiological findings. For example, a Swedish study examining paediatric infectious disease hospitalisations excluded 11% of sinusitis, 23% of mastoiditis, and 33% of bacterial meningitis cases after validation using medical records.[159] However, the overall incidence of paediatric infectious diseases would only be affected if infectious diseases were misclassified as non-infectious. Additionally, we expect the misclassification of infectious disease hospitalisations to be independent of the exposures and, therefore, non-differential.

Confounding

Confounding refers to the distortion of the association between an exposure and an outcome by a third variable. To qualify as a confounder, a variable must be associated with the exposure in the source population, be a risk factor for the outcome, and not be affected by either the exposure or the outcome.[97]

In Studies II, III, and IV, there is a risk of residual confounding due to factors such as daycare attendance, vaccinations, and genetic

predispositions. We do not expect these factors to be significant confounders. Nevertheless, some of them could have been accounted for by using, for example, sibling comparison designs.[160] Additionally, high pregnancy BMI and pregnancy smoking are likely associated with other related behaviours. For instance, high pregnancy BMI is very likely linked to an unhealthy diet; therefore, the aim should be to promote a healthy diet, not merely weight loss. Similarly, pregnancy smoking is likely associated with smoking after pregnancy, as well as paternal smoking. Consequently, interventions should aim to prevent all forms of environmental tobacco smoke exposure.

Conclusion

In this thesis, we found complex inequalities in paediatric infectious diseases related to maternal education, household income, sex/gender, maternal migration status, and their combinations, with maternal education as the main driver. Additionally, we found that high pregnancy BMI was associated with an increased risk of paediatric infectious diseases, that pregnancy smoking was associated with an increased risk of lower respiratory infections, and that EBF until 4 months was associated with a decreased risk of paediatric infectious diseases. Finally, we showed that high pregnancy BMI, pregnancy smoking, and low EBF mediated approximately half of the association between maternal education level and paediatric infectious diseases, with low EBF identified as the main mediator. Consequently, interventions targeting these health-related behaviours may reduce inequalities in paediatric infectious diseases.

Implications for policy and future research

Implications for policy

In this thesis, we identified pregnancy BMI, pregnancy smoking and low EBF as potential targets for policies addressing inequalities in paediatric infectious diseases.

Taxation of foods high in fat, salt, and sugar may reduce inequalities in overweight and obesity.[43] Sweden has no taxes on unhealthy foods. Consequently, implementing these types of taxes might reduce inequalities in overweight and obesity. Additionally, it is important to ensure equitable access to obesity care.

Targeted smoking cessation support can reduce inequalities in smoking.[161] According to Swedish guidelines, all pregnant women who smoke should be offered smoking cessation treatment.[80 81] However, there is a strong socioeconomic gradient in smoking cessation. For example, in women who smoke three months before pregnancy, continuous smoking during pregnancy varies from 47 per cent in women with less than 10 years of education to 13 per cent in women with more than 12 years of education.[90] This indicates that pregnant women from lower socioeconomic groups need extended smoking cessation support.

There is some evidence suggesting that BF support tailored to the needs of women from vulnerable groups can reduce inequalities in BF.[162 163] In contrast, population-based BF promotion interventions do not seem to reduce inequalities.[164 165] Consequently, women from lower socioeconomic groups may need extended breastfeeding support.

Implications for future research

Further research may examine how interventions can reduce inequalities in health-related behaviours, particularly if extended smoking cessation support can reduce inequalities in smoking cessation and if extended BF support can reduce inequalities in breastfeeding. It would also be interesting to (i) examine whether the effect of socioeconomic circumstances on the risk of paediatric infectious diseases is mediated by environmental factors, for example, ambient air pollution, (ii) evaluate how new vaccines and other preventive strategies, such as varicella vaccination and RSV-prophylaxis, affect inequalities in paediatric infectious diseases, and (iii) explore inequalities in obesity care before and during pregnancy.

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Appendix

Table A1: Swedish studies examining socioeconomic circumstances and paediatric infectious diseases

Study	Study design	Population	Outcome	Findings
Silfverdal et al.[63]	Case-control study	Örebro County 1987–1992 n = cases/controls 54/139	Invasive *Haemophilus influenza* <6 years of age	Socioeconomic level: High/middle = OR 1.0 Low = OR 1.28 (0.6–2.9)
Hjern et al.[166]	Cross-sectional study	Sweden 1996–1997 n = 2142	Use of antibiotics Physician visits for infection 1–5 years	Parental education 0–9 years and/or 'worker': Visited a physician for infectious diseases: No= OR 1.0 Yes = OR 0.6 (0.4–0.8) Use of antibiotics: No = OR 1.0 Yes = OR 0.8 (0.6–0.97) Parental country of birth: Visited a physician for infectious diseases: Sweden = OR 1.0 Outside Sweden = 1.2 (0.9–1.7) Use of antibiotics: Sweden = OR 1.0 Outside Sweden = 0.9 (0.7–1.2)

Hjern et al.[11]	Cohort study	Stockholm 1990–94 PY = 820 871	Lower respiratory infections 0–4 years	0–1 years: Maternal education: 0–9 years = OR 1.4 (1.3–1.6) 10–12 years = OR 1.0 University = OR 0.8 (0.7–0.9) Social welfare: Yes = OR 1.4 (1.2–1.5) No = OR 1.0 2–4 years: Maternal education: 0–9 years = OR 1.2 (1.1–1.4) 10–12 years = OR 1.0 University = OR 1. (0.9–1.1) Social welfare: Yes = OR 1.1 (0.97–1.3) No = OR 1.0 Mediation analysis by smoking and family size 0–1 years: Maternal education: OR decreased from 1.4 to 1.2 Social welfare: OR decreased from 1.4 to 1.3 Mediation analysis by smoking and family size 2–4 years: No effect

Jansson et al.[167]	Cohort study	Malmö 1998–1999 n = 8265	Acute bronchiolitis hospitalisations <1 year	The admission rate differed between residential areas by more than fourfold and correlated with the percentage of immigrants (r = 0.640, p < 0.05)
Bremberg[168]	Ecological study	Stockholm 1987–1998 n = around 250 000	Infections disease hospitalisations 0–18 years	The relative inequality did not differ significantly between the recession years (1991–1996) and adjacent time periods
Nilsson and Laurell[169]	Ecological study	Malmö 1998–1999 n =17490	PNSP 0–5 years	No correlation between socioeconomic factors and PNSP cases
Lind J et alt.[13]	Cohort study	Sweden 2006–2012 n = 752 078	Gastroenteritis hospitalisations <5 years	Maternal education in years: ≤9 = HR 1.42 (1.37–1.48) 10–12 = HR 1.14 (1.11–1.18) 13–14 = HR 1.07 (1.02–1.11) ≥15 = HR 1.0 Household income: Q1 = HR 1.29 (1.24–1.34) Q2 = HR 1.11 (1.07–1.16) Q3 = HR 1.08 (1.04–1.13) Q4 = HR 1.06 (1.02–1.11) Q5 = HR 1.0 Parental country of birth: Non-European = HR 1.32 (1.27–1.37) European = HR 1.13 (1.06–1.21) One Swedish-born = HR 0.97 (0.93–

				1.01) Two Swedish-born = HR 1.0
Mangrio et al.[17]	Cross-sectional study	Malmö 2003–2006 n = 7266	Antibiotic use 0–7 months	Parental country of birth: Both Sweden = OR 1.0 One Sweden = OR 1.09 (0.91–1.32) Outside Sweden = OR 1.73 (1.44–2.07) Maternal educational level: >12 years = OR 1.0 10–12 years = OR 1.15 (0.99–1.34) ≤9 years = OR 1.25 (0.98–1.60)
Nejat et al.[18]	Cohort study	Stockholm 2000–2009 PY = 4 153 442	Tuberculosis 0–17 years	90 of 147 cases (61%) were foreign-born. Highest incidence in Somalia-born children: 451 (334–595) cases per 100 000 PY
Rhedin et al.[53]	Case-control study	Stockholm 2011–2014 n = cases/controls 93/240	Pneumonia ≤5 years	University degree in ≥1 parent = cases 58% vs. controls 69%, p = 0.03

Schollin Ask et al.[95]	Cohort study	Stockholm 2011–2015 n = 537 479	Viral gastroenteritis 2 months– <2 years	Inpatient care: Maternal education: Primary = HR 0.99 (0.74–1.31) Secondary = HR 1.15 (0.84–1.57) Tertiary = HR 1.0 Paternal education: Primary = HR 0.99 (0.77–1.25) Secondary = HR 0.81 (0.59–1.12) Tertiary = HR 1.0 Social welfare: Yes = HR 0.90 (0.58–1.39) No = HR 1.0 Foreign-born parents: Yes = HR 0.96 (0.77–1.20) No = HR 1.0 Outpatient care Maternal education: Primary = HR 1.25 (1.12–1.39) Secondary = HR 1.15 (1.02–1.31) Tertiary = HR 1.0 Paternal education: Primary = HR 1.18 (1.07–1.29) Secondary = HR 1.14 (1.01–1.28) Tertiary = HR 1.0 Social welfare: Yes = HR 1.39 (1.17–1.64) No = HR 1.0 Foreign-born parents: Yes = HR 1.15 (1.06–1.25) No = HR 1.0

Zylbersztejn et al.[15]	Cohort study	Sweden 2003–2012 n = 1 012 682	Respiratory infection mortality rates per 100,000 child-years 31 days–4 years	Household income: 31–364 days: Q1 = 28 (21–36) Q2 = 15 (10–21) Q3 = 7 (4.1–12) Q4 = 11 (6.9–17) Q5 = 11 (6.9–17) 1–4 years: Q1 = 4.6 (3.2–6.6) Q2 = 3.6 (2.4–5.4) Q3 = 4.2 (2.9–6.1) Q4 = 3.1 (2.0–4.8) Q5 = 2.9 (1.9–4.6)
Hertting et al.[19]	Cohort study	Stockholm 2015–2016 342 138 PY	Infectious disease hospitalisations < 18 years	Asylum seekers vs. residents: Gastroenteritis = RR 7.0 (3.8–13) Varicella = RR 20 (5.9–66) Viral infection = RR 2.9 (0.9–9.0) Upper respiratory infections = RR 6.4 (2.8–14) Influenza = RR 8.6 (3.2–24) Pneumonia = RR 3.6 (1.8–7.2) Empyema = RR 145 (20–1028) Skin abscess = RR 5.0 (1.6–16) Urinary tract infections = RR 1.1 (0.1–7.5)

Schollin Ask[16]	Cohort study	Sweden 2006–2013 n = 751 646	Gastroenteritis 0–4 years	Maternal education: 0–9 years = HR 1.42 (1.37–1.48) 10–12 years = HR 1.14 (1.11–1.18) 13–14 years = HR 1.07 (1.02–1.1) ≥15 = Ref Household income: Q1 = HR 1.29 (0.24–1.34) Q2 = HR 1.11 (1.07–1.16) Q3 = HR 1.08 (1.04–1.13) Q4 = HR 1.06 (1.02–1.11) Q5 = HR 1.0 Parental country of birth: Non- European = HR 1.32 (1.27–1.37) European = HR 1.13 (1.06–1.21) One Swedish born = HR 0.97 (0.93–1.01) Two Swedish born = HR 1.0
Videholm et al.[14]	Cohort study	Sweden 1997–2013 n = 1 406 547	Sepsis and bacterial meningitis 28 days and <2 years	Maternal education in years: Sepsis: ≤9 = IRR 1.27 (1.01–1.59) 10–12 = IRR 0.95 (0.80–1.13) 13–14 = IRR 0.93 (0.73–1.18) ≥15 = IRR 1.0 Bacterial meningitis: ≤9 = IRR 1.70 (1.23–2.36) 10–12 = IRR 1.23 (0.96–1.59) 13–14 = IRR 1.46 (1.06–2.02) ≥15 = IRR 1.0

Strandell et al.[12]	Cohort study	Sweden 1998–2015 n = 1 641 747	Bacterial pneumonia 0–1 years	Maternal education in years: ≤9 = IRR 1.39 (1.26–1.53) 10–12 = IRR 1.12 (1.04–1.20) 13–14 = IRR 1.04 (0.95–1.15) ≥15 = IRR 1.0

Studies were included based on a PubMed search restricted to children (0–18 years) using the following terms: infect* AND ('ethnic*' OR 'socio*' OR 'deprivation' OR 'poverty' OR 'income' OR 'education' OR 'migration') AND Sweden. The search yielded 1523 articles; 39 articles were reviewed in full text, 3 articles were added manually, and 16 articles were included. IRR, incidence rate ratio; HR, hazard ratio; OR, odds ratio; PNSP, penicillin-non-susceptible *Streptococcus pneumoniae*; PY, person-year; RR, relative risk; Q, quintile.

Table A2: Infectious disease categories, groups and ICD-10 codes

Categories:	**Groups:**	**ICD-10 codes:**
Upper respiratory infections	Upper respiratory infections	J00, J01, J02, J03, J04, J05, J06, J32, J340, J36, J37, J390, J391
	Ear infections	H600, H601, H602, H603, H608, H609, H62, H65, H66, H67, H680, H70, H730, H750, H830, H940
Lower respiratory infections	Acute lower respiratory infections	A481, A482, B59, J09, J10, J11, J12, J13, J14, J15, J16, J17, J18, J20, J21, J22
	Chronic lower respiratory infections	J40, J41, J42, J440, J47, J85, J86, J988
	Tuberculosis	A15, A16, A17, A18, A19, N740, N741, J65
Enteric infections	Enteric infections	A00, A01, A020, A029, A03, A04, A05, A06, A07,A08
	Enteric symptoms	A09, I880, K528, K529, R11
Genitourinary infections	Kidney infections	N00, N05, N10, N136, N151
	Urinary tract infections	N300, N341, N351, N37, N390

	Reproductive system infections, male	N410, N411, N412, N413, N431, N45, N481, N482, N490, N49, N51
	Reproductive system infections, female	N70, N71, N72, N73, N74, N751, N764, N87
Infections of perinatal period	Perinatal infections	P002, P027, P23, P35, P36, P37, P38, P39
Skin and soft tissue infections	Skin infections, typical	A46, L00, L01, L02, L03, L04, L050, L08
	Skin infections, other	B86, T009, T633, T634, T793
	Connective tissue infections	M021, M023, M03, M600, M630, M631, M632, M650, M651, M680, M710, M711, M896
Neurological and eye infections	CNS viral infections	A801, A802, A803, A804, A809, A811, A812, A818, A819, A82, A83, A84, A85, A86, A87, A88, A89
	CNS general infections	G00, G01, G02, G030, G039, G04, G05, G06, G07, G08, G09, G610, A321, A390
	Eye infections	B30, H000, H03, H043, H050, H100, H102, H103, H109, H130, H131, H160, H190, H191, H192, H220, H440, H451
Bloodstream infections	Sepsis	A391, A392, A393, A394, A395, A398, A399 A40, A41, A483, A021, A327

Digestive infections	Oral infections	K02, K044, K046, K050, K052, K053, K113, K122
	Gastrointestinal tract infections	K230, K231, K25, K26, K27, K28, K293, K294, K295, K35, K36, K37, K61, K630, K632, K650, K678, K908, K930
	Hepatic infections	K750, K770, K830
	Viral hepatitis	B15, B16, B17, B18, B19
Other infections	HIV/AIDS	B20, B21. B22, B23, B24
	Sexually transmitted infections	A50, A51, A52, A53, A54, A55, A56, A57, A58, A59, A60, A63, A64, N290
	Breast infections	N61
	Osteomyelitis	M462, M463, M464, M465
	Joint infections	M00, M01
	Heart and circulatory infections	B332, I00, I01, I02, I05, I06, I07, I08, I09, I301, I33, I38, I39, I400, I410, I411, I412, I430, I716, I790, I791

	Neoplasms from infection	C11, C161, C162, C163, C164, C165, C166, C168, C169, C210, C211, C220, C46, C53, D002, D013, D06
	Postoperative infections	T802, T814, T826, T827, T835, T836, T845, T846, T847, T857, T874
	Adverse effect of ID treatment	R761, R762, T36, T37, T485, T487, T490, T495, T496, T499, T788, T789, T880, T881, T887
	Other bacterial infections	A20, A21, A22, A23, A24, A25, A26, A27, A28, A30, A31, A320, A328, A329, A33, A34, A35, A36, A37, A38, A42, A43, A44, A480, A484, A488, A49, A65, A66, A67, A68, A69, A70, A71, A74, A75, A77, A78, A79, B95, B96
	Other viral infections	A90, A91, A92, A93, A94, A95, A96, A98, A99, B00, B01, B02, B03, B04, B05, B06, B07, A08, B09, B25, B26, B27, B33, B34, B97
	Other mycoses	B35, B36, B37, B38, B39, B40, B41, B42, B43, B44, B45, B46, B47, B48, B49
	Other protozoan infections	B50, B51, B52, B53, B54, B55, B56, B57, B58, B60, B64
	Other IDs	B65, B66, B67, B68, B69, B70, B71, B72, B73, B74, B75, B76, B77, B78, B79, B80, B81, B82, B83, B85, B87, B88, B89, B94, B99, E033, E321, F024, F071, I88, T64

AIDS, acquired immunodeficiency syndrome; CNS, central nervous system; HIV, human immunodeficiency virus; ID, infectious disease; ICD-10, International Classification of Diseases, 10th Revision.

Appendix Table A3: Associations between intersectional strata and hospitalisations for paediatric infectious diseases

	All infections	**Upper respiratory infections**	**Lower respiratory infections**	**Enteric infections**	**Genitourinary infections**
Strata:	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)
Non-immigrant, F, ≥13, Q5	1 ref	1 ref	1 ref	1 ref	1 ref
Non-immigrant, F, ≥13, Q4	1.07 (1.04–1.10)	1.15 (1.08–1.22)	0.96 (0.91–1.01)	1.10 (1.04–1.16)	1.08 (0.99–1.17)
Non-immigrant, F, ≥13, Q3	1.06 (1.03–1.09)	1.22 (1.14–1.30)	0.93 (0.88–0.98)	1.09 (1.03–1.15)	1.03 (0.93–1.13)
Non-immigrant, F, ≥13, Q2	1.08 (1.05–1.12)	1.25 (1.17–1.34)	0.91 (0.86–0.97)	1.12 (1.05–1.19)	1.15 (1.03–1.27)
Non-immigrant, F, ≥13, Q1	1.02 (0.98–1.07)	1.03 (0.94–1.13)	0.85 (0.79–0.92)	1.14 (1.06–1.24)	1.05 (0.92–1.20)
Non-immigrant, F, 10-12, Q5	1.13 (1.09–1.17)	1.20 (1.12–1.29)	0.99 (0.93–1.06)	1.15 (1.08–1.23)	1.10 (0.99–1.23)
Non-immigrant, F, 10-12, Q4	1.16 (1.12–1.19)	1.34 (1.26–1.42)	1.04 (0.99–1.10)	1.16 (1.10–1.23)	1.14 (1.04–1.25)
Non-immigrant, F, 10-12, Q3	1.19 (1.15–1.22)	1.38 (1.30–1.46)	1.04 (0.99–1.09)	1.19 (1.13–1.25)	1.22 (1.12–1.33)
Non-immigrant, F, 10-12, Q2	1.22 (1.19–1.26)	1.42 (1.34–1.51)	1.05 (1.00–1.11)	1.25 (1.19–1.32)	1.21 (1.11–1.32)
Non-immigrant, F, 10-12, Q1	1.23 (1.19–1.27)	1.38 (1.29–1.47)	1.02 (0.97–1.09)	1.29 (1.21–1.37)	1.26 (1.14–1.39)
Non-immigrant, F, ≤9, Q5	1.23 (1.12–1.35)	1.45 (1.21–1.74)	1.14 (0.97–1.35)	1.11 (0.92–1.33)	1.33 (1.01–1.76)
Non-immigrant, F, ≤9, Q4	1.41 (1.32–1.51)	1.63 (1.43–1.85)	1.26 (1.11–1.42)	1.36 (1.20–1.54)	1.35 (1.10–1.66)
Non-immigrant, F, ≤9, Q3	1.42 (1.34–1.50)	1.85 (1.66–2.06)	1.28 (1.15–1.42)	1.31 (1.17–1.46)	1.41 (1.18–1.68)
Non-immigrant, F, ≤9, Q2	1.49 (1.41–1.56)	1.76 (1.60–1.93)	1.39 (1.27–1.51)	1.45 (1.33–1.59)	1.20 (1.03–1.42)
Non-immigrant, F, ≤9, Q1	1.49 (1.43–1.54)	1.84 (1.71–1.98)	1.27 (1.19–1.36)	1.50 (1.40–1.61)	1.12 (0.99–1.28)
Non-immigrant, M, ≥13, Q5	1.24 (1.21–1.27)	1.45 (1.37–1.52)	1.33 (1.28–1.39)	1.04 (0.99–1.09)	0.82 (0.75–0.89)
Non-immigrant, M, ≥13, Q4	1.29 (1.25–1.32)	1.64 (1.56–1.73)	1.31 (1.25–1.37)	1.07 (1.02–1.13)	0.90 (0.82–0.98)

Non-immigrant, M, ≥13, Q3	1.33 (1.29–1.37)	1.71 (1.61–1.81)	1.24 (1.18–1.30)	1.15 (1.09–1.21)	0.85 (0.77–0.94)
Non-immigrant, M, ≥13, Q2	1.32 (1.28–1.36)	1.65 (1.55–1.76)	1.24 (1.17–1.31)	1.19 (1.12–1.27)	0.82 (0.74–0.92)
Non-immigrant, M, ≥13, Q1	1.25 (1.20–1.30)	1.60 (1.48–1.73)	1.17 (1.09–1.25)	1.21 (1.12–1.30)	0.73 (0.63–0.85)
Non-immigrant, M, 10-12, Q5	1.35 (1.30–1.39)	1.68 (1.58–1.79)	1.36 (1.29–1.44)	1.17 (1.10–1.25)	0.82 (0.73–0.92)
Non-immigrant, M, 10-12, Q4	1.44 (1.40–1.48)	1.94 (1.83–2.04)	1.43 (1.37–1.50)	1.21 (1.15–1.28)	0.97 (0.89–1.07)
Non-immigrant, M, 10-12, Q3	1.48 (1.44–1.52)	1.95 (1.85–2.06)	1.41 (1.34–1.47)	1.24 (1.17–1.30)	0.96 (0.88–1.05)
Non-immigrant, M, 10-12, Q2	1.46 (1.42–1.50)	1.95 (1.85–2.06)	1.37 (1.31–1.44)	1.30 (1.23–1.37)	0.81 (0.73–0.89)
Non-immigrant, M, 10-12, Q1	1.50 (1.45–1.55)	1.98 (1.86–2.10)	1.36 (1.29–1.43)	1.40 (1.32–1.48)	0.87 (0.78–0.97)
Non-immigrant, M, ≤9, Q5	1.54 (1.41–1.67)	1.97 (1.68–2.30)	1.58 (1.37–1.83)	1.34 (1.14–1.58)	1.12 (0.83–1.50)
Non-immigrant, M, ≤9, Q4	1.61 (1.52–1.72)	2.30 (2.06–2.57)	1.61 (1.45–1.78)	1.32 (1.17–1.49)	0.78 (0.60–1.01)
Non-immigrant, M, ≤9, Q3	1.65 (1.56–1.74)	2.11 (1.91–2.34)	1.80 (1.65–1.96)	1.39 (1.25–1.54)	0.82 (0.66–1.03)
Non-immigrant, M, ≤9, Q2	1.81 (1.73–1.90)	2.33 (2.14–2.54)	1.75 (1.62–1.89)	1.66 (1.53–1.81)	0.91 (0.76–1.09)
Non-immigrant, M, ≤9, Q1	1.80 (1.73–1.86)	2.40 (2.24–2.57)	1.71 (1.61–1.82)	1.73 (1.62–1.85)	0.90 (0.79–1.04)
Immigrant, F, ≥13, Q5	0.88 (0.83–0.93)	0.75 (0.66–0.87)	0.85 (0.77–0.95)	0.93 (0.83–1.04)	0.99 (0.83–1.18)
Immigrant, F, ≥13, Q4	1.01 (0.95–1.08)	1.06 (0.92–1.22)	0.75 (0.66–0.85)	1.08 (0.96–1.22)	1.23 (1.02–1.49)
Immigrant, F, ≥13, Q3	1.01 (0.94–1.08)	1.04 (0.90–1.20)	0.82 (0.73–0.93)	1.11 (0.99–1.26)	1.11 (0.91–1.35)
Immigrant, F, ≥13, Q2	1.02 (0.97–1.07)	1.10 (0.98–1.23)	0.80 (0.72–0.88)	1.05 (0.95–1.16)	1.27 (1.09–1.47)
Immigrant, F, ≥13, Q1	1.00 (0.96–1.05)	1.08 (0.98–1.19)	0.79 (0.72–0.86)	1.12 (1.03–1.22)	1.12 (0.97–1.29)
Immigrant, F, 10-12, Q5	1.05 (0.95–1.16)	1.02 (0.82–1.26)	0.90 (0.75–1.07)	1.24 (1.05–1.47)	1.21 (0.91–1.61)
Immigrant, F, 10-12, Q4	1.18 (1.09–1.27)	1.29 (1.11–1.50)	1.00 (0.88–1.15)	1.28 (1.12–1.47)	1.66 (1.37–2.03)
Immigrant, F, 10-12, Q3	1.24 (1.16–1.32)	1.48 (1.30–1.67)	1.02 (0.91–1.14)	1.22 (1.09–1.38)	1.21 (0.99–1.47)

Immigrant, F, 10-12, Q2	1.18 (1.12–1.24)	1.39 (1.26–1.54)	0.99 (0.91–1.09)	1.26 (1.15–1.38)	1.38 (1.20–1.60)
Immigrant, F, 10-12, Q1	1.24 (1.19–1.30)	1.33 (1.22–1.45)	1.01 (0.94–1.09)	1.38 (1.28–1.49)	1.40 (1.24–1.58)
Immigrant, F, ≤9, Q5	1.11 (0.94–1.32)	0.97 (0.67–1.42)	1.29 (0.99–1.69)	0.95 (0.68–1.33)	1.28 (0.79–2.08)
Immigrant, F, ≤9, Q4	1.23 (1.10–1.38)	1.05 (0.80–1.36)	1.11 (0.90–1.37)	1.31 (1.06–1.61)	1.07 (0.73–1.56)
Immigrant, F, ≤9, Q3	1.44 (1.31–1.57)	1.66 (1.40–1.98)	1.43 (1.23–1.65)	1.48 (1.26–1.74)	1.27 (0.96–1.68)
Immigrant, F, ≤9, Q2	1.34 (1.27–1.42)	1.62 (1.45–1.80)	1.17 (1.06–1.30)	1.35 (1.22–1.49)	1.23 (1.03–1.47)
Immigrant, F, ≤9, Q1	1.48 (1.42–1.54)	1.65 (1.52–1.79)	1.15 (1.07–1.24)	1.80 (1.68–1.93)	1.39 (1.23–1.58)
Immigrant, M, ≥13, Q5	1.00 (0.95–1.06)	1.13 (1.01–1.27)	1.01 (0.92–1.11)	0.91 (0.82–1.02)	0.77 (0.64–0.93)
Immigrant, M, ≥13, Q4	1.20 (1.13–1.27)	1.48 (1.32–1.67)	1.14 (1.03–1.26)	1.13 (1.00–1.27)	0.96 (0.79–1.18)
Immigrant, M, ≥13, Q3	1.19 (1.12–1.26)	1.55 (1.38–1.75)	1.05 (0.95–1.17)	1.14 (1.01–1.28)	0.82 (0.65–1.02)
Immigrant, M, ≥13, Q2	1.27 (1.21–1.33)	1.58 (1.44–1.74)	1.09 (1.00–1.19)	1.27 (1.16–1.39)	0.86 (0.72–1.02)
Immigrant, M, ≥13, Q1	1.26 (1.21–1.32)	1.56 (1.44–1.70)	1.02 (0.94–1.10)	1.29 (1.19–1.39)	0.96 (0.83–1.11)
Immigrant, M, 10-12, Q5	1.34 (1.23–1.46)	1.54 (1.30–1.82)	1.39 (1.21–1.61)	1.22 (1.03–1.43)	0.88 (0.64–1.20)
Immigrant, M, 10-12, Q4	1.40 (1.31–1.51)	1.87 (1.65–2.13)	1.31 (1.16–1.47)	1.29 (1.13–1.47)	0.99 (0.77–1.26)
Immigrant, M, 10-12, Q3	1.45 (1.37–1.54)	1.85 (1.65–2.06)	1.35 (1.22–1.50)	1.38 (1.24–1.54)	0.96 (0.78–1.19)
Immigrant, M, 10-12, Q2	1.55 (1.48–1.62)	2.07 (1.90–2.25)	1.32 (1.22–1.43)	1.47 (1.35–1.60)	1.06 (0.90–1.24)
Immigrant, M, 10-12, Q1	1.52 (1.46–1.58)	2.04 (1.89–2.19)	1.27 (1.19–1.36)	1.70 (1.59–1.82)	0.93 (0.81–1.07)
Immigrant, M, ≤9, Q5	1.41 (1.21–1.64)	1.35 (0.98–1.84)	1.52 (1.20–1.94)	1.46 (1.12–1.90)	1.27 (0.80–2.03)
Immigrant, M, ≤9, Q4	1.65 (1.48–1.83)	1.70 (1.38–2.11)	1.61 (1.35–1.92)	1.75 (1.46–2.11)	1.33 (0.95–1.88)
Immigrant, M, ≤9, Q3	1.72 (1.59–1.86)	2.45 (2.12–2.82)	1.42 (1.23–1.64)	1.71 (1.48–1.98)	1.06 (0.79–1.43)
Immigrant, M, ≤9, Q2	1.77 (1.68–1.86)	2.30 (2.09–2.53)	1.66 (1.52–1.80)	1.78 (1.63–1.95)	1.13 (0.94–1.35)
Immigrant, M, ≤9, Q1	1.80 (1.74–1.87)	2.24 (2.09–2.41)	1.58 (1.49–1.69)	1.98 (1.85–2.11)	1.01 (0.88–1.16)

Year of birth:					
Per year	0.98 (0.97–0.98)	0.96 (0.96–0.96)	0.99 (0.99–1.00)	0.96 (0.96–0.97)	0.98 (0.97–0.98)
AUC	0.56 (0.56-0.56)	0.59 (0.59-0.59)	0.55 (0.55-0.55)	0.57 (0.57-0.57)	0.56 (0.55-0.56)
ΔAUC model 2*	<0.01	<0.01	<0.01	<0.01	<0.01
ΔAUC model 1†	0.02	0.03	0.04	0.01	0.03

Analyses included 1785 588 children, with 249 644 overall, 59 928 upper respiratory, 73 465 lower respiratory, 62 985 enteric, 18 750 genitourinary and 71 673 other infectious disease hospitalisations.
*AUC model with intersectional strata variable and year of birth compared with the AUC model with sociodemographic variables (maternal education, household income, sex/gender, maternal migration status) and year of birth.
†AUC model with intersectional strata variable and year of birth compared with the AUC model with year of birth only.
AUC, area under the receiver operating characteristic curve; ΔAUC, incremental change in AUC value; CI, confidence interval; F, female/girl; M, male/boy; OR, odds ratio.