

The Norwegian Family Based Life Course (NFLC) study: data structure and potential for public health research

Øyvind Næss · Dominic Anthony Hoff

Received: 9 November 2011 / Revised: 31 May 2012 / Accepted: 1 June 2012 / Published online: 27 June 2012
© Swiss School of Public Health 2012

Abstract

Objectives To present details of the Norwegian Family Based Life Course Study.

Methods All Norwegians participating in censuses from 1960 to 2001 were included. In addition to the personal identity number, we used household and family information from the 1960 census to link family members together. The NFLC study is further linked to other health registers and surveys.

Results The proportion included and alive in 1960 increased from 67 % among those born in 1900 to more than 90 % for those born after 1940. In all, 5,266,270 were included. This combined family linkage approach gave 85 % parental linkage for those born in 1940 that dropped to 20 % of those born in 1930. The proportion with misclassified parents was less than 0.5 %. In all, 3,564,582 individuals were linked to their parents.

Conclusions The NFLC is one of the largest follow-up of individuals over several decades in their life course. The comprehensive multigenerational, family linkage within the database contributes to large-scale use of various designs for investigating life course determinants.

Keywords Data linkage · Longitudinal studies · Socioeconomic factors

Introduction

Public health researchers have increasingly emphasized the need to take the full life course into account in understanding the origin of many chronic diseases and health inequalities in adulthood (Ben-Shlomo and Kuh 2002; Lynch and Davey Smith 2005). As many chronic diseases may take many years to develop, only focusing on social and biological determinants in adulthood has been seen as inadequate. This comes from a plethora of research on diseases or health-related states like coronary heart disease, chronic obstructive pulmonary disease (Naess et al. 2007), obesity (Howe et al. 2012) and diabetes type 2 (Smith et al. 2011). For example, premature mortality due to cardiovascular disease has been linked to a number of risk factors through the life course, including intrauterine growth retardation (Lawlor and Ben-Shlomo 2004), rapid growth in infancy (Davey Smith et al. 2001), childhood cognitive development (Batty et al. 2005), childhood overweight/obesity, their associated cardiometabolic risk factors and tracking of these into adulthood (Bao et al. 1995; Kvaavik et al. 2003; Lauer and Clarke 1990; Sundstrom et al. 2011), and access to preventive and curative medical services later in the disease process (Korda et al. 2011).

Early life interventions have been highlighted in efforts to prevent many chronic diseases and reduce social inequalities in health. The evidence for this comes from historic cohorts like the 1946 and the 1958 birth cohorts in the UK. More recently, new cohorts have been established like the Millennium Cohort and the Norwegian and Danish birth cohorts (Andersen and Olsen 2011; Magnus et al. 2006). These

This article is part of the special issue “Life course influences on health and health inequalities: moving toward a Public Health perspective”.

Ø. Næss (✉) · D. A. Hoff
Division of Epidemiology, National Institute of Public Health,
Oslo, Norway
e-mail: oyvind.nass@medisin.uio.no

Ø. Næss
Institute of Health and Society, University of Oslo,
Oslo, Norway

provide valuable insight but have limitations in terms of sample size; also, they are still young and preclude investigation of outcomes in adulthood. The sensitive and critical periods for a child born today may not be of similar magnitude and importance as for a child born in 1940, which calls for comparison of life course processes in various generations. Precluding the life course processes to individual level is considered inadequate, because the social patterning of risk over the life course may vary at population level, either over historical time or in different populations (Krieger 2001). Formal testing of the models by more robust and/or alternative approaches has been only recently started by researchers. But also, to compare and detect differences between various life course models, large sample sizes may often be needed. Novel methodological developments for investigating early life influences more robustly often requires large sample sizes and a flexible data structure (De Stavola et al. 2006; Lawlor and Mishra 2009).

Nordic countries are in a fortunate position to conduct public health and etiologically oriented epidemiological research due to the personal identity code (PIN) (Olsen et al. 2010; Rosen 2002). This makes it possible to link all individuals to various health databases and surveys. These codes were established in the 1960s; as time since the introduction of the PIN passes and novel approaches to investigate the data become appreciated, the scientific value of using population-based linkages is likely to increase. We have established a large-scale linkage on Norwegian data, the Norwegian Family Based Life Course Linkage (NFLC) with an explicit aim to construct life course data that could take maximal advantage of Norwegian registers. This involved extending the lifetime as much as possible to include older birth cohorts and to link families for intergenerational studies.

The NFLC linkage and others using register linkages in Norway and other Nordic countries may have large potential for researchers when they plan future studies. Nevertheless, given the comprehensiveness of the database and the challenges in communicating the potentials and limitations inherent in the database, we aim here to give a more detailed presentation. We will discuss this in light of some examples of novel methodological developments in life course epidemiology.

Methods

Population

The population was based on a selection of all Norwegians who participated in the census in 1960, 1970, 1980, 1990 or 2001 and who had received a personal identity number. This comprises in all 6,272,827 individuals. To make the presentation clearer in terms of comparison with the Norwegian population and linkage across populations, we will only present those who were born in Norway ($n = 5,266,270$; see Table 1). Those born outside Norway prior to the 1960 census and thus lacking Norwegian birth data would provide missing family information by using data from the 1960 census. Those who emigrated between census time points (1960 and later) and who did not participate in the following census were excluded. Finally, those who were born and died during census time points were not included (1960 census and later).

Family linkage

The Norwegian personal identity code, an 11-digit code, was introduced in 1964 and applied to the 1960 census

Table 1 Number of included records by year of birth in the database compared to liveborn in Statistics Norway total per decade in Norway

Year of birth ^a	Liveborn, Statistics Norway	Emigrated	Deaths	Included in the database	% Included	Norwegian census total ^b
1900–1909	640,643	–	–	428,276	67	–
1910–1919	615,045	–	–	475,418	77	–
1920–1929	573,253	–	–	472,591	82	–
1930–1939	442,522	–	–	386,203	87	–
1940–1949	595,299	–	–	547,488	92	–
1950–1959	628,024	–	–	594,680	95	–
1960–1969	650,762	10,052	11,106	629,604	97	3,457,504
1970–1979	579,197	4,666	7,582	566,949	98	3,797,043
1980–1989	527,707	4,999	5,017	517,691	98	4,098,116
1990–2001	659,530	8,641	3,519	647,370	98	4,198,267
Total	5,911,982	18,358	27,224	5,266,270	89	–

^a Those born in Norway prior to 1960 had to participate in the 1960 census in order to be included in the database

^b Total number of Norwegian-born citizens living in Norway at census time according to census data from 1960, 1970, 1980, 1990 and 2001

(Vassenden 1987). Because of limited resources at that time, the identity of the spouse, mother and father was not established for everyone. Emphasis was placed on individuals living with young families. In order to extend this family linkage for older age groups, we used household and demographic variables from the 1960 census to identify parents and children within families. Each family and household had unique codes. The variable family status included single, single mother or father with offspring and married or partner with or without offspring. By using this information, the oldest person(s) were identified as mother(s) and/or father(s). The age difference between the identified mother and/or father and the other household/family members should be larger or equal to 15 years. The age limit for sexual relations in Norway has been 16 years for several decades. As a consequence, we discovered that there were few official data, prior to the 1970s, officially identifying parents under the age of 15 years. We only excluded those parents who were underaged and who we could only identify by using the 1960 census data (no pin number). Underaged parents who were identified by official pin numbers by Statistics Norway were included in the database, consequently almost all mothers/fathers born after 1960 were included if they fulfilled the other selection criteria.

For the older age groups (born before 1960), we found that a considerably higher degree of the population were born in Norway and this meant that a higher percentage of the population who were alive in 1960 could be included in the database. There was little immigration to Norway prior to the mid-1970s, especially from non-Western countries. As a consequence, the linkage is not representative to all Norwegian-born inhabitants residing in Norway today, due to a higher rate of non-Norwegian-born residents living in Norway, many for a limited time period due to work commitments. For those who were not born in Norway, relevant personal information, in addition to parental information from other registries, will often be missing, thus excluding these people from the database.

Variables

The database is a linkage of censuses and register information from the National Personal Registry, the Educational Registry and the Cause of Death Registry. At each census, all individuals were asked a number of questions related to household, family, residential area, place of birth, length of education and housing conditions. In 1990, the census was a combination of register information and questions asked to a random 10 % sample. In 2001, the census was only based on register data. Table 2 presents an overview on some of these variables. The other registers include panel data on several socioeconomic and demographic variables.

Table 2 Some of the socioeconomic and demographic variables available from the censuses and registers in the database

	Census				Register
	1960	1970	1980	1990	Yearly by January the 1st
Education	*	*	*	*	1970
Income		*	*	*	1968
Wealth					1993
Migrations					1964/67
Housing data		*	*	*	
Occupation and labor	*	*			
Marital status and spouse identity	*	*	*	*	1967
County	*	*	*	*	1990
Municipality	*	*	*	*	1990
Area	*	*	*	*	1990
Country of birth	*	*	*	*	1990
Household identity	*	*	*	*	1990

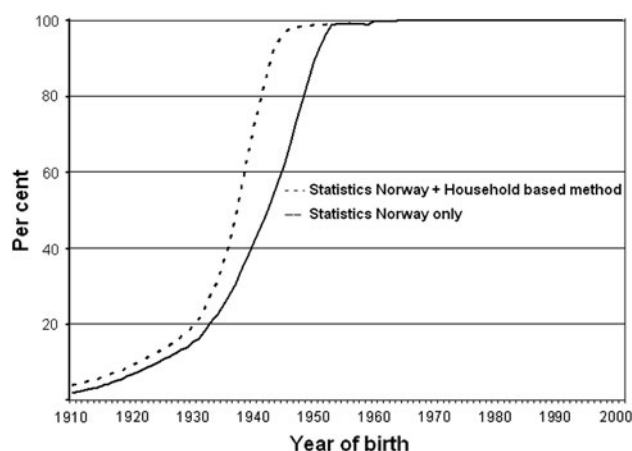


Fig. 1 Percent by year of birth born in Norway from 1910 to 2000 that could be linked to mother within the database using personal identity code from Statistics Norway only and by also including a household-based method in the 1960 census

Results

For birth cohorts who were liveborn in Norway, the proportion included in the linkage and alive at the census in 1960 increased from 67 % among those born in 1900 to more than 90 % for those born after 1940 (Table 1). For the family linkage using the PIN from Statistics Norway, the proportion that could be linked to their mother was 100 % for those born after 1952, but dropped rapidly for older cohorts (Fig. 1). By adding our household-based method using variables from the 1960 census, older cohorts could be linked. The identity of mother could be found for 85 % of those born after 1940 compared to 45 % using the

PIN method only. Most of the decrease in the combined method was for those born between 1930 and 1940, as only 20 % of those born in 1930 could be linked to their mother.

The pin code method and the household-based method for family linkage were compared for those individuals where both methods provided information, mainly for those who were born between 1952 and 1960 (Table 3). At most, 0.5 % was misclassified with a non-matching pin number and household-based pin number. There was no tendency of larger misclassification in the oldest cohorts or for any combination of linkage to mother only, father only or both mother and father. In most cases where the household-based pin method and the pin codes provided by Statistics Norway did not match, we found this was due to parents having offspring with new partners. Based on the 1960 Census, they were linked to their household at that time, whereas based on official statistics they linked to their former family (those born after 1952 in most cases).

Amongst those who were included in the database from the 1960 census ($n = 3,407,024$), the mean age was 35.0 years, 67.8 % lived in a family unit consisting of a father, mother and children, 79.1 % had primary education only and 49.9 % were male. Amongst those who were not Norwegian born and therefore who were not included in the database from the 1960 census ($n = 61,116$), the mean age was 40.0 years, 52.5 % lived in a family unit consisting of a father, mother and children, 72.8 % had primary education only, and 55.5 % were female indicating that it was more common for Norwegian-born men to marry foreign-born wives compared to Norwegian-born women to marry foreign-born men amongst this excluded sample.

Figure 2 presents the number of siblings in three birth cohorts: those born from 1940 to 59, 1960 to 79 and 1980 to 01. Larger families with more children were more common in the oldest cohorts. The number of families with only a single child has doubled in the same period. In total, 3,564,582 individuals could be linked to both mother and father in the database (not tabulated). A total of 1,575,516

offspring had a complete combination of both parents and all four grandparents (not tabulated). Looking at grandparents separately for mothers and fathers gives 1,808,624 offspring with complete information on mothers and both grandparents and 1,774,283 offspring with similar data on fathers; 1,900,691 cousins could be linked on the premise that they shared the same grandmother.

Further linkages

The NFLC is currently linked to the Cause of Death Registry, the Medical Birth Registry, the Sickness and Disability Registry and the Cohort of Norway (CONOR) (see Table 4; Naess et al. 2005, 2008b). The Cause of Death Registry includes information on date and cause of death from 1 January 1960 onward through 2009. The Medical Birth Registry was established in 1967 and includes detailed information on pregnancy and birth for all born in Norway. The Sickness and Disability Registry was established in 1992 and includes panel data on each cause of sick leave, permanent disability benefit with ICD-code and other social security benefits as well as labor market participation. CONOR is a collection of Norwegian regional health surveys of adults conducted from 1994 onward, including information on 173,236 individuals with sampling rate averaging a range between 50 and 80 % for the cohorts (Naess et al. 2008a). People were asked about their health and exposure to various health risks. CONOR also includes blood pressure, pulse and height and weight. Triglycerides and cholesterol levels in serum were also collected. Blood (EDTA) was collected and frozen in biobanks for later DNA extractions. By using the life course database linked to CONOR, we have been able to identify 48,401 siblings within CONOR, sharing the same mother and 46,131 sharing the same mother and father. A total of 19,840 participants within CONOR could be internally linked both to their mother and father participating in CONOR (full trio).

Table 3 Number of correctly identified records and percent (%) within the database born prior to 1960 by year of birth using the household census-based method compared to the official family linkage method based on personal identity numbers from Statistics Norway

Year of birth	Paternal ($n = 905,990$)		Maternal ($n = 960,814$)		Paternal and maternal ($n = 870,580$)	
	Wrong (%)	Correct (%)	Wrong (%)	Correct (%)	Wrong (%)	Correct (%)
<1910	1 (0.1)	725 (99.9)	2 (0.2)	1,307 (99.8)	0 (0.0)	275 (100.0)
1910–1919	6 (0.1)	4927 (99.9)	10 (0.1)	7141 (99.9)	3 (0.1)	3,044 (99.9)
1920–1929	19 (0.1)	14,753 (99.9)	42 (0.1)	18,147 (99.8)	16 (0.1)	11,505 (99.9)
1930–1939	133 (0.3)	48,526 (99.7)	189 (0.3)	54,811 (99.7)	129 (0.3)	43,516 (99.7)
1940–1949	920 (0.3)	287,330 (99.7)	934 (0.3)	314,301 (99.7)	1,000 (0.4)	271,670 (99.6)
1950–1959	2,443 (0.4)	546,207 (99.6)	1,805 (0.3)	562,125 (99.7)	2,748 (0.5)	536,674 (99.5)
Total	3,522 (0.4)	902,468 (99.6)	2,982 (0.3)	957,832 (99.7)	3,896 (0.4)	866,684 (99.6)

Only those individuals born in Norway within the database where parental identity could be retrieved by both methods are compared

Fig. 2 Number of siblings within families in the database born in Norway, stratified by birth cohort born from 1940 to 2001

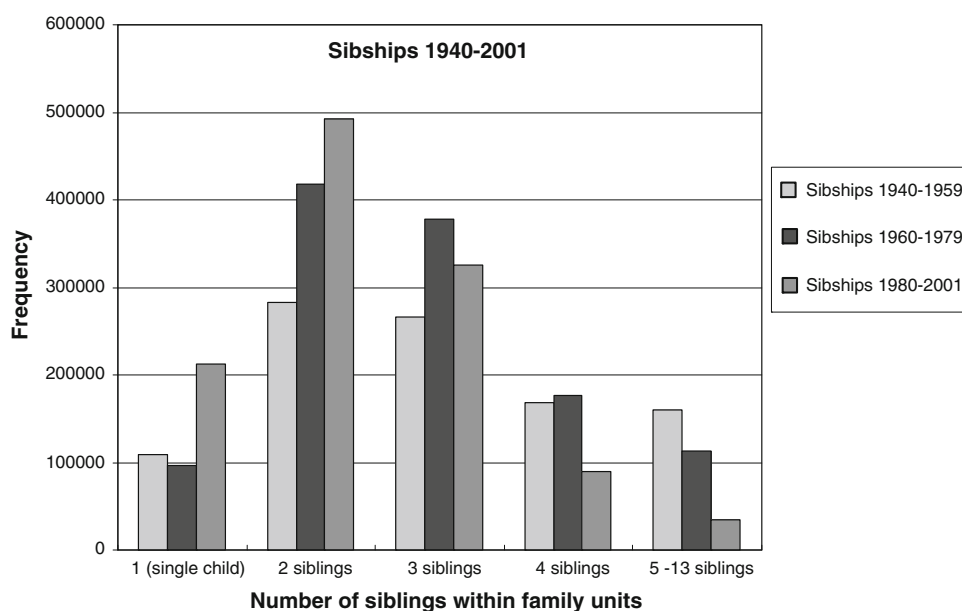


Table 4 Current and planned linkages to health surveys and health registers in the database with number of records and time span covered

	1960	1970	1980	1990	2001	2010
The Norwegian Family based life course database +						
<i>Linkage 1:</i>						
The Cause of death registry (n=2,024,166)						
The Medical birth registry (n=2,517,812)						
The Sickness and disability registry (n=1,252,923)*						
The Cohort of Norway (n=173,236)						
<i>Linkage 2:</i>						
The Cause of death registry (n=2,024,166)						
The Conscript registry (n=1,226,030)						
Tbc Screening with height and weight (n=2,001 719)						
Norwegian 40-year olds surveys (n=429,245)						
Norwegian county surveys (n=94,034)						
The Cohort of Norway (n=173,236)						

*Individuals have multiple sick leaves

Another linkage has been currently established including the Norwegian Military Conscript Registry, TBC screening including height and weight, the Norwegian 40-year-old surveys and the Norwegian county studies. The first will include cognitive ability as well as height and weight, whereas the latter two include information focusing mainly on cardiovascular risk factors.

Discussion

The NFLC linkage is one of the largest worldwide follow-up of individuals over several decades in their life course.

The comprehensive multigenerational and family linkage within the database provides large-scale use of family-based designs for investigating life course determinants.

Strengths and weaknesses of the database compared to other data sets

The main comparative strengths of this database are the large size, long time span, extensive family linkage and the possibility of linking many registers together, including biomedical data. In addition to this, the comprehensive and flexible nature of the linkage enables researchers to select more specific birth cohorts to advance the field of

longitudinal research. A large proportion of those born in 1940 having survived to 1960 could be linked to their parents (Fig. 1) and with follow-up of disease end points to 2010 this would soon cover a full lifetime. Previously, this has given us the opportunity to investigate the impact of paternal occupational class for 20 single causes of death in adulthood (Naess et al. 2007). Furthermore, as the database includes all Norwegians who participated in censuses from 1960 to 2001, historical trends can be studied in more detail. A recent study from the database showed that educational inequalities in mortality had increased from 1960 to 2001 (Strand et al. 2010). The future inequalities in common diseases like cardiovascular diseases in contemporary children will not be fully explained by what has been learned from contemporary cohorts of adults (Galobardes 2010). Thus, replication of descriptive trend studies including younger birth cohorts will be needed to understand better what will be the important proximal and distal determinants in future generations. The data includes repeat measurements of socioeconomic exposures from the censuses over the life course where occupation was coded into occupational class according to the Ericsson Goldthorpe scheme (Table 2). This has been exploited in earlier studies to investigate if repeated experiences of disadvantage through the life course add risk cumulatively for mortality from cardiovascular diseases and chronic obstructive pulmonary diseases (Naess et al. 2004). The multigenerational linkage makes it possible to extend parental offspring associations to grandparents (Modin and Fritzell 2009).

Several weaknesses should be mentioned: There is a possibility of paternal and maternal misclassification, especially for the oldest birth cohorts (Table 3). Even if the comparison between the family linkage methods using the PIN code and the household data from the 1960 census gave surprisingly few classifications of mothers and fathers, we do not know for certain if this is the case for all those for whom we could produce a family linkage, but lacked PIN code. However, we have no reason to believe that this would be of greater magnitude for those. We do not know for sure if they were biological parents even if they belonged to the same household, i.e., social parents. For the older cohorts born before 1960, growing up in a divorced family was uncommon, for example the rate of divorce was constant from 1940 to 1970 at 4 per 1,000 marriages. Since then, the rate has increased to 12 (Mamelund et al. 1997). The core part of the database includes several socioeconomic variables, but lacks more detail on specific early life exposures. Also, the field of life course epidemiology may benefit from more use of modeling physiological or health function rather than only relying on the onset of impairment or disease (Singh-Manoux et al. 2011). The linkages with other health surveys give

additional information on exposures, mostly from repeated measurements from early to late adulthood, like body mass index, blood pressure, cholesterol and self-reported health-related behaviors. Inherent in most life course studies especially when people are followed up into old age, there is a possibility of selection bias when sampling is based on those who have survived to a certain age (Glymour 2007). Compared to official records in Statistics Norway, this is most likely to be of importance for those born before 1940, as the proportion having survived to 1960 drops for older cohorts (Table 1). The measurements and procedures for data collection have changed somewhat across the cohorts and for some items of the registers. To what degree this causes bias may vary.

Data challenges for increasing prevention and policy relevance

The scientific approaches coming from life course epidemiology has become part of normal science in a wide range of subjects and disciplines, such as further developments in early life programming to understand what determines healthy aging. Examples of the first could be to understand better if the childhood obesity epidemic was mainly driven by intrauterine programming of the fetus or the postnatal environment (Ebbeling et al. 2002). Evidence for this would need to be based on various approaches, but a recent study from Sweden used comprehensive record linkage between sibling data, birth registry data on weight change during pregnancy and BMI from conscripts (Lawlor et al. 2011). The demographic developments in Western societies with a shift toward an increase in the proportion of elderly challenge researchers to investigate how previous life course trajectories interact with current health as people enter old age, which will be highly relevant to the concept of ‘compression of morbidity’ (Blane et al. 2008). A way to investigate this more in detail is to compare different generations as they enter old age, which will normally not be feasible within one single cohort study.

Researchers are faced with methodological and analytical challenges when analyzing datasets that are suited to life course analyses. This often implies using repeat measures of exposure and/or outcomes. It allows one to examine the dynamic processes of health states to tease out to see if critical or sensitive periods exist. The repeated recording of residential area has been used to disentangle the relative importance of residential area at various time points through the life course for various causes of death in a multilevel framework (Naess et al. 2005, 2008b). This has involved developing and applying novel statistical approaches that require large data to take individual’s migration history into account. It has also involved looking at area effects of deprivation and variation on mortality

being largely explained by people's individual life course socioeconomic position (Naess et al. 2005). This and other similar studies, finding smaller variability and effect at area level than individual level, could suggest that in the current environment where chronic diseases are predominant, even if such diseases seem to cluster in deprived areas, the individual level life course trajectory may explain more of the total variance than the residential areas over the life course (Merlo et al. 2009). However, this may be disease and context specific. The current health policy for primordial and primary prevention of chronic diseases has paid attention to life course epidemiology by emphasizing critical periods (Marmot et al. 2010), but may also question the levels of influence, e.g., individual, family and area or state where interventions are most likely to be influential and which would need comprehensive linkages such as this one.

Testing life course models

To draw causal inferences from associations between early life exposures can be challenging because these occur many years before the outcome and are related to other exposures through the life course. Several new analytical solutions have been suggested: Mendelian randomization using genes known to be related to certain phenotypes and randomly assorted during the process of meiosis and gamete formation have been used in an instrumental variables approach (Davey Smith 2003). Strengthened causal inference has also been suggested when comparing maternal and paternal offspring associations. The association between birth weight and cardiovascular disease risk may be due to shared genes or socioeconomic environment rather than intrauterine programming (Davey Smith 2008). If the association between cardiovascular disease risk in parents and offspring birth weight is similar for both mothers and fathers, it would be consistent with shared factors. This approach has been used to investigate if childhood obesity originates in utero. Family-based designs such as discordant sibling analysis have been used to adjust for early life confounders shared in the family. Within a life course framework, this could also be used to disentangle the impact of the early life socioeconomic environment for inequalities observed in adulthood or using the partner's identity for later life changes. At population level, life course determinants may be historically contingent and change in future generations (Krieger 2001). The recent decline in CVD mortality illustrates this because the decline could be linked to the historic decline in smoking. In future, other determinants of CVD mortality may become more important at population level.

Conclusions

The scientific value of the NFLC is likely to increase as the life span of the population covered increases. The comprehensive multigenerational and family linkage within the database provides large-scale use of various designs for investigating life course determinants. To facilitate collaborative research using the data, researchers would benefit from understanding the complex nature of the data. This database may be accessed through a formal collaboration with the Norwegian Institute of Public Health.

References

- Andersen AM, Olsen J (2011) The Danish National Birth Cohort: selected scientific contributions within perinatal epidemiology and future perspectives. *Scand J Public Health* 39:115–120
- Bao W, Threefoot SA, Srinivasan SR, Berenson GS (1995) Essential hypertension predicted by tracking of elevated blood pressure from childhood to adulthood: the Bogalusa Heart Study. *Am J Hypertens* 8(7):657–665
- Batty GD, Mortensen EL, Nybo Andersen AM, Osler M (2005) Childhood intelligence in relation to adult coronary heart disease and stroke risk: evidence from a Danish birth cohort study. *Paediatr Perinat Epidemiol* 19(6):452–459
- Ben-Shlomo Y, Kuh D (2002) A life course approach to chronic disease epidemiology: conceptual models, empirical challenges and interdisciplinary perspectives. *Int J Epidemiol* 31:285–293
- Blane D, Netuveli G, Montgomery SM (2008) Quality of life, health and physiological status and change at older ages. *Soc Sci Med* 66(7):1579–1587
- Davey Smith G (2003) 'Mendelian randomization': can genetic epidemiology contribute to understanding environmental determinants of disease? *Int J Epidemiol* 32:1–22
- Davey Smith G (2008) Assessing intrauterine influences on offspring health outcomes: can epidemiological studies yield robust findings? *Basic Clin Pharmacol Toxicol* 102:245–256
- Davey Smith G, Greenwood R, Gunnell D, Sweetnam P, Yarnell J, Elwood P (2001) Leg length, insulin resistance, and coronary heart disease risk: the Caerphilly Study. *J Epidemiol Community Health* 55:867–872
- De Stavola BL, Nitsch D, Silva ID, McCormack V, Hardy R, Mann V et al (2006) Statistical issues in life course epidemiology. *Am J Epidemiol* 163(1):84–96
- Ebbeling CB, Pawlak DB, Ludwig DS (2002) Childhood obesity: public-health crisis, common sense cure. *Lancet* 360(9331):473–482
- Galobardes B (2010) Closing the gap in a generation: what more research do we need? *Int J Public Health* 55(5):453–455
- Glymour MM (2007) Commentary: selected samples and nebulous measures: some methodological difficulties in life-course epidemiology. *Int J Epidemiol* 36(3):566–568
- Howe LD, Tilling K, Galobardes B, Davey Smith G, Gunnell D, Lawlor DA (2012) Socioeconomic differences in childhood growth trajectories: at what age do height inequalities emerge? *J Epidemiol Community Health* 66(2):143–148
- Korda RJ, Clements MS, Dixon J (2011) Socioeconomic inequalities in the diffusion of health technology: uptake of coronary procedures as an example. *Soc Sci Med* 72(2):224–229

- Krieger N (2001) Theories for social epidemiology in the 21st century: an ecosocial perspective. *Int J Epidemiol* 30(4):668–677
- Kvaavik E, Tell GS, Klepp KI (2003) Predictors and tracking of body mass index from adolescence into adulthood: follow-up of 18 to 20 years in the Oslo Youth Study. *Arch Pediatr Adolesc Med* 157(12):1212–1218
- Lauer RM, Clarke WR (1990) Use of cholesterol measurements in childhood for the prediction of adult hypercholesterolemia. The Muscatine Study. *JAMA* 264(23):3034–3038
- Lawlor D, Ben-Shlomo Y (2004) Pre-adult influences on cardiovascular disease. In: Kuh D and Ben-Shlomo Y (eds) *A life course approach to chronic disease epidemiology*, 2nd edn. Oxford University Press, Oxford
- Lawlor DA, Mishra GD (2009) *Family Matters. Designing, analysing and understanding family-based studies in life-course epidemiology*. Oxford University Press, New York
- Lawlor DA, Lichtenstein P, Fraser A, Langstrom N (2011) Does maternal weight gain in pregnancy have long-term effects on offspring adiposity? A sibling study in a prospective cohort of 146,894 men from 136,050 families. *Am J Clin Nutr* 94(1):142–148
- Lynch J, Davey Smith G (2005) A life course approach to chronic disease epidemiology. *Annu Rev Public Health* 26:1–35
- Magnus P, Irgens LM, Haug K, Nystad W, Skjaerven R, Stoltenberg C (2006) Cohort profile: the Norwegian Mother and Child Cohort Study (MoBa). *Int J Epidemiol* 35(5):1146–1150
- Mamelund SE, Brunborg H, Noack T (1997) Divorce in Norway 1886–1995 by calendar year and marriage cohort, Statistics Norway, Oslo-Kongsvinger
- Marmot M et al (2010) *The marmot review: fair society, healthy lives. Strategic review of health inequalities in England post-2010. The marmot review*, London
- Merlo J, Ohlsson H, Lynch KF, Chaix B, Subramanian SV (2009) Individual and collective bodies: using measures of variance and association in contextual epidemiology. *J Epidemiol Community Health* 63(12):1043–1048
- Modin B, Fritzell J (2009) The long arm of the family: are parental and grandparental earnings related to young men's body mass index and cognitive ability? *Int J Epidemiol* 38(3):733–744
- Naess O, Claussen B, Thelle DS, Davey Smith GD (2004) Cumulative deprivation and cause specific mortality. A census based study of life course influences over three decades. *J Epidemiol Community Health* 58(7):599–603
- Naess O, Leyland AH, Davey Smith G, Claussen B (2005) Contextual effect on mortality of neighbourhood level education explained by earlier life deprivation. *J Epidemiol Community Health* 59(12):1058–1059
- Naess O, Strand BH, Davey Smith G (2007) Childhood and adulthood socioeconomic position across 20 causes of death: a prospective cohort study of 800 000 Norwegian men and women. *J Epidemiol Community Health* 61(11):1004–1009
- Naess O, Sogaard AJ, Arnesen E, Beckstrom AC, Bjertness E, Engeland A et al (2008a) Cohort profile: cohort of Norway. *Int J Epidemiol* 37(3):481–485
- Naess O, Claussen B, Davey Smith GD, Leyland AH (2008b) Life course influence of residential area on cause-specific mortality. *J Epidemiol Community Health* 62(1):29–34
- Olsen J, Bronnum-Hansen H, Gissler M, Hakama M, Hjertnæs A, Kamper-Jorgensen F et al (2010) High-throughput epidemiology: combining existing data from the Nordic countries in health-related collaborative research. *Scand J Public Health* 38(7):777–779
- Rosen M (2002) National Health Data Registers: a Nordic heritage to public health. *Scand J Public Health* 30(2):81–85
- Singh-Manoux A, Marmot MG, Glymour M, Sabia S, Kivimaki M, Dugravot A (2011) Does cognitive reserve shape cognitive decline? *Ann Neurol* 70(2):296–304
- Smith BT, Lynch JW, Fox CS, Harper S, Abrahamowicz M, Almeida ND et al (2011) Life-course socioeconomic position and type 2 diabetes mellitus: the Framingham Offspring Study. *Am J Epidemiol* 173(4):438–447
- Strand BH, Groholt EK, Steingrimsdottir OA, Blakely T, Graff-Iversen S, Naess O (2010) Educational inequalities in mortality over four decades in Norway: prospective study of middle aged men and women followed for cause specific mortality, 1960–2000. *BMJ* 340:c654
- Sundstrom J, Neovius M, Tynelius P, Rasmussen F (2011) Association of blood pressure in late adolescence with subsequent mortality: cohort study of Swedish male conscripts. *BMJ* 342:d643
- Vassenden K (1987) *Folke-og boligtellingsene 1960, 1970 og 1980. Dokumentasjon*, Central Bureau of Statistics of Norway, Oslo, 87/2