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**Fetal Origins, Childhood Development, and Famine:  
A Bibliography and Literature Review**

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FETAL ORIGINS, CHILDHOOD DEVELOPMENT, AND FAMINE: A  
BIBLIOGRAPHY AND LITERATURE REVIEW<sup>1</sup>

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## ABSTRACT

The human costs of famines outlast the famines themselves. An increasing body of research points to their adverse long-run consequences for those born or in utero during them. This paper offers an introduction to the burgeoning literature on fetal origins and famine through a review of research on one well-known case study and a bibliography of published work in the field generally.

## FETAL ORIGINS, CHILDHOOD DEVELOPMENT, AND FAMINE

Famine historiography has been rather slow to absorb the implications of research claiming that famines might have long-run consequences for the health of those born during them—the fetal origins hypothesis (FOH)—for the human cost of famines. There is a literature on the long-term of famine in terms of individual and collective trauma, but the kinds of link posited by versions of the FOH are missing, for example, in recent accounts of famine in China, Greece, Ireland, Bengal, and elsewhere (Dikötter 2010; Ó Gráda 1999; Hionidou 2004; Maharatna 1996). Economists too have been rather slow to employ famines as testing grounds for the FOH, although in recent years they have been making up for their earlier disinterest (Almond and Currie 2011). Here, by way of an introduction to the literature on severe malnutrition, famine and the FOH, I offer a review of studies published on the case study that has attracted most research so far, followed by a bibliography of work, mostly published, on FOH and famine from a range of disciplines. The bibliography contains separate sections on three case studies that have attracted a lot of attention: the Dutch Hungerwinter of 1944-45, the Finnish Male Birth Cohort Study (Osmond *et al.* 2007), and the Swedish Överkalix project ([http://en.wikipedia.org/wiki/%C3%96verkalix\\_study](http://en.wikipedia.org/wiki/%C3%96verkalix_study)). It should be noted that the Finnish and Swedish studies refer more to the

consequences of severe malnutrition than to those of famine specifically.

Two preliminary points about the costs implied by versions of the FOH seem worth making at the outset. First, the added cost imposed by FOH-related factors is likely to be small compared to the demographic cost in terms of lives lost. In the case of the Netherlands in 1944-45, for example, there might have been at most 450 cases of schizophrenia among survivors relative to twenty thousand or so lives lost to famine. Second, even though these added costs must be borne in mind, they do not necessarily mean that survivors on average would have been better off had there been no famine.

In the literature on the fetal origins hypothesis (FOH) the Dutch Hunger Winter of 1944-45 holds a special place. The resulting famine is the *locus classicus* for famine-related research on the link between fetal exposure to malnutrition, on the one hand, and adult health and disease susceptibility, on the other. It is far better documented and more extensively researched than any other historical episode employed as a 'natural experiment'. Even allowing for the broad interest in the FOH, the extent of the specialist literature based on the Hunger Winter is extraordinary (see Section A of the bibliography below).

The following pages offer a brief review of a literature that originated in the early 1970s with the work of husband-and-wife team

of Zena Stein and Ezra Susser. It bears noting that the original focus of South African-born Stein and Susser was on cognitive development rather than heart disease, as in the early research of David Barker and his team. Stein and Susser were not the first scholars to be interested in the link between fetal origins and cognitive development, but they began early: Susser dated their initial interest back to 1952 (Stein *et al.* 1972, 1975: vi-vii). In 1967 Stein and Susser realized that the subjects of Clarence Smith's analysis of fetal growth during the Dutch Hunger Winter (Smith 1947) would by then have reached adulthood. The idea of a study that would overcome two difficulties with testing any version of the FOH—that of being able to study the required individual nutritional levels in 'free-living populations' and the long interval between fetal exposure and adulthood—germinated with an initial research proposal and visit to the Netherlands in 1968. Collaboration from the Dutch government and military was secured, along with research funding from the U.S. National Institutes of Health.

The project yielded its first fruits in 1972 (Stein, Susser, Saenger, and Marolla 1972a, 1972b) and the much-cited *Famine and Human Development: the Dutch Hunger Winter of 1944/45* followed in 1975. These works compared measures of cognitive development for Dutch conscripts exposed to famine *in utero* to conscripts born before and after the famine and to conscripts born in non-famine regions of the Netherlands. *Famine and Human Development* (1975) concluded with an acceptance that 'poor prenatal nutrition cannot be considered a

factor in the social distribution of mental competence among surviving adults in industrial societies' (1975: 236; compare Stein *et al.* 1972: 708), an outcome which Stein *et al.* described as 'negative'. Much later Zena Stein would relate how many colleagues were 'furious with us because it would have been much more satisfactory (in terms of social justice) to have found that food did matter' (Willcox and Stein 2003; see too Davey Smith and Susser 2002: 35). But they attributed their finding that 18-year old army recruits who had been *in utero* during the famine were *more* intelligent<sup>2</sup> than both recruits from non-famine areas and recruits born just before and after the famine in the affected area (see Figure 2a) to selection bias. This was because more resilient and better-resourced households, whose children were on average brighter, experienced less of a fertility decline during the famine than the remained. So what Stein and Susser initially deemed a disappointing outcome was due to the famine's impact on the social composition of famine birth cohort.<sup>3</sup>

The Dutch famine struck towards the end of World War II, when access to outside food supplies in the German-occupied, heavily urbanized western Netherlands was severely restricted for several months (Banning 1946; Dols and van Arcken 1946; Sellin 1946; Smith 1947; Hart 1993; Trienekens 2000; Futselaar 2009). The famine-affected

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<sup>2</sup> From scores based on Raven's widely-used tests.

<sup>3</sup> As noted below this finding has been contested by de Rooij, Wouters, *et al.* 2011.

area is shown in Figure 1. There is some slight ambiguity about precisely how long the accompanying famine lasted<sup>4</sup>, but Figure 2b, based on data in Stein *et al.* (1975: 244-46), tracks birth weights by month in the worst-affected part of the country and offers quite a precise guide to the famine's duration and intensity.

Although mortality was already rising before the famine, which makes estimating excess mortality tricky, it is likely that the famine resulted in about twenty thousand excess deaths in a population of 4.5 million. Age and gender were better predictors of death during the famine than socio-economic class. Thus in the Hague, for example, nearly four-fifths of all deaths from malnutrition in the first half of 1945 were of people aged 55 years and above. Those aged 20-39 years accounted for less than four per cent of the total (Banning 1946) [see Figure 3]. This must not be taken as evidence that the elderly suffered relatively more: the contrary is true, since they were also more vulnerable in non-crisis years.

Moreover, nearly two-thirds of those who died in the Hague at the famine's peak during the first three months of 1945 were male. The gender gap in mortality was much greater than usual during famines. The wartime context must partly account for the marked male disadvantage. There was a class aspect to the famine too; in the

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<sup>4</sup> Lumey *et al.* (2010) refer to a 'well-defined period lasting approximately six months'; but Stein, Zybert, and Lumey (2004) refer to 'an acute severe famine of seven months' duration', whereas Lumey and Stein ('In utero exposure', 1997) refer to 'a severe 5-month famine'. Scholte, van den Berg, and Lindeboom (2010) refer to '4 months', while Ralf Futselaar (2009) refers to the famine being at its most intense for about nine weeks (p. 47).

Hague the female share of deaths was 31.5 per cent in working-class households, 40.2 per cent in middle-class households, and 46.8 per cent in the relatively small number of upper-class households. Those who suffered most, relatively speaking, were prime-age males from working-class households. Deaths in working-class households were also more likely to be from malnutrition, although the share of all deaths in the Hague due to malnutrition was also significant in middle-class households, reaching two-fifths at the peak in April (Banning 1946; Human Mortality Database).

Food ration entitlements before and during the Hunger Winter are well documented, but a shortcoming of Dutch research on the FOH is that individual-level consumption levels at different stages of the famine are not. That the average mother was malnourished may be inferred by the low weights of full-term births during the famine but there is no hard evidence on how badly off individual mothers were. This is quite a serious but unavoidable shortcoming of the Dutch studies. The self-administered questionnaire on which Abeelen *et al.* base their 2011 paper contains questions about 'experience of hunger and weight loss' during the famine to which the women who agreed to participate could answer 'hardly', 'little', or 'very much'. The respondents were living in Utrecht between 1993 and 1997. Of those aged 0-9 years during the Hunger Winter 2,084 were categorized as having 'no' exposure, 1,557 'moderate' exposure, and 639 as severe' famine exposure. Given the severity of the crisis, these rates seem

implausibly modest, but Abeelen *et al.* do not distinguish between women born in the famine area and those born outside it. As Lumey and van Poppel (2010) complain, it is 'difficult' to establish the validity of such self-recall measures of individual-level nutrition (compare Kesternich *et al.* 2011).

Stein *et al.* (1975) made the point at the outset that 'Nutritional deprivation confined to the prenatal period may be too brief to produce much effect' and concluded that 'post-natal learning [might be] an attractive explanation for a great part of [the] differences among social strata' (1975: 236). However, the short duration of the Dutch famine is critical for research purposes because it permits analysis of the impact of exposure by trimester, a feature that research on the Hunger Winter has focused on from the start.

Between 1975 and 1990 Susser and Stein focused mainly on issues other than the Hunger Winter, and produced just a few papers related to it (e.g. Stein and Susser 1985; Susser 1989). With the arrival in Columbia in the early 1990s of Lambert H. Lumey from the Netherlands and the involvement of psychiatrist Ezra Susser (son of Stein and Susser), however, the output of the Columbia-based group began to grow. In an important paper published in 1992 Lumey identified 3G birth-weight effects using Hunger Winter data. The main focus in this period was on birth-weights, but Ezra Susser also co-authored two linked papers about the link between fetal origins and schizophrenia.

In 1995 Nigel Paneth and Mervyn Susser challenged the Southampton group led by David Barker to produce 'a much more careful and specific a priori formulation of the component parts of the baby's nourishment hypothesis' and then to subject this to rigorous testing and go further than loose hypothesis (Paneth and Susser 1995). Perhaps this was the gauntlet that prompted Barker to invade Columbia University's territory and to begin work on the Hunger Winter with a group of scholars based mainly at the Amsterdam Medical Centre of the University of Amsterdam?

Barker's FOH focused initially on the risk of coronary heart disease, not on cognitive or neural development (see Barker and Osmond 1986; Barker and Martyn 1992; Barker 1995). The first papers emanating from the AMC/Southampton group in 1998-99 concerned glucose tolerance, obesity, and hypertension rather than cognitive development or mental disease. Then in November 2000 Barker's Amsterdam group published a paper in *Heart* (Roseboom *et al.* 2000) which, according to one of its Dutch co-authors, provided 'the first evidence that undernutrition during gestation increases the risk of coronary heart disease'

[<http://news.bbc.co.uk/2/hi/health/1027845.stm>]. In another paper published in 2001 Barker's group claimed to have found 'the first evidence in humans that maternal undernutrition during gestation is linked with the risk of CHD in later life' (Roseboom, van der Meulen, *et al.* 2001). However, they added that the timing of malnutrition during

gestation was crucial, and that adult health might be compromised even though birth weight was unaffected, giving the FOH 'a new dimension'.

These papers cited the work of Susser, Stein, and their colleagues sparingly, and at the outset relations between the Columbia and AMC/Southampton groups seem to have been rather tense. Meanwhile research groups based at the Utrecht medical school (e.g. Elias *et al.* 2004) and at the Maastricht University Medical Center (e.g. Hughes *et al.* 2009, 2010) have also been active in the field. In the past the Utrecht group, which has recently been collaborating with the AMC (e.g. van Abeelen *et al.* 2011), has focused more on cancer and on early childhood rather than fetal origins, and the link between fetal and early childhood exposure with cancer has also been the main focus of Maastricht researchers.

- 1 *FERTILITY*. Lumey and Stein (1997) found that prenatal exposure had no effect on age at menarche, the proportion having no children, age at first delivery, or family size'. But the AMC group (2008) dispute this, finding that 'women who were exposed to the Dutch famine of 1944-1945 *in utero* are more reproductively successful than women who were not exposed to famine during their fetal development; they have more offspring, have more twins, are less likely to remain childless and start reproducing at a younger age' (Painter, Westerdorp, *et al.* 2008). They speculated

that under-nutrition in utero followed by better nutrition in early infancy or childhood might produce 'a female phenotype characterized by greater reproductive success'.

- 2 *HEART DISEASE*. As noted above the AMC/Southampton group have highlighted this aspect (e.g. Painter, de Rooij *et al.* 2006; Roseboom, van der Meulen, *et al.* 2000). But the literature reviews by Lumey *et al.* (2010) and Lumey and van Poppel (2010) conclude that research outcomes on CHD are 'inconclusive', having been 'reported for only one of the birth cohort studies for the Netherlands'. Research on the remote northern Swedish parish of Överkalix (Bygren, Kaati, and Edvinsson 2001) identifies 2G impact on CHD through lack of food during father's slow growth period (as compared to 3G impact on diabetes through too much food in grandfather's slow growth period).

A 2011 paper from the AMC/Utrecht group (van Abeelen *et al.* 2011) supports the link between exposure to famine and CHD and claims to be the first to find a lower risk of stroke in adulthood in exposed women. However, 'exposed' here refers not to fetal origins but to women of all ages (0-9, 10-17, and 18+ years).

- 3 *3G EFFECTS*. The AMC group failed to find 'transgenerational effects of prenatal exposure to famine on birthweight or on

cardiovascular and metabolic disease rates' (Painter, Osmond, *et al.* 2008), but claimed to have identified a link between G2 exposure in utero and increased G3 neonatal adiposity and poor health in later life. They claimed that their results were 'the first direct evidence' of 3G effects. This elicited a swift protest from Lumey and Stein (2009) that previous work of theirs had been ignored. To be fair, in an important paper published more than a decade earlier they had compared birth weights of children born to mothers exposed (or not) during the Hunger Winter, and the analysis led them to speculate that there might be 'long-term biological effects, even into the next generation, of maternal intrauterine nutrition, which do not correspond to the effects on the mothers' own birthweights' (Lumey and Stein 1997). Their finding that mothers exposed to the Hunger Winter early in gestation gave birth to underweight babies had a broader resonance, and was taken up by anthropologist Jared Diamond.<sup>5</sup>

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<sup>5</sup> Diamond (2000) puts it like this: 'The implication is that babies were somehow affected by the starvation of their grandmothers many decades earlier. This result might have been easier to understand if the mothers themselves had been underweight at birth or were small as adults. Neither was true. Recall that starvation in the first or second trimester produced babies with normal birth weights. Only third-trimester starvation led to small babies. Yet, paradoxically, when these small babies later became mothers, they gave birth to normal-size babies. It was the women who were themselves normal size at birth who became mothers babies. Yet, paradoxically, when these small babies later became mothers, they gave birth to normal-size babies. It was the women who were themselves normal size at birth who became mothers of underweight infants. Somehow the grandmothers' suffering programmed their children in utero so that the

4 *COGNITIVE FUNCTION*. Both the Columbia and AMC/Southampton groups have recently produced papers on the link between prenatal famine exposure and adult cognition. The former (Groot *et al.* 2011) found no link between fetal exposure and cognitive functioning at age 59—except for the faint possibility of a link with early pregnancy exposure—but the latter (de Rooij *et al.* 2010) identify an impact on selective attention in middle age. Using the Stroop task, they find that ‘at age 56 to 59, men and women exposed to famine during the early stage of gestation performed worse on a selective attention task, a cognitive ability that usually declines with increasing age’. The former corroborates research going back to Stein and Susser (who in 1972 found that cognitive performance in 19-year old male conscripts was unaffected by exposure to the famine before birth) but the claims for accelerated aging in the latter are novel and, naturally, have been taken up in the media<sup>6</sup>.

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grandchildren would be affected. This astonishing result will undoubtedly inspire experiments aimed at identifying the still-unknown cellular mechanism.’

<sup>6</sup> ‘World War II Dutch famine babies’ brains ‘aging faster’ , 13 September 2010 [<http://www.bbc.co.uk/news/health-11286462>].

5 *HEIGHT*: Much of the work on FOH and famine focuses on height, because this is the most easily identifiable outcome in the available data. For instance, Dercon and Porter (2010) report that children who aged under three years at the peak of the 1984 Ethiopian famine were at least 3 cm shorter in adulthood than those at less vulnerable ages (compare too Khoroshinina (2005) on the impact of the Leningrad blockade-famine). However, height has not been the focus of Dutch work.

Van den Brandt *et al.* (1997) report a positive association between height and the risk of breast cancer. So might growth stunting due to the Hunger Winter have reduced the incidence of cancer? One Dutch study that claims that fetal exposure had an effect on height is van Noord and Arias-Careaga (1995). This is the work of an Utrecht scientist and his Spanish co-author, based on results in the latter's dissertation. It found that height was reduced among women severely exposed to the famine which, van Noord and Arias-Careaga speculate, would square with an expected effect of a reduction in breast cancer risk.

6 *MENTAL DISORDERS*. Ezra Susser and Lin (1992) found that cohorts exposed to severe deprivation during the first trimester 'showed a substantial increase in hospitalized schizophrenia for women but not for men', concluding that their findings suggested gender-specific effects of early prenatal nutrition on the risk of

schizophrenia. The issue was taken up again in a more ambitious study by Susser *et al.* (1996), which found that the impact of early prenatal nutritional deficiency was not confined to women.<sup>7</sup> Moreover, they found that only those born between October 15<sup>th</sup> and December 31<sup>st</sup> 1945, i.e. when the famine was at its peak, were affected; babies born between August 1 and mid-October 1945 (and thus conceived between the beginning of November 1944 and mid-January 1945) were unaffected (Susser *et al.* 1996: Figure 1). Next, Hoek *et al.* (1999) investigated the evidence for schizophrenia spectrum personality disorder, as distinct from schizophrenia *per se*, in men conceived at the height of the Hunger Winter. They found that the exposed cohort was much more likely to develop a schizoid personality disorder by age 18 years (see too Hoek *et al.* 1996).

- 7 *CANCER*. The Utrecht group claims that malnutrition increased the risk of breast cancer for women aged 2 to 33 years during the Hunger Winter (Elias *et al.* 2005). Painter *et al.* (2006) found an increased incidence of breast cancer among those conceived during the Hunger Winter, but the difference was not statistically significant. Another Maastricht paper (Hughes *et al.*

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<sup>7</sup> According to St. Clair *et al.* (2005) and Xu *et al.* (2009), the Great Leap famine doubled the risk of schizophrenia in adults, an outcome that 'almost exactly replicate[s] the Dutch findings'. Given the vast differences in economic contexts and scale of the two famines, exact replication is surely almost coincidental.

2009) claims to be the first to establish a link between 'severe environmental condition in adolescence or early adulthood' and the incidence of colorectal cancer in later life. Lumey *et al.* (2010) cite but do not discuss three papers on breast, prostate, and colon cancer by the Maastricht group (Dirx *et al.* 1999, 2001, 2003) and by van Noord and Kaaks (1991) because they were concerned with post-natal rather than *in utero* exposure.

Heijmans *et al.* (2008) and Tobi *et al.* (2009) found that fetuses exposed to famine early in pregnancy had lower rates of DNA methylation—a biochemical process which is important in the development of most kinds of cancer--than their than their same-sex siblings. The rate of methylation influences how much gene cells will synthesize. Almost simultaneously, the Maastricht group (Hughes *et al.* 2009) reported finding that exposure to famine in adolescence and young adulthood may produce persistent epigenetic changes that increase the risk of colorectal cancer.

- 8 *THE TRIVERS-WILLARD HYPOTHESIS.* Since bearing male children exacts a greater toll on mothers and male infants are less likely to survive, it is sometimes argued that, for evolutionary reasons, during famines the proportion of males born falls (e.g. Gibson and Mace 2003). This is what is known in the literature as the Trivers-Willard effect. In general the evidence in favour of the

hypothesis is thin. The Columbia group denies its presence in the Netherlands during the Hongerwinter on Hunger Winter (Stein, Zybert, and Lumey 2004; Cramer and Lumey 2010) and—see Figure 4—it is absent also in Ireland in the 1840s, in Leningrad in the 1940s, and in Finland in the 1860s (Ó Gráda 2009: 107-108).

9 *GENDER*. The literature on early childhood points to some gender differences in links between treatments and subsequent cognitive functioning. On the FOH and gender differences in general, the survey by Lumey *et al.* (2010) is quite equivocal, but it posits a gender effect on DNA methylation (citing Tobi *et al.* 2009). The Överkalix project highlights how parental food supply (poor in the case of the father, good in the case of the mother) reduced the risk of death from heart disease (Kaati *et al.* 2002), but its focus is on the pre-growth spurt period rather than on *in utero*.

10 *SELECTION*. As noted above, Stein *et al.* (1972, 1972) attributed their 'negative' result to selection bias. However, this problem—in a sense the original sin of studies like those described here (Bozzoli, Deaton, and Quintana-Domeque 2009)—has been rarely addressed. An exception is the important study by Lumey and Stein (1997) of the impact of exposure on birth weights of mothers and their children, in which they controlled for selection

by making 'pairwise comparisons of parity-specific birth weights with siblings'.<sup>8</sup> Similarly the study of obesity by Stein, Rundle et al. (2009), that of DNA methylation by Heijmans, Tobi, *et al.* (2008), and that of cognitive functioning at age 59 by de Groot, Stein, *et al.* (2011) use siblings as controls. See too Lumey, Stein, *et al.* (2007); Stein, Kahn, *et al.* (2007). Using siblings as controls gets rid at least of selection due to time-invariant factors. This is not a feature of Southampton/Amsterdam studies.

Nearly four decades on, there is an amusing quality to the complaint of a reviewer of *Famine and Human Development* that Zena Stein and her co-authors had wrung 'the last ounce of juice from the subjects of the House of Orange' (A. M. Thomson, *Science*, (1976) 188: 832). Today the FOH/Hongerwinter enterprise rolls on, and with no signs of slackening. New research alliances are being formed: some of David Barker's Southampton team have spread their wings to Helsinki, while the Columbia group have plans to collaborate with the Umeå University group responsible for the northern Swedish Överkalix studies. The recent interest in epigenetics has probably broadened interest in their work; variants of the term 'epigenetic' entered the literature on the Hunger Winter only in the late 2000s.

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<sup>8</sup> Their conclusion (1997: 819) anticipates later findings: 'Long-term health effects after undernutrition may occur in the absence of a birth-weight effect, and may not be apparent even in its presence'.

Several of the principals have provided useful summaries of findings to date (e.g. Lumey and van Poppel 1994, 2010; Lumey, Stein, and Susser 2011). There seems to be broad agreement on a link between exposure and obesity (already noted in 1976 by Ravelli-Stein-Susser in a frequently-cited paper, although that study referred to males only) and on later life implications for the risk of diabetes, heart disease, and schizophrenia. The initial mutual hostility between the Columbia and AMC groups, and their tendency not to cite each other's work, has given way to healthier competition. But on some key issues, rival groups are producing conflicting results from seemingly very similar databases, and it is not clear whom to believe. I will mention two examples. In a recent paper Stein, Rundle, *et al.* (2009) could not replicate the 'couch-potato' finding of Lussana *et al.* (2008) that gestational exposure led to a preference for high fat foods. They put the 'discordant' findings down to 'sampling variability' (2009: 1559). And Lumey *et al.* (2010: 246) attribute the conflicting findings of the Columbia and Amsterdam groups regarding the impact of exposure on fertility to 'minor differences in the selected study populations and in the definitions of famine exposure'.<sup>9</sup> Unfortunately, in epidemiological research replicability is not as straightforward as in the social sciences,

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<sup>9</sup> In a very useful recent survey Lumey and van Poppel (2010b) lament the 'diffuse and conflicting' nature of the findings of different studies, which they attribute to 'limited sample size and chance observations'. For that reason they must still be considered 'exploratory and hypothesis generating'. Lumey and van Poppel counsel 'common analytic strategies across comparable studies to further explore specific hypotheses'.

and research teams guard their databases jealously. From a social science and economic-historical perspective this is a regrettable outcome.

Both the AMC/Southampton and Columbia groups have broadened their focus considerably. A recent paper 'explores' the impact on sexual differentiation, on the basis that animal experiments have found that 'underfeeding of the mother can result in feminization of the male offspring', but finds that prenatal exposure to famine did not affect sexual orientation in men or in women'. But they add that 'the small sample size of participants with non-exclusively heterosexual identification (possibly due to underreporting of homosexuality) may have reduced our power to detect any differences.' There is a paper on irritable bowel syndrome which finds that 'exposure to severe wartime conditions *in utero was not associated* with the prevalence of IBS in adulthood', but that early-life exposure to severe wartime conditions *was*.

Research outcomes on the FOH and the Hunger Winter have a broad appeal. They have prompted advice to women not to try to lose weight during pregnancy, to the claim that it 'may have accelerated brain ageing'.<sup>10</sup> And Jared Diamond has warned that we ignore the lessons of the Dutch Hunger Winter 'only at our children's, and our grandchildren's, expense'.

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<sup>10</sup> 'Birthweight link to lifelong health: <http://www.bbc.co.uk/news/health-14576961>;  
<http://www.bbc.co.uk/search/news/?q=famine%20brain%20ageing>.

Finally, it is striking how little involvement there has been so far by Dutch economists and economic historians in FOH studies of the Dutch famine. This seems a pity, since some of the Dutch research has been rather insensitive to worries that matter more in the social sciences such as the small size of samples, various selection issues (likelihood to respond to questionnaires, socio-economic background of parents), the role of historical context, and replicability. The field seems ripe for interdisciplinary collaboration.

The rest of this paper consists of a bibliography of published work on the FOH and famine. It is in four sections. Section A contains both general items and case studies. Section B lists work so far on the Dutch Hunger Winter. Section C refers to studies based on the Finnish Cohort Finnish Male Birth Cohort. Section refers to papers published on the Swedish Överkalix Study. Work continues apace!

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*B. Studies using Dutch Hunger Winter Databases (by date):*

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### C. Studies Based on Finnish Male Birth Cohort (by year of publication):

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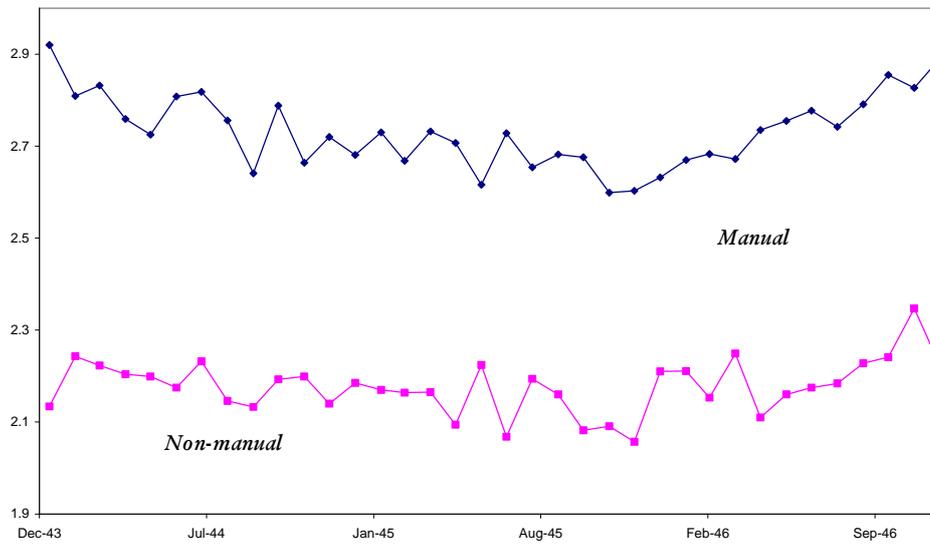


Figure 2a. Raven Test Scores by Monthly Birth Cohort in the Western Netherlands, 1944-46

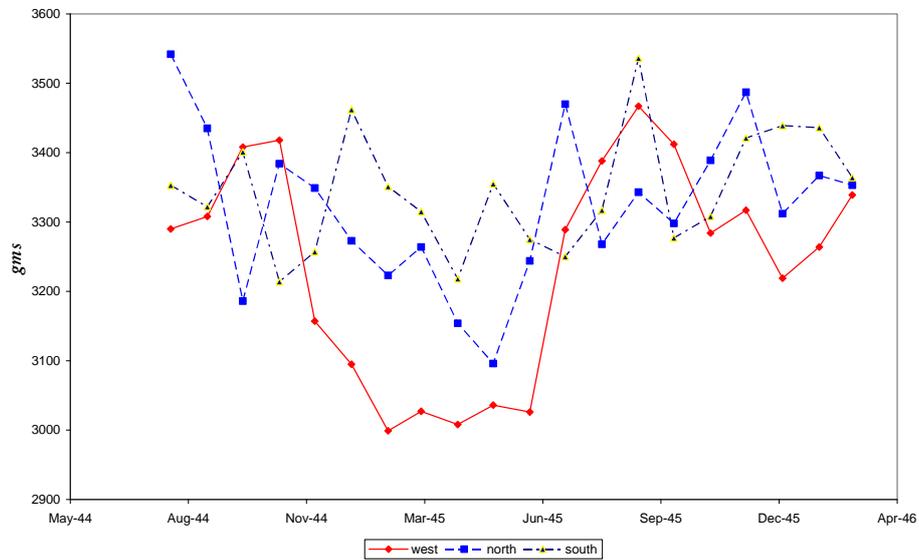


Figure 2b. Birth Weight by Month, 1944-46

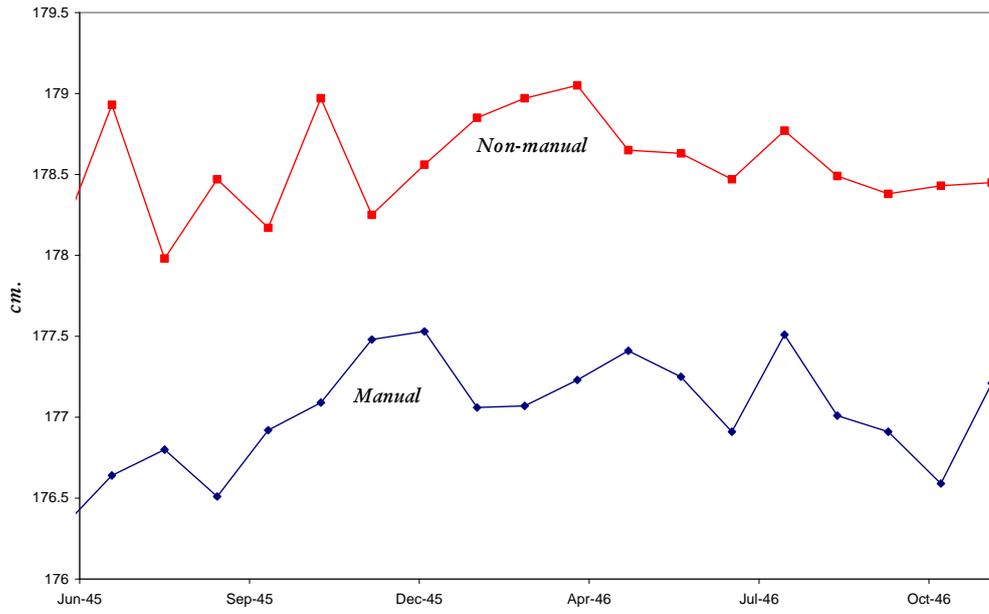
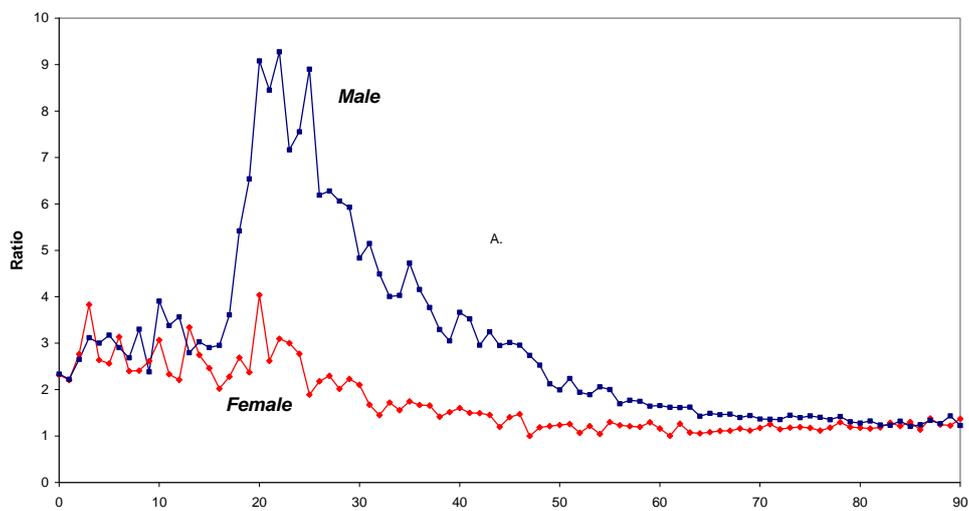


Figure 2c. Adult Height by Month of Birth, 1945-46

**A. 1945 Relative to 1939**



**B. 1945 Relative to 1943**

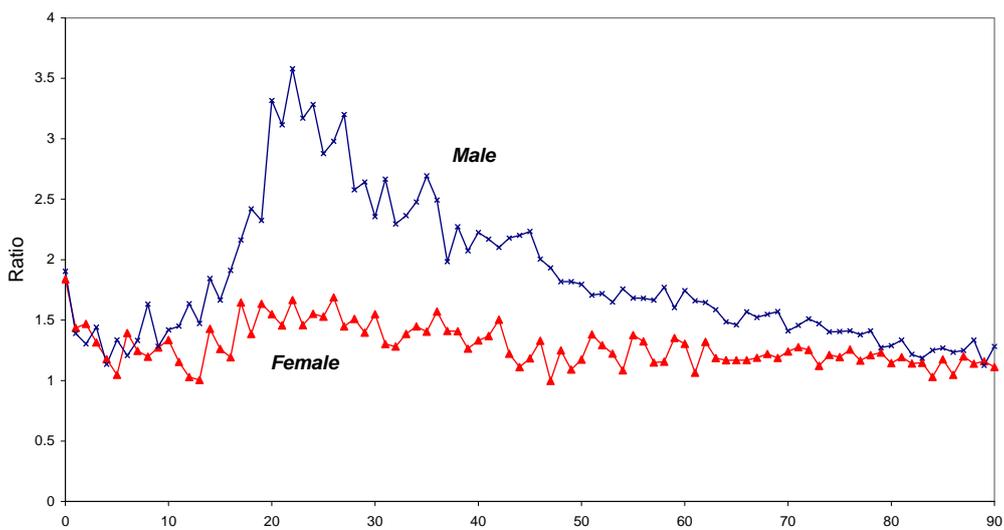
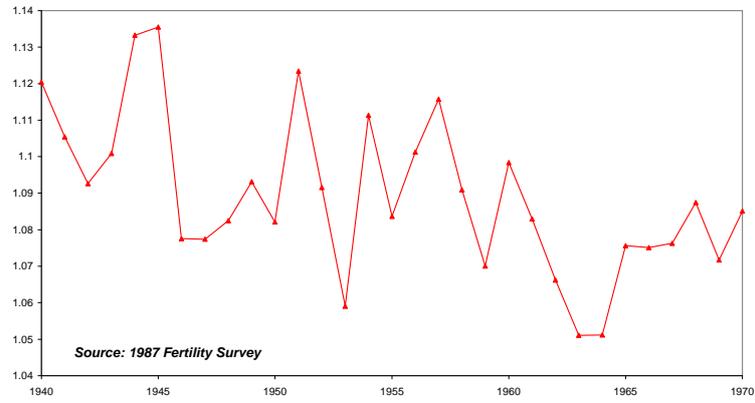


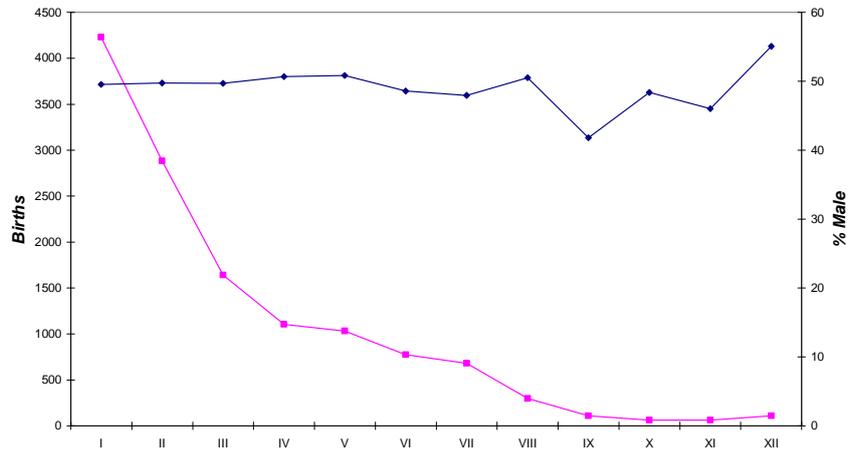
Figure 3. Mortality by Age in the Netherlands before and during the Hunger Winter

Source: Human Mortality Database

**M/F Ratio in China, 1940-70: All Births**



**Leningrad 1942: Total Births and Percentage Male**



**Finland: Percentage Male Births 1860-1875**

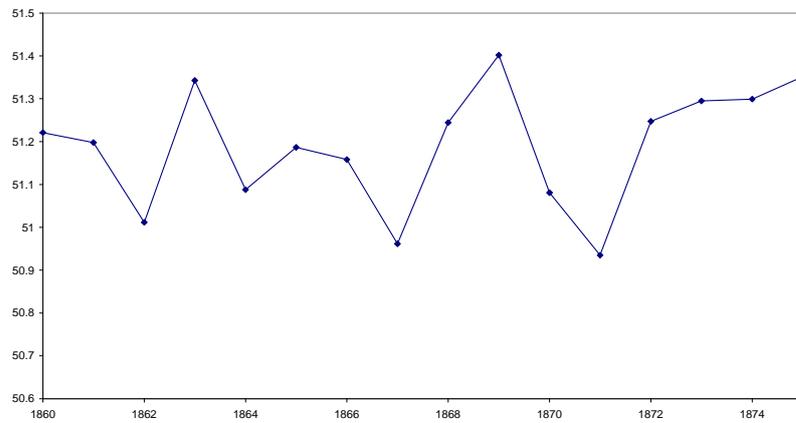


Figure 4. Births, Gender, and Famine

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