

**The War Machine
and Global Health**

***A Critical Medical
Anthropological Examination
of the Human Costs of Armed
Conflict and the International
Violence Industry***

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CHAPTER ONE
THE ECHOES OF WAR
Effects of Early Malnutrition on Adult Health
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A Biocultural Approach to War

The direct and indirect mechanisms through which war affects health are numerous. One pathway is the violence of malnutrition, a frequent by-product of war. "High-intensity conflict" (defined, following Cohen and Pinstrup-Andersen [1999], as a war with more than 1,000 deaths per year) regularly leads to severe food insecurity and malnutrition. If this occurs during growth and development in childhood, infancy, or even prenatally, there exists a high likelihood of long-term or even permanent costs to health. The developmental origins of health and disease hypothesis, or DOHaD (Barker 1998; Gluckman and Hanson 2005), within the context of war is well supported by a body of evidence that strongly indicates that physiological stressors, particularly malnutrition, occurring in utero or in infancy predispose an individual to developing chronic diseases later in life, including cardiovascular disease, diabetes, obesity, and osteoporosis. Using examples from World War II and the first and second Indochina wars, this chapter examines how war-induced famine has long-term and possibly irreversible effects on the development of some of the aforementioned chronic diseases.

While it is necessary to incorporate a healthy dose of biology here, the focus will not be physiological. Rather, my aim is to link different levels of analysis by employing a hybrid of critical, biocultural, and evolutionary perspectives to demonstrate that warfare consistently creates a suite of conditions that are harmful to human biology and health. Studies on the

health effects of war typically have fallen under the aegis of epidemiology, public health, relief agencies, or human rights groups. Some anthropologists have advocated a greater role for the discipline in investigating the broader impacts of war on human biology and health. For example, Farmer (2003:241) has called for "mak[ing] room in the academy for serious scholarly work on the multiple dynamics of health and human rights, on the health effects of war and political-economic disruption, and on the pathogenic effects of social inequalities. . . . By what mechanisms do such noxious events and processes become embodied as adverse health outcomes?" (emphasis added). In order to account for these mechanisms, multiple disciplines are needed, as none is sufficient by itself to describe how ecological and political-economic disruptions, in this case resulting from war, get "under the skin" (Goodman and Leatherman 1998:20). Anthropology, given its multiple subdisciplines, may be uniquely poised to examine war from a biocultural perspective.

While it has been difficult to live up to the ideal of holism in anthropology (Wiley 2004), collaboration is essential if the goal is to not only understand the health costs of war but ultimately to reduce its frequency. It is clear that theoretical overlap toward the study of human biology and health exists among the traditional subdisciplines of anthropology. Critical cultural and medical anthropologists have correctly pointed out the need to account for the role that human agency, political economy, inequality, historical contingency, and wider global forces have on local realities and health (Baer et al. 2003; Singer 1998). Similarly, a number of biological anthropologists have called for a holistic, transdisciplinary approach to human biology that goes beyond looking at single stressors and broadly defines the environment as the totality of its evolutionary, ecological, and social components, including social inequality (Leatherman et al. 1993; Little and Haas 1989; Thomas et al. 1989). Biological anthropologists are particularly well suited to study the effects of stressors on biology, and warfare arguably creates conditions (at least in the short term from an evolutionary standpoint) that rival any of the ecological stressors traditionally studied in the discipline, such as hypoxia at high altitudes, circumpolar temperatures, or infectious diseases in tropical latitudes. They may also address the ways that populations adapt to conditions created by war or whether the concept of adaptation is even appropriate under such

circumstances (Bogin et al. 2007; Ellison and Jasienska 2007; see also Singer 1989).

For these reasons, it becomes clearer that a hybrid of approaches to the study of war and health has great utility. Ecological and evolutionary approaches are likely to reveal consistent biological responses to war. However, critical and biocultural perspectives remind us that rather than focusing solely on biological variables and risking overgeneralizations when looking at the health effects of war, it is also necessary to account for local historical circumstances. In some cases, the general health status of a population may already be poor prior to war's inception because of poverty or structural violence exacerbated by inequality or colonialism (e.g., Laos during the first and second Indochina wars). In other cases, the health of a population may be good prior to the onset of war, then rapidly deteriorate once war begins and recover fairly quickly after the cessation of military conflict (e.g., the Netherlands during World War II). In either scenario, what they share in common is that war inexorably makes things worse, and the health effects may reverberate for decades for fundamental biological reasons that will be discussed here.

War, Food Insecurity, and Malnutrition

Throughout history, armed conflict has served as both cause and consequence of food insecurity and famine. In the seminal two-volume study *The Biology of Human Starvation* by Ancel Keys et al. (1950), the forty-fifth chapter was devoted to the effects of famine on childhood growth and development. A meta-analysis conducted for the purposes of this essay reveals that Keys et al. cited eighty-eight studies in the chapter pertaining to famine (after excluding a subsection on seasonality and family), sixty-one of which (69 percent) dealt with famine resulting from war. The fact that the majority of citations pertained to war may be reflective of the historical period, given that most of the authors' research took place during and just after World War II (indeed, much of their research on the effects of starvation was conducted on conscientious objectors to the war). An alternative interpretation is that when conducting a literature review on famine, military conflict reliably provides examples from which to draw.

The reasons for this are many. Food often has been used deliberately as a weapon in war, and its provision has served as a means to win the "hearts and minds" of a population (Macrae and Zwi 1994). Conversely, withholding food through the destruction or theft of crops or through the interception or obstruction of food aid or trade routes has been used to deprive an adversarial military or a civilian population suspected of supporting one's opposition (Levy et al. 2000; Roland 1992; Vincent 1994). Food blockades, sieges of cities, and "scorched-earth" tactics meant to deny food to civilian and military populations have been documented in ancient history from Mesopotamia, Egypt, and the siege of Troy to the "winning of the West," from Native Americans to Leningrad and the Netherlands in World War II, to Ethiopia, Eritrea, Angola, and Mozambique in the 1980s and 1990s (Carmean 2002; Macrae and Zwi 1994; Stein et al. 1975; White 2005). In addition to direct attacks on crops and livestock, food production may be diminished through the reduction of the farming population via targeted killings, genocide, terrorization, forced recruitment, enslavement, and forced displacement or through contamination of farmland by indiscriminant bombing, chemical weapons, or land mines (Cohen and Pinstrop-Andersen 1999; Cuny and Hill 1999).

For refugees, food acquisition is tenuous, particularly for those who have fled their homes but remain "internally displaced" (i.e., remain within their country of origin), since finding sanctuary from nearby military conflict may be difficult and delivery of food aid may be problematic. In fact, in the early 1990s, internally displaced persons had higher mortality rates than did displaced persons who crossed international borders, though, admittedly, reliable data are difficult to collect (Toole and Waldman 1993).

Displaced individuals are at several disadvantages with regard to obtaining adequate food, leading to quantitative and qualitative nutritional deficiencies. First, as involuntary migrants, refugees are often forced to flee without warning or time to plan for evacuation. Second, long journeys to safety, usually on foot, are physically draining. For children who have lost one or both of their parents, the chances of finding food or making the journey are even harder. Third, on reaching safety, displaced persons are often trapped in less fertile areas with fewer natural resources than their former homes. In subsistence economies, agricultural cycles broken by war mean that it could take months to grow crops to fruition. For the poor,

there are even fewer options since they have few liquid resources available to exchange for food possessed by nondisplaced local populations, and food prices are often increased during war, making it even harder to acquire adequate nutrition. Fourth, massive displacement during war leads not only to the loss of one's own home but also to the large-scale destruction of social support and trade networks, thus removing any safety net. Fifth, despite the Geneva Convention's call for the protection of civilian victims of war, refugees crossing international borders are often seen as burdens by host countries, possibly affecting the provision of food aid (Toole and Waldman 1997). Sixth, conditions for displaced people are often crowded and unsanitary, promoting the spread of air- and waterborne infectious disease, which has an interactive effect with malnutrition (Yip and Sharp 1993). Finally, food aid rations for refugees, particularly in past decades, have been inadequate in total calories and micronutrients. Mason (2002) traced the history of international food relief efforts and reported that prior to the late 1980s, caloric recommendations were as low as 1,200 to 1,800 kilocalories per person per day, rising to 1,900 kilocalories in 1989, 2,100 in 1997, and then 2,400 in 2000. In effect, more realistic needs of refugees were recognized, causing rations roughly to double over two decades, though refugees from earlier decades likely suffered a cost to their health as a result of the earlier recommendations.

Childhood Growth during War: Adaptation or Accommodation?

Biological anthropologists, epidemiologists, physicians, and humanitarian aid workers have long recognized that physical growth during childhood is to a great extent a reflection of the larger environment, including available nutrition, exposure to infection, and social and economic inequality (Bogin 1999). In the words of the pediatrician and auxologist James Tanner (1986:3), childhood growth is a mirror for "the material and moral condition" of a society. In various populations around the world, anthropometric markers such as height and weight repeatedly have been demonstrated to increase over generations when conditions conducive for health improve. The opposite is also true; wherever the "material and moral condition" is in decline, including during war, childhood growth usually declines as well.

In the study by Keys et al. (1950) mentioned previously, the growth of children living in war-induced famine conditions were compared to those in nonfamine conditions from the same country (e.g., children in postwar conditions vs. prewar conditions or children in war zones vs. those in safe zones). The majority of studies (41 of 61, or 67.2 percent) found that war had a negative impact on either height or weight. Fifteen studies (24.6 percent) found no effect on growth, while just two studies (3.3 percent) showed positive effects, the reasons for which are unclear. Three showed contradictory effects (i.e., some age-groups or sex groups were negatively affected, while others had no effect or even positive effects). Additional studies have shown that during World War II, childhood growth declined sharply in many countries, including China, Japan, the Soviet Union,

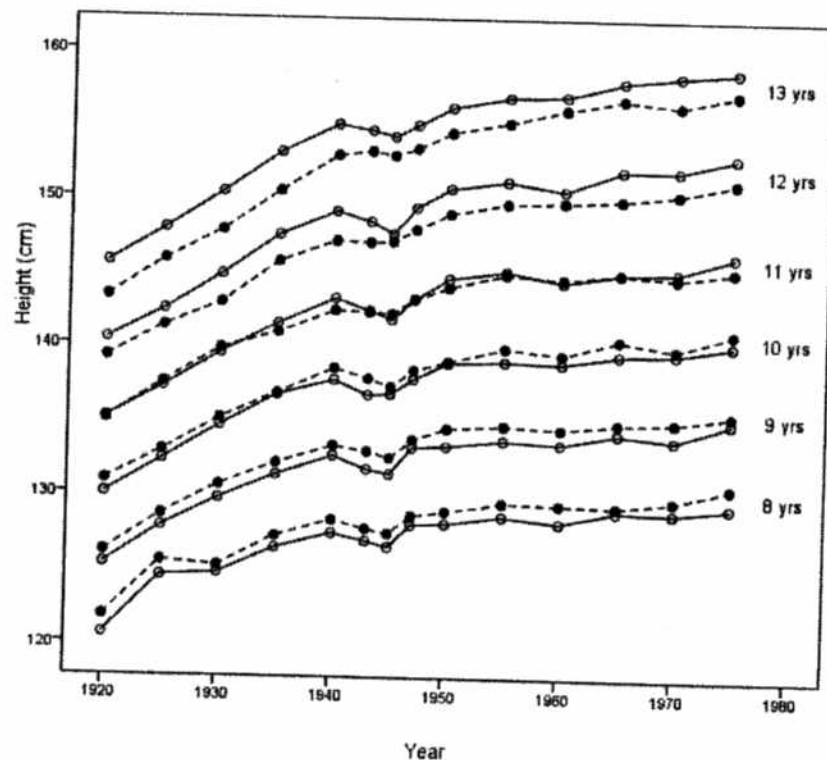


Figure 1.1. Height of schoolchildren aged 8 to 13 years in Oslo, Norway, from 1920 to 1975. Height increased over time but decreased in the mid-1940s during World War II. Boys are solid circles; girls are open circles. Data from Brundtland et al. (1980).

Germany, France, Italy, and Belgium (Kimura 1984; Markowitz 1955; Vlastovsky 1966). For example, data from Brundtland et al. (1980) on Norwegian children reveal that height increased from 1920 to 1940, then declined during the war, after which it began to increase again.

Similar patterns have been found in more recent decades as well, where rates of acute malnutrition ranged from 12 to 81 percent among refugee children from many war-torn countries from the late 1980s to the mid-1990s, including Afghanistan and various African nations (Toole and Waldman 1997).

Military conflict also has had a negative impact on other markers of growth and development. A delay in the age at menarche, or first menstruation, has been observed in Croatia and Bosnia during the 1990s war in the former Yugoslavia and in South Korea during the Korean War, likely because of a combination of nutritional and psychological stress (Hwang et al. 2003; Prebeg and Brali 2000; Tahirović 1998). Admittedly, it is necessary to avoid overgeneralizing the effects of war and to take into account the local circumstances that a given conflict has on any particular population (e.g., the duration and intensity of the war, the prewar political-economic conditions that may exacerbate stresses brought on by war, or whether specific groups fare worse than others). Overall, however, the trend appears to be consistent: war is wanton in its destruction and is detrimental to growth and development and, by extension, health. For example, while the poor and disadvantaged groups may be hit the hardest by war, Bogin and Keep (1999) reported that height declined among Mayan and Ladino children from all social classes in Guatemala from 1974 to 1984, a period that included some of the harshest fighting of the civil war and led to economic instability, a drop in food production, and the targeting of civilians by the military.

A deeper look into the biology of growth and development reveals why they frequently decline during war. Reducing the rate of growth is a predictable mechanism for the body to cope with a shortage of nutrients by forgoing the development of new tissues in favor of maintaining those already present (Bogin 1999). From an evolutionary perspective, natural selection should favor genes that provide an organism some physiological and morphological plasticity as a means of responding to rapidly changing environmental conditions over time scales shorter than a lifetime rather than directing it down a rigid, predetermined genetic pathway

with no room for deviation (Piersma and Drent 2003). This has led Ridley (2003:174) to refer to plasticity as natural selection's "master stroke" because it increases an organism's chances for survival and reproduction by allowing genes to "flexibly calibrate" themselves and their protein products by responding to unforeseen circumstances. The process of reducing growth rate, however, should not be viewed as a purely adaptive one. A more appropriate concept may be "accommodation," defined as a biological response that favors the survival of the individual but also leads to significant losses in some important functions (Frisancho 1993:7). For example, children with low weight for their age, even at moderate deficits, have a higher risk of mortality, perhaps because of a reduced immunological capacity to stave off infection (Pelletier 1994).

Similarly, a low height for age, or "stunting," is not innocuous or cost free (e.g., see Pelto and Pelto's [1989] critique of the "small-but-healthy" hypothesis). Stunting typically begins in infancy, when growth rates are rapid, nutritional requirements per unit of body weight (particularly protein) are at their highest, and infection as a result of weaning is common. When the nutritional situation is poor, such as during war or in the case of poverty, a smaller body may have lower absolute caloric requirements and be easier to maintain than a larger body, thereby increasing the probability of survival and, ultimately, the opportunity to reproduce (Bateson et al. 2004). However, stunting is also correlated with compromised immunocompetence and cognitive performance as well as decreases in child physical activity (Martorell 1989). The potential for catch-up growth during childhood does exist, but this prospect decreases significantly when stressful conditions are severe and prolonged, as they often are during war (Tanner 1990:128). Thus, malnutrition's effects may last into adulthood and lead to shorter final height, a reduced capacity for physical work, and possibly impaired fertility and higher infant mortality rates (Martorell 1989). Furthermore, these biological outcomes not only are the result of social disruption but also play an integral role in a vicious circle of perpetuating health inequalities and the reproduction of poverty (Leatherman 1998). In this light, it is clear that biological responses to malnutrition are not adaptive in the strictest sense but rather lead to a costly compromise in terms of health.

The Echoes of War: The Enduring Effects of Prenatal Malnutrition

The DOHaD model reveals another important mechanism through which war-related malnutrition has enduring effects on health. Evidence from epidemiological studies and experimental studies on animals demonstrates that prenatal malnutrition may predispose the individual to developing various chronic diseases in adulthood, including non-insulin-dependent (type 2) diabetes, obesity, coronary heart disease, stroke, hypertension, and osteoporosis (Barker 1998; Joseph and Kramer 1996; Langley-Evans 2006). Such effects are not limited to prenatal malnutrition experienced at the level of famine and may be triggered by more modest levels of malnutrition (Jackson 2000) or even psychological stress and an increase in stress hormones experienced by the mother during gestation (Seckl and Meaney 2006). This is the result of a fairly consistent series of physiological responses made by the fetus, though the costs to health may take decades to become fully apparent. Thus, it seems that "earlier environmental influences can have an echo throughout life" (Gluckman and Hanson 2005:19). It has been argued that the DOHaD also may be critically important in reducing the burden of chronic diseases in populations with high poverty rates or who face various forms of discrimination (Kramer et al. 2006; Moore 1998). However, it is the intensity and duration of prenatal malnutrition accompanying war and the resulting "echoes" that are of interest here.

A synthesis of biocultural and evolutionary perspectives helps elucidate why prenatal malnutrition increases the probability for health problems later in life. First, as is true of the child, malnutrition has an adverse effect on the physical growth of the fetus. Data from various countries show that war-related food insecurity coincides with significant negative effects on birth weight, sometimes by as much as 1,100 grams (Keys et al. 1950; Markowitz 1955; Skokić et al. 2006; Steckel 1998; Vlastovsky 1966). Importantly, qualitative differences exist between fetal and child responses to malnutrition. Since the organs of the fetus are still in their initial stages of development, the fetus has a much greater degree of plasticity to modify its metabolism on receiving maternal "signals" regarding the external nutritional environment. If the situation outside the womb is

poor, fetal muscles may become resistant to the hormone insulin, effectively prioritizing scarce amounts of glucose for the brain and red blood cells and leaving the individual better equipped to deal with nutritional deprivation (Hales and Barker 1992). This "thrifty phenotype" is adaptive in that it increases the probability for survival of the fetus during malnutrition, though it may come at the expense of the development of other organs and also lead to a predisposition for non-insulin-dependent diabetes later in life, particularly if the postnatal nutritional environment improves. Prenatal malnutrition may also elevate the risk for other conditions later in life, including obesity and cardiovascular diseases (Barker 1998; Yajnik 2000). Therefore, it seems more appropriate to view these fetal responses as "making the best of a bad start" rather than as adaptations per se (Jones 2005).

Furthermore, it has been argued that in response to malnutrition, a fetus may alter its growth trajectory and physiology not only to avoid immediate negative energy balance but in expectation of the future nutritional environment as well. According to Bateson et al. (2004:420), "The pregnant woman in poor nutritional condition may unwittingly signal to her unborn baby that it is about to enter a harsh world. If so, this 'weather forecast' from the mother's body may result in her baby being born with characteristics, such as a small body and a modified metabolism, that help it to cope with a shortage of food." Gluckman and Hanson (2005) have referred to this idea as the "predictive adaptive response" model, whereby prenatal metabolic adjustments continue to be adaptive into the postnatal period but only when nutritional conditions remain constant. The implication is that intrauterine malnutrition at critical periods of development may "program" physiology and, as a result, have long-term effects on health (Lucas 1998). It is likely that these long-term adjustments to metabolism would have served our hunter-gatherer ancestors well since nutritional conditions would have changed slowly as a result of natural ecological fluctuations.

Over the past few thousand years, however, humans increasingly have gained a greater ability to induce dramatic environmental and social changes on a very brief time scale, as is the case when a high-intensity war commences and ends within the life span of an individual. In a scenario where a fetus experiences nutritional deprivation prenatally, inducing the development of a thrifty phenotype, there exists the potential for a mis-

match should the postnatal social conditions improve and the nutritional situation shift from that of deprivation to one of excess. Although fetal metabolic adaptations may eventually lead to the aforementioned chronic diseases later in life, this is consistent with evolutionary theory and was likely favored by natural selection because genes (in this case those enhancing plasticity in fetal development) that have harmful effects later in life may be favored by selection if they increase the probability of reaching the age of reproduction (Williams 1957).

It is likely that such fetal plasticity was favored by natural selection deep in mammalian history, given that similar patterns have been observed in other species. In addition to humans, prenatal malnutrition has been associated with postnatal insulin resistance, reduced muscle mass (sacrificed in favor of other tissues), and elevated body fat (for enhanced energy storage) in rats, sheep, and guinea pigs (Budge et al. 2005; Kind et al. 2005). With this in mind, it is plain that both genes and environment play a critical role in the development of chronic diseases and that both operate in an inextricably intertwined fashion rather than as oppositional forces (Ridley 2003). Social or environmental factors may create conditions of nutritional deprivation in utero, but the response of the fetus is one that

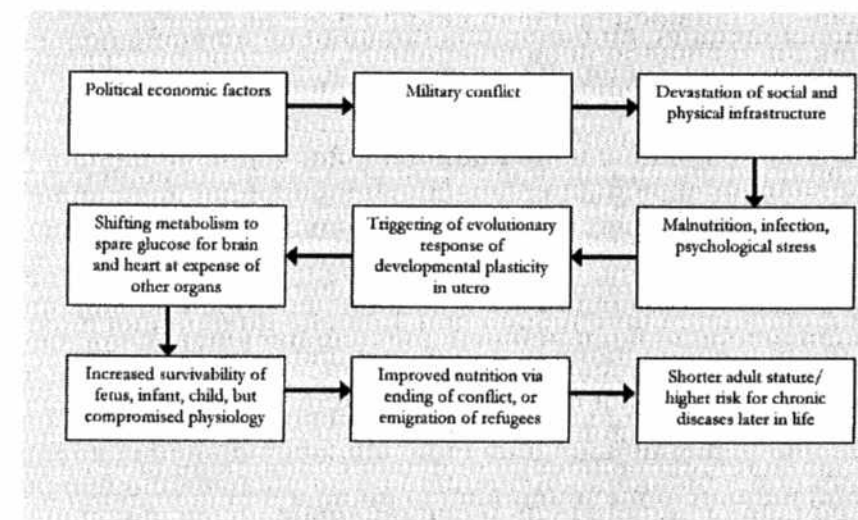


Figure 1.2. Chain of causality illustrating the pathway from war, via prenatal malnutrition and fetal plasticity, to the development of chronic diseases and smaller body size in adulthood.

has been established by natural selection in the distant past. Therefore, it seems appropriate to view the developmental origins of chronic disease as multifactorial, influenced by various levels of causality.

The final sections of this chapter attempt to link different levels of analysis by discussing specific case studies of famine resulting from wars in Europe and Southeast Asia and their long-term effects on adult health.

Famine and World War II

A great deal of research has been conducted on the health effects of prenatal famine in the Netherlands and in the Soviet Union during World War II, both of which resulted from Nazi food blockades on the civilian population. In the case of the Netherlands, the importation of all food and supplies was restricted in the western part of the country (including the six largest cities of Amsterdam, Rotterdam, Utrecht, Leiden, Haarlem, and The Hague) from October 1944 to liberation by Allied forces on May 7, 1945, a period referred to as the "Hunger Winter" (Stein et al. 1975). The blockades were implemented to break a work strike by Dutch railroad workers who had been urged to resist Nazi occupation by the queen-in-exile, Wilhelmina. In November 1944, the rations per person in the famine region were 1,200 calories per day, but this figure had declined to 580 calories by February 1945. After Nazi withdrawal, the nutritional situation quickly improved, with rations rising to over 2,000 calories a day.

The immediate effects of the Hunger Winter were an estimated 20,000 deaths from famine-related causes, a decline in fertility and birth weight, an increase in perinatal mortality, and an increase in birth defects such as spina bifida and anencephaly (Neugebauer et al. 1999; Smith 1947; Susser et al. 1998). Moreover, given the well-delineated historical and geographical parameters of the famine, it was possible to assess its long-term effects on survivors. By using place and date of birth, researchers have been able to determine not only whether a person was exposed to famine prenatally but also the *timing* of exposure. In one of the earlier studies to look at the impact of the famine on adult health, Ravelli et al. (1976) examined the military draft records of more than 300,000 19-year-old Dutch males, 31 percent of whom were exposed to famine either pre- or postnatally. They found that the cohort exposed to famine during the first two trimesters of gestation had the highest rate of obesity by early

adulthood, and a significantly higher rate than unexposed control subjects born outside of the famine area ($p < 0.0005$). Later studies with a smaller sample ($n \sim 740$) reported that the famine had various deleterious effects on health at age 50 years, such as impaired glucose tolerance (a precursor for diabetes), coronary heart disease, hypertension, and higher serum cholesterol (Painter et al. 2005; Roseboom et al. 1999).

Similar patterns were found in Russian adults born during the siege of Leningrad from 1941 to 1944. At that time, food supplies were prevented from reaching the city, leading to the deaths of up to 1 million people (of a population of 2.4 million), mainly from starvation (Stanner et al. 1997). At the height of the siege from November 1941 to February 1942, daily rations provided a meager 300 calories per day, with virtually no protein. In a study of 549 men and women 52 or 53 years old, blood pressure was higher among those exposed to the siege prenatally or in infancy when compared to a control group that was born outside the city limits and thus spared from famine. There was also an interactive effect between prenatal and postnatal conditions in that among women who were prenatally exposed to the Leningrad famine, obesity had a stronger effect on raising blood pressure than in women exposed in infancy or not at all. A larger study of more than 5,000 people found that those who were exposed to the siege as children had higher blood pressure compared to unexposed individuals and that men had higher mortality from ischemic heart disease and stroke (Koupil et al. 2007). This illustrates that both pre- and postnatal conditions can have long-term effects in the programming of cardiovascular diseases, such as during conditions of extreme psychological stress and nutritional deprivation resulting from war.

Wars in Southeast Asia: The Past Is Not Past

The first and second Indochina wars (1945–1954 and 1958–1975, respectively) devastated civilian life in Vietnam, Cambodia, and Laos, with enduring costs to health. Following World War II, France tried to regain control over its former colony in Indochina, which had been disrupted by the war at home and by Japanese military expansion throughout Asia (Stuart-Fox 1997). However, communist-inspired independence movements, in particular the Vietminh and Pathet Lao, mobilized against the French returning as a colonial power. According to LaFeber (2007:36),

the United States hoped that France would be able to “recover its colonial hold and milk Vietnam’s wealth (notably in food products and rubber plantations)” in order to help it regain its strength and be better able to repel communism in Europe. After France’s defeat by the Vietminh in the decisive battle at Dien Bien Phu in May 1954, the United States became more committed to military intervention in the region. This was in the hopes of “containing” communism and preventing its spread from Indochina throughout Asia, which would have meant the loss of potential trading partners, decreased access to raw materials, and significant loss of profits.

The immediate costs of the war, which began in Vietnam but quickly spilled into Laos and eventually Cambodia, were enormous. For the United States, more than 58,000 U.S. military personnel were killed, while roughly 3 million Vietnamese died from war-related causes between 1965 and 1975 alone, representing 7.7 percent of the population (Allukian and Atwood 2000; U.S. National Archives 2007). Additionally, 10 million people were displaced from their homes in South Vietnam alone, while in the North, American air strikes damaged roads and railways, all 29 provincial capitals, and 2,700 of 4,000 villages. In neighboring Laos, of a population of 3 million people, 200,000 died, and 750,000 were made internal refugees (Stuart-Fox 1997), while an estimated 1.7 million Cambodians died during the genocide of 1975–1979 (Kiernan 1996).

Many effects of the war lasted beyond well beyond the official period of conflict. Environmental destruction from conventional bombs and dioxin-containing defoliants such as Agent Orange destroyed rubber tree plantations, mangrove forests, and shrimp and fish stocks in Laos and Vietnam (Allukian and Atwood 2000). These factors, along with unexploded ordnance littering villages and nearby fields, made it difficult to farm both during and after the war (Morikawa 1998). Laos alone was subjected to more than half a million bombing missions that dropped 2.1 million tons of bombs at the cost of \$7.2 billion (Stuart-Fox 1997:144). This calculates as two tons for every inhabitant, giving Laos the infamous distinction of being the most heavily bombed nation, per capita, in the history of warfare.

These historical events certainly became embedded “under the skin” in the people of Vietnam, Cambodia, and Laos. As recently as 1995, blood levels of dioxin were 10 times higher in southern than northern

Vietnamese as a result of the spraying of defoliants decades earlier, thus leading to an elevated risk for various cancers (Schechter et al. 1995). There is also some suggestive evidence that prenatal malnutrition or other early biological insults resulting from the wars in Southeast Asia may have increased the risk for chronic diseases in those who remained in the region or elsewhere in the refugee diaspora. In a study of 490 Cambodian adult refugees in California, 99 percent suffered from “near-death due to starvation” under the Khmer Rouge between 1975 and 1979 (Marshall et al. 2005). This illustrates how widespread hunger was during the period and suggests that the DOHaD model might be applicable to the region. In fact, King et al. (2005) found that rates of diabetes among adults in Cambodia’s Siem Reap and Kampong Cham provinces were much higher than predicted given the rural agricultural lifestyle of the country, leading the authors to speculate that the period of mass starvation under the Khmer Rouge “may have left a grim legacy” (1639).

Thus far, the only studies that have directly tested the DOHaD hypothesis on the effects of war on early malnutrition and later chronic disease in Southeast Asian populations have been among refugees from neighboring Laos, in particular Hmong and Lao living in the United States or French Guiana (Clarkin 2008, 2009). There is consensus among scholars that food insecurity and human suffering in Laos were most intense in certain areas and among certain populations, namely, rural farmers and highland minority groups (including the Hmong) in the northeast and southern parts of the country, while the towns on the western border along the Mekong River were largely protected (Stuart-Fox 1997:139). For instance, infant mortality rates were as high as 50 percent in war-torn areas, while persons displaced from war-zone areas in the northeast were reported to be starving, with protein, caloric, and micronutrient deficiencies (Weldon 1999:125; Yang 1993:51).

Using birthplace as a proxy for prenatal nutrition, Clarkin (2008, 2009) compared the height and body fat percentage of 378 Hmong and Lao adults (mean age 34.3 and 43.5 years, respectively) who were born between 1945 and 1980 in either a war zone or a safe zone. Birthplaces were categorized as “war zone” if nearby conflict (including fighting from guerilla troops, regular ground troops, or American air strikes) likely led to food insecurity in the nine months prior to birth. Retrospective life history interviews revealed that most Hmong were from the northeast of Laos,

while most Lao were from larger towns along the Mekong River, in particular the capital of Vientiane. After cross-checking birthplaces against the historical record, it was determined that 30.5 percent of Hmong were born in a war zone versus just 3.0 percent of Lao. In addition, 38.8 percent of Hmong and 3.0 percent of Lao were displaced from their homes by war during infancy, while the Hmong were displaced from their homes an average of 2.5 times versus 1.0 for the Lao. Finally, the Hmong were also more likely to have a close relative die during their own childhood than the Lao, including a sibling (76.8 vs. 40.4 percent) or a parent (24.2 vs. 11.1 percent). Thus, it was clear that, at least in this relatively small sample, the primarily rural highland Hmong suffered more from the war than did the lowland Lao.

Accordingly, the biological data reveal predicted patterns. After excluding the very small number of Lao who were born in a war zone for purposes of maintaining statistical validity, three groups were compared: 1) Hmong born in a war zone, 2) Hmong born in a safe zone, and 3) Lao born in safe zone. For both sexes, height was greatest among the Lao,

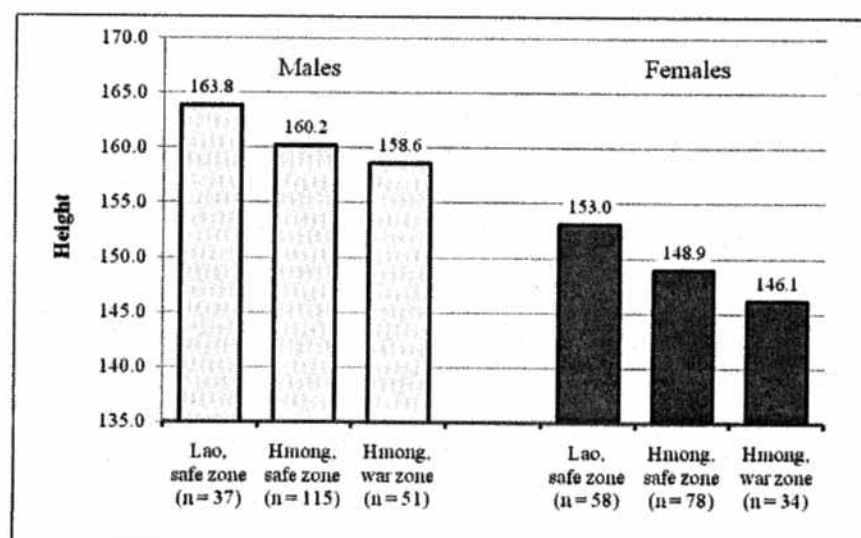


Figure 1.3. Average (mean) height of 373 Hmong and Lao adult refugees by place of birth. Males are represented by light-colored bars, females by dark bars. All Lao adults in this study were born in towns and villages that were generally protected from the war, while the Hmong were more likely to be born in areas in proximity to military conflict. Results indicate that being born in a war zone was associated with significantly shorter adult height ($p = 0.009$).

then Hmong born in safe areas, followed by Hmong born in a war zone ($p < 0.001$).

This was not due solely to prenatal exposure to malnutrition but was likely the result of the Hmong from war zones having more experiences with malnutrition (or other stressful experiences such as infection or psychological stress) throughout infancy and childhood. Conversely, the Lao in this sample were likely more protected from these stresses, while the Hmong born in safe zones may have been spared at least during the prenatal period of life but likely had intermediate exposure to war-related stressors throughout childhood. The opposite pattern was seen with regard to obesity: namely, Hmong born in a war zone had the highest body fat percentage, followed by Hmong in a safe zone and Lao born in a safe zone ($p = 0.019$, after controlling for age and sex). This finding of higher body fat in adulthood, following prenatal exposure to malnutrition, mirrors the pattern found in Ravelli's (1976) Dutch Hunger Winter study. It is also consistent with the "thrifty phenotype" concept, where a fetus modifies its metabolism in order to cope with postnatal nutritional scarcity. After the war, it is likely that the rapid transition away from food insecurity to one of adequacy or even excess after migrating to French Guiana or the United States has led to a mismatch between expected and actual nutritional conditions, increasing the risk for obesity and its concomitant sequelae, such as diabetes and cardiovascular disease.

Conclusion

More than a decade ago, Leatherman and Goodman (1998) proposed that anthropologists, particularly biological anthropologists, focus their efforts at better understanding how social and economic processes affect human biology, which they termed the "biology of poverty." Similarly, the discipline today may be well positioned to address the "biology of warfare" by delineating the ways that military conflict impacts human health in various societies. One might envision greater collaboration between the various subdisciplines of anthropology on the causes and consequences of war, including the study of growth patterns or infectious disease epidemiology within war zones (modern or historical) or retrospective studies testing whether refugees disproportionately suffer from chronic diseases in comparison to nonrefugees. By illustrating the interplay between

evolution, developmental plasticity, and wider social events, biological anthropology can make a unique contribution to the understanding of how warfare contributes to disparities in health. Unfortunately, many opportunities for research exist.

It is possible that dissemination of these findings could cause political leaders considering war a reason to pause, if only briefly, to consider the likelihood that negative health consequences would linger for decades after hostilities have officially ended. In the hierarchy of arguments against war, priority certainly must be given to the immediate loss of life among combatants and civilians. Beyond this, important consideration should be given to the myriad ways that war-induced stressors permanently embed themselves in the biology of survivors of war, often leading to future pathology. This includes not only the more well-known phenomena of physical trauma resulting in injury and psychological distress leading to posttraumatic stress disorder or major depression but also the less cited problem of early malnutrition leading to disruptions in physical growth and development. Such disruptions *predictably* lead to impairments in final adult height, muscle mass, work capacity, immunocompetence, cognitive performance, and fertility as well as elevated risk for chronic diseases in adulthood. Although it could be argued that chronic conditions such as coronary heart disease, stroke, and diabetes are certainly not limited to populations suffering from war or severe malnutrition, the evidence strongly suggests that these greatly exacerbate the problem. Furthermore, chronic diseases are among the leading causes of death worldwide and therefore cannot be considered trivial. Reducing the frequency of war and, by extension, malnutrition is likely to have the side benefit of reducing the global burden of chronic diseases as well as other incalculable benefits to health.

Biological anthropologists (e.g., Thomas 1998) have noted that studies of human adaptability need to make the connections between sociopolitical processes and their downstream biological manifestations more explicit in order to increase the relevance of the subdiscipline. What an adaptability perspective adds to the study of war and health is that it provides an awareness not only that there is a correlation between ecological and economic destruction brought by war and a higher predisposition for chronic disease but also of why that correlation exists. For fundamental biological reasons, developmental plasticity allows the fetus to respond to malnutrition to increase its immediate chances of survival and possibly in

expectation of future nutritional scarcity. However, plasticity has limits, and fetal accommodations are suboptimal compromises that become programmed, or "locked in," and made a permanent part of an individual's physiology. It is also important to remember that plasticity is triggered by environmental events and is intertwined rather than separate from larger social forces. Quite simply, our bodies have memories, and events such as war and political-economic disruption trickle down into our cells, leading to the embodiment of war experiences and elevating risk for adverse health outcomes.

Finally, although it is risky to speak in generalities about war, it is a safe conclusion that, regardless of the motives or politics of the participants, warfare is quite efficient at harming human health. Whether its effects are intentional or incidental, war disrupts every facet of a society to such a predictable degree that this must be considered an integral component of armed conflict. Thus far, there are many studies of war leading to a decline in nutrition and physical growth of children, but only a few examples of war-related prenatal famine leading to chronic disease have been documented (the Netherlands, Leningrad, and Laos). It is likely that wars of the past produced similar long-term health effects, though these may be lost to time. According to the historians Will and Ariel Durant (1968:81), "War is one of the constants of history. . . . In the last 3,421 years of recorded history only 268 have seen no war." The importance of the Durants' calculation is not to suggest that war is inevitable or to dehistoricize it since it is clear that peaceful societies and periods of history do exist (Fry 2006). Rather, it is significant because historically the calculation of health costs of war have rarely gone far beyond the traditional casualty statistics of deaths and injuries and been expanded to include such things as impaired growth and development or an increase in chronic diseases. It is certain that wars' victors have grossly underestimated the damage done by war over human history.

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CHAPTER TWO WAR AND THE PUBLIC HEALTH DISASTER IN IRAQ

Scott Harding and Kathryn Libal

Public health is directly shaped by war, conflict, and capitalism, yet exploring the connections between these processes remains neglected in scholarship and policymaking arenas. Such inquiry is vital to better understand how public health systems can be rebuilt following the cessation of violent conflict, which is critical to promoting individual well-being and supporting families and communities. Anthropologists have much to offer regarding the interconnections between health, war, and political economy. While the ethnography of war and political conflict are topics of lasting interest to anthropologists (Lubkemann 2008; Nordstrom 2004; Schepher-Hughes and Bourgois 2004), medical anthropology has not deeply examined the connections between war and health (Inhorn 2008). In her presidential address to the Society for Medical Anthropology in 2007, Marcia Inhorn signaled “chagrin over medical anthropology’s relative apathy” in the face of ongoing wars in the Middle East (2008:418). She asserts:

As a discipline, we have been faint of heart and lacking moral courage in this arena. In so doing, we have turned away from the brutal realities, the embodied suffering, the psychological devastation, the sexual violence, and the refugee aftermath of war (2008:421–422).

The history of Iraq illustrates that war is not an episodic and limited phenomenon; rather it is often a long-term, structural process with different phases, some more visible than others. Instead of an event