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Dying of Hunger

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Before one can answer the question of mortality rates through hunger, one must first operationalise the term itself.That is to say, there is some confusion in the literature when it comes to the meaning of hunger. Is the concept, for examplesynonymous with starvation? – Or does it perhaps,embrace a wider more inclusive definition encompassing a broader range of clinical malnutritional disorders? Whichever the definition used, one would ultimately expect that mortality attributable to hunger or starvation would be readily identifiable; and quantifiable.However,in reality it is not that easy. The result of all this confusion is the fact thatmortality numbers resulting 'directly' from hunger or famine, have been and still are notoriously problematical to estimate.

The problem is not new either, historically, comorbidityvis-à-vis hunger, disease and malnutrition was not particularly well understood; this resulted in people invariably attributingfood and malnutritionaldeaths simply to the catchall of hunger and malnutrition (Hionidou 2002). Furthermore, poor methodologies as well as errors in reporting have resulted in a number of compounded problems of accounting. Among the many issues(historically) was a general lack of official or unreliable records. This is especiallyso for some of the poorer countries whereby average crude 'mortality' rates, in non-famine years,were often used as the foundation for the basis of mortality calculations in years of famine; i.e. differences being allocated to famine. While this is a crude simplification, it is one that illustrates the point well. All theseinaccuracies though, according to Devereux (2000), has not-surprisingly, resulted in a lack of confidence in historical estimates(Watts 2013).

But what of the present?These days, not too much has changed, and rather attributing 'pure' hunger deaths as a class on its own, it has become customary to talk of mortality due to mal- or undernutrition as–in terms of hunger and hunger-related deaths. However this practice tends to miss the point and still does notdirectly operalisethe important singular concept of mortality due to hunger. Moreover, it does nothing to answer the question of the numbersattributable to 'hunger' deaths. Subsequently, if we were to put aside the term 'hunger' for the moment and substitute it with 'starvation', to represent acute or prolonged and severe absence of nutrients such as vitamins, minerals and overall energy intake, then the definition becomes more readily identifiable. Unfortunately though, the figures attributable to 'starvation'are not. In truth it can be said, death directly from starvation or systemic atrophy is not common. The following demonstrates.

The main cause of death in cases of undernutrition, or instances of famine, is not presently, nor has it ever been through actual starvation (Mokyr and Grada 2002; Vanhaute 2011). This apparent and long-standing paradox caused researchers to rethink the situation of starvation and hunger, and to look elsewhere for alternative or coincidental factors that might interact with malnutrition in mortality (Ó Grada 2008). After much research, widespread consensus now agrees that this dichotomy exists as a result of the interaction between malnutrition and morbidity or disease (Pelletier 1994; FrongilloJr. 1999; Caulfield, Onis et al. 2004; Gibson 2012). What researchers

agreed was the there is a reduction inthe body's immune system under conditions of malnutrition. This means any nutrients that work in harmony to protect against infection (under these conditions are out of balance or simply not bio-available; it is this imbalance that contributes to the onset of disease. This increased susceptibility has been implicated in many,usually non-fatal morbidities, such as dysentery, diarrhea,dropsy (oedema, sometimes spelt 'edema'), respiratory infections (including pneumonia and tuberculosis etc.), and to a lesser extent fever (influenza) as well as other opportunistic diseases like cholera etc. (Watkins and Menken 1985; Hionidou 2002; Mokyr and Grada 2002; Caulfield, Onis et al. 2004; WHO 2010). Of course this works in reverse too, certain diseases such as those considered in secondary Protein Energy Undernutrition (PEU) work against the body in its ability to properly absorb or utilisethe ingested nutrients. What all this means is there is synergistic relationship between hunger, malnutrition, disease and mortality rates (Elamin 2008; Gibson 2012).

The question remains that if hunger is not the main cause of death when considering malnutrition and starvation in particular – then exactly how do people die of hunger or hunger related diseases? Taking starvation in adults to its logical conclusion and depending on a person's existing weight and current condition etc., starvation is more or less fatal between approximately 7 to 12 weeks (Altun, Akansu et al. 2004; Morley 2007). The process itself is more or less straight forward. As the body's metabolic processes convert food into energy, so a total withdrawal of food causes the body to begin to catabolise2 its own stores of energy. This is analogous to the body ingesting itself. Firstly the body requires glucose as one of its main sources of energy. As such the first thing to happen when food is withheld is the body converts the glycogen reserves in the liver and indirectly from the muscles into glucose (via glycogenolysis and gluconeogenesis). In this way during starvation the body first utilises liver glycogen to balance blood sugar (glucose) levels within about 24 hours, after this, muscle glycogen is used. Muscle glycogen cannot be directly converted into glucose, however through glycogenolysis glucose can be secreted into the blood in an attempt to restore balance (Blakemore and Jennett 2001; Ophardt 2003; Gibson 2012). This whole process might take up to a few days. Once the body's glucose is depleted, it then looks to the fatty deposits (the adipose cells). Indeed, for every pound of fat the body stores are equivalent to about 4086 kcal of energy. Thus, body fat depletioncould last for several weeks assuming sufficient water supply and depending on the individual's initial condition.During all this the individual maintains essential bodily functions although, as fat metabolisation continues, so the body generally adaptsto lowered caloric intake by decreasing its basal metabolic rate. Nevertheless, even with the body's response, fat stores ultimately continue to dwindle and the body becomes emaciated (Altun, Akansu et al. 2004). Fat depletion causes the cheeks to appearhollow,while the eyes appear sunken, bones protrude and all the while the skin becomes pallid, dry and inelastic (Thomas 2007).

At this juncture, once all the body's fat is used up, the body initiates the final and catastrophic process of protein catabolism. This process metabolises the body's muscle mass into useable energy. This muscle atrophy is a further attempt by the body to keep vital organs and the immune system functioning properly. However, at this point, without immediate nutrient intake, a person's body becomes counter-productive to his own needs, becoming lethargic and apathetic. By now the heart muscle and other organs (except the brain) begin to shrink whilst the skin begins to hang loosely off the body. Simultaneously, the pulse is already slowing and importantly,blood pressure is dropping. Anaemia,– a situation where the blood lacks enough healthy red blood cells or hemoglobin, commonly develops. This is sometimes accompanied with signs of oedema (extracellular fluid collection); all the while, the sufferer's sense of thirst is impaired. Further malabsorption of nutrients and water often manifests as diarrhea; which may be an ongoing symptom and one that further exacerbates dehydration. All the while concurrent indicators of vitamin and mineral deficiencies (particularly vitamins B and C) might also present;although, by this time the patient is now quite likely suffering psychological and mental disorders. At about the same time, perhaps 7-12 weeks into starvation, if left untreated then death through literal starvation might result directly from multiple organ failure, vascular collapse, severe sepsis or ventricular fibrillation (UdallJr, Bhutta et al. 2002; Altun, Akansu et al.

2004; MedlinePlus 2010).

More likely however, and even in such extreme circumstances, it is well understood that if diarrhea (often associated with extreme starvation) hasn't already become the terminal event, then due to the person's much weakened state, they are far more liable to succumb to associated nutritional comorbidities. These might well include conditions like pneumonia, tuberculosis or some other opportunistic disease long before atrophy or catabolism (starvation) takes its toll (FAO/WHO 1951; Latham 1997; Watts 2013).

Therefore, in answer to the question of how many people die of hunger (in this case - starvation) the truth is: not that many; in fact, rarely do people actually starve to death (Checchi, Gayer et al. 2007; Walker, Bryce et al. 2007; Gibson 2012).

If death by starvation is not the greatest killer of the hungry, it then follows that by studying, the types and causes of malnutrition and their contribution to mortality more closely; a fuller more complete picture would be generated. This would be helpful in thequest of unraveling hunger's co-morbidities and subsequent mortality rates in the overall picture of food insecurity. Commonly however, in literature there is a predilection of solely referring to deficiency disorders, and quite often researchers fail to capture the full picture. This misses the point of malnutrition and serves to reinforce existing perceptions of the one-dimensionality of malnutrition. That said, to be fair, the majority of the disorders of malnutrition are indeed brought on by deficiency rather than excesses. Understanding the overall big picture of hunger, malnutrition and its associated co-morbidities however can help in valuable timely and targeted intervention.

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