Those courageous boys: 73 years after the Minnesota starvation experiment. A psychiatrist's view

And its importance in the understanding of eating disorders and anorexia nervosa in particular

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ABSTRACT

Introduction. At a time when most of the Western world was feeling the hardships of war and the tide was turning toward an Allied advantage, an experiment was conducted with conscientious objectors who volunteered to be starved and renourished in a controlled programme.

Development. Intended as preparation for the refeeding of European populations in countries to be liberated, the Minnesota starvation experiment made a crucial contribution to the understanding of eating disorders, mainly anorexia nervosa, shedding light on both the physiological and the mental processes of the disease.

Conclusions. This study examines the immediate and the often disregarded long-term consequences and compares them to non-experimental eating disorders, analysing the common and divergent aspects, suggesting connections with present advances in neuroanatomical and biochemical eating disorders research, and remembering the volunteers who made it possible.

KEYWORDS

Starvation, anorexia nervosa, eating disorders, World War II, volunteer medical research, medical ethics

Introduction

In late May 1944, only ten days before D-day, a brochure appealing for volunteers for a medical experiment was distributed at conscientious objectors' labour camps across the USA. Over 400 young men applied, with 36 finally being selected; these volunteers could have had little idea of the immense influence of their decision on the study and understanding of human behaviour under starvation, and of eating disorders in particular.

The leaflet, entitled "Will you starve that they be better fed?," alluded to the European population suffering from food shortage and malnourishment as a result of the war, and asked young men to go through a period of controlled starvation followed by refeeding, in preparation for the immense task expected to be found in liberated countries. By this stage, the Second World War was turning in the favour of the Allies, and the US government was concerned about the need to feed the thousands of people either in concentration camps or who were simply going through hard times, when food was often unavailable. With the Normandy landings (June 6, 1944) fast approaching, the issue of designing a refeeding strategy became increasingly important. In fact, that winter would be one of the hardest in some countries. In the Netherlands, where it became known as the *Hongerwinter* ("Hunger

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Winter"), thousands of people died of starvation, with Allied relief not reaching parts of the country still held by the Nazis. The experiment was performed at the Laboratory of Physiological Hygiene at the University of Minnesota, and was conceived by the Laboratory's founder, Dr Ancel Keys, a physiologist who had left the Mayo Foundation to pursue practical field work in Minnesota. Several years earlier, in 1941-1942, he had developed the extremely popular (if not especially loved) ready-to-eat, pocket-portable "K-ration." A decade later, he would also become famous for contributing to the study of cardiovascular risk factors and the Mediterranean diet. He probably followed his own advice, as he lived to over 100 years of age. Many people first learned about the Minnesota experiment through the article published in *Life* magazine in July 1945.[1]

Information was gathered using a Medline and wider Internet search, plus extensive reading on the experiment itself, long-term follow-up reports, and eating disorder research. The article is intended less as an extensive review than as an analysis of the influence of the Minnesota report on the clinical study and understanding of eating disorders.

Development

The experiment: controlled semi-starvation

Keys and his team selected 36 men from over 400 applicants and designed a three-stage programme including a six-month semi-starvation period (beginning February 1945). It consisted of a diet similar to that of war-ridden countries, based on cabbage, potatoes, turnip, beans, and a small amount of pasta or brown bread in two daily meals cut to a half-normal 1500-calorie regime, to achieve a 25% reduction in body weight.

During the first three months, diets for all men were standardised, anthropometric measures taken for each participant, and psychological tests were administered. Next, a six-month starvation period was started, during which regular treadmill walking was added to the reduced caloric intake, while coordination and other psychological parameters were tested. The men were allowed to continue their college studies if they wished, and to leave the centre (in pairs, to prevent cheating). This was followed by a three-month period of refeeding on one of four different diets, designed according to the belief of the times that vitamin supplementation would accelerate their improvement; this was found not to be the case. Nor did protein content have such an effect; simple caloric intake proved to be determinant. The men were kept under medical follow-up, and were offered accommodation at the Laboratory after release from the experiment, as the scientific team were conscious of potential physical and mental sequelae.

The results of the starvation experiment led to a short guidance book, *Men and Hunger,* published in 1946 to assist personnel aiding war-starved populations,[2] and later a full two-volume report entitled *The Biology of Human Starvation* (1950), which is still available from the University of Minnesota Press.[3] The sad paradox is that the experiment was completed too late for its findings to be applied in the liberated countries: semi-starvation took place between 1945 and 1946 and refeeding was completed in October 1946, more than a year after peace was established in Europe on V-day (8 May 1945).

However, more impressive than the facts are the men themselves and their testimony, recorded in personal diaries during the experiment. When it ended they continued with their lives and personal goals; not unsurprisingly, many of them would take war relief posts and afterwards began careers as teachers, priests, or diplomats. They never forgot the experiment (nor, it seems, their ideals); when they were once and again interviewed about those months they provided invaluable information, personal details and even recordings in which they speak with modesty and honesty alike not only about what they went through, but also their motivation: starving so that others could be better fed.

"I wanted to do something that had a little more punch to it. I wanted to risk my life in some way and be of service," explained volunteer Marshall Sutton. "The difference between us and the people we were trying to serve […]: we knew the exact day on which our torture was going to end," stated Sam Legg. "I am proud of what I did. My protruding ribs were my battle scars," said Henry Scholberg. As starvation advanced,

Figure 1. Volunteers Gerald Wilsnack, Marshall Sutton, and Jasper Garner relax in the sun during their daily routine as participants in the Ancel Keys starvation experiment, c.1944. ©Minneapolis Newspaper Collection, Hennepin County Library Special Collections.

belts were adjusted and ankles swollen; they were also aware of the psychological changes it was causing: "[…] food became the one central and only thing really in one's life. And life is pretty dull if that's the only thing," said Harold Blickenstaff. Marshall Sutton explains how "it changed our personalities. We were always apologising to each other for something we didn't mean to do. […] We had periods of elation. Periods of deep depression. And our difficult traits came to the surface" (Figure 1).[4]

The Minnesota experiment and eating disorders: similarities and differences

Keys and others favoured the Quetelet body mass index (BMI) for measuring the relationship between weight and height; it continues to be commonly used for monitoring weight changes today. During starvation, BMI dropped to a range of 14.9-18.6 (mean 16.4), compared to a previous range of 18.4-25.4 (mean 21.9).

Aside from the well-known physical effects (weakness, decreased heart rate, coldness, swollen joints, hair loss, dizziness, and constipation, with hearing being the only improved measure), the main psychological changes observed in the volunteers also occur in eating-disordered patients. These include obsessiveness with food, weight and related activities; food crumbling and rumination; decreased concentration; bad dreams; depression; loss of libido; addiction to chewing gum; and kleptomania. Some men reported viewing people with normal weight as obese, whereas others did not perceive themselves as severely underweight but acknowledged that others were (perceptual distortion).[5] There was a case of

psychosis and another of self-mutilation. During refeeding, seven men also reported concern about the body areas where fat was reappearing.

All these symptoms are to be found in patients with eating disorders, especially restrictive-type anorexia nervosa. The mental crises described above resulted in some drop-outs, including transient psychiatric hospitalisation. Thirty-two of the 36 volunteers completed the experiment.

After eight months of rehabilitation, normal previous weight was restored for the 21 participants followed up, with five showing overweight (considered as BMI > 30 at the time, nowadays lowered to > 24) if not obesity. When free access to food was restored, many volunteers initially lost control over their eating and their sense of satiety, and ate continuously, not stopping despite feeling full, and always had to carry some food with them; six (32%) reported binging, and two vomited involuntarily during this refeeding phase. Some participants asked to stay at the Laboratory premises for some time as a safety measure before returning home. Many anorexic patients show a similar course, progressing to purgative-type anorexia and eventually to bulimia nervosa over the years. Their sense of satiety and hunger recognition often becomes distorted; however, one must account for the influence of these patients' will to ignore hunger stimuli and to continue with the desired restriction.

In psychometric testing, it is worth noticing that although the men's perceived psychological performance worsened, "no objective loss in intellectual ability and no faults in memory or logic were observed."[3] The Minnesota Multiphasic Personality Inventory (MMPI), which was issued a year before the study started and is still widely used by psychiatrists and psychologists today, showed increased scores for what became known as the "neurotic triad" or "semi-starvation neurosis," namely hypochondriasis, hysteria and depression, while scores for psychotic subscales remained within normal limits, except for the cases mentioned above (it should be noted that the MMPI did not include validity scales or corrections at that time; these were added later). For three of the four drop-outs, the MMPI showed increased scores on the psychopathic subscale, congruent with increased anger, aggressiveness and impulsivity, and for the psychotic subscales. Though the latter did not reach abnormal values, scores were consistent with pre-psychotic clinical features in the two men who had to be hospitalised. Female patients with bulimia nervosa show a similar MMPI impulsive/compulsive profile to these cases.[6]

We have addressed the similarities between the starvation experiment and eating disorders. Now, we shall discuss the differences.

First and most obviously, participants' gender. Sex could account for different personality traits and physical endurance.

Secondly, we should mention ideology: all participants were conscientious objectors, with most having pacifist and religious ideals (many were affiliated with pacifist churches, including three Quakers). Age might also have had some effect, as the volunteers' age range was 22 to 33, while eating disorders usually start at younger ages, and tend to increase in prepubertal boys and girls.

We should also consider personality (premorbid personality traits) and inner conflicts. All men were tested and had to be considered mentally healthy to be selected to enter the study. On the other hand, most eating-disordered patients display obsessive, avoidant and/or impulsive personality traits that facilitate the disclosure of the disorder, persist over time, and can also be found in relatives (this point is discussed in greater detail below). Although the feeling of inadequacy and self-sacrifice may be shared to some extent, these men wished not to starve per se, but to help others; therefore, when they could avoid extra physical activity, such as climbing stairs, they all gladly did so; they acknowledged feeling "hungry all the time," and when they were given access to food they wilfully took it; they did not fight against their natural instincts regarding food and exercise as soon as they were allowed to act freely. They wanted to show their commitment, and shared the persistence observed in patients, but they were not deeply dissatisfied with their bodies in the same way as patients. *Eating-disordered patients develop the need for their dietary symptoms from existing inner self-esteem and developmental conflicts*, and fight to

repress their natural feeling of hunger, often becoming both physically and mentally hyperactivated to attenuate self-consciousness and suffering, in the search for an alleged better but ever-distant goal. The Minnesota volunteers laid on the campus grass as soon as they were released from the treadmill routine, whereas anorexic and bulimic patients seek extra physical activity, especially after an imposed meal arousing feelings of guilt and self-deprecating thoughts, capable sometimes of evolving into external voices. The Keys volunteers were not previously especially critical of their own bodies, nor did they *fear* regaining weight (although some did show concern in selective areas during refeeding), a key diagnostic feature for anorexic patients. On the other hand, eating-disordered patients are highly critical about both their physical and mental/personality traits. The Minnesota volunteers' lack of previous obsessive traits, deep self-dissatisfaction (other than feeling different or despised for not wanting to use weapons), and/or impulsive or agressive drives (in a sample composed of pacifist men) represents a crucial difference and explains such a difference in outcomes (prognosis).

The final difference is related to psychopharmacological treatment: today, it is common to use antidepressants, benzodiazepines, and even antipsychotic medication to control anxiety, insomnia, or hyperactivity and to help diminish dysphoria, depressive moods, and obsessive thinking, which biochemical research has linked to serotonin and dopamine imbalances. This is further discussed later in the article.

Long-term consequences and follow-up

The experiment ended and the volunteers returned to their everyday lives. Many took months to recover their normal eating pattern, whereas others took years. A minority took as long as five years to feel that they had regained their normal eating behaviour and control weight previous to the experiment. Ten reported that "their perceptions and perspectives regarding food were permanently altered by the study experience."[7,8] The men's sense of camaraderie and good nature were steadily restored when they were refed. The survivors continued meeting for 50 years, and a 57-year follow-up study has recently been published.[8] Even at the age of 95, volunteer Daniel Peacock said that he would undergo the whole experience again, "providing he was young again." These men believed in what they did, and believed in helping others as long as they did not have to kill. Starvation, on the other hand, was not uncommon at the time, and affected both civilians and military personnel at various times, and not only in concentration camps, as testified by many resistance members and commandos (Dutch troops, Norwegian heavy water saboteurs).

Finally, it is important to note that the ethics underlying the experiment were acceptable only in those hard times, with a world war being fought; the study would not be allowed today. We should acknowledge this as an advance in human rights to preserve human beings from unnecessary suffering; sadly, patients often do not think the same.

Conclusions

The Minnesota experiment provides an accurate description of both the physical and the mental changes taking place in starvation, which also occur during the development of restrictive-type anorexia nervosa, while refeeding events (eg, overeating) are also reported in bulimia nervosa and binge-eating disorder or, from a wider perspective, in non-restrictive phases of eating disorders: impulsivity often lays behind restriction and rigidity, and both anorexia and bulimia frequently interchange clinical features as they evolve, emerging as poles on a symptomatic continuum where rigidity (restriction) and impulsivity (overeating, purging) alternate.

The starvation experiment demonstrated the possibility of empirically inducing eating disorders, which while not exactly the same as those observed in patients, do show a high degree of similarity. Twenty years later, the activity-based anorexia rodent model demonstrated that anorexia nervosa can be induced in mice and rats with restricted access to food simply by giving them free access to running wheels.[9] This results in loss of cerebral volume (mainly reduced astrocytes in the cerebral cortex, hippocampus, and corpus callosum) and reduced serotonin transporter activity and reuptake, and suggests dysregulation of the mesolimbic reward pathways.[10-13] Mice with increased ghrelin (obese or IgG-treated mice), on

the contrary, seem to be at less risk of activity-based anorexia, due to the hormone's orexigenic effect.[14]

Research on eating disorders has advanced since those days, with progress on neuroanatomical, biochemical, and genetic grounds.

Several authors hypothesise serotonergic and dopaminergic system dysregulation in eating disorders, although results are far from conclusive. Serotonin is involved in mood, appetite, and circadian feeding and sleep rhythms; its release stimulates carbohydrate intake and insulin release and causes satiety, turning appetite towards proteins at the next meal. Dopamine intervenes in arousal, reward and motivation, execution, cognitive flexibility, and movement.[15] One of the most relevant studies was carried by Frank et al.,[16] who report reduced serotonin 5-HT2A receptor binding, persisting after refeeding, in patients with anorexia nervosa,[16] pointing to a possible trait-marker. Selective serotonin reuptake inhibitors, which correct the serotonin imbalance, can ameliorate obsessive and depressive symptoms; dopamine-receptor blockade by antipsychotic drugs can contribute to reverting such other symptoms as hyperactivity. Maintained serotonin dysfunction could explain the tendency towards binge-eating and bulimia.

In view of the Minnesota data, we could hypothesise that the volunteers experienced decreased cerebral serotonin levels (which would account for obsessive and depressive symptoms), with less evidence of dopaminergic dysfunction, as neuropsychological performance was preserved and the men reported feeling tired and less alert, rather than overactive, although they did display "periods of elation."[4,5] Maintained restriction and unrestored serotonin imbalance without treatment may explain the transient loss of the sense of satiety, thus predisposing to the binge eating and vomiting reported by some participants during the refeeding stage. Restriction was limited to six months; as neuroanatomical findings in eating disorders are related to disease duration, this would be compatible with an almost complete recovery in most of the volunteers.

Reports from such a collaborative and generous group of volunteers have also provided extraordinarily extensive follow-up over six decades, as well as raising awareness of the long-term effects of starvation (if mild in these cases); however, little detailed information is available on this subject (an area of particular interest to the author). In recent eating disorders research, some studies report lasting impairment of both brain structure and neuropsychological performance (see next point), a highly relevant if not conclusive issue. Neuroanatomical changes are mainly found in fronto-striatal and limbic circuits, although the temporal and parietal cortices, the thalamus, and the amygdala have also been implicated.[17] Frontal lobe atrophy and ventricular enlargement (mainly due to grey matter loss) are the most common findings in patients with anorexia nervosa. White matter also seems to be reduced, although to a lesser extent, attaining full recovery in the medium term (2-5 months after weight restoration).[18-20] While the limbic system is involved in emotional processing, motivation, reward, arousal, and memory and self-preservation instincts (autonomic connections), the frontal lobes and fronto-striatal circuits guide decisions on eating choices; it is therefore logical to consider that the restriction and cognitive rigidity found in anorexia nervosa are related to frontal cortical atrophy, which has been proposed as a maintenance factor for low weight.[21] Reduced temporal lobe volume has been found to correlate with high scores for obsessive symptoms.[22] Refeeding seems only partially to revert this alteration, making these cases more prone to chronicity.[23-25] In functional neuroimaging studies, increased frontal and temporal lobe activity (measured by blood flow) have been observed in low weight and acute phases.[26] This could be related to overactivation, rigidity and difficulties in decision-making when subjects are submitted to changing paradigms (poor set-shifting ability). Some PET studies have found brain glucose hypometabolism, with relative hyperactivity in the caudate nuclei, which is involved in the reward circuitry.[27] It is likely that the Minnesota volunteers did not particularly experience such effects, as starvation was restricted to six months, although it is also possible that the psychometric measures used were focused not on perception and flexibility but on executive performance and speed.

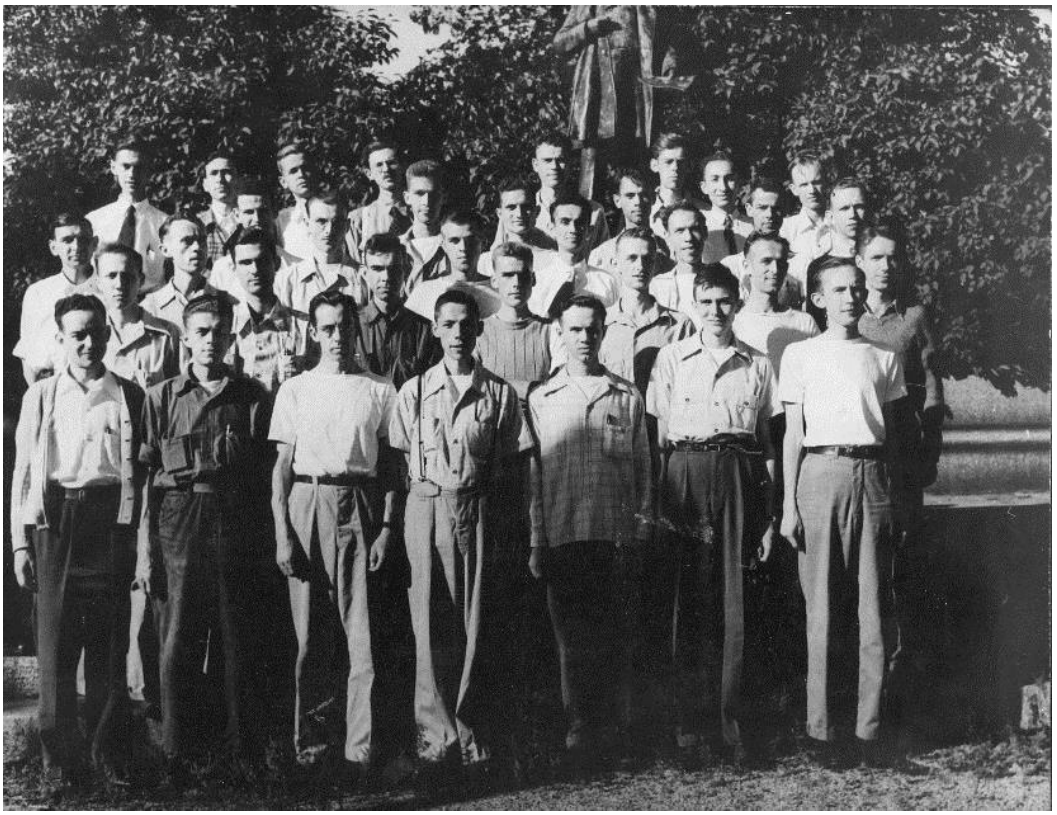

Figure 2. Those courageous boys. ©Wallace Kirkland. The LIFE Picture Collection. Getty Images

Neither do we have an idea of a possible family linkage for developing symptoms, or premorbid predisposing traits in the study volunteers, which are not covered by Keys team, as have since been reported in studies on eating-disordered patients regarding personality traits, cognitive style, and to a lesser extent, biochemical and genetic findings.[28,29] Identifying *trait* and *state markers* is one of the most engaging fields in eating disorders research, prevention and treatment design.

However, we can identify determinant differences between the Minnesota volunteers and eating-disordered patients that explain the diverging outcomes. Most volunteers recovered in the medium term with no reported relapse. On the contrary, and depending upon various factors (eg, illness onset/duration and treatment strategies), a high relapse rate has been reported in patients with eating disorders, reaching 50% in anorexia nervosa.[30] When the study concluded, these men had fulfilled their goal, whereas patients do not, and continue to be in need of symptomatic behaviours.

The experiment also proved, among many other issues, that starvation causes mental changes and that refeeding and dietary normalisation are necessary to revert these changes and achieve psychological improvement. We should also take into account that this is the best treatment both in states of starvation and eating disorders.

The study also provides a limited exploration of the processes of mind that may lead to the development of psychoses.[31]

Performance in the various psychological tests administered at the time showed no significant impairment overall, although the MMPI did reflect neurotic and pre-psychotic changes. This could be explained by the relatively brief duration of starvation as compared to eating-disordered patients. Findings from more recent studies on neuropsychological profiles identify specific changes in perception and executive functions: impaired mental flexibility in set-shifting tasks and general decision-making, and poor global perspective with over-attention to detail in visual perceptive tasks (weaker central coherence), showing a correlation with BMI.[32,33] These changes become more evident with longer illness duration, and persist after refeeding and weight normalisation.[28] Some of these findings have been replicated in first degree relatives.[28,34,35] The brain's processing of rewarding and distasteful (aversive) stimuli, as well as face perception and judgement, also appear to be altered in patients with anorexia nervosa, who show better verbal than non-verbal performance and decreased learning from reinforcement compared to healthy controls, both before and after weight gain.[36-38] That is, they present a rigid cognitive style and emotional problems, with frequent difficulties in identifying and expressing their emotions (alexithymia); however, other studies have failed to find specific impairments in anorexic patients.[39,40] The Minnesota volunteers were well aware of the negative emotions induced by starvation and of the difficulty of managing them, even if their good nature meant that they were "always apologising to one another" for things they did not mean to say or do.

While neuroanatomical, biochemical and genetic studies have taken a central role with time and scientific progress, the starvation experiment remains an invaluable source of information for clinical descriptions, including the subjective experiences and thoughts of the men who valiantly and consciously volunteered. While it was completed too late to succeed in its initial goal of designing refeeding strategies, it did dispel some false beliefs regarding diet and improve understanding and awareness of the development of eating disorders. It should be remembered as having truly helped others by shedding light on the physical and mental processes of starvation and eating disorders (Figure 2).

Conflicts of interest

The author has no conflicts of interest to declare.

To the memory of Lew Ayres.

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