"The True Fiasco:
Efforts to Combat Protein Malnutrition in Uganda and the World, 1950-1974"

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With the 1974 publication of "The Great Protein Fiasco," more than two decades of research on protein malnutrition as "the most serious and widespread problem in the world" became the one of the greatest embarrassments of nutritional science and international public health. The global emphasis upon protein malnutrition quickly evaporated and more recent efforts to mitigate severe childhood malnutrition have been left with little if any knowledge of prior research and programming. As an extension of a larger historical investigation of nutritional science and applied nutritional programs in Uganda, the proposed paper examines the scientific research underlying initiatives to combat severe protein malnutrition. While many interpreted the "Great Protein Fiasco" as an indictment of the biomedical science implicating protein, I argue that it was the interpretation of that evidence in the international arena that "The Great Protein Fiasco" called into question. Revisiting the assessment that the scientists got it wrong allows for a more accurate appraisal of how "erroneous worldwide generalizations" together with narrow definitions of the problem shaped an extensive and expensive international public health program that aimed to close the so-called "worldwide protein-gap," with significant unintended consequences. Through an examination of WHO, FAO and UNICEF reports, scientific publications and oral and archival evidence gathered in Uganda and the UK, the local and the international are kept in a single frame in order to recast "The Great Protein Fiasco" as a cautionary tale emerging from a formative period in the growth of global science and medicine.

Introduction

In 1974, Dr. Donald S. McLaren published an article in *The Lancet* entitled "The Great Protein Fiasco."[1] His critique of the undo emphasis placed upon protein malnutrition by international agencies linked to the United Nations sparked heated debate and controversy. Opposition to McLaren's attack of the widely held view that there was an "impending protein crisis" of global proportions stemmed from sectors with significant stakes in the establishment and from a critical misunderstanding of his argument. Rather than a "single-handed protest," McLaren merely made public the growing conviction that undernutrition or marasmus was far more prevalent than protein deficiency or kwashiorkor and that the international preoccupation with protein had been a grave mistake.[2] McLaren's article was therefore seminal to the subsequent and decisive shift away from protein malnutrition as a predominant focus of international endeavors to improve nutritional health. What McLaren exposed was the crucial importance of accurately assessing public health problems prior to the deployment of limited resources. While many interpreted the "Protein Fiasco" as an indictment of the biomedical science implicating protein, it was the interpretation of that evidence in the international arena that McLaren's critique called into question. The interpretive error, as McLaren has repeatedly argued, was "the extrapolation by others of the atypical experience in Africa to the rest of the developing world."[3] Revisiting the "Protein Fiasco" reveals that in the period following the Second World War, notions of a global "protein gap" or "crisis" contributed to crystallizing conceptions of the "developing" or "Third World." As the former "tropics" became "developing" or "Third World" regions, the science of nutrition replaced the paradigms of tropical medicine. As I seek to demonstrate in this essay, however, the central tenets of tropical medicine remained intact and their reformulation in the universal language of nutritional science suggests the limits of univeralism as part of twentieth-century efforts to secure global health and wellbeing.

The idea of the 'tropics' took shape in the context of European exploration and imperialism from the seventeenth through the early twentieth century. What began as positive perceptions of tropical wealth and exuberance ultimately gave way to negative views of the 'tropics' as dangerous and primitive places incompatible with the civilization of temperate climes. Drawing upon the importance of heat in the Hippocratic tradition, the hot and humid climate of the 'tropics' became closely associated with illness and disease.[4] The rise of tropical medicine as a disciplinary specialization served to underscore the idea that a close relationship existed between climate and disease thereby casting the 'tropics' in a particularly negative light. Thus, at the heart of tropical medicine was a very firm belief in environmental determinism. As an integral component of the imperial project, tropical medicine and negative views of the 'tropics' were also critical to growing ideas of racial difference and the hardening boundaries between Europeans and their colonial subjects.[5] The very high rates of illness and mortality experienced by European merchants, soldiers and explorers, particularly in West Africa during the era of the slave trade gave rise to ideas of racial immunity in which 'tropical races' were predisposed to labor in hot and humid conditions.[6] In inextricably linking race, place and disease, tropical medicine became a powerful expression of the racial and cultural superiority that Europeans believed justified their conquest of the supposedly primitive and 'backward' people of the 'tropics.' Like Christianity and 'civilization,' tropical medicine was viewed as part of the advanced science and technology that aided in the uplift of colonial populations, particularly with the advent of germ theory and the renewed imperialism of the late nineteenth-century.[7]

In addition to underpinning colonization and notions of biological race, the environmental determinism of tropical medicine meant that tropical diseases were by definition caused by 'natural' as opposed to socio-economic and political forces. As a result, the discipline of tropical medicine concealed the fact that many of the most prevalent forms of illness in 'tropical' and 'sub-tropical'

regions were products of poverty and poor sanitation. Combined with the focus upon vector-borne diseases like malaria, yellow fever and sleeping sickness, tropical medicine tends therefore to favor "disease-specific" campaigns or "vertical" rather than "horizontal" programs that aim to improve general health and wellbeing.[8] In the context of a colonial medical framework dominated by the discipline of tropical medicine, the growing interest in African nutritional health from the inter-war period onward was a potential watershed in the history of colonial medicine. Not only did malnutrition directly implicate poverty and the political economy of ill-health, but the universal physiology and biochemistry of nutritional science was inherently free from the racial and cultural presuppositions so central to tropical medicine. However, as several historians have shown, this potential turning point was effectively foreclosed when malnutrition was defined as a problem of African 'ignorance' and 'backwardness.' The 'betterment' policies that were then implemented focused upon improvements in agricultural production and the preservation and preparation of food in Africa.[9] As Michael Worboys explains, attributing malnutrition to "inadequate knowledge . . . allowed evidence of the recent origins of the problem to be ignored. Colonial malnutrition was rapidly and readily reconstructed from . . . an epidemic problem to an endemic one, for which colonialism had little responsibility and over which it could exercise little control."[10]

The following analysis traces the origins of the "Great Protien Fiasco" in order to broaden the critique initially launched by McLaren in 1974. Thus while McLaren was rightly concerned with the worldwide prevalence of protein malnutrition relative to what he and others argued was a far greater problem of insufficient calories or undernutrition, my aim is to consider how the problem of protein malnutrition was defined. I begin by documenting why protein became the focus of nutritional research in Uganda in order to highlight the high mortality rates associated with severe protein deficiency and the role of effective therapeutic measures in making protein the focus of international attention. I then examine how biomedical practitioners in Uganda interpreted protein

malnutrition as an endemic problem caused by the inadequacies of the African environment as well as the ignorance of African mothers and their backward cultural practices. As Worboys suggested twenty years ago, this interpretation made poor nutritional health a static feature of African culture and the natural environment rather than a result of colonial impoverishment. Conceptions of environmental determinism, cultural backwardness and racial difference remained pervasive particularly as they became obscured by the universal language of nutritional science. As protein malnutrition in Uganda was transformed into a global "protein gap" or "crisis," these conceptions of the problem became "erroneous worldwide generalisations," to borrow McLaren's terminology.[11] More than an issue of undo emphasis, this analysis of the "protein fiasco" suggests that nutritional science played a fundamental part in shaping "erroneous" perceptions of the "developing" world. According to David Arnold, "the dark side of the tropics that once haunted naturalists, novelists, geographers and physicians has been shunted off on to our notions of the Third World," and it is this that constitutes the most problematic and enduring impact of "The Great Protein Fiasco."[12]

Protein Malnutrition & Uganda as an International Center of Nutritional Research

Despite common perceptions that Africa has always been the recipient of scientific knowledge produced elsewhere, reconsidering the "Great Protein Fiasco" requires that we first invert the paradigm and focus upon the critical contributions research centers in Africa have made to biomedical knowledge and practice around the world. In the case of protein malnutrition, biomedical practitioners working in East, West and South Africa were instrumental to advancements in kwashiorkor research over a period spanning more than forty years. The story of how Uganda became an international center of nutritional research begins, not in Uganda itself, nor in the British metropole, but in the capital of present-day Ghana. Moreover, as Donald McLaren pointed out in his famous critique of the "Protein Fiasco," "it took the appointment of the first woman medical

officer to the Gold Coast in the person of Cicely Williams to give us the first substantial description in 1932 of 'a deficiency disease of infants' in which "some amino acid or protein deficiency cannot be excluded'."[13] Williams' protein hypothesis quickly came under direct attack by the prevailing experts in the still new science of nutrition who held firm to the view that the condition was merely a form of the vitamin-B deficiency known as pellagra.[14] The very idea that children in a British colony suffered from a diet deficient in one of the basic food groups, rather than a newly discovered vitamin or mineral, not only challenged the accepted diagnosis but was "politically objectionable" and may have been a factor in Williams' subsequent transfer to Malaysia.[15] Not easily discouraged, Williams' remained an ardent advocate of the protein hypothesis throughout her life and particularly in her role as the first Head of Maternal and Child Health at the World Health Organization (WHO) following her internment during World War II.

Nearly two decades elapsed before Williams' protein hypothesis became the accepted wisdom and the intervening period was marked by much controversy and debate. At the center of disputes over the protein etiology was the therapeutic failure of dietary protein. In fact, the inability to treat severely malnourished children with protein rich food served to disprove not only the protein theory, but the very notion that kwashiorkor was a form of malnutrition altogether. Despite the failure of dietary therapy, a number of biomedical practitioners continued to suspect that malnutrition played a role in the etiology of the condition, and in the 1930s and 1940s they fed severely malnourished children brought to hospitals around the world a variety of dietary mixtures in desperate attempts to save their lives. When vitamin therapies proved ineffective, they experimented with other components of a nutritious diet. First, on an extensive list that included liver extracts, hog's stomach, brewer's yeast and enzyme-digested whale meat, was cows milk. Long considered an ideal form of protein for young children, the curative failure of milk was particularly problematic for proponents of the protein theory.

What the early controversy over the protein hypothesis highlights is the significance of therapeutic efficacy to conceptions of etiology. In part, the close relationship between ideas of causation and cure are integral to the logic of biomedical science. The point that must be stressed in this instance, however, is the fact that what motivated biomedical practitioners in their ongoing endeavor to determine the etiology of the condition was the extremely high mortality rates that they observed in their pediatric wards. The physician who became one of the key proponents of the protein theory, Dr. Hugh Trowell, reported that while posted to the hospital in Nairobi in the early 1930s he lost more than three-quarters of his kwashiorkor patients and the WHO cited mortality rates in Africa of up to 90 percent as late as 1950.[16] The helplessness and frustration that came with the inability to alleviate the suffering and save the lives of these young children is palpable in the period prior to the development of effective therapeutic measures. As Trowell, who had been searching for a cure for over a decade, explained, "Still we were losing 40% of our cases in hospital. And you expect a person to get better if you are giving all the known vitamins, and protein, and we were still having this enormous mortality."[17] Such sentiments were exacerbated by the failure of the vitamin therapies recommended by the nutritional scientists based in the metropole. When a bottle of the newly discovered nicotinic acid was flown to Nairobi from the United States in the mid-1930s, Trowell, "went down to meet the plane," hopeful that the new B-vitamin "was going to cure all the pellagra." [18] Of the ten children that Trowell tried to treat with niacin, "eight died," and in his own words he recalled, "I could have wept."[19] Trowell pointed to the pivotal role of curative efficacy when he admitted regretfully, "If we'd stuck to protein we'd have done better."[20]

Following so many years of therapeutic failure when a breakthrough was finally made, shortly after the end of the Second World War, the reverberations were felt far and wide. The breakthrough in question had to await the arrival of a new pathologist, Dr. Jack Davies. Like Trowell, Davies joined the staff at the Makerere Medical School on Mulago Hill due to his combined

interest in both African medical training and biomedical research. The remarkable standard of medical training achieved at the medical school was a key component of Makerere's advancement to university status and as Makerere became the center of knowledge and learning serving all of East Africa, the Makerere-Mulago medical complex became a focal point of ongoing biomedical research, beginning with Trowell's early work on kwashiorkor.[21] Unlike his predecessor, Davies agreed to conduct thorough autopsies on Trowell's severely malnourished patients. These autopsies quickly revealed that in children who died of kwashiorkor, the pancreas had stopped secreting digestive enzymes. This discovery provided the long awaited pathological evidence substantiating the protein theory, as enzyme synthesis is dependent upon a supply of protein. Even more importantly, however, the pancreatic atrophy explained why children suffering from severe malnutrition could not be easily cured through food alone. Without sufficient protein for the production of the digestive enzymes needed to breakdown and absorb essential nutrients, even protein rich foods like milk were of limited therapeutic value. Although they were finally armed with an explanation for years of therapeutic failure, skepticism of the protein etiology continued as it took several years to devise an effective form of treatment. The delay, as Davies explained was due to the fact that they, "had no special high protein material to feed the children."[22]

The development of a high protein formula suitable for children suffering from acute protein deficiency had to await the arrival of a "giant in the field of nutritional science," Dr. Rex Dean.[23] As a leading member of Professor R.A. McCance and Dr. Elsie Widdowson's team in the Medical Research Council's (MRC) Department of Experimental Medicine at Cambridge University, Dean was at the forefront of British nutritional science.[24] The aim of Dean's work in Uganda was to build upon the success he achieved alleviating undernutrition in German schoolchildren and children who had been orphaned during the Second World War. In Germany, Dean found that it was possible to observe excellent rates of growth using mixtures of plant proteins "rivaling milk in

nutritive value"—even apparently in children between the ages of six months and two years, "an age group which" as Dean pointed out, "is particularly difficult to feed well without milk."[25] For Dean to continue his research on vegetable-protein substitutes required a population suffering from chronic malnutrition as well as the laboratory facilities and infrastructure required for complex biochemical analysis—at the time the Makerere-Mulago medical complex was an obvious choice. In the context of severe protein malnutrition in Uganda, Dean's work had a clear application and Dean was sent to the Protectorate in order to apply his knowledge and expertise toward the future prevention of kwashiorkor in Africa. Yet, "on arrival in Uganda one clinical issue that disturbed Dean greatly was the high mortality in cases of kwashiorkor and marasmus that seemed acceptable in the hospital wards."[26] And while he never lost sight of his goal to prevent malnutrition, Dean's major contribution to applied nutritional science was the development of a highly effective curative therapy for kwashiorkor. Ironically, Dean's "High Protein Therapy" was a milk-based formula that mixtures of vegetable proteins could never rival.

Dean turned to milk, in spite of his original mandate, simply because the most inexpensive and accessible source of protein turned out to be a waste product in the manufacture of butter in Europe and the United States. As Dean's successor, Roger Whitehead explained, "dried skimmed milk was readily available as an aid food, but in the 1950s few knew how the high-quality protein could best be used."[27] According to Whitehead, skim milk powder "just got dumped. The World Food Program used to put it at the frontier and the Uganda Government would then ship it in."[28] At the time, skim milk was valued for more than its low cost and ready supply, however. In addition to delivering almost twice as much protein as the full cream variety, the successful results achieved with skimmed milk led many to surmise that children suffering from kwashiorkor were unable to digest fat.[29] Yet Dean found that even skim milk was often poorly tolerated. A simple mixture of dried skimmed milk and glucose proved highly effective in alleviating many of the most egregious

symptoms, including the edema, anorexia, apathy and skin rash, but very often led to an extremely dangerous side-effect in already severely malnourished children—diarrhea. In one twelve-month old Ganda child, for instance, the loose stools that developed in response to the therapy led to such dangerous levels of weight loss that her treatment had to be stopped. Although she later made a full recovery, Dean cited her case as indicative of the fact that even skimmed milk fell short of the desired therapeutic outcome.[30]

The solution that Dean devised in order to further perfect the skim-milk formula served to further substantiate the now widely held belief that the condition was caused by protein malnutrition. As Dean quickly discovered the digestive upset was easily avoided by reducing the amount of skim milk in the mixture during the initial stage of treatment. In its place, Dean supplemented the formula with Casilin, a commercially produced preparation of calcium caseinate containing an eighty percent concentration of milk protein. Despite the added cost, this new therapeutic formula was a resounding success that was interpreted at the time as evidence that children suffering from kwashiorkor were so protein deficient they were unable to digest carbohydrates. More than a decade passed before scientists discovered that the diarrhea was actually due to lactose-intolerance, lowering the amount of skim milk in the formula had inadvertently lowered the level of lactose.[31] In the meantime, Dean focused upon finding an alternative means of augmenting the formula with a more palatable source of calories and by 1956 Dean and his colleagues were reporting that their efforts to further perfect the High Protein formula had met with stunning success.[32]

Success, measured in terms of therapeutic outcome, was translated as direct evidence in support of Williams' original hypothesis. In an article published in 1952, Dean wrote, "The mixture of skimmed milk and concentrated milk protein was the most consistently successful of the various diets which had been tried at Mulago Hospital. It has been used in about 70 cases, often in the most

difficult circumstances, and often when treatment with the skimmed milk alone has proved unsatisfactory. . . . the introduction of the concentrated milk-protein diets has probably halved the death rate from kwashiorkor at Mulago.[33] Writing after working in Uganda for less than two years, Dean's figures underestimated the efficacy of his "High Protein Therapy;" the ten to twenty percent mortality rate consistently achieved through Dean's therapeutic regimen from 1952 onward constituted a reduction of at least half of the 40 to 90 percent mortality observed not five years earlier.[34] By 1954, Trowell, Davies and Dean could claim that, "any very high rate, such as that of 35 percent . . . should now suggest a failure of therapeutics."[35] Biochemical measures of recovery and rehabilitation were equally compelling. The total levels of protein found in the blood, for instance, doubled within one week of treatment and reached expected levels for healthy children around the third week.[36]

Yet, mortality rates and serum protein levels only reveal part of the story. The phenomenal pace of recovery, particularly in the initial phase of treatment, had an equal, if not greater impact upon perceptions of the condition and its underlying cause. Even in very severe cases nearly all of the most pronounced symptoms disappeared or began to improve within seven to ten days. The anorexia that often made intragastric tube feeding necessary began to subside so rapidly that, as Dean pointed out, "it was unusual to have to continue tube-feeding for more than two days." In fact, Dean wrote, "the return of appetite was one of the most clear signs of the beginning of recovery. The loss of the oedema was also prompt, and the skin lesions, however extensive they might be, had always begun to heal by the end of the week. About the same time the demeanour of the child began to change; it lost its apathy and started to take an interest in its surroundings."[37] Although a full recovery involved the resumption of weight gain and growth at rates that would facilitate the "catch-up" required for a stunted child to reach the weight and height considered "standard" for her age, only in exceptional cases was it possible to keep a child in the hospital long

enough to observe this final phase of rehabilitation.[38] The few children who were treated for extended periods, however, demonstrated rates of accelerated growth capable of eventually reversing their stunted stature. For example, a child named Bandiho weighed five kilograms below the American standard when she began her therapy, but grew three and a half times more quickly than considered "normal" and began to approach the "standard" weight for her age after a year of hospital treatment.[39] Given the excessive mortality associated with kwashiorkor prior to Dean's "High Protein Therapy," Whitehead later wrote that, "Dean was indeed a pioneer. His clinical and nutritional methods for the therapy of kwashiorkor and marasmus have had an impact throughout the third world."[40]

Defining the Problem of Protein Malnutrition in Uganda

In addition to effective therapeutic measures, biomedical scientists working in Uganda also made critical contributions to worldwide perceptions of protein malnutrition. With the etiology more or less established, Dean and his team at the MRC Malnutrition Research Unit that he established on Mulago Hill in 1953, turned their attention to prevention. Successful implementation of preventive measures entailed first establishing the sequence of events that "typically" led to the development of kwashiorkor –or the "natural history of the disease."[41] The feature of kwashiorkor's "natural history" that was taken to be the most revealing was the average age of incidence. In Uganda, the incidence pattern was so striking and consistent that kwashiorkor became exclusively associated with children in the weaning phase of life. Figures collected in the 1950s, by several different biomedical practitioners working in Uganda indicated that more than 85 percent of children suffering from the condition were between the ages of six months and three years.[42] In fact, the evidence was so clear that the difficulty of ascertaining the exact age of a child was considered unimportant. A great deal was concluded from the fact that the severe manifestations of the

syndrome occurred within a narrow window of time in a young child's life. Evidence that acute kwashiorkor was exceptional in infants under six months old was attributed to the "efficiency" of breastfeeding and it was frequently observed that exclusively breastfed infants in Buganda exhibited "excellent growth" and development.[43] All of that changed, however, at the age of six months, when breast milk was no longer sufficient as the sole source of nourishment. Explanations of the subsequent decline in the nutritional health of Ganda children centered upon the food onto which children were weaned and the culture of weaning in the south-central kingdom of Buganda.

The fundamental issue, according to biomedical perspectives, was that children in Buganda were weaned onto foods that contained very little protein. The predominant staples of Ganda cuisine are the green cooking bananas or plantains, known as *matooke*, followed by sweet potatoes and cassava. As it turns out, cassava, sweet potatoes and plantains contain very little protein particularly as compared to other staples including millet, sorghum and wheat. The very low ratio of protein to calories meant that the "sheer size of a meal that might have adequate" nutrients was far too large for the small stomach of a very young child.[44] As in many African societies the definition of food in Buganda was synonymous with the primary staple; *matooke* best fulfilled the purposes of eating, which were to satisfy hunger and fill the stomach. Yet, what most troubled biomedical personnel was the cultural importance of *matooke* in Buganda. In a statement revealing a sense of exasperation, the MRC Unit's nutritionist, Ingrid Rutishauser wrote, "everything about '*matoke*' is ritual of an intensity that is almost religious."[45] Dean, in his first treatise on kwashiorkor after arriving in Uganda, concluded that the problem of protein malnutrition "can be explained by a fondness for matoke."[46]

The cultural centrality of *matooke* was only the tip of the iceberg, as a wide range of customary practices and cultural beliefs came under intense scrutiny. First and foremost, parents in Buganda were faulted for not ensuring that their young children consumed sufficient quantities of

protein. Ignorance of the unique nutritional needs of young children was the most frequently cited reason that children were not explicitly fed the high protein components of the diet. Cultural taboos proscribing certain foods for women, did not officially apply to children, but in practice meant that most of the meat, fish and eggs were reserved for men.[47] Rutishauser was especially critical of the diet prepared for children once they had been weaned. "It is the custom," she wrote, "for young children after weaning to have the same foods as the adults in the family, the idea of preparing food specially for the children being completely alien to the Baganda."[48] The "adult" diet was considered inappropriate for the needs of very young children because the high-protein relishes and sauces were not offered to young children in a manner guaranteeing their consumption. The problem was that children in Buganda were not actively "fed" and were not considered physically capable of consuming adult food in the customary fashion. Protein deficiency in Buganda was understood, therefore, to be at least partly due to the way in which food was consumed, using the hands to dip an edible portion of the steamed *matooke* into the sauce or relish. Yet, the manner in which a meal was served was also criticized and while it was noted that the practice of serving individual portions was becoming increasingly common, especially for the father or the head of the household, the customary practice of sharing food was viewed as yet another factor limiting the amount of protein consumed by young children in Buganda.

In addition to scrutinizing the cultural beliefs and practices surrounding food in Buganda, biomedical practitioners were equally critical of weaning and child-rearing practices more generally. Chief among them was the practice of sending a child to live with a member of the father's clan as soon as the child had been weaned.[49] As part of the naming ceremony through which children became official members of their father's clan, the practice also clearly served to cement the kinship ties that structured Buganda's patriarchal clan-based society. Yet, rather than condemn the patriarchal purview of the clan, the biomedical community blamed Ganda mothers who they

portrayed as insensitive and indifferent to the health and welfare of their children. Mothers were criticized for arbitrarily choosing the date to cease breastfeeding and the subsequent change in their attitude towards their children. As the psychologist Dr. Marcelle Geber explained, "The mother does not only stop giving him the breast, but often behaves as though she is deliberately trying to effect a separation"[50] "she no longer carries him on her back, or sleeps with him, no longer consoles him, but laughs at him if he cries or merely tells him to stop crying."[51] The "maternal deprivation" that a newly weaned child supposedly felt as a result of the "abrupt" weaning practices in Buganda was thought to contribute to the anorexia of malnourished children –a refusal to eat might, some hypothesized, be an attempt to express the distress of the sudden separation from the mother. According to a number of the doctors and scientists, weaning practices in Buganda constituted a psychological upset that precipitated the onset of severe protein malnutrition.

Whether the problem of protein malnutrition in Buganda was the result of weaning practices, customary ways of serving and consuming food or the high value placed upon *matooke*, protein deficiency was clearly understood as an endemic problem. This perception was reinforced by evidence that a mild form of protein deficiency was pervasive in the region. X-rays taken of the knee and hand bones of children with kwashiorkor demonstrated that bone development had been arrested long before the onset of their condition, suggesting that a mild form of malnutrition preceded the acute stage of the syndrome.[52] Biochemical measures provided further evidence that this was the case. For example, urea is released when the body breaks down or synthesizes protein and as expected very low levels of urea were found in the blood of severely malnourished children. What Dean and his colleagues were not expecting was that the levels of urea rose so far above the standards previously observed that it appeared the children undergoing kwashiorkor therapy were breaking down protein in order to build body proteins at levels that had never been seen before.[53]

Similar results found by a team conducing nitrogen balance studies indicated that protein

malnutrition was a widespread problem affecting large segments of the population. Like urea levels, nitrogen balance studies allow scientists to quantify the amount of protein used by the body. Nitrogen retention is only expected during a period of substantial growth, pregnancy, recovery from an illness or following protein starvation. The fact that all of the adult male subjects of the study retained nitrogen for the entire duration, which in some cases was as long as 170 days, initially indicated an error in the methodology. When the possibility of systematic error (and severe liver damage) was ruled out, however, the combination of nitrogen retention, weight gain and the loss of intercellular fluid associated with edema left only one possible conclusion:

> in spite of the prolonged high protein feeding, we never succeeded in "saturating" our subjects with protein; put in another way, given sufficient protein, they were still able to form new body tissues for as long as we were able to observe them. . . . If our subjects were indeed capable of protein synthesis to the degree observed, the most obvious explanation is that when treatment was begun they had suffered from very severe and prolonged protein deficiency, lasting perhaps from the time of weaning.[54]

Data collected from presumably healthy and relatively well-off children in order to establish "normal standards" of growth was interpreted as clear evidence of an endemic shortage of dietary protein in Buganda. These growth charts demonstrated that the height and weight of Ganda infants compared favorably with their American counterparts through the first four to five months of life. By the age of six months, however, both indicators of nutritional health dropped dramatically to well below the tenth-percentile of the American standard. What was most troubling was that these Ganda children continued to be underweight and stunted for their age; they never recovered the ground they lost. The view that protein malnutrition was an endemic problem became the basis of a new explanation for racial difference. Thus one physician wrote, "a large proportion of 'normal' African children suffer from mild kwashiorkor during the weaning period. It is probable that among many African children the ground lost during weaning is never fully recovered. Not only are African school children and adults subnormal in stature, but they also carry some serious scars for life from

the attack of malnutrition during weaning. . . . The 'danger period during weaning' is therefore a subject of profound importance not only for the individual child but also for African society as a whole."[55] Dean, however, was far less subtle: "I am prepared to support the idea of one essential pathology, having its origin in a dietary insufficiency in early childhood, which might give rise to most of these African peculiarities. There are so many remarkable differences between an African and a European. How many of them," he rhetorically asked, "can be explained by a fondness for matoke?"[56]

Defining protein malnutrition as endemic made protein deficiency a cultural problem capable of explaining racial inferiority. Instead of acknowledging the economic imperatives operating against the inclusion of sufficient protein in the diet of young children, biomedical practitioners interpreted the incidence of kwashiorkor as a natural feature of the environment that in many ways echoed longstanding colonial conceptions of the region and its inhabitants. From the arrival of European explorers and missionaries in the late nineteenth-century, Buganda became known as the Banana Kingdom. Endowed with remarkably reliable and ample annual rainfall, this 'fertile crescent' that wrapped around the north-western shores of Lake Victoria was particularly well-suited to the cultivation of bananas, a factor that facilitated settled agriculture, surplus production, population growth and state-formation. Moreover, once planted plantains can provide annual sustenance for nearly a decade meaning that unlike millet, sorghum and maize, maintaining a family's supply of plantains did not require the same labor-intensive inputs associated with annual planting, harvesting and crop rotation. Ignoring the need to frequently prune, weed and fertilize, the idea that banana cultivation was a very simple and straightforward task that left Ganda men free to engage in hunting and politics became the widespread view of colonial administrators and observers. As Richard Reid argues, these assumptions reinforced "the nineteenth-century European notion that such natural abundance made Africans inherently lazy, or at least unwilling to progress, and

prevented the development of a work ethic."[57]

Echoing conceptions of the tropical environment, such views justified colonization, forced labor and the spread of cash crop production in Buganda. The fact that the extensive production of cotton displaced the cultivation of protein-rich crops, like millet, by the early 1930s, was entirely lost upon biomedical practitioners working in Uganda during the 1950s and 1960s.[58] Instead, peasant production of cash-crops in Buganda, the center of a thriving colonial economy, led most to presume that poverty played no part in the problem of protein malnutrition. The apparent wealth of Ganda farmers masked the fact that paying for school fees and a growing list of necessities might preclude the purchase of high-protein foods like meat and fish.

Moreover, defining protein deficiency as endemic, as an unchanging problem that went back to time immemorial, meant that the impact of economic factors was entirely missed. The scarcity of imports and the wartime inflation that led to a dramatic rise in the cost of living provides a case in point. According to Gardner Thompson, during the Second World War, goods in high demand were fetching prices five to ten times their legal price and the cost of locally grown produce increased by between forty and eighty percent. As the evidence from this period indicates, inflation had the greatest impact upon the high-protein components of the diet. Not only did beef prices soar with frequent reports of profiteering on the part of butchers, but many Ugandans responded by selling beans and groundnuts. In 1942, for instance, the Chief Secretary in Entebbe replied to a request for groundnuts from Nairobi by stating that there were no groundnuts available in Uganda and another official later reported that a number of Indian firms were secretly sending groundnuts to Kenya by hiding them among other items.[59] Although, the extreme economic circumstances of wartime did not persist, they are indicative of the ways in which socio-economic factors, like fluctuating cash-crop prices, directly affected the availability of protein-rich foods even within households of relative means.

The Worldwide Protein Crisis

In October of 1949, an Expert Committee on Nutrition formed by both the United Nations Food and Agricultural Organization (FAO) and the WHO held its first meeting in Geneva. Kwashiorkor, cited as "one of the most widespread nutritional disorders in tropical and sub-tropical areas," was high on the agenda and Trowell was even apparently asked to prepare a memorandum on the condition for the committee's consideration.[60] The Committee resolved to conduct an investigation of kwashiorkor and the subsequent WHO report on "Kwashiorkor in Africa" became the seminal study in the growing international focus on protein malnutrition.[61] From the outset the members of the Committee entertained the possibility that kwashiorkor was an exotic condition confined to the tropics and they specifically recommended that WHO investigation "define the part played by . . . tropical parasitism."[62] The failure to establish a correlation between parasitic infections and kwashiorkor did not, however, succeed in disrupting the tropical medical framework and a new environmental determinism and narrow definition of the syndrome prevailed. Despite the universal insights that emerged from the scientific study of kwashiorkor, the problem of protein malnutrition was increasingly associated with emerging concepts of the "developing" or "Third World." Narrowly defining protein deficiency, in this way, favored technical rather than political or economic solutions and vertical rather than comprehensive public health programs.

One of the primary aims of the WHO's investigation of "Kwashiorkor in Africa" was to rectify the fact that the condition remained "ill-defined." The fundamental features identified in the report were previously documented by the various doctors who encountered the condition around the world. The WHO inquiry combined the evidence from across the continent to create a unified account of kwashiorkor that was to have a lasting impact on international perceptions of the problem of protein deficiency. The fact that the evidence was drawn from as far north as Dakar and as far south as South Africa, for instance, confirmed that kwashiorkor, as a single syndrome, had

widespread prevalence in Africa. Moreover, the evidence drawn from areas in South Africa, where parasitic diseases were not a complicating factor, ruled out the possibility that the condition was merely the manifestation of tropical diseases like malaria or hookworm anemia. According to the authors of the study, the existence of kwashiorkor in the absence of parasitic infection proved that the nutritional etiology was dominant.

In establishing that kwashiorkor was, in fact, a form of malnutrition and not, therefore, an exotic infection only found in the tropics, the growing international focus upon protein deficiency represented a distinct shift away from tropical medicine. Yet, the potential to see poor nutritional health as a universal human problem was effectively foreclosed by a series of moves through which kwashiorkor was narrowly defined as a condition of African childhood. Prior to the investigation, the nutritional research connected to kwashiorkor concerned adults as well as children. The symptoms of kwashiorkor observed in adult cases were not previously regarded as entirely distinct from the infantile syndrome and while acknowledging that "a syndrome very similar to kwashiorkor is undoubtedly encountered in other age-groups and even in adults," the authors of the WHO report decided to confine the term kwashiorkor solely to children in the late-breastfeeding, weaning and post-weaning phases of life.[63] The rationale for narrowly defining the syndrome to very young children was that the protein needed for growth and development meant that protein requirements were higher during the weaning phase of life than at any other point in the life cycle.[64] It was for this reason that very young children were particularly susceptible to severe protein deficiency and exhibited rates of mortality not seen in protein deficient adults. The pronounced prevalence and severity of kwashiorkor among young children made them the focus of medical attention and nutritional research from the outset. Yet, the WHO defined the extremely high mortality associated with kwashiorkor in young children as a central feature of the syndrome and thereby effectively excluded adult forms of protein deficiency, making kwashiorkor only universal during a brief period

of childhood.

In a section of the WHO report devoted solely to the cause of kwashiorkor, the authors noted that, "The areas, therefore, in which protein deficiency and kwashiorkor are likely to occur are those in which cassava, plantains, bananas and sweet potatoes constitute the staple foods."[65] The fact that all three staples were indigenous to and principally grown in the tropical and sub-tropical regions and supply the lowest ratio of dietary protein as compared to carbohydrate was compounded by the insufficient availability of animal protein. On this point the report was emphatic, "In general, and with few exceptions, the territories of Central and tropical Africa are deficient in livestock. Intake of animal protein is low everywhere."[66] The authors of the report further attributed the high prevalence of kwashiorkor among African children to the fact that cow's milk, which is "normally the most convenient source of protein for the child during the post-weaning period, is not available in the tropics in Africa. Its absence," they stressed, "is probably among the important causes of the prevalence of the syndrome."[67] The definition of kwashiorkor outlined by the WHO advanced an explanation of poor nutritional health in Africa that echoed the environmental determinism of tropical medicine more than the universal physiology of nutritional science.

The first and most obvious implication of suggesting that the prevalence of kwashiorkor in Africa was due to an overall lack of protein on the continent was that kwashiorkor was therefore an endemic rather than an epidemic problem and not a recent development or an effect of colonization. Casting the problem in this way allowed for a recognition of the significant role of poverty without demanding a political solution. At various points, the authors of the investigation even drew attention to the political economy of protein deficiency in Africa. They repeatedly acknowledged that much of kwashiorkor was the result of impoverishment and specific references were made to evidence that clearly indicated how economic conditions accounted for the prevalence

of kwashiorkor in Africa. They noted, for instance, that even in areas where cow's milk could be obtained, poor families could not usually afford to buy milk or other sources of animal protein. What is more, they attributed a noticeable reduction in the prevalence of kwashiorkor in the Gold Coast Colony to rising standards of living.[68] Moreover, the authors were explicitly critical of how colonial policies, like the forced cultivation of cassava, contributed to the prevalence of kwashiorkor in Africa as cassava is contains the lowest ratio of protein as compared to other staples.[69] Their indictment of colonial efforts to increase the production of cash crops was far less conclusive, however. They qualified their critique of policies that put the needs of the imperial economy above those of individual Africans by concluding that whether or not consumption or cash crops were the priority of agricultural directives, "The policy adopted should take account of the real needs and interests of the African in each individual territory."[70] The WHO could, therefore, point to colonial policies that were detrimental to nutritional health and to the ways in which poverty increased the incidence of protein deficiency, without calling colonialism into question.

The apparent endemicity and inevitability of kwashiorkor in Africa also opened the way for a lengthy discussion of how African ignorance and African cultural feeding practices accounted for the prevalence of protein malnutrition, while simultaneously suggesting that Africans were victimized by the lack of protein on their continent. Thus while the report blamed African mothers ignorant of proper weaning practices and the special nutritional needs of children in the weaning phase of life, the low levels of protein found in their breast milk indicated that insufficient supplies of protein were made available to lactating women in Africa as a result misguided cultural practices and the inevitable shortages of the tropical environment. As the authors of the investigation concluded, "It would not be too far-fetched to attribute to that protein deficiency, at least in part, the backwardness of the African people."[71]

The findings of this seminal report inaugurated a period that was later referred to as the "protein decade."[72] The report itself was the focus of the second meeting of the Joint FAO/WHO Expert Committee on Nutrition and although it was decided that further surveys were necessary, many recommendations were made concerning the need to increase the production of high protein foods. Following the committee's recommendation, delegations were sent to Central America and then to Brazil to assess the prevalence and features of kwashiorkor.[73] The third Joint FAO/WHO Expert Committee on Nutrition which was held in the Gambia in 1952, focused entirely upon nutrition in mothers, infants and children and the emphasis was largely on protein malnutrition. In 1953, a conference, sponsored by the FAO and WHO was held on "Protein Malnutrition" at the MRC Unit in Jamaica, with the aim of further delineating the biochemical, pathological, clinical and epidemiological aspects of protein deficiency.[74] In Africa, further attention was drawn to protein malnutrition at a series of Inter-African Conferences on Food and Nutrition sponsored by the FAO, WHO and the Commission for Technical Co-operation in Africa South of the Sahara (CCTA). And in November of 1957, the Uganda Government together with the WHO and the FAO, sponsored a "Nutrition Seminar for English-speaking countries and territories in Africa South of the Sahara."[75] As the head of the department of nutrition in Bombay stated at a conference in 1960 on *Progress in Meeting Protein Needs of Infants and Preschool Children*, "we have moved from the era of vitamin research to the era of protein research."[76]

Through these international forums the evidence of protein deficiency emerging from Uganda and other regions of the world came to be seen as an "impending protein crisis." Nearly a decade after the publication of his investigation of "Kwashiorkor in Africa," Autret proclaimed that the "deficiency of protein in the diet is the most serious and widespread problem in the world." In 1955, the growing belief that one of the most pressing problems facing humanity was the "world protein gap" led the WHO to form a Protein Advisory Group. The US National Research Council

also established a Committee on Protein Malnutrition and through these bodies a considerable level of funding and expertise was devoted to further research on protein malnutrition and increasingly on the production of high protein foods that could be used in the prevention of protein deficiency. Research efforts led to an FAO and UNICEF program to produce an odorless fish powder or "fish protein concentrate" that could be added to bread and efforts were also made to produce similar products using soybeans, cottonseeds and groundnuts.[77] These vertical programs took a "single-nutrient" approach to malnutrition that as McLaren argued was a tragic waste of resources. The cost of this grave mistake, McLaren insists, must be measured not only in terms of the financial investment but in the numbers of children who were "lost in the unchecked scourge of malnutrition."[78]

Conclusion

At many levels kwashiorkor, as a severe form of protein malnutrition, has not been a top public health priority for some time. On the one hand, scientists began to question the role of protein deficiency as the underlying cause of the condition nearly forty years ago. Subsequent efforts to determine the elusive etiology of kwashiorkor have centered upon the possible role of toxic food molds and most recently a lack of antioxidants. Yet, as the German Professor of Human and International Nutrition, Michael Krawinkel, observed in 2003, "Kwashiorkor is still not fully understood." What is most disturbing about this gap in our knowledge is as Krawinkel states, "Kwashiorkor still has a high case-fatality rate in many places, and it is still true that most children die after initiation of treatment. This situation," Krawinkel continues, "requires us to reassess continually our knowledge about the disease and our approach to its management."[79] What is most surprising about this need for further investigations to determine the cause of the condition so that

we may devise more effective forms of treatment is the fact that kwashiorkor was once the chief focus of international efforts to improve nutritional health.

As a result of McLaren's critique of the "Great Protein Fiasco," the pendulum swung in the opposite direction and calorie deficiencies became the focus of international efforts to combat "protein-calorie malnutrition." In broadening McLaren's critique, I have endeavored to show that the emphasis upon protein was not the problem, rather it was how the problem of protein malnutrition was understood. The shift away from protein did not necessarily entail a shift away from conceptions of malnutrition as endemic to and inherent in our conceptions of the "developing" or "Third World." Interpreting evidence of malnutrition as an endemic feature of the natural environment and the result of cultural beliefs and customary practices obscured the socio-economic and political factors that account for the prevalence of poor nutritional health across the globe. Moreover, these interpretations contained and served therefore to reinforce perceptions of racial difference, reifying the divisions that cut across our globe despite the universal pretentions of nutritional science.

The "vertical" single-nutrient programs of the protein era failed, first and foremost, to improve nutritional health, but also suggest the dangers of inaccurately assessing the underlying cause of public health problems. Doctors involved in the effort to close the protein gap in the 1950s and 1960s, distributed skim milk powder to mothers across the "developing" world. The result was a rise of bottle feeding in regions of the world still lacking access to safe drinking water, which consequently led to growing rates of diarrheal diseases and severe undernutrition or marasmus. Professor Derrick Jelliffe who ironically had been among the initial physicians at Mulago to recommend the distribution of dried skim milk as a preventive measure later led the international campaign to promote breastfeeding. With the benefit of hindsight Jelliffe acknowledged that, "As far as mothers of young children are concerned, [the distribution of dried skim milk] can only have

appeared as an endorsement of bottle feeding, with a resulting displacement effect on breast-feeding." Yet at the time, this was not recognized as the "nutritional tragedy" that it was.[80] Instead, biomedical practitioners interpreted the rise of bottle-feeding and the growing prevalence of marasmus as independent evidence that the real problem had always been undernutrition, or calorie deficiency rather than kwashiorkor. It was presumed that the proponents of the protein crisis had got it wrong, when in reality what they missed was the highly dynamic and shifting prevalence of malnutrition and the unintended consequences of their own interventions. Narrow definitions of the problem precluded the development of effective preventive measures engendering a vertical approach that was thereby implicated in the growing problem of undernutrition among very young children around the world. Rather than call attention to the crucial importance of evaluating how severe malnutrition was approached and understood, McLaren's critique served to obscure the relationship between efforts to prevent kwashiorkor and rising rates of marasmus. As interest in protein malnutrition evaporated, "the protein fiasco" could be forgotten leaving more recent efforts to contend with severe malnutrition in young children perilously lacking knowledge of the mistakes made in previous times.

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[1] Donald S. McLaren, "The Great Protein Fiasco," *Lancet* 304 (July, 1974): 93-96.
[2] Donald S. McLaren, "The Great Protein Fiasco Revisited," *Nutrition* 16 (2000): 464-465. Although I do not address it in this paper, the misunderstanding of McLaren's critique of the "Protein Fiasco" was due in large part to nutritional research indicating that even severe protein malnutrition (kwashiorkor) was primarily caused by calorie/energy rather than protein deficiency, i.e. protein-calorie malnutrition (PCM).
[3] McLaren, "The Great Protein Fiasco Revisited," 464. For the original articulation of this argument see McLaren, "The Great Protein Fiasco," 95.
[4] David Arnold, "The Place of 'The Tropics' in Western Medical Ideas Since 1750," *Tropical Medicine and International Health* 2:4 (1997): 303-313; Nancy Leys Stepan, *Picturing Tropical Nature* (Ithaca, New York, 2001);
[5] Mark Harrison, "The Tender Frame of Man": Disease, Climate, and Racial Difference in India and the West Indies," *Bulletin of the History of Medicine* 70:1 (1996): 68-93; Mark Harrison, *Climates and Constitutions: Health, Race, Environment and British Imperialism, 1600-1850* (1999).

[6] Philip Curtin, *Death by Migration: Europe's Encounter with the Tropical World in the Nineteenth Century* (Cambridge, 1989); Kenneth F. Kiple, *The Caribbean Slave: A Biological History* (Cambridge, 1984); Warwick Anderson, "Immunities of Empire: Race, Disease, and the New Tropical Medicine, 1900-1920," *Bulletin of the History of Medicine* 70:1 (1996): 94-118.
[7] David Arnold, "Introduction: Disease, Medicine and Empire," in *Imperial Medicine and Indigenous Societies* (New York, 1998), 7; Arnold, "The Place of 'The Tropics' in Western Medical Ideas."
[8] Michael Worboys, "Tropical Diseases," in *Companion Encyclopedia in the History of Medicine*, eds., William F. Bynum and Roy Porter (London, 1993): 512-513.
[9] Michael Worboys, "The Discovery of Colonial Malnutrition Between the Wars," in *Imperial Medicine and Indigenous Societies*, ed. David Arnold (New York, 1988), 208-225; *Nutrition in the Colonial Empire*, Report of the Economic Advisory Council's Committee on Nutrition in the Colonial Empire (London, 1939); Diana Wylie, *Starving on a Full Stomach: Hunger and the Triumph of Cultural Racism in Modern South Africa* (Charlottesville, 2001); Cythnia Brantley, *Feeding Families: African Realities and British Ideas of Nutrition and Development in Early Colonial Africa* (Portsmouth, 2002).
[10] Worboys, The Discovery of Colonial Malnutrition," 222-223.
[11] McLaren, "The Great Protein Fiasco," 95.
[12] Arnold, "The Place of 'The Tropics' in Western Medical Ideas," 312.
[13] Donald S. McLaren, "The Great Protein Fiasco," *Lancet* (July 13, 1974): 93; McLaren quotes from C. D. Williams, "A Nutritional Disease of Childhood Associated with a Maize Diet," *Archives of Disease in Childhood*, 8 (1933). Williams first referred to the condition as kwashiorkor in her 1935 article: C. D. Williams, "Kwashiorkor: A Nutritional Disease of Children Associated with a Maize Diet," *Lancet*, (Nov. 16 1935).
[14] H. S. Stannus, "A Nutritional Disease of Childhood Associated with a Maize Diet -and Pellagra-Archives of Disease in Childhood," *Tropical Diseases Bulletin*, 9 (1934).
[15] R. G. Whitehead, "Kwashiorkor in Uganda" in *The Contribution of Nutrition to Human and Animal Health*. eds. Widdowson and Mathers (Cambridge: Cambridge University Press, 1992).
[16] Hugh Trowell, "Food, Protein and Kwashiorkor (Presidential Address)," *Uganda Journal* 21 (1957): 84; J. F. Brock and M. Autret, "Kwashiorkor in Africa," World Health Organization: Monograph Series, 8 (1952), 24. Average mortality rates had already begun to fall and were in the order of 30 to 40 percent when the WHO conducted its study in 1951.
[17] Interview with Hugh Trowell by Elizabeth Bray MSS.Afr.s.1872 (144B) RHL: 33-34.
[18] Interview with Hugh Trowell by Elizabeth Bray MSS.Afr.s.1872 (144B) RHL: 21.
[19] Trowell, "Food, Protein and Kwashiorkor," 85.
[20] Interview with Hugh Trowell by Elizabeth Bray MSS.Afr.s.1872 (144B) RHL: 21.
[21] According to Carol Sicherman's sources, "the Medical School had been 'imperceptibly transformed into a university faculty of medicine'" by 1939: *Becoming an African University: Makerere, 1922-2000* (Trenton, 2005), 152; Iliffe, *East African Doctors*; Odonga, *The First Fifty Years*.
[22] "Davies, Dr. J.N.P" Personal Memoir, MSS.Afr.s.1872(40), RHL, 76.
[23] According to Roger Whitehead, Dean was "one of the great giants of nutritional science," and "one of the very first professional scientists" in Uganda. Interview with Dr. Roger Whitehead, Kampala, Uganda, 9 December 2003.
[24] Together with Elsie Widdowson, Dr. R.A. McCance founded one of the two main schools of nutritional science in Britain and the *McCance and Widdowson's Composition of Foods* not only remains the authoritative source of information on the nutritional value of foods in the UK, but continues in the sixth edition, published posthumously, to bear their names. E. M. Widdowson, "Obituary

Notice: R. A. McCance (9 December 1898-5 March 1993)", *Proceedings of the Nutrition Society*, 52 (1993), 385.
[25] R.F.A. Dean, "Plant Proteins in Child Feeding" *Medical Research Council Special Report Series*, No. 279 (London, 1953), see "Preface"; Members of the Department of Experimental Medicine, Cambridge, and Associated Workers. "Studies of Undernutrition, Wuppertal 1946-9," *Medical Research Council Special Report Series*, No. 275 (London, 1951).
[26] R.G. Whitehead, "Kwashiorkor in Uganda" in *The Contribution of Nutrition to Human and Animal Health.* eds. E.M. Widdowson and J.C. Mathers (Cambridge: Cambridge University Press, 1992), 307.
[27] Whitehead, "Kwashiorkor in Uganda," 307.
[28] Interview with Dr. Roger Whitehead, Kampala, Uganda, 9 December 2003.
[29] Trowell, Davies and Dean, *Kwashiorkor*, 186-187; Brock and Autret, "Kwashiorkor in Africa," 34.
[30] R.F.A. Dean, "The Treatment of Kwashiorkor with Milk and Vegetable Proteins" *British Medical Journal* (1952): 792.
[31] Whitehead to Himsworth, 21 November 1964, PRO FD 12/274; Interview with Dr. Paget Stanfield, Edinburgh, Scotland, 27 November 2003; Interview with Dr. Roger Whitehead, Kampala, Uganda, 9 December 2003.
[32] R.F.A. Dean and B. Weinbren, "Fat Absorption in Chronic Severe Malnutrition in Children" *Lancet* (1956); R.F.A. Dean and M. Skinner, "A Note on the Treatment of Kwashiorkor," *Journal of Tropical Pediatrics* (March, 1957).
[33] Dean, "The Treatment of Kwashiorkor with Milk and Vegetable Proteins," 792.
[34] Dean, "The Treatment of Kwashiorkor with Milk and Vegetable Proteins," 792.
[35] Trowell, Davies and Dean, *Kwashiorkor*, 113.
[36] R.F.A Dean and Ruth Schwartz, "The Serum Chemistry in Uncomplicated Kwashiorkor," *British Journal of Nutrition* 7 (1953).
[37] Dean, "The Treatment of Kwashiorkor with Milk and Vegetable Proteins," 792-793.
[38] I. Schneideman, F.J. Bennett and I.H.E. Rutishauser, "The Nutrition Rehabilitation Unit at Mulago Hospital-Kampala: Development and Evaluation, 1965-67" *Journal of Tropical Pediatrics* 17:1 (1971).
[39] Trowell, Davies and Dean, *Kwashiorkor*, 206-207.
[40] Whitehead, "Kwashiorkor in Uganda," 308.
[41] Trowell, Davies, Dean, *Kwashiorkor* (London: Academic Press, 1982).
[42] Trowell, Davies, Dean, *Kwashiorkor* (London: Academic Press, 1982): 47; H.F. Welbourn, "Backgrounds and Follow-Up of Children with Kwashiorkor" *Journal of Tropical Pediatrics* (December, 1959): 85; Latimer K. Musoke, "An Analysis of Admissions to The Paediatric Division, Mulago Hospital in 1959" *Archives of Disease in Childhood*, 36 (1961): 308. The percentages cited are compiled from the above sources by the author.
[43] H.F. Welbourn, "The Danger Period During Weaning" *The Journal of Tropical Pediatrics* (June, 1955); D.B. Jelliffe and R.F.A. Dean, "Protein-Calorie Malnutrition in Early Childhood" *Journal of Tropical Pediatrics* (December, 1959): 96; R.F.A. Dean "Biochemical Changes Caused by Protein Deficiency in Young Children" *Clinica Chimica Acta* 5 (1960): 186.
[44] H.F. Welbourn, "The Danger Period During Weaning (Part II)" *The Journal of Tropical Pediatrics* (September, 1955).
[45] I.H.E. Rutishauser, "Custom and Child Health in Buganda: IV. Food and Nutrition" *Tropical and Geographical Medicine* 15 (1963): 140.
[46] Dean to Williams, 19 June 1952, Wellcome Library PP/CDW L.1; R.F.A. Dean, "Observations on African Children," Wellcome Library PP/CDW L.1: 8.

[47] Rutishauser, "Custom and Child Health," 142 & 145.
[48] Rutishauser, "Custom and Child Health," 145.
[49] H.F. Welbourn, "Custom and Child Health in Buganda: II. Methods of Child Rearing" *Tropical and Geographical Medicine* 15 (1963): 126.
[50] M. Geber, "The Psycho-Motor Development of African Children in the First Year, and the Influence of Maternal Behavior" *Journal of Social Psychology* 47 (1958): 194.
[51] M. Geber and R.F.A. Dean, "Gesell Tests on African Children" *Pediatrics* (December, 1957): 1062.
[52] P.R.M. Jones and R.F.A. Dean, "The Effects of Kwashiorkor on the Development of the Bones and of the Hand" *Journal of Tropical Pediatrics* (September, 1956); P.R.M. Jones and R.F.A. Dean, "The Effects of Kwashiorkor on the Development of the Bones and of the Knee" *Journal of Pediatrics* (1959).
[53] R.F.A Dean and Ruth Schwartz, "The Serum Chemistry in Uncomplicated Kwashiorkor," *British Journal of Nutrition* 7 (1953).
[54] E.G. Holmes, R.E. Jones, M.W. Stanier, "The Protein Metabolism of East Africans with Hookworm Anaemia and other Conditions," PRO FD 1/1873.
[55] Welbourn, "The Danger Period During Weaning" *The Journal of Tropical Pediatrics* (June, 1955): 35.
[56] Dean to Williams, 19 June 1952, Wellcome Library PP/CDW L.1; R.F.A. Dean, "Observations on African Children," Wellcome Library PP/CDW L.1: 8.
[57] Richard Reid, *Political Power in Pre-Colonial Buganda: Economy, Society & Warfare in the Nineteenth Century* (Athens, Ohio, 2002): 24-25.
[58] L.P. Mair, *An African People in the Twentieth Century* (London, 1934): 111.
[59] Gardner Thompson, *Governing Uganda: British Colonial Rule and its Legacy* (Fountain, 2003); Gardner Thompson, "Colonialism in Crisis: The Uganda Disturbances of 1945" *African Affairs* 91 (1992); R.M.A van Zwanenberg and Anne King, *An Economic History of Kenya and Uganda, 1800-1970* (MacMillan Press, 1975).
[60] "Joint FAO/WHO Expert Committee on Nutrition: Report on the First Session," World Health Organization Technical Report Series No. 16 (Geneva, 1950), 15; Interview with Hugh Trowell by Elizabeth Bray, RHL MSS.Afr.s.1872 (144B): 56-57. According to Trowell's testimony, he was not informed of the request until the WHO arrived in Uganda to investigate kwashiorkor.
[61] Brock and Autret, "Kwashiorkor in Africa".
[62] "Joint FAO/WHO Expert Committee on Nutrition: Report on the First Session," 15.
[63] Brock and Autret, "Kwashiorkor in Africa", 12.
[64] The only comparable exceptions being during pregnancy, lactation and when recovering from illness.
[65] Brock and Autret, "Kwashiorkor in Africa", 57.
[66] Brock and Autret, "Kwashiorkor in Africa", 61.
[67] Brock and Autret, "Kwashiorkor in Africa", 58.
[68] Brock and Autret, "Kwashiorkor in Africa", 66.
[69] Brock and Autret, "Kwashiorkor in Africa", 62.
[70] Brock and Autret, "Kwashiorkor in Africa," 64.
[71] Brock and Autret, "Kwashiorkor in Africa", 32-33.
[72] K. J. Carpenter, *Protein and Energy: A Study of Changing Ideas in Nutrition* (New York: Cambridge University Press, 1994): 160. This widely cited quote (see Wylie and Brantley) is originally from J.F. Brock, "Dietary Proteins in Relation to Man's Health" *Proc. Internat. Congr. Nutr.* (1961)

[73] M. Autret and M. Behar, "Sindrome Policarencial Infantil (Kwashiorkor) and its Prevention in Central America," FAO Nutritional Studies No. 13, (1954); J.C. Waterlow and A. Vergara, "Protein Malnurition in Brazil," FAO Nutritional Studies No. 14, (1956).
[74] J.C Waterlow ed., *Protein Malnutrition*, Proceedings of a Conference in Jamaica, Sponsored Jointly by the FAO, WHO and the Josiah Macy Jr. Foundation, New York (Cambridge University Press, 1953).
[75] "Nutrition Seminar for English-speaking countries and territories in Africa South of the Sahara" Organized Jointly by the Uganda Government, WHO and FAO at Kampala, Uganda, 25-29 November 1957.
[76] "Progress in Meeting Protein Needs of Infants and Preschool Children," Proceedings on an International Conference held in Washington, D.C. August 21-24, 1960: 541.
[77] Carpenter, *Protein and Energy: A Study of Changing Ideas in Nutrition* (1994).
[78] McLaren, "The Great Protein Fiasco," 95.
[79] Michael Krawinkel, "Kwashiorkor is Still Not Fully Understood," *Bulletin of the World Health Organization* 81:12 (2003): 910.
[80] Jelliffe and Jelliffe, *Human Milk in the Modern World*, 234.