The Uses of Rickets: Race, Technology, and the Politics of Preventive Medicine in the Early Twentieth Century

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The Uses of Rickets: 
Race, Technology, and the Politics of Preventive Medicine 
in the Early Twentieth Century

A Thesis Submitted to the 
Yale University School of Medicine 
In Partial Fulfillment of the Requirements for the 
Degree of Doctor of Medicine

by

M. Allison Arwady

2008
Rickets, the bone disease classically caused by Vitamin D deficiency, was one of the most common diseases of children 100 years ago. It has been recognized as a disease of urban living and linked to issues of race and culture for generations. This paper uses unpublished patient records from 1904 to 1909 and archival and published materials from multiple community-based trials, including the New Haven Rickets Study (1923-1926), to explore how the definition, diagnosis, and treatment of rickets shifted in the first decades of the twentieth century in the United States.

Before 1910, as evidenced by patient records, neither the diagnosis nor the treatment of rickets had been standardized. The disease was frequently presented as a disease of African-Americans or Italian immigrants and used to reinforce racial stereotypes, to promote the assimilation of immigrants into majority cultures, and to call for behavioral change. In the second and third decades of the twentieth century, as clinicians and scientists unraveled the twin roles of diet and sunlight exposure in the disease’s etiology, both diagnosis and treatment became more standardized. But this standardization—including exchanging bedside diagnosis for X-ray technology and promoting general preventive measures—altered the perceived prevalence and even the definition of the disease. By the mid-1920s, rickets was promoted as universal, at times invisible to non-experts, but present to some degree in nearly every young child regardless of race or class. It was thus used to promote the young disciplines of preventive medicine, pediatrics, and public health.

Rickets therefore provides an excellent window into the early politics of preventive health in the United States and a relevant historical counterpoint for current debates over the role of race and ethnicity as risk factors for disease; the use of diagnostic technology in defining disease; and the promotion of targeted interventions for today’s so-called “lifestyle” diseases.
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First, I would like to thank John Warner for his kind support, helpful questions, and ongoing commitment to promoting student research in the humanities at the medical school. The entire History of Medicine Department at Yale (including Naomi Rogers and, previously, Susan Lederer) has welcomed me warmly from the beginning, tolerated my ever-shifting schedule, and provided necessary historical and social context for my clinical training.

I thank the Office of Student Research for giving me a short-term stipend to help support a productive year of clinical tuberculosis research in Botswana—and also for giving me the academic freedom to work on a separate history of medicine research project for my thesis.

Finally, my fascination with the history of public health and the roots for this project date from my time at the Mailman School of Public Health at Columbia University, where Ron Bayer, David Rosner, and Amy Fairchild taught me how archival materials can inform and complicate current debates.

This thesis is a work in progress rather than a finished document. I hope to continue working on this and related topics as I enter the next phase of my clinical training at Yale.
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Introduction

One hundred years ago, rickets was a common disease of children in the cities of the northern United States. “Rickets,” an orthopedic surgeon at Yale wrote in 1898, “is by far the most frequent cause of all deformities which have come under my observation.”\(^1\) Until 1925, remembered a Yale pediatrician, most admissions to any American non-contagious children’s ward were to treat either diarrhea or results of vitamin D deficiency (that is, rickets).\(^2\) Many--if not most--children living in large northern cities exhibited at least mild forms of the disease in the 1920s.\(^3\) A postmortem study conducted in Dresden between 1901 and 1908 found that 96 per cent of the infants and 90 percent of the children under four who came to autopsy had signs of rickets.\(^4\)

African American and Italian children were understood to be particularly at risk. “It may be safely stated,” wrote pediatrician Alfred Hess in the 1917 *Journal of the American Medical Association*, “that over 90 per cent of the colored babies [in New York City] have rickets.”\(^5\) The Director of Social Welfare of the Association for the Improvement of the Condition of the Poor (AICP) in New York wrote in 1924, “Rickets presents a problem of paramount importance among Italians.”\(^6\) Children raised on “artificial feed” were also believed to be at increased risk. L. Emmett Holt, the author of the most influential early pediatrics textbook,\(^7\) wrote in 1897 that in his six years of practice in New York City, he had “yet to see an infant reared solely on canned condensed milk who did not exhibit the signs of rickets to a greater or lesser degree.”\(^8\) After following 250 infants for four years in the Hebrew Infant Asylum, Hess declared in 1922 that

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\(^1\) Charles Alling Tuttle, “A Short Paper on Rickets,” *Transactions of the New York State Medical Association for the Year 1898*, p. 202. Tuttle, a Yale-affiliated physician, read his paper on October 19, 1898 at the New York State Medical Association meeting. It was subsequently reprinted in both the *Yale Medical Journal* and *Pediatrics*.


rickets was virtually universal in bottle-fed infants.9 Breast-fed infants, however, were not
immune. In a 1922 study, fully half of a group of breast-fed infants in Baltimore developed
rickets.10 A 1926 New Orleans study of 197 breast-fed infants found that all showed signs of
rickets by the age of one and a Cincinnati study from the same year demonstrated that a focus on
prenatal nutrition did not protect offspring from rickets.11

“Rickets is a national public health problem in every country,” concluded the 1921 book
*Preventive Medicine and Hygiene.* “It is a world scourge preventing the normal development of
the young, leaving them damaged not only in their bones, but also in their mental and moral
faculties. It is particularly marked among the poor of large cities, who are ill fed and badly
housed.”12

Finally, rickets, while rarely immediately fatal, predisposed children to more lethal
diseases. “While not a fatal disease *per se*, rickets adds very greatly to the danger from all acute
disease in infancy, and even to some degree also to those of later life,” Holt’s 1902 textbook
warned.13 The chest deformities often present may have made children more susceptible to
pneumonia and other respiratory diseases. A 1921 Johns Hopkins study noted that 11 of 22
children who had presented with a severely rachitic thorax had died.14 Calcium deficits meant that
seizures and tetany sometimes accompanied rickets. Even as rickety children outgrew the most
obvious signs of illness, as adults they were often smaller in stature, which caused particular
problems for women in childbirth. Harrison suggests that the contracted pelvises seen in women

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Examinations In Its Diagnosis and Incidence,” *Am. J. Diseases Children,* 1922, 24:327.
Association,* 1922, 79: 2210.
14 E. A. Park and J. Howland, “The Dangers to Life of Severe Involvement of the Thorax in Rickets,” *Johns Hopkins
who had had rickets as children may have fueled the growth of medical obstetrics and use of childbirth instruments in the eighteenth and nineteenth centuries.15

Rickets has been used to promote varied ideas and agendas over the years, from prohibition to prenatal care, and from racial degeneracy to radiology. The disease was common enough to invite analogy and wide speculation about diet and environment before the science of vitamin D had been uncovered. In addition to medical uses, for example, a University of Virginia pathologist hypothesized in 1928 that the dinosaurs developed rickets when vast clouds of volcanic dust obscured the sun, leading directly to their extinction because their massive skeletons could no longer support their own body weight.16 An 1822 article in a sporting journal railed against the use of opium with the cautionary tale, “It is said that the Turks who have given themselves up to the immoderate use of opium, are known by a kind of rickets which in time this poison never fails to produce.”17 A 1922 Anthology of Sex Knowledge warned readers that they were at increased risk of having rickety children if they married too young (under age 20 for women, under 24 for men) or too old (over 40 for women, over 50 for men).18 By the late 1920s, when the science was clearer, rickets could be defined colloquially as “the disorder of the mineral chemistry of the body that makes babies grow up into bow-legged flappers and unsymmetrical sheiks.”19

Framing Rickets

Historians have explored the “social construction of disease” for at least a generation.

Even the most traditional medical schools now supplement lessons on biological mechanisms and

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16 Marjorie MacDill, “Dinosaurs Died of Rickets,” The Science News-Letter, 1928, 14: 63-64. The writer noted that because bony brontosauruses could be over 80 feet long, “Rickets in such a creature would be worse than the proverbial sore throat in a giraffe.”
pathological processes with occasional discussions on the ways societies understand and define disease. It is now taken for granted that emerging biological threats have cultural and societal influences. As Charles Rosenberg wrote in his classic argument,

Disease is at once a biological event, a generation-specific repertoire of verbal constructs reflecting medicine's intellectual and institutional history, an aspect of and potential legitimation for public policy, a potentially defining element of social role, a sanction for cultural norms, and a structuring element in doctor/patient interactions.20

The disease of rickets is no different. At one level, it is a biological affliction, a softening of the bones of children classically caused by Vitamin D deficiency. But rickets is also an excellent case study for exploring the shifting definitions of disease in the United States. One hundred years ago, for example, the word “vitamin” did not exist; even the idea of negative causality (disease as the result of deficiency rather than invader) was largely suspect. Rickets was used to advance the field of radiology and to legitimate public policy in the relatively young specialty of pediatrics and the growing field of public health.

Medically Defining Rickets in the 17th Century

The first medical definitions of rickets date from over 350 years ago. Daniel Whistler, an Englishman studying medicine in the Netherlands, described rickets (in Latin) for his University of Leyden medical school thesis in 1645. Francis Glisson, a Professor of Physic at the University of Cambridge, published a more complete description of *De Rachitide* in 1650, which was translated to English in 1651.21 Rather than discussing the unresolved debate over the scientific claim to originality, this paper shows five main themes in Whistler’s paper, which were echoed in Glisson’s work. These themes played, and continue to play, a prominent role in subsequent descriptions of rickets.

First, Whistler emphasized the novelty, or newness of the disease. “It was, so far as I am aware, unknown to the ancients,” he wrote, continuing that it was “unknown to us until some twenty-six years ago…” Glisson, similarly, wrote, “This is absolutely a new Disease, and never described by any of the Ancient or Modern Writers or in their practical Books which are extant at this day, of the Disease of Infants.” Many physicians and historians have challenged this claim, pointing to much earlier physical evidence of rickets, even if the disease was not yet named. The important point is that disease definitions must describe something new—even if it is a new recognition of how old patterns are pathologically related.

Second, Whistler claimed that rickets was geographically specific to England and pathologically inherent to English people. Scurvy, he wrote, was endemic to the Baltic coast, bronchocele was common in the “Alpine people,” and venereal disease ran rampant in India, while rickets was “a peculiar and domestic scourge to our English infants.” Glisson, likewise, considered it specific to England, referring to “our particular climate” and “the very nature of the English temperament” as reasons for the disease’s prevalence. In part through wide reprinting of Glisson’s work, rickets became colloquially known as the “English disease” for centuries. At the turn of the twentieth century in the United States, rickets was understood to be primarily an African-American or Italian disease rather than an “English disease,” but the practice of linking the disease to geography and especially race had not changed.

Third, Whistler addressed environment and behavior, as well as class differences in the etiology of rickets, blaming both an unhealthy environment among the poor and deficient parental behavior (“vile habits”) among the wealthy. “The disease is most frequent in the ranks of the highest citizens, next amongst the dregs of the populace, least of all amongst those of moderate

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23 Francis Glisson, *De rachitide sive morbo puerili, que vulgo The Rickets dicetur* (London, 1650), translated in Clarke.
means,” he wrote. “The cause in the first group I take to be intemperance of the parents and the fact that the infants are entrusted to the care of hired wet nurses; in the second, as well as mistakes in diet, lack of fires and a daily environment of dung and dirt and the use of cold and badly-aired napkins.”

His biological explanation for the disease used the then-common language of heat and cold, but he linked this biology back to class-influenced behavioral choices. Just as those cold napkins used in the poor families could “extinguish…a native heat inherently weak,” warm red-haired wet nurses hired by wealthier families would produce milk that was “more cooked” and thus dispose the infants to rickets. Debates over environment and behavior, obviously, played central roles in the public health interventions of the early 20th century, with wealthy and impoverished families blamed for different choices or living conditions that predisposed their children to rickets.

Fourth, Whistler included a hereditary component (the “virtue of the seed”) as well as parental behavior or conditions at conception. Some infants, he wrote, developed rickets because their parents suffered from gonorrhea, scurvy, or scrofula, all considered inherited diseases. However, other cases of rickets suggested that the infant’s parents had “engaged in immature and immoderate frictions” (presumably masturbation) or in “over-hasty or enormous lovemakings or…indulged over-liberally in drinking.” Some 350 years later, the debate over inheritance used different language, but rickets was clearly linked to concepts of racial and cultural degeneracy.

Finally, Whistler added scientific language, writing about this disease in Latin for the first time and therefore recognizing it as a formal disease, worthy of research. Though as he described, “the whole bony structure is as flexible as softened wax, his proposition for a more formal name focused on the disease’s systemic symptoms. He chose the weighty-sounding Latin

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27 Whistler, De morbo… Translated in Smerdon, p. 409.
28 Ibid., p. 408.
29 Ibid., p. 408.
name of *Paedosplanchnosteocaces*. Glisson’s choice of the simpler name *Rachitis* may have been part of the reason why his became the more enduring document. In a typical statement, a historian wrote, “From then on, having required the respectability of a Latin name, the disease was recognized more widely and all subsequent authors refer to Glisson’s masterpiece.” This attention to naming and defining disease, coupled with an emphasis on expert scientific knowledge and technology, is central to understanding how rickets was used in the early twentieth century to promote various agendas.

It is tempting to dismiss red-haired wet nurses and over-hasty lovemakings with an indulgent smile. But to do so diminishes the bald fact that this rough framework and rhetoric is still used to explore etiology and define disease today. These are what we now call “risk factors” and we talk about them daily in the hospital: geography and race, environment and behavior, genetics and diet. Although they are now entered into equations of risk, modified by technological advances and buoyed by the polysyllabic language of science, they are not as “objective” or immutable as they seem.

Often, risk factors are biologically valid. It is, for example, epidemiologically true that living in a more northern climate or having darker skin both increase the risk of vitamin D deficiency and rickets. There is also a valid biological explanation for why this is so: less direct sunlight exposure or more melanin in skin mean that less vitamin D is produced by the body. But in the years before this biology was understood, before the concept of a vitamin (and especially a UV-ray dependent vitamin) was accepted, doctors drew different conclusions from the epidemiological differences.

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Rickets at Bellevue 1904-1909

Ultimately, clinicians make treatment decisions at the bedsides of individual patients. Any understanding of clinical treatment of rickets in the early part of the twentieth century should go beyond physicians’ stated generalities to study specific, individual cases. This paper examines one complete casebook from Bellevue Hospital, which contained 80 individual patient records of children admitted to the First Division between 1904 and 1909. Conveniently, the hospital still filed records by patient disease at that time, so all 80 patients carried a primary diagnosis of rickets (generally marked “rachitis”). The records were filed in chronological order and included 76 medical patients along with four surgical patients (all from 1904).

The rickets patients were admitted to a hospital with a tradition of innovation. Bellevue opened the first maternity ward in the U.S. in 1799. Doctors used the world’s first hypodermic syringe there (1856) and performed the first caesarean section in a U.S. hospital (1887). The hospital boasted the country’s first outpatient department (1867), hospital-based ambulance service (horse-drawn; 1869), and school of nursing based on the teachings of Florence Nightingale (1873). Of particular note, in 1874, Bellevue opened the first children’s clinic in the nation. Bellevue was New York’s largest hospital in the first years of the twentieth century (and remained so until the late twentieth century). It had 1200 beds as early as 1870 and approximately 900 when the rickets patients were admitted between 1904 and 1909, with many more children visiting its outpatient clinics.

Bellevue also had a long tradition of providing care for “the endless stream of poor, sick people cast off by the city.” As the first public hospital in the U.S., it opened in 1736 as an

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32 Rickets 1904-1909 from Casebooks, 1877-1916, Bellevue Hospital, New York (nine volumes). First Medical Division. Casebooks accessed at Special Historical Collection, Personal Papers and Manuscripts (Bellevue Hospital), Health Sciences Library, Columbia University, New York.
almshouse with six beds. For approximately the first 175 years of its existence, Bellevue filled the two major functions of hospitals before the twentieth century—housing victims of epidemics and serving as an official and then unofficial almshouse, with a greater focus on providing shelter and meals for those with no other options than on offering trained nursing or medical care.

Bellevue of the early twentieth century also promoted a relatively new focus on curing rather than caretaking. A new building had been constructed in 1904 and some of the most prominent names in the new specialty of pediatrics taught at Bellevue’s medical school. The hospital’s president bragged in 1910 that the “hospital of the present day has developed into a complex institution, having for its prime object the relief of sickness.” The fact that a hospital’s prime object is the relief of sickness now seems redundant, but it illuminates the shift away from the idea of hospitals as last-resort poorhouses. As germ theory gained acceptance and new medical technology became available in hospitals, some physicians started encouraging even their better-off patients and in New York, the use of hospital-based services increased by 85 percent between 1890 and 1905.

Bellevue, however, continued to care mainly for those who could not afford admission at the more selective hospitals. A 1905 article provided a detailed tour of the “mother of hospitals and training schools—the dear old gray pile we call Bellevue.” As they had been for generations, the author noted, Bellevue’s “gates are always open to every ambulance and...[its] nine hundred beds are often supplemented with cots to afford shelter to the sick and needy. Other hospitals pick and choose, Bellevue receives all! Other hospitals have pay rooms and wards, Bellevue gives all for nothing!” Although the children admitted with rickets only had their race or nationality recorded sporadically, other sources indicate that many of Bellevue’s patients were

40 Ibid.
immigrants. As the nurse wrote in 1905, Bellevue was filled with “foreigners—a few French, many Germans, Italians, Russians, Slavs, Hungarians, Greeks, Armenians, Swedes, Hebrews, etc.” 41 In particular, she noted that the pediatric wards where the rickets patients would have been admitted right at this time were filled with “babies of all nationalities and colors.” 42

The casebook illuminates as much about hospital practices in the first decade of the twentieth century as it does about rickets. For a hospital that had moved away from the concept of an almshouse to the relief of sickness, many of the rickets patients stayed a long time, seemingly longer than was necessary. The mean length of stay for the 80 patients was 29 nights, but some patients stayed much, much longer. For example, a 15-month-old slept at Bellevue for 96 consecutive nights in 1907, a one-year-old stayed 99 nights in 1908, a four-year-old surgical patient remained for 112 nights in 1904, a three-year-old stayed for 151 nights in 1904, a two-year-old stayed 240 nights in 1906-07, and, in the longest case, a 17-month-old lived at Bellevue for 242 consecutive nights in 1904. These patients lived in the wards long after any formal treatments had finished, still having their temperature taken twice a day and their ounces of urine measured, even while they took no medication and underwent no recorded procedures. The only comments in their charts, day after day, are of the “resting comfortably” and “doing well” variety. Just a few years later, as the cost of hospital stays increased concurrently with the increasingly trained staff and the ever-growing range of expensive diagnostic and therapeutic technologies, the idea of keeping a relatively healthy child in the hospital for 242 nights became unthinkable.

But between 1904 and 1909, children with nowhere else to go apparently sometimes stayed at Bellevue. Notes on individual records highlight the problem. On Record 46, in the history of a 15-month-old admitted in 1908, the doctor wrote, “Mother is very unintelligent; doesn’t know anything about child. She is unable and too poor to look after child.” Despite spending 37 nights in the hospital, this child received no medication, and no special diet beyond

41 Ibid., p. 296.
42 Ibid., p. 294.
milk, beef juice, and orange juice. Record 43 was noted “illegitimate,” while Record 52, a two-year-old, had a “wicked disposition.” In Record 68, the physician wrote, “Father deserted wife and child. Mother had to work.” The only history note on Record 50, a one-year-old who called Bellevue home for 99 nights, was the poignant, “History unobtainable—not visited by relatives nor friends.” With its high prevalence, murky etiology, and nonspecific diagnostic techniques, rickets may have served as a catchall diagnosis for admitting and housing such children on a longer basis. It was undoubtedly very common. A 1963 letter to the editor of *Pediatrics* remembered, “Before the introduction of widespread vitamin D prophylaxis, all the babies over 3 or 4 months on the infant’s ward at Bellevue Hospital had rickets during the late winter and spring. The only variation was in degree.”

**Debating Etiology**

For years it seemed unlikely that rickets was nutrition-related simply because it appeared so early in a child’s life, while most children were still nursing and should therefore have been exempt from any deficiencies at the family’s supper table. Human milk was food *designed* for a baby, available to infants from poor and wealthy families alike, and the idea that this food was lacking in any way seemed antithetical. However, by the late nineteenth century, many practitioners believed that rickets was caused primarily by a problem in diet. The 1902 edition of Holt’s pediatric textbook explained that the disease was “principally seen in children living in crowded tenements” because that was where “the effects of improper food are most strikingly shown.”

Most doctors and public health workers believed that rickets was caused, at least in part, by environmental deficiencies. These evils were most often expressed as forms of home pollution, in crowded, uncleanly tenement homes where infants received no fresh air and little

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exercise. The emphasis on impure air and lack of exercise was logical, since the afflicted infants could not leave their homes for fresh air on their own or exercise. The spontaneous improvement of most cases of rickets once children were old enough to exercise on their own lent credence to this miasma-like theory. Turn-of-the-century theories about the higher prevalence of rickets in blacks and Italian immigrants focused on differences in diet, cleanliness, hygiene, and customs.

Even as the work on vitamins became central, some writers continued to question the focus on nutrition and vitamins. As John Lawson Dick argued in 1919, "The weight of evidence shows that defective housing and overcrowding ... with the loss of fresh air, sunshine, and exercise which these conditions entail, are the essential factors in producing rickets."45

Dick’s 1922 book *Rickets: A Study of Economic Conditions and Their Effects on the Health of the Nation* again downplayed diet and emphasized poverty and poor hygiene as the most important risk factors in the disease.46 He questioned the conclusions of recent scientific studies, including Mellanby’s successful induction of rickets in puppies fed deficient diets, arguing that the changes in the animal bones were not the hypoplastic changes of “true rickets.” Dick’s book won great admiration in the preventive medicine community, with one review calling it “a milestone in the history and literature of this disease,” and “a treatise that should prove of great value to the cause of preventive medicine and national hygiene.”

In retrospect, it is easier to understand the patterns around this unusual environmental etiology. Unlike the great infectious disease killers, rickets surfaced in the dark of winter rather than the heat of summer. People living in northern cities regularly had more limited sunlight exposure than people living in rural, or more Southern areas. Medical missionaries from the 1890s wrote with surprise about the almost complete lack of rickets in China, India, Morocco, and Japan, despite high incidences of tuberculosis, unvarying diets, and poor sanitary conditions in those countries.

The disease’s prevalence in cities remained the best indication of its etiology as an environmental disease. Increased levels of dust, smoke, and moisture in the city’s air absorbed more of the ultraviolet radiation crucial for protection against rickets, making the rays that did reach the ground between the shadows cast by tall buildings less effective. At least historically, people who have lived in cities have spent less time outside than people who live in the country. Rickets, therefore, was indeed more likely to be found in these “civilized communities…where children are kept too much indoors.”

Advancing Etiology: Scientific Research on Rickets

The discovery of vitamins in the second and third decades of the century revolutionized nutritional science. Between 1918 and 1928, scientists determined that rickets was caused by a deficiency in an “anti-rachitic substance,” and eventually named this substance Vitamin D. Vitamin D promotes absorption of calcium and phosphorus, which are needed for proper bone mineralization, particularly in the rapidly growing skeletons of young children. The body can obtain Vitamin D from two very different sources. First, as with other vitamins, it can be ingested in the diet. It is found in the highest levels in fish (and particular, in cod liver oil, and today, in Vitamin D-enhanced milk). But secondly, the skin can convert ultraviolet rays from the sun into Vitamin D.

The scientific advances in rickets research were in part the results of dramatic shifts in the culture and funding of medical research that had occurred around the turn of the century. In 1890, few American medical schools supported research activities, in part because funding was not available. In that year, the combined capital funds of all of the medical schools in the United States was not even $250,000, as compared to the almost $12 million held by all of the

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48 In experiments between 1918 and 1920, Edward Mellanby proved that dietary deficiencies were central to rickets by successfully inducing the disease in animals. In 1921, Elmer McCollum found a substance in some fats (like cod liver oil) that cured and helped prevent rickets. Though the substance had not been biochemically identified, its clear anti-rachitic action led to it being dubbed vitamin D, as the fourth identified vitamin. Adolf Windaus discovered the steroid precursor of vitamin D within a few years, and won the 1928 Nobel Prize in Chemistry for his work.
theological schools (who educated half as many students). But with the turn of the century, new attention to public health also led to more monetary support for the science of medicine. Just in 1901, for example, Congress appropriated funds for a national Hygienic Laboratory to investigate infectious and contagious diseases; The Rockefeller Institute for Medical Research opened with a gift from John D. Rockefeller; and New York City’s Health Department established its first laboratory, which eventually contributed to Alfred Hess’ work on rickets. This support started to bear fruit in the second decade of the twentieth century, concurrent with the flood of scientific investigations of rickets. The United States’ first Nobel Prize in medicine was awarded to a scientist working at the Rockefeller in 1912. This was the same year the Flexner Report advised more stringent science requirements for medical students, and the same year Casimir Funk published his influential book promoting the theory of vitamins.

Hess wrote that after the 1918 discovery that rickets could be cured with ultra violet radiation, the disease, “which for two and a half centuries had awakened but a fitful interest in the clinician,” became the “object of intense investigation in many of the biological, chemical and physical laboratories both in this country and abroad.” Biologists turned to animal research, chemists studied bone composition, and physicists explored ultraviolet radiation, all in an effort to better treat rickets.

An environmental disease like rickets might not have been solved without extensive animal experimentation. Ethically, rickets could not be induced in babies, and practically their environments could not be easily controlled. As it became “to a certain extent irrelevant whether these processes occurred in a laboratory animal or an Irish immigrant,” animal experimentation earned more monetary and public support, and experimenters used rats, dogs, cows, and chickens

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52 Numbers, p. 139.
to study rickets. Experiments treating animals with cod liver oil and ultraviolet light exposure became increasingly popular, as more people believed that the conclusions could be directly applied to humans. This, of course, is the ultimate example of the shift from specificity to universalism, wherein the treatment of a disease or the understanding of a physiological process is cross-applied not only among humans, but between humans and animals.

During a time when the revolutionary implications of the germ theory of disease still dominated the medical profession’s attention, the argument that rickets was caused by a vitamin deficiency met with some resistance. As Goodwin and Tangum described, academic medicine in the early twentieth century “lacked the vocabulary to integrate the public health observations of vitamin deficiency into a pathophysiology dominated by the germ theory.” In 1919, a report of the British Medical Research Committee stated:

> It is difficult to implant the idea of disease as due to deficiency. Disease is so generally associated with positive agents—the parasite, the toxin, the *materies morbi*—that the thought of the pathologist turns naturally to such positive associations and seems to believe with difficulty in causation prefixed by a minus sign.

If vitamin deficiency, or “negative causality” explanations for rickets had been hard for clinicians to swallow, the environmental explanations implicating a lack of sunlight met with still more skepticism. By the late 1920s, however, the improbable had been proven in carefully designed experiments, and clinicians had no choice but to accept the results. An article in the August 1927 edition of *The Scientific Monthly* commented on Dr. Hess’s experiments reported earlier that year in *JAMA*. His work had shown conclusively that while the milk of a nursing mother could not cure rats with rickets, if that woman had been exposed to the rays of an ultraviolet mercury vapor lamp, her milk did cure the rats’ rickets. “These developments are really surprising when viewed coldly,” the article stated simply, continuing that this “ridiculous

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idea...would have been dismissed as half-mystic nonsense by the practical investigator just a few years ago.” The article continued,

> It is easy to imagine the merry ridicule one would have met a few years ago by suggesting to some practical husbandryman that holding a lamp over a goat a few minutes every day would make it a much better goat or to the practical physicians that certain lamplight might act therapeutically on nursing women. Yet these are to-day known facts.56

Intellectual curiosity about the “great and peculiar difficulties”57 of the etiology of rickets was not limited to a handful of scientists working in isolated laboratories on the fringe of scientific relevance. No lesser prize than the 1928 Nobel in chemistry went to Windhaus for his work on the structural changes in the provitamins that produced the antirachitic factor. Subject reviews in the 1920s often cited 150 papers or more. A small 31-page booklet published in 1929 by the Mead-Johnson Company on the use of irradiated ergosterol in rickets included 295 scientific references in the bibliography.58 Only 26 of these 295 references dated from before 1925 (and most of those dealt with general yeast or ergosterol studies rather than specifically rickets-related research).

Even after the science of rickets was clearer and treatment more standardized, rickets continued to be a major public health concern. In 1928, Hess wrote, “Although decreasing in incidence and severity, rickets still undoubtedly is the most common nutritional disorder of early childhood in the temperate zones.”59 A physician recalled in the American Journal of Public Health that as late as 1940, rickets was “still probably the most common disease of early childhood.”60

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Debating Etiology: Urbanization and Ancient Civilizations

By the first decade of the twentieth century, rickets had long been recognized as an urban disease. “It is not common in the country,” a 1902 textbook offered, “but is exceedingly frequent in most large cities.” A 1906 textbook declared that rickets was “the direct result of domestication and a penalty that man pays for the artificial structure which he has created.”

Review articles often linked the “English Disease” in Britain to the cultural changes that accompanied the industrial revolution, as the economy shifted from agriculture to manufacturing, most notably in the late eighteenth to early nineteenth century. As explained in a typical article, “Rickets in epidemic proportions was a consequence of the industrial revolution which created the factory city with atmosphere laden with dust and coal smoke which form an efficient ultraviolet filter. Adaptation of the infant to the city has only been possible with the advent of nutritional science…” Physicians pointed to increasing urbanization and the growth of large cities in the U.S. as American risk factors. “Rickets is a disease preeminently of civilized countries although most prone to develop in large industrial centres,” a 1921 article stated. “It is in the latter that the neglect of child hygiene is greatest as not infrequently the women of the household work in the factories and the children have to be shut up in close quarters for many hours.”

This explanation was repeated throughout the 1920s. Rickets, for example, was generally considered “one of the consequences of the complex conditions of modern life.”

Rickets had been presented as a sign of progressive degeneracy for centuries. As early as 1822, an English writer used the physical manifestations of rickets and scurvy to illuminate the “progressive unhealthiness of mankind,” which he blamed on the corrupting influence of...

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64 Nine U.S. cities had populations of at least 100,000 in 1860, 50 cities did in 1910.
European civilization. “The rickets, a disease which for three centuries has been a scourge to Europe, is as yet unknown within the boundaries of Chili, and lame or deformed persons are very rarely to be met with,” he argued in his article “Back to Nature.” He linked the deformities of rickets and scurvy to decadence:

Scurvy…broke out for the first time in Augustus's reign, at which period we know how luxurious the Romans had become. Not long after, Seneca remarks in one of his epistles, that the Romans had acquired an ambling unsteady gait, from their high living and effeminacy.

As the environmental etiology of rickets became clear in the 1920s, physicians, historians, and archaeologists cast their thoughts backward, looking for evidence of rickets in the ancient past and advancing behavioral theories for the disease’s changing incidence. In 1927, a doctor named J.H. Foote looked for—and found—stigmata of rickets (open fontanelles, distended abdomens, square heads, rib beading) in children in German and Dutch paintings dating back to 1470. He scoured these medieval paintings with a particular goal: to demonstrate, as a popular science writer wrote in 1928, “that rickets is not one more thing that can be blamed on our degenerate times.”

Writers of the 1920s then imagined ancient behaviors. Soranus of Ephesus, writing around 110 AD, had concluded, “country children have straighter limbs than those raised in cities.” The 1928 popular science article quoted Soranus at much greater length: “Frequent examples of such deformity are seen in Rome, due in most cases to ignorance and improper care of the infant. City women devote much less time and care to their infants than those in the rural districts, hence the more frequent occurrence of these deformities among the infants of city dwellers.”

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70 Foote, p. 167.
71 MacDill, p. 164-5.
liver oil into their protesting offspring regardless of kicks and howls, will regard with interest the
dig at the parental carelessness of the ancient Roman ladies.”72 Finally, Foote pointed to the
ancient practice of swaddling infants to keep babies’ legs straight as evidence of early rickets.73
The absence of rickets in Greek and Roman artwork, he believed, was both because those art
traditions celebrated idealism over realism and typically depicted gods rather than mortals, and
because their societies’ relative lack of clothing and sunny climates would have helped prevent
the disease.

In 1929, Hess reported that long-buried Persian skeletons uncovered from ancient
battlefields demonstrated more signs of rickets and “softer skulls” than long-buried Egyptian
skeletons. He speculated that this was because the Egyptians shaved their hair and wore fewer
clothes, while the Persians wore turbans and covered their bodies.74 An archaeological excavation
of a Native American burial mound in Kentucky recovered an arm bone “curved to the point of
deformity” of a pre-Columbian Indian youth, which, as the popular science writer wrote,
“demonstrates that there was at least one aborigine that could have done with tasty doses of cod
liver oil during his papoosehood.”75

**Debating Etiology: Race as a Risk Factor**

African-Americans and Italians were the most common groups targeted for rickets
intervention. Echoing the experts who promoted rickets as a disease of neglect and poverty, *The
Century Book for Mothers: A Practical Guide in the Rearing of Healthy Children* declared rickets

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72 MacDill, p. 165. Later in the article, she also wrote humorously: “The up-to-date female parent, brandishing her cod liver oil bottle, will probably say that the old Greek physicians were only following the common custom of trying to blame it all on the women anyway.”

73 Soranus of Ephesus wrote (in translation), “Since swaddling is an important reinforcement and a preventive of deformities, it is best not to free the infant from its protection until the body has become strong enough to remove all fear of the appearance of irregularities of form. The infant's feet may become crooked from unwise attempts at walking.”


75 MacDill, p. 164.
“a disease which in well-cared-for children rarely reaches a pronounced degree.”

Rickets, the authors concluded, is “undoubtedly due to defective feeding.” They advised their readers, “The striking bony changes spoken of are usually seen among the poor, and in this country most of the cases are found in the children of the negroes and Italian immigrants.”

As historian Rima Apple wrote, “Admitting that the condition was not a general health problem in the United States, investigators were concerned for specific groups here, especially African Americans and immigrants in urban cities.”

Discussion at the American Pediatric Society meeting in 1894 shows the tendency to associate rickets with a particular racial or ethnic group. George Acker, a Washington, D.C.-based physician, presented a review of “Rickets in Negroes,” while Irving Snow, a physician at Buffalo Fresh Air Mission Hospital, described the high incidence of rickets in Italians. This paper looks carefully at each article below.

**Rickets in African-Americans**

Acker echoed the common belief that higher disease rates in African-Americans stemmed from inherent physical differences between races and not just differences in cultural or nutritional practices. “The negro race presents so many peculiarities,” he wrote, “and there are such physical differences between it and the white race.” For example, conventional wisdom held that excessively sweaty heads in infants indicated rickets, and Acker proposed that the “negro has a greater number of cutaneous glands than the white,” meaning that more sweating—and, therefore,
more rickets—would be detected. He linked the higher rates of rickets to changes in living conditions, rather than any change in geographic location, and waxed nostalgic about the supposed health benefits of slavery:

It was to the interest of the master to see that the young were well taken care of…the slaves were fed on food most conductive to their health. Good warm clothing was provided. They had healthy homes and regular hours of work. Care was taken of them when sick and their wants were attended to. Thus the parents were most likely to produce healthy offspring.

In contrast, once freed from this system, he argued that former slaves chose unhealthy behaviors, out of ignorance or worse. “Since their emancipation the negroes have been compelled to take care of themselves, and look after their own interests,” he noted. “Any one who has been brought in contact with them knows of their shiftless, improvident ways.” Although Acker mentioned unsanitary apartments, poor food, and unclean air, most of his explanations for the increased incidence of rickets in African-Americans included similarly broad generalizations about race characteristics. Without the “restraining influences” previously found under slavery, he argued, African-Americans chose to live in “small, damp and dirty habitations” and exhibited a “want of care and cleanliness.” He advocated teaching hygiene in colored schools, noting, “If left to themselves they exhibit a total disregard for the laws of sanitation and hygiene.”

Rather than focusing on unfortunate individuals’ circumstances, Acker pointed to a fundamental unhealthiness of the whole race. “The negro naturally occupies the lowest stratum of society and is consequently subjected to the deteriorating influences that attach to indulgence and low propensities,” he wrote. “His stamina or power of resistance to disease is therefore more or less defective as a general rule.” Acker used language that foreshadowed eugenics, warning, “The race is undergoing serious physical decay.” He argued that producing inexpensive but properly prepared milk “should engage the attention of the health authorities, for we now have a

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82 Acker, p. 141.
83 Acker, p. 137.
84 ibid.
85 Acker, p. 138.
86 ibid.
87 ibid.
prolific race of unhealthy, degenerated beings who will prove a menace to the interests of the country.”

Acker’s paper is quoted at length because it illustrates how disease—in this case, rickets—can be used to call attention to perceived differences between races. He started with a biological fact, traced it back to what he perceived to be its root causes, and generalized about behavior and living circumstances of a particular subgroup in society. He also suggested that the physical signs of disease could be outward signs of more general defects.

Psychiatry lectures given to medical students at Johns Hopkins were published in 1900 as *A Treatise on Mental Diseases*. The Hopkins professor of psychiatry taught that the “negro” race was “still in process of mental development,” and that physical diseases were both a sign and symptom of this delayed mental development, coupled with poor living conditions. The delayed development of the race was not surprising, he taught, because, “The negro has been thrown upon his own physical and mental resources and has entered the strife for existence as an inferior; he is syphilited, alcoholized, his food is oftentimes unsuitable — the prevalence of rickets among negro children being an attestation of this fact — his surroundings are usually unhygienic, and tuberculosis finds in him an easy prey.” In this case, rickets was linked directly to “unsuitable food,” rather than some immutable predisposition, but the physical evidence of disease was linked to larger struggles of the race.

A 1913 article about the health of the “Negro race” pointed to the 25 years after emancipation and directly linked increased rates of disease to social and emotional factors. “For the Negro race it was a time of storm and stress, of unsettled political tendencies, of chimerical ambitions and social unrest,” the author wrote. While he noted that African-Americans had inadequate access to hospitals or medical care, he also pointed out that the economic problems across the race had “lower[ed] its vital resistance,” which then “made it an easy prey for the

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88 Acker, p. 142.
inroads of disease.” Physical disease was then compounded by “nervous tension as the race, with varying success, strove to adjust itself to the larger life of individual and racial freedom.” These emotional, social, and physical difficulties “increased continually because of ignorance and of poverty, of ill-advised schemes of emigration and of overcrowding in large cities,” which then led to a high infant mortality rate. Therefore, he concluded, “The fecundity of the race was diminished while that of the white race increased. Rickets became the characteristic infantile disease of the race; pulmonary tuberculosis of its youth.”

Many physicians believed that public health measures that would work for white Americans would not work as well for black Americans. Even the Hopkins psychiatry professor, quoted earlier, who linked the higher rates of rickets directly to unsuitable food in African-Americans, rather than some immutable predisposition, apparently did not believe that improving the food would solve the problem of rickets in African-Americans. He prescribed a combination of educating mothers and finding an “economical and safe” anti-rachitic food to combat rickets, stating that this combination could “eradicate the disease from the whites, and to a slight degree diminish it among the negroes.”

Rickets in Italian-Americans

Racial links to rickets were not limited to African-Americans. Many writers discussed the increased incidence of rickets among Italians living in the U.S. Irving Snow noted that in his medical practice in Buffalo, 70 percent of the Italian infants had rickets, as compared with 12 percent of the “children of other nationalities.” Rather than focusing on a multitude of physical differences, as Acker had for African-Americans, Dr. Snow noted that this high incidence of

91 P. 173.
rickets was in fact the “only pathologic difference between them and the rest of the community.”

He speculated that the various causes of rickets, including “city life, improper feeding, acute or chronic digestive disorders, bad air, crowding in dwellings, uncleanliness, [and] infectious disease,” may in fact “act with far greater frequency and virulence” on first-generation Neapolitans. Why would this be the case? First, like Acker, Snow referenced cultural stereotypes and wrote, “It goes without saying that from our standard of living their dwellings are dirty, overcrowded, and poorly ventilated.” The problems went beyond infrastructure to personal hygiene and behavior, as he added, “Concerning the hygiene of the skin, the Italian babies were never as clean or as well cared for as the average American, Irish, or German.” There was some hope, though, since “to say that they were, as a class, filthy would be untrue.”

However, had the problem been a simple one of overcrowding or a lack of fresh air, he pointed out that more rickets would also have been expected in recent Irish or Polish immigrants. In fact, more Italian mothers used the “ideal infant food, breast-milk,” which should have helped protect their children from the rickets that could accompany an “artificial diet.” Therefore, Snow concluded, there must be something inherently different about the Italians. More fundamentally, he believed that the increased incidence of rickets was rooted in the move from sunny Sicily to bleak Buffalo. Rickets, he felt, was the sign of a “physical deterioration of a southern race in a northern climate.” He included average temperature and precipitation charts for both regions in his article and argued that in the clear skies of Italy, “the misery, ignorance, and neglect of hygiene amid which the children are reared are in some degree offset by the bright, warm sunshine.” Just as plants should not be moved too quickly from their traditional environment, Snow argued it was “reasonable to suppose that serious changes in physique seen in the first

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92 Dr. wrote, “An Explanation of the Frequency of Rickets Among Neapolitan Children in American Cities.” P. 160.
93 P. 161.
94 P. 165.
95 P. 165
96 P. 161.
97 P. 169.
generation would follow a change of residence from an out-of-door life in a semi-tropical country to the gloomy skies and long winters of the North Atlantic and lake cities.”

It was, however, not the parents who bore the brunt of the change in environment. He believed that adults could move successfully to the “tropics or the Polar Seas,” but that their children would then “endure very badly any marked change in the air to which their ancestors have for centuries been accustomed.” As he concluded, “It is certain that any race not inured to our bleak northern climate must not only change its dress and regimen of life, but undergo a kind of cellular transformation in order to be in harmony with its environments and to preserve a vigorous physique in the second and third generations.”

Debating Etiology: Assimilation/Americanization

When a Yale orthopedic surgeon defined rickets in 1898 as “a constitutional malady acquired through mal-assimilation,” he was referring to a pathological process that would be termed malabsorption today. (The disease, he continued, was “characterized by impaired nutrition and alteration in the growing bones.”) However, the medical term “mal-assimilation” acquired an important social meaning in this period. Rickets was just one of many diseases associated with immigrants in the minds and practices of public health workers in the early twentieth century. Many health workers believed that higher disease incidence among immigrants was partly attributable to poor assimilation to the majority culture.

Promoting assimilation (also called “Americanization”) was particularly important for rickets once the clear links to behavior (and particularly sunlight exposure) made the disease understood as an unspoken choice, a disease that could be both prevented and cured relatively

98 P. 169
99 Proceedings P. 160.
100 Charles Alling Tuttle, “A Short Paper on Rickets,” Transactions of the New York State Medical Association for the Year 1898, p. 202. Presented to the New York State Medical Association in October 1898 and subsequently reprinted over the next two years in both the Yale Medical Journal and Pediatrics.
easily. Rickets prevention work also provided an easy entrée to the homes of immigrants, which was where most “race assimilation” education took place.

The war drives of World War I followed on the heels of the new field of social work with its neighborhood-based settlement houses, pulling women, in particular, into the homes of others. At a large “Americanization Conference” hosted by the U.S. Office of Education in 1919, a speaker called for a “new science of race assimilation” and declared that the U.S was the best possible laboratory for this new science. She pointed to the thousands of women who had been pulled into the war-measure drives as the best possible “scientists,” and inadvertently showed how common this well-meaning intrusion into the lives of immigrants had become. The speaker described her experience of seeking out a Russian infant “who was very sick with rickets” at home:

I climbed seven flights of stairs on a hot August day. I rapped on the door, and a woman opened it a crack, looked at me, and said, ‘Good God, what do you want?’ Do you know what was the trouble? She had been investigated, surveyed, and recruited by numberless people, each with some special thing which they were intent in putting over.

However, the speaker was able to overcome the mother’s natural suspicion; once the woman “understood” that the visitor was there to help the infant, she was “ready to listen.” Disease, once again, led to admittance. The speaker was able to provide a happy ending to the Americanization Conference: “the baby was finally left with the hospital until it was cured.”

S. Josephine Baker, the head of New York’s Bureau of Child Hygiene from 1908 to 1923, often drew distinct links between working with infants and the process of Americanization, in which, as she wrote, the nation “assimilate[s] the vast hordes of alien races that are coming to our shores and being made part of us.” Foreigners, she believed, needed to be given “the kind of mental and moral, social and physical surrounding that will make them fit to be Americans.” As for babies, “When they open their eyes for the first time on this world they are in the same

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position of requiring adjustment to their environment as the immigrant is who gets his first
glimpse of his new life on Ellis Island; only with the baby we have a great advantage. Because his
environment is much more limited, we can control it to a much greater extent, as we do not have
to eradicate any preconceived opinions or ideas.”

Equating immigrants with infants certainly shows patronization, but on the whole the
Bureau’s departmental programs, including those for rickets, acted on the principle that
immigrant mothers, with some Department of Health intervention and education, could do a
beautiful job raising and attending children. In 1922, the same year that the Bureau sponsored the
study on rickets in the Italian district, Baker wrote an article in the *Ladies Home Journal.* “In my
experience,” she stated plainly, “nearly all mothers are fine when they are given half a chance to
know how to be.” She wrote later that many mothers who received visits were “a little flattered
to have an American lady take all that trouble about little Giovanni, and were likely to go out of
their way to learn and to cooperate.”

This faith in immigrant mothers stands in some contrast to the conclusions reached by
Katrina Irving in her book *Immigrant Mothers.* Irving argues that many of the women working
with immigrant mothers during this time period (particularly in the context of settlement houses)
created and publicized the image of a completely incapable immigrant mother, “hapless and
refractory,” because of a “sentimental regime of intelligibility” and the “need to legitimate their
own activities.” She argues, “Depictions of immigrant mothers unable to fulfill their maternal
functions constructed a domain for the authoritative expertise of the newly developed profession of
social work.” Certainly, there was a condescending attitude in the Bureau’s belief that all
mothers in certain tenement districts needed education and reform in raising their infants, but the

103 Ibid, p. 42
105 Ibid, p. 86.
106 Katrina Irving, *Immigrant Mothers: Narratives of Race and Maternity, 1890-1925* (Urbana and Chicago: University of
107 Ibid, p. 84.
Bureau won its legitimacy by publishing ever-improving infant mortality statistics rather than by emphasizing poor or immigrant mothers’ incapability.

In fact, Baker actually held up the compliant tenement mother as an example for wealthier women to aspire to. She pointed to the “curious fact” that it was eminently possible to improve the infant death rate in tenement districts but that not much could be done for sickly wealthy babies. “Sometimes it really looked as if a baby brought up in a dingy tenement room had a better chance to survive its first year, given reasonable care, than a baby born with a silver spoon in its mouth,” she wrote. Inherent in these statements was a challenge and a reproof for her middle- and upper-class readers. Baker subtly led her readers from comfortable beliefs that immigrant mothers in tenements had been ignorant but could not help it to more uncomfortable conclusions: that perhaps they, as, for example, *Ladies Home Journal* readers, were ignorant but could help it. “The truth as it stands today,” Baker stated baldly in 1922, “is that it is not poverty that kills babies, but ignorance, and that this ignorance is largely an attribute of our so-called ‘more intelligent classes.’”

Baker was not alone in chastising “so-called” educated women for their supposed lack of knowledge in raising infants. A “troublesome obstacle that the pediatrist [pediatrician] encounters to-day,” a Philadelphia physician wrote in 1905, is the “general ignorance and helplessness of the young mother. She may be skilled in letters, arts and sciences as a college graduate, but know little or nothing regarding the essential hygiene of early life.” And what should these so-called more intelligent women be doing differently? For Baker, the answer was clear: they should be accepting and acting on the advice of the Bureau of Child Hygiene, which knew the scientifically proven way to raise babies. In the same *Ladies Home Journal* article that faulted the ignorance of the “more intelligent classes”, Baker bemoaned the reluctance of many upper-class women to

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breastfeed. In comparison, she noted, “Owing to their natural inclination and their willingness to accept advice, foreign-born mothers almost universally nurse their babies.”  

Showing how little things have changed in some ways, historian Roberta Bivins published a recent article on postcolonial medicine in the former colonizers. As a case study, she wrote about vitamin D deficiency in South Asian communities in Britain since the 1960s. After showing the debates over adding vitamins to chapatti, the tendency to blame higher rates of rickets on cultural practices of covering skin, and even the unofficial naming of the disease as “Asian Rickets,” she concluded, “diet frequently became a proxy or shorthand for culture (and religion, and race), while disease justified pressure to assimilate.”

**Debating Etiology: Inheriting Rickets and Links to Eugenics**

An important factor in this debate over race degeneracy was whether or not rickets could be passed down from parents. The similarity of rickets symptoms to those observed in congenital syphilis, which also affected the long bones and teeth, lent some credence to the theory that rickets, too, could be congenital. When the Surgeon General for the U.S. Army in the Civil War divided diseases and injuries into five classes, rickets was listed in class four, the “developmental” diseases (along with atrophy, deformity, and congenital malformation), rather than in class one, the “dietic” diseases, where it should have stood next to scurvy. An English physician writing in 1894 about epidemics was adamant: “The congenital nature of rickets is not only an empirical fact, based upon experience, but it is a doctrine of rational pathology.”

Even after the links to nutrition had become more clear, writers continued to link rickets to syphilis (and other so-called “moral” diseases). The History of Prostitution, published in 1895, warned of the “para-syphilitic affections” found in the children of syphilitic mothers. The most

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prominent problems included, “organic and dystrophic troubles, arrest of development (physical and mental), hydrocephalus, rickets, epilepsy, infantile paralysis, tabes, etc.” New York State’s 1912 *Eugenics and Social Welfare Bulletin* blamed a combination of imperfect food (“artificial diet”) and unhygienic conditions for rickets, but argued that “parental syphilis, tuberculosis and alcoholism are predisposing causes by reducing the child's resistance.”

The debate over inheritance had special significance to promoters of eugenics, who often invoked rickets as a physical example of degeneracy. A professor of zoology at the University of California published a frequently cited paper in 1921 (6 years before the infamous *Buck v. Bell* case), which argued that mental illness in parents did not necessarily lead to mental illness in children. Physical illnesses, including rickets, could manifest instead: “What the parents transmit to the children is not insanity, but a vicious constitution which will manifest itself under various forms in epilepsy, hysteria, scrofula, rickets, etc.”

Both Margaret Sanger, in the hugely influential *The Case for Birth Control* and Michael Guyer, in the popular *Being Well-born: An Introduction to Eugenics*, quoted verbatim the same study by a Zurich psychiatrist, which had found that the descendents of alcoholics were more likely to be the victims of murder, to become prostitutes, and to have physical defects—including rickets. Both Sanger and Guyer included this quote: “The offspring tainted with alcoholic blastophthoria suffer various bodily and physical anomalies, among which are dwarfism, rickets, a predisposition to tuberculosis and epilepsy, moral idiocy, and idiocy in general, a predisposition to crime and mental diseases, sexual perversions, loss of suckling in women, and many other misfortunes.”

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118 Margaret Sanger, *The Case for Birth Control: To Aid the Court in its Consideration of the Statute Designed to prevent the Dissemination of Information for Preventing Conception* (Margaret Sanger, 1917), p. 230.
Debating Diagnosis: Clinical Signs

Traditionally, doctors diagnosed rickets using a set of clinical signs, including enlarged epiphyses (at the ends of the long bones), beading of the ribs (often referred to as the “rachitic rosary”), and craniotabes (skull depressions), in addition to the bowed weight-bearing long bones seen in advanced disease. These signs could be subtle and the symptoms nonspecific in mild disease, meaning that different clinicians might assess the same infant differently.

Occasionally, physicians admitted difficulties with diagnosing rickets clinically. In 1905, a dermatologist exploring a possible link between infantile eczema and rickets reexamined a sample of his own young patients more systematically, looking specifically for rib beading, joint enlargement, head sweating during sleep, and belly prominence. He found fewer signs of rickets than he had originally recorded, and concluded that he could not completely trust his older clinical records, making it difficult to provide “definite results.” Specifically, he wrote, “I am convinced that my statistics as regards the presence of rickets would be modified in the direction of lessening the percentage, had I to examine the cases again.” In particular, he wrote, rib beading and joint enlargements proved difficult to evaluate:

The fat wrist of a young baby with the skin attached lightly close over the wrist itself, and the flesh bulging out above, often looks exceedingly like an enlarged lower end of the fore-arm bones, and, I am sure that, in my earlier cases, I have frequently mistaken it for such. Again, I am inclined to think that many babies have a very slight prominence at the junction of the ribs with the cartilages, which does not necessarily indicate rickets. In my desire not to minimise the presence of rickets, I am afraid I have marked these as beaded ribs.120

Debating Diagnosis: Rickets at Bellevue, 1904-1909

The rachitic children admitted to Bellevue between 1904 and 1909 underwent very few tests. A handful had urinalysis or blood work results in their records, and two post-mortem

autopsy reports are included, but no tests, including X-rays, were given as a matter of course. Bellevue definitely had X-ray facilities available (the 1905 article specifically mentions the X-ray room, next to the museum and near the demonstrations of sterile technique), and two of the patients with rickets who underwent surgery had X-rays, but the great majority did not.

The clinical records clearly show the lack of standardization in diagnosis and the subjective nature of diagnosing even the general nutritional state, regardless of the presence of rickets. Record 48, a 7-month old in the hospital in March of 1908, highlights this problem. One doctor wrote, “well nourished, somewhat rachitic looking” while a different examiner wrote about the same child a few days later, “poorly nourished.” Some vital signs were recorded carefully, but these signs had nothing to do with rickets specifically, so that each child’s individual improvement cannot be assessed beyond the occasional clinical note. The chart of each child, without fail, included a detailed temperature record, with temperatures recorded twice a day, along with pulse and respiration rate, and total daily output of urine and excrement, in ounces. In fact, the temperature charts are the *only* records on file for all 80 patients. We may not know the patient’s age, history, progress, or secondary diagnoses, but we know his or her temperature on every night spent in the hospital. Weight, on the other hand, is rarely recorded, which seems surprising because these infants had recognized (even at the time) nutritional deficiencies. Clinicians generally wrote “+” signs to indicate degree of disease, with children given between one and four “+” signs for each of the standard clinical signs. These were recorded at or near admission, but do not seem to have been recorded again, so there is little way of knowing which children’s clinical signs improved dramatically while under treatment.

Rickets diagnosis was complicated by the fact that, often, children came to the attention of the physician with a more pressing disease. The Bellevue casebook is cross-indexed at the front with 18 secondary diagnoses that at least one of the 80 rachitic children carried concurrently. These ranged from pseudoleukemia to eczema, imbecility, convulsions, and measles, and many affected multiple children. Nine additional diseases are listed as
“complications of rachitis,” including whooping cough, bronchitis, curvature of spine, and tetany. Despite these other serious diseases, clinicians made the decision to diagnose the children with rickets as their primary problem. We cannot know for sure why these children had rickets entered as their primary diagnosis, though a doctor at another hospital discussing records for children with rickets in 1894 noted, “We do not enter the patient with the diagnosis of rachitis unless the disease is very severe.” At his hospital, and assumedly at Bellevue, mild cases of rickets were entered as a secondary diagnosis, since “The case is usually admitted for some acute trouble of the digestive and respiratory organs or tuberculosis and treated under that diagnosis.”

Eight of the 80 Bellevue rickets patients died in the hospital, highlighting inconsistencies in recording disease. The mean age for the 67 patients with age recorded was 19 months. However, every patient who died was younger than that. Ranging in age from 6 weeks to 14 months, their average age was only 6.9 months. They were not only younger, but sicker. While the average length of stay across all 80 patients was 29 nights, none of the children who died at Bellevue lived longer than 10 nights, and the mean was only 7.6 nights. Clearly, these patients were already very sick when they were admitted, and although they were filed under rickets as the primary diagnosis, all must have had another problem. After all, rickets in and of itself was not a fatal disease. Notes on their daily medication and status sheets show their poor conditions: “Very poorly nourished” says one; “Vomited green fluid…child died in tetany,” says another. In fact, a post-mortem report included for one child did not mention rickets at all; the autopsy showed that the child died of gastroenteritis.

122 Record 5.
123 Record 18.
Rapid Adoption of X-ray Technology

German physicist Wilhelm Roentgen first detected electromagnetic radiation in the X-ray range in November 1895. He captured the first permanent human image (of his wife’s hand wearing her wedding ring) in late December, and submitted his first paper, including that image, just after Christmas in 1895. By January 1, 1896, he had sent reprints of “A New Kind of Rays: A Preliminary Communication” to a few scientists in Europe. The concept of seeing inside a living body immediately captured the imagination of both popular and scientific writers and, as Bettyann Kevles argued, helped revolutionize Victorian notions of bodily privacy.124 Journalists—attracted, in particular, by the sensational skeletal hand image, which could be reprinted with no scientific interpretation—fueled the explosion of popular interest. The Vienna Presse trumpeted the news on January 5 (just 10 days after Roentgen first submitted his work to a scientific publisher), the Frankfurter Zeitung followed on January 7, the New York Times on January 16. Articles often quoted his wife as saying she had “glimpsed her death,” highlighting the traditional connection between skeletons and death.

The general public accepted the new technology rapidly. By the second week of January, the German emperor had summoned Roentgen to Berlin for a personal demonstration and other European royalty followed suit, requesting personal X-rays and showering honors on the physicist. Rumors swirled around the new technology and cartoonists depicted more nefarious possibilities within days. By February 1896, advertisements in London hawked X-ray proof underclothing, and shortly thereafter a New Jersey Assemblyman introduced a bill prohibiting the use of X-rays in opera glasses.125 Even in more conservative settings, the technology was accepted remarkably quickly: X-rays of a dislocated bone were admitted as evidence in a Denver

court case in March 1896. Initially the new technology was seen as a new kind of popular photography, with, for example, $15 public X-ray booths set up in St. Louis by June 1896.

The medical community hoped to claim the technology as their own and quickly found medical applications. Versions and translations of Roentgen’s original article appeared in seven major medical journals, including *Nature*, *Science*, and *JAMA* by mid-February 1896. In February of 1896, articles advised using the films for dental studies, while articles in April demonstrated that radiographic wrist development could help determine bone age. Within a year of Roentgen’s original article, more than 1000 articles and 49 books had been published on the subject. By May 1896, the first scientific journal specifically designated for radiology was being published out of London, *Archives of Clinical Skiagraphy*. Within a decade (in 1906), the *American Journal of Roentgenology* was founded. In 1901, just five years after his original paper, Roentgen won the first Nobel Prize given in physics. The American Medical Association officially recognized a Section for radiology in 1925.

The biggest appeal of the new technology was its “supposed objectivity.” X-rays clearly showed problems in the epiphyseal ends of long bones and allowed for positive rickets diagnoses. Even better, lines on X-rays showed new deposits of calcium, giving a visual indication of the healing of rickets when a patient was under appropriate treatment. Just as in tuberculosis, where the “radiographic image itself would eventually come to represent the disease,” X-ray studies of rickets in both research and clinical practice came to prove the existence of rickets and to test proposed treatments. Even more important, X-rays both required expert opinion and could find disease before it had appeared clinically. A Boston physician in

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127 Dewing, p. 85.
128 Dewing, p. 31.
130 Lerner, p. 389.
1915 published an article in *JAMA*, for example, explaining that he had uncovered more than 500 cases of rickets using the new technique of X-ray diagnosis.\(^{131}\)

But radiology had to fight hard for its status as a recognized medical specialty.\(^{132}\) As Eugene Caldwell, the Director of X-ray at Bellevue, warned the New York State Medical Society in 1909, “The almost irradicable impression of radiographs as a picture or photograph which anyone can properly examine, interpret and criticize, has been a great hindrance to the progress of roentgenology and its proper recognition.” He warned that without training, “laymen and medical men alike” provided “overconfident interpretations” that could be dangerous.\(^{133}\) Caldwell had been an electrical engineer when news of Roentgen’s discovery spread. He purchased an X-ray apparatus in 1897, started work as an “X-ray photographer,” and founded “probably the first X-ray laboratory in New York City.”\(^{134}\) Notably, he was soon hired both to take films and to teach roentgenology at Bellevue, despite his lack of medical training.\(^{135}\) The instructor then became a student, formally enrolling in Bellevue’s medical school to earn his M.D. degree in 1902, reflecting the argument that formal medical expertise was necessary to properly interpret the images.\(^{136}\) The original X-ray photographer at Massachusetts General Hospital, Walter Dodd, followed a strikingly similar course, also earning his M.D. degree in the program where he taught.\(^{137}\)

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\(^{136}\) Caldwell published the respected book *The Practical Application of Roentgen Rays in Therapeutics* in 1903. He continued to work and conduct research at Bellevue, including developing the first portable X-ray. Unfortunately, the cumulative exposure to years of radiation led to his early death of “X-ray burns,” chronic skin changes that looked like burns.

Hess and Unger’s Community Study, 1917

By 1917, with cod liver oil considered a well-established therapeutic agent, Hess decided to “investigate whether its usefulness could not be extended to the prophylactic treatment of rickets.”138 Hess and Unger’s classic 1917 study “Prophylactic Therapy for Rickets in a Negro Community” moved research into the community, rather than waiting for cases to come to the hospital. The study gave cod liver oil to infants, regardless of whether they had signs of rickets, in one of the first community-based public health experiments in the world. It was done in the Columbus Hill district in part because the section had been thoroughly surveyed by the Henry Street settlement and by the National League for the Study of the Urban Conditions of the Negro.139 Details about the residents’ economic status, living conditions, morbidity, and mortality painted a grim picture. Infant mortality across New York City in 1915 was measured at 94 per thousand births among white infants and 202 per thousand births among black infants. Columbus Hill, with its population of about 9,000, had the highest infant mortality of any neighborhood in the city--314 per thousand births.140

The results of this community-based study were outstanding. Hess and Unger demonstrated a dramatic improvement in rickets incidence by giving cod liver oil to 80 black infants prophylactically, feeding the children cod liver oil three times daily. More than four-fifths of the infants who took the oil for six months never developed rickets, while 15 of the 16 infants who did not take the oil showed some signs of rickets, “though all of them lived under the same conditions and many in the very same families.”141

Hess and Unger published the study results in the Journal of the American Medical Association (JAMA) and, in light of their results, suggested offering cod liver oil at the baby

stations that had been recently established around New York City. *JAMA* also published a letter from the commissioner of the New York City Department of Health responding to the suggestion. The commissioner complained that too many people wanted to use the health department to “promote or exploit some special substance” and argued that it would be “just as logical to dispense sugar or milk or limewater.”\(^ {142}\) Such a negative reaction suggests that the health commissioner had been down this road before. Clearly, other people had already appreciated the infrastructure of milk stations for reaching communities of young children and the commissioner wanted to protect the city’s prized investment. However, Hess’ results proved too conclusive. After some debate, the city started educating mothers and providing cod liver oil at cost through milk stations, in one of the earliest preventive, prophylactic health campaigns for a non-infectious disease.

“The prevalence of rickets, among negroes and Italians, has generally been attributed to racial susceptibility,” the editors of *American Medicine* wrote in November 1917. However, they felt the experiments by Hess and Unger, “demonstrate conclusively that, so far as the colored race is concerned, the development of rickets is based upon some dietetic insufficiency which appears to be satisfied by the administration of cod-liver oil.” Diet, not inherent racial susceptibility, was the culprit.

The writer continued, “Inasmuch as the economic status of the negro population is distinctly bad and the degree of education is notably inferior, it is natural that the infant mortality rate should be far higher for the black race than for the white. If, however, groups similar, economically and educationally, were selected among the blacks and whites the difference would probably be far less than is evident in the infant mortality rate for 1916 of 193.3 for New York City negro infants as against 91.2 for the white babies.\(^ {143}\)


The idea of using cod liver oil to help prevent rickets and not just to treat it was not a novel concept in 1917. It was still relatively untested, but animal experiments had given some promising results. As an editorial in *American Medicine* wished, “It is to be hoped that cod-liver oil will prove itself to be as effective in preventing rickets as orange juice is in inhibiting the appearance of scurvy.” Assuming ongoing investigations continued to prove successful, the editorial advocated using “the widespread organization of infant welfare stations… for attacking the problems of rickets along preventive lines” by dispensing cod liver oil “at cost, or at public expense.” The editors pointed to precedents for using this kind of public health framework to dispense a particular prophylactic or treatment in the example of giving quinine for preventing malaria or oil of chenopodium for hookworm.  

However, the editors explicitly stated what many others had hinted at, that cod liver oil would not work well for African-American infants, as the problems were too great and the population too poor and ignorant. “Too great dependence, however, must not be placed upon cod-liver oil as a means of attacking the infant mortality rate among negro children,” the editorial concluded. “The relation of ignorance and poverty and the concomitant inadequacies of the maternal and infantile dietary will continue to challenge the attention of those striving to reduce the infant mortality rate of the negro population.”

**AICP and Bureau of Child Hygiene Community Study, 1922**

The Association for Improving the Condition of the Poor (AICP) founded the Mulberry Health Center in New York City in 1918 to serve the Italian section between Canal and Houston Streets and Bowery and Broadway (pop. 40,000). The Center focused on preventive maternal and young child health, and John Duffy’s single mention of rickets in his two-volume history of public health in New York City came in a discussion of this center. Initially, the AICP began

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Long-term sequelae of rickets meant that one in four preschoolers in the district had “serious orthopedic defects,” and the AICP had noted an “alarming” mortality of children under five from pneumonia and other respiratory diseases “in part due to the deformed chest and the low muscle tone which follows severe rickets.” The area was nearly all Italian, and rickets were prominent, the AICP wrote, because “Italians are perhaps of all races most inclined to keep their babies indoors throughout the winter months.”

The AICP approached the Bureau of Child Health in 1922, suggesting a study in the district involving the routine administration of cod liver oil. Beginning that September, nurses targeted all babies three to nine months old who were registered at the baby health stations in the Mulberry District, with a goal of making cod liver oil administration the standard of care for mothers. However, this broad goal proved ineffective—mothers rarely give the oil, and the clinical data was “not high quality.” So, beginning in January 1923, the investigators focused more attention on the study by limiting it to just four months, with a goal of registering 200 infants. (As the report noted, “It was hoped that of these 200, at least 100 could be counted on to take the oil regularly and that the balance would serve as a control group.”) The committee also decided to advise giving the cod liver oil three times a day, to promote its importance. Finally, the investigators agreed that it would be “important to have X-ray plates made of the wrists of the children both at the beginning of the experiment and at its close because it was generally agreed that for purpose of such a study a diagnosis based upon X-ray findings is infinitely preferable to one based upon signs usually noted by physicians in a physical examination of the baby.”

The investigators managed to enroll 150 infants in a 3-week enrollment period, then attempted to visit these infants at home three times each week to encourage the use of the cod liver oil.
liver oil and examine the bottles (which, as the committee noted in an understatement, “called for the greatest amount of tact and perseverance”).\textsuperscript{150} Despite this frequent attention, the mothers of 36 of the 150 infants either refused to purchase the cod liver oil or did not give it to their infants regularly. The investigators estimated adherence by dividing the group based on how many four-ounce bottles of cod liver oil the mothers purchased. Those who purchased at least three bottles and who generally reported giving the oil daily were compared to the group who bought two to three bottles but did not necessarily give it daily, and finally to those who purchased one or no bottles.

Although the investigators had made arrangements for the infants’ initial radiographs to be taken at Bellevue Hospital, only 23 of the 150 actually had enrollment X-rays taken “because of the difficulty of persuading mothers to take these babies so far from home.” But by the end of the four-month study, arrangements had been made for infants to have X-rays taken at a dispensary in the Mulberry District, and 104 of the 150 families obliged. Hess read the radiographs at the end of the study and “indicated presence or absence of rickets,” with a small “doubtful” category for indeterminate cases. Of the 104 infants with a final X-ray, 35 had taken the oil regularly, 45 irregularly, and 24 had not taken it.\textsuperscript{151} Even among the 35 infants taking cod liver oil regularly, 7 were found to have definite rickets (and 3 more had “doubtful” rickets). In contrast, among the 24 infants who had not taken the oil, 16 had definite rickets (and 1 more had “doubtful” rickets), and among the 45 who had taken it irregularly, 27 had rickets (and 1 more had “doubtful” rickets).

The investigators presented the use of X-ray in diagnosis as the major strength of their study. They pointed out that the number of cases observed was larger than any prior study, and called attention to the frequent home visits (so the “statements of the mothers were carefully checked”), but the fact that the “appraisal of results was based upon X-ray findings and not

\textsuperscript{150} Gebhart, p. 572.
\textsuperscript{151} P. 572,
counted on clinical diagnoses,” was most important.152 “Diagnosis was based on radiographic findings and not on clinical examinations,” the study reported proudly, even stating, “There was in fact very little correlation between the radiographs and clinical findings.” The final report suggested two reasons for the divergent X-ray and clinical findings. First, with a clear nod to the need for experts and a mention of the X-ray’s objectivity: “The diagnosis of rickets in early infancy calls for greater skills than the average physician, not especially trained in this field, is likely to possess, so that their findings are not the equivalent of an objective test.” Second, in a more debatable conclusion, “it has been pointed out that the rachitic ‘rosary’, enlarged epiphyses, etc., can be observed clinically long before defective bone formation can be portrayed by the X-ray. Similarly these signs may persist for some time after the X-ray indicates that the rickets has healed.”153 Other investigators found that the X-ray changes came before the clinical changes; the fact that this study drew the opposite conclusion suggests how variable even the most basic conclusions could be.

The study concluded, in a somewhat convoluted sentence, “The fact that 71 per cent of those taking the oil regularly showed no rickets, whereas only 30 per cent of those who took no oil did show rickets is ample evidence that the measure was effective.” In their final conclusion, the investigators wrote that they had demonstrated that, “mothers can be induced by means of systematic home visiting to give cod liver oil to their babies for the three winter months and that at least 70 per cent of such babies can be prevented from developing rickets in a district where this disorder is practically universal.”154

New Haven Rickets Study: Changing Prevalence Through Technology

Just a few years later, in October 1923, the New Haven rickets study began. This study, co-sponsored by the U.S. Children’s Bureau and the Pediatric Department of the Yale School of

152 p. 574.
153 p. 573.
154 P. 574.
Medicine, also had support from the New Haven Health Department, New Haven Medical Association, Department of Public Health of the Yale School of Medicine, and the Visiting Nurse Association. It was known, Eliot wrote in 1925, that cod liver oil and sunlight could “influence the cure of rickets,” and this was designed instead to be a wide-scale study of prevention. It was fundamentally a demonstration study, with a goal of reaching all children in a catchment area, regardless of parental involvement or income.

The study lasted three years and focused on one district of New Haven with 13,500 people, “one third of which were negroes, and two thirds a mixed population composed of Italians, Irish, Polish, and Americans.” Three physicians, three public health nurses, two social investigators, a roentgen-ray technician and a secretary made up the team. When infants were born in the district, their mothers were instructed to begin cod-liver oil supplementation and sunbaths (if possible) in their first month of life. Mothers of any infant born between March 1 and September 1 were instructed to begin outdoor sunbaths within the first month of life; others were to allow their children to have sunlight exposure through open windows. Nurses visited New Haven homes that had new infants, demonstrating how to give the oil and sunbaths to babies, then returned frequently “to see whether the instructions are being carried out.” Once per month, mothers were instructed to bring in their infants “for physical and roentgen-ray examinations in order that rickets may be discovered as early as possible.”

Archived minutes and discussion from all of the advisory committee meetings for the New Haven Rickets Study (beginning with the first meeting, in October 1923) help illuminate how the study evolved over its three years, and how preliminary diagnostic findings altered some

of the participating physicians’ fundamental understanding and definition of the disease. As with any new study, much of the early committee discussion addressed practical details of space, equipment, records, and recruitment. A typical discussion for 1923, at the third meeting of the advisory committee, included proposals for lantern slides for the “local moving picture theatres.” An interesting decision was made at the fourth meeting, the following month, to “lay no emphasis on Rickets” when discussing the study with mothers. The committee suggested instead saying or writing in promotional materials, “The U.S. Children’s Bureau and the New Haven Board of Health are working for the health of the children in your neighborhood. Your baby and children can be weighed and measured to see if they are growing strong and growing straight.” This wording promoted the physical exam for health that was central to preventive medicine, with the implication that healthy children would measure up to new, explicitly defined standards.

The New Haven Visiting Nurse Association cards recorded citizenship, years in the U.S., years in New Haven, and name of church for the “man” and “wife” of the household, along with date and place of birth, physical condition or date of death, occupation, and insurance status for man, wife, and child. The nurses also recorded the “home condition,” including whether the child lived in a house or tenement, whether it was rented, and whether the home used a milk dealer. Nurses marked the number of rooms in the home, then whether each was “overcrowded, clean, or dirty”; in “sunshine, light, dim or dark;” with “good, fair, or poor” ventilation; and with “good, fair, or poor” sanitation. They were also expected to record if a family was “destitute or partially destitute,” whether it was known to a social agency, and to assess familial “habits” (e.g. conscientious or careless; intelligent or ignorant).

An even more detailed form, apparently used for the initial visit for children enrolled in the intervention arm included 49 questions. These forms recorded whether either parent had

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158 Administrative papers related to the New Haven Rickets Study are available at Archives and Manuscripts, Yale University. These advisory committee meeting minutes and discussion are available, with other forms, in the Winslow papers. Winslow, a prominent pediatrician at Yale, was a vocal member of the advisory committee.

159 Charles-Edward Amory Winslow Papers (box 36, folders 99-101). Yale Manuscripts and Archives.

syphilis or tuberculosis, whether there were lodgers in the home, and even whether the mother had had help with housework during her confinement. They captured more details of housing (question 14, for example, asked about the number of “dark” rooms, their use, how many feet to the opposite wall or the opposite building out the window, and question 15 asked about the number of “sunny” rooms, their use, their exposure, and the exact number of hours of sun).

Nurses recorded the position of the bed, whether there was a porch, whether there was a yard (and if so, its size, and whether it was sunny, dry, dark, or wet), whether the house was “clean, dirty, or filthy,” whether screens were in place, and where the toilet was located. Finally, details about the infant included his or her temperament (excitable, phlegmatic), intelligence, how many hours the child spent in the sun, where the child napped, his or her development and birth history, and every detail of feeding (even including, for example, which month green vegetables versus white vegetables were introduced into the child’s diet).^161

Using the new X-ray definitions, the New Haven Rickets Study found rickets in almost every child examined. Even among the 216 children in the “demonstration” (the ones with all of the attention of the study, including heavily promoted cod liver oil and sunbaths), a full 96% were found to have rickets. In fact, this nearly universal prevalence in the demonstration infants was even higher than the prevalence in the control groups—which, at 89% to 91%, in the different groups, was almost universal there, as well.^162 On first glance, this suggests that the demonstration was in fact a failure—if the goal was preventing rickets, only 4% of the children successfully avoided the disease, and more children not in the demonstration escaped a rickets diagnosis.

The researchers therefore redefined study success. Instead of total prevention of rickets, the final reports instead focused on prevention of moderate or severe (“marked”) rickets. Here,

^161 Advisory committee minutes, medical examination forms, and other administrative papers from the New Haven Rickets Study can be found in the subject files in the Charles-Edward Amory Winslow Papers (box 36, folders 99-101). Yale Manuscripts and Archives.

^162 Ibid., p. 660, chart 4.
the numbers looked better, as classified by X-ray. No infants in the demonstration developed marked rickets and only 4.3 percent developed moderate rickets “shown by roentgen ray.” In contrast 23% of the first control group and 34% of the second control group developed either moderate or marked rickets. The New Haven Rickets Study reports finally concluded that the treatment “has been successful in keeping them from developing advanced rickets.” They also subdivided the study infants into those with and without “cooperative mothers,” as well as other subgroups and drew more positive conclusions.

By the 14th meeting (February 1925) the committee had moved on to new debates about how to best diagnose the rickets that was, after all, the study’s primary outcome. Specifically, the committee discussed “whether the diagnosis of clinical rickets was becoming too refined or not.” Fault was assigned to the clinical diagnosis procedure rather than the radiographic findings, as a “certain percentage of infants are being given a diagnosis of rickets on clinical findings only.” The committee speculated, “[T]he evidences upon which the clinical diagnosis is now being made may not in reality be rickets.” On reviewing the X-rays for these cases, in several cases “though the X-rays do not definitely show rickets, at the same time they are not absolutely normal.” A new category was added: children were now recorded as having or not having rickets, but could also carry the label of “probably” and the more complicated label “no rickets on physical exam” (though noted on X-ray). At the 15th meeting (April 1925), Dr. Eliot reported that all X-rays had been reviewed “and that the number showing clinical rickets but no definite X-rays had been reduced greatly.” Although on this retrospective review, rickets was now more clearly seen on X-ray, “the degree of rickets is very slight in this group [c]ompared with that seen by children living outside the district and who have taken no antirachitic treatment.” Eliot argued that X-rays were necessary to find rickets at the earliest possible time. “The importance of the fact that

163 Ibid.
definite clinical signs of rickets do not appear until after the roentgen-ray evidence cannot be
overemphasized when considering the problem of prevention,” she wrote in 1925.165

The nearly universal X-ray finings for rickets contrasts with how X-rays for tuberculosis
ruled tuberculosis out in patients as often as it ruled it in, helping to refine the disease. A typical
description of an early use of the technology for tuberculosis described why the X-ray was useful.
“Physical signs of increased density at the apex and rales, if not confirmed by an X-ray
examination, may be questioned in cases where the history and other evidence do not bear out
these signs,” the doctor wrote. While the X-ray in rickets mainly found disease where no clinical
complaints existed, ruling in the disease, the X-ray in tuberculosis helped refine clinical signs and
symptoms, at times ruling out the disease. As the physician wrote in 1899, “I have seen seven
cases where an X ray examination prevented my taking too unfavorable a view, though the
physical signs indicated beginning tuberculosis. In none of these has tuberculosis developed, and
in such of them as I could test with tuberculin there was no reaction.”166

Eliot became well known for her expertise in reading the X-rays of children with rickets.
A physician who had trained under Dr. Eliot at Yale remembered that in 1934, a Canadian
physician was finishing a large rickets study, “and decided that he needed to have Martha Eliot
review critically all the roentgenograms before making his final report.”167 This doctor packed
three trunks of films, took the train to New Haven, and arrived “at the same time as a monumental
snow storm.” As the former resident recalled, house staff were sent to pick up the films on
snowshoes so Eliot could complete the readings before leaving for Washington.

Using X-ray rather than clinical presentation as the gold standard for diagnosis yielded
nearly universal disease. Each month, investigators took X-rays of both forearms of the infants.
This repeated examination schedule meant that more rickets was diagnosed. These problems

165 Ibid.
166 Francis H. Williams, “Roentgen Ray Examinations in Incipient Pulmonary Tuberculosis,” The Medical News, 1899, 75: 356.
extended to the X-rays themselves, since the study classified “a doubtful roentgenogram” in control groups as negative, while in the demonstration group the doubtful X-ray was “usually followed in a month or two by a positive one.” As the committee concluded, “The total incidence of rickets in the demonstration group (96 per cent.) is undoubtedly higher than in the control groups, because, as a result of repeated examinations, evidence of transient rickets by the roentgen ray, and slight rickets by clinical signs, has been found more frequently than in the control groups in which only one examination has been made.”

Still, this study’s results were jarring, as the disease previously associated with risk factors like race and poverty was now universal. As Eliot wrote, “Our investigations have shown that a slight degree of rickets is well-nigh universal in our climate and in our state of society.” She even broached a controversial subject, writing, “The very intimate association of rickets with growth, its early appearance regardless of season, and its universality raise the question…whether this slight degree of rickets must not be considered normal.” However, Eliot quickly clarified that she could not even discuss that provocative question yet, stating, “Before this question could be answered, it would be necessary to study the amount of rickets existing in a group of young infants born and living in the tropics.” This idea of studying a group of infants in the tropics was not idle speculation. Concurrently with the later years of the New Haven Rickets Study, Eliot and others from the federal Children’s Bureau investigated rickets in Puerto Rico.

**Changing Prevalence Through Technology: Puerto Rico**

Fred Fleagle, an American sociologist who was the dean of the “University of Porto Rico” in 1917, portrayed rickets and malnutrition as the natural condition of Puerto Rico. Fleagle’s book *Social Problems in Porto Rico* discussed the 26,572 deaths that had been recorded

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169 Ibid.

169 Ibid., p. 661.

170 Ibid., p. 661.

in the previous year. Of these 26,572 deaths, 1,271 had been attributed to rickets, making it the fourth most common cause of mortality, after “diarrhea and enteritis under two years” (3,485 deaths), tuberculosis (2,125) and malaria (1,290). Fleagle commented on this “exceptionally high death rate” from rickets and described a study that found that Puerto Rican university students had smaller chests than their American counterparts, suggesting a “general softening of the bones in early childhood.” The 1918 Annual War Reports of Porto Rico had targeted rickets as well as a sign of degeneration and a good target for the principles of eugenics. The report advised, “[T]he best sanitary efforts should be directed toward making good the lives of those born under the handicap of family diseases; under the stigma of degeneration including everything from rickets to lunacy, and then toward full entrance into the field of eugenics…”

Intrigued by this high death rate in the sunny tropical climate, and looking to assess whether rickets was truly universal in sunnier parts of the world, Eliot and others from New Haven and the U.S. Children’s Bureau brought an X-ray machine to Puerto Rico and completed a “detailed clinical, chemical and roentgenologic” study of 584 infants. Eliot gave a lecture during the inaugural year of the School of Tropical Medicine of the University of Porto Rico in March of 1927, describing her rickets work in both New Haven and Puerto Rico. In sharp contrast to New Haven, where the X-ray had diagnosed rickets in all but a few infants, in Puerto Rico, the investigators found only three mild cases of the disease and one very severe, despite the reportedly high mortality rate. In fact, the one very severe case turned out to be an illegitimate child who was “such a source of shame” that he had been hidden in the house. When the child

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172 Fleagle, p. 77. Rickets caused more deaths than typhoid, whooping cough, tetanus, cancer, meningitis, epilepsy, acute bronchitis, chronic bronchitis, bronco-pneumonia, pneumonia, “diarrhea and enteritis two years and over,” infantile tetanus, “lack of care in infancy,” congenital debility in children, unciniarisis [hookworm], smallpox, or diphtheria.
174 Roland W. Moskowitz, Rickets Including Osteomalacia and Tetany (Lea & Febiger, 1975), p. 55. The high mortality from rickets was described by other authors, too: the 1919-1920 death rate per 100,000 in Puerto Rico was 186 per 100,000 for tuberculosis, 121.2 for malaria—and 108.9 for rickets.
was brought into the sun, “lesions were well on the way to complete healing by the end of 3 weeks.”

The same preventive medicine experts who had redefined rickets in New Haven as a nearly universal disease now used the same X-ray machines in Puerto Rico to shift the definition of rickets in that setting from a common and deadly disease to a rare, relatively innocuous disease. A 1957 *Pediatrics* article described Eliot’s Puerto Rico work as one of her most influential studies. “Rickets had been listed on death certificates with great frequency as a cause of death among Puerto Rican infants,” the article noted, “yet rickets on a tropical island, noted for its sunshine, seemed out of place, to say the least.” The article praised the investigators for “carefully investigating the accuracy of a medical diagnosis which has an unusual incidence, in order to have a more realistic appraisal of problems in the area.” It concluded succinctly, “As one might expect, ‘rickets’ disappeared promptly from the death certificates.”

**Standardizing Treatment**

**Treatment at Bellevue, 1904-1909**

The most striking aspect of the children’s records at Bellevue is the simple lack of consistency. There seems to have been no standard treatment for children with rickets. Of the 80 patients, only 24--30 percent--received any cod liver oil (often abbreviated C.L.O. in the medication lists) at all. Castor oil was given as often as cod liver oil, and calomel, Bashman’s mix, barley water, brandy, beef juice, sodium bromide, whiskey, antipyrine, pot. bromide, ferric iodine, syrup of wild cherry, “special cough mist,” and turpentine were among other treatments dispensed. Six patients received orange juice, which was listed under medications instead of under diet. (Orange juice was a known specific treatment for scurvy, a disease eventually discovered to be caused by vitamin C deficiency.) Often, there is not consistency even within a

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patient’s treatment. A 22-month-old, Record 49, was not given cod liver oil until her fifth day of an eight-night stay. Record 11 had been in the hospital for more than three months before taking any cod liver oil. Record 4 received cod liver oil only on the last day of her stay. Some children took cod liver oil for a day or two and then never again.

No patterns are obvious for determining which patients received cod liver oil; its use is spread sporadically through the years and even the same doctors seem to have used it only occasionally. Clearly, the oil’s efficacy had not been proven or widely accepted in clinical practice. In at least some cases, patients on cod liver oil improved quickly and noticeably, as would be expected. Record 16, a one-year-old who was admitted November 10, 1904, and given cod liver oil daily was discharged to her mother on December 4 with the note “under proper feeding and cod liver oil has gained 10 lb in 25 days.” Curiously, though, a 13-month-old with the same primary diagnosis of rickets, as well as specific “rachitic signs” noted in his chart, was in the same ward at the same time (admitted November 16, 1904, and discharged November 19) as the one-year-old who improved so much on the daily dose of cod liver oil. He never received the oil once, or in fact any medications, and was instead given only milk and limewater for the three nights he spent in the ward. Another child admitted December 2, 1904, two days before the one-year-old was released, never received cod liver oil either. Even cases with severe rickets did not receive cod liver oil with any consistency. Record 12, whose history sheet read “Rachitic signs ++++” (the most pluses given) was never given cod liver oil.

Even most of the patients who eventually died in the hospital, suggesting they were very ill on arrival, never received any medication. A 5-month old was given only castor oil and barley water until he died. A 6-month old received milk with sugar, barley water, and brandy. A 6-week-old (who must have been too young even to have rickets clearly diagnosed) took barley water
with sugar added. No medication notes are listed at all for a fourth child. Only two of the eight children who died received cod liver oil (along with barley water, castor oil, and orange juice).\(^{178}\)

Some hospitals may have had more standard treatment regiments. Ten years before these children were treated, a doctor from Children’s Hospital in Washington, D.C. (while arguing that cod liver oil was only useful to suspend the phosphorous he believed was the crucial ingredient), mentioned his hospital’s standard treatment. For 20 years, he explained, children with rickets had been given a “phosphatic emulsion,” using “cod liver oil combined with yolks of eggs, New England rum, dilute phosphoric acid, glycerine, and flavored with oil of bitter almonds and orange water.”\(^{179}\) If Bellevue had a similar concoction, it was not given in practice.

**Cod Liver Oil**

Cod liver oil had, of course, been used for centuries as a non-specific treatment for a range of diseases.\(^{180}\) Generations of children had learned to hate the taste and smell of the black oily liquid, and then grown up to be parents who in turn hated to force it down their children’s throats. Occasional papers before 1900 pointed to its efficacy for rickets and most textbooks of the early 1900s mentioned it as a treatment option, but its status as a general folk remedy led many in the scientific community to reject it as a specific antidote for rickets.

Many of the country’s most well known physicians, including those most important in the founding of pediatrics as a separate discipline, did original research on rickets while practicing or teaching at Bellevue. Some of them advocated cod liver oil specifically. The preeminent early example is Job Lewis Smith, who entered medical practice at Bellevue just before the Civil War.\(^{181}\) Smith published many papers investigating infectious disease, tetanus, and rickets, and

\(^{178}\) Records 5 and 69.
\(^{179}\) (1894 peds proceeding 176).
\(^{180}\) probably first in Norway for rheumatism
\(^{181}\) Smith spent most of his career at Bellevue, where he was first named Clinical Professor of Morbid Anatomy in 1861 and then Clinical Professor of Diseases of Children in 1876.
was the primary organizer and founder of the American Pediatric Society.\footnote{Semi-Centennial Volume of the American Pediatric Society 1888-1938 (New York: American Pediatric Society, 1938), Introduction.} As early as 1893 he advocated using cod liver oil in treating rickets (with references to experiments with cod liver oil on rachitic lion cubs at the London Zoo). However, showing the gap between scientific and clinical medicine and the lack of a standardized approach to treating rickets, this advice on cod liver oil was not taken at the institution where he had won his fame. As Hess (who later worked at Bellevue himself) wrote,

> It is a sad commentary on the fallibility of clinical medicine that a therapeutic action as well defined as cod-liver oil could not be conclusively established by means of bedside observation, carried out in many countries over a period of one hundred years, but that physicians had to await the proof of its specificity from non-clinical sources.\footnote{Hess, \textit{Rickets} 406.}

Researchers instead proposed many other substances. In 1899, many physicians believed that a deficiency of lime led to rickets, and physicians advocated close monitoring of inorganic salt intake.\footnote{Louise E. Hogan, \textit{How To Feed Children: A Manual for Mothers, Nurses, and Physicians}, 3rd ed. (Philadelphia: J.B. Lippincott, 1899), p. 86.} At the American Medical Association (AMA) convention in 1908, one physician stated that he had cured 198 out of 200 rickets cases using phosphorus, making it much more effective than cod liver oil. However, in the ensuing discussion, a Boston physician countered that he had found little benefit with phosphorus, even in those patients who had been given so much phosphorus that he “feared they would light up if rubbed too hard.”\footnote{M. P. Hatfield, \textit{A Compendium of Disease of Children} (Philadelphia: Blakiston, 1891), p. 63. Cited in Mary Theodora Weick, “A History of Rickets in the United States,” \textit{The American Journal of Clinical Nutrition}, 1967, 20: 1236. A menu recommended for a rachitic child between age one and two in the 1890s: “Breakfast—Milk and crackers or toast, or cracked wheat. 11 AM—Milk and lime water. 2 PM—Chop, gravy, and stale bread; cauliflower. 6 PM—Like breakfast, or egg, if not meat at noon. 8 PM—Milk and lime water.”}

Sixteen years later, the topic was still being debated at AMA conferences. At the 1924 conference, Howland called cod liver oil “as specific an agent as we have in the whole field of

\begin{thebibliography}{9}
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\item 183 Hess, \textit{Rickets} 406.
\item 185 M. P. Hatfield, \textit{A Compendium of Disease of Children} (Philadelphia: Blakiston, 1891), p. 63. Cited in Mary Theodora Weick, “A History of Rickets in the United States,” \textit{The American Journal of Clinical Nutrition}, 1967, 20: 1236. A menu recommended for a rachitic child between age one and two in the 1890s: “Breakfast—Milk and crackers or toast, or cracked wheat. 11 AM—Milk and lime water. 2 PM—Chop, gravy, and stale bread; cauliflower. 6 PM—Like breakfast, or egg, if not meat at noon. 8 PM—Milk and lime water.”
\item 186 T. S. Southworth, “The importance of the early recognition and treatment of rachitis,” \textit{Journal of the American Medical Association}, 1908, 50: 89. The group discussion that followed this paper presentation was also discussed in Weick, p. 1236.
\end{thebibliography}
medicine,” but was still met with resistance.187 The AMA continued to list phosphorus as a specific treatment for rickets until 1928.188 As Harrison wrote, “Many pediatric authorities as late as the early 1920’s considered cod-liver oil of no greater therapeutic benefit in rickets than other fats; one eminent pediatric authority, for example, recommended olive oil instead as being much more palatable than cod-liver oil and in his opinion equally effective.”189

Advice books for mothers were similarly circumspect. A 1914 publication of the Federal Children’s Bureau stated that appropriate food, sunshine, and fresh air could help prevent and cure rickets but did not specifically mention cod liver oil.190 Only in 1929 (after both Viosterol and irradiated whole milk were on the market), did the popular “Infant Care” publication of the Children’s Bureau start officially recommending cod liver oil.191 The suggested treatments for rickets in popular literature before the 1920s were as varied as they were ineffective. Mothers swaddled babies tightly to keep their limbs straight, rubbed and massaged their muscles, and fed them a wide variety of supplements. A Kellogg booklet for mothers encouraged expectant mothers to eat whole grains to help prevent rickets in their unborn children, and advised giving rickety children prunes and wheat germ to cause them to have at least four bowel movements each day.192 A 1916 booklet by a chiropracter promised, “by means of the right adjustment of the spine, massages, hygienic nutrition, water and pure air, you can prevent rachitis from attacking those children who are predisposed to it.”193

The slow universal acceptance of cod liver oil was likely due in part to variations in the quality of the cod liver oil sold by unregulated companies. In a 1926 study, for example, all but

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189 Harrison 735.
190 Mary T. Weick. 1236.
191 Weick, 1238.
one of 36 infants studied developed rickets, though all had received cod liver oil.\textsuperscript{194} On investigation, the Department of Agriculture found little to no actual vitamin D in the preparation that had been sold as cod liver oil.\textsuperscript{195} This explains why many studies discussed the particular brand or provenance of the oil they had used.

**Treatment: Cod Liver Oil and Consumerism**

By 1921, three vitamins (vitamins A, B, and C) had been isolated, though the biochemical secrets of the rickets-preventing vitamin D still proved elusive.\textsuperscript{196} A 1923 article in the *American Journal of Public Health* lamented the “surprising flood of literature” that had led to significant “confusion regarding vitamins” in both the public and the medical and public health professions. The article pointed out the field was both exciting and “intensely interesting,” but that in the preceding five years, approximately 3000 “fairly serious” papers about vitamins had been published, along with “a perfect fog of other contributions.”\textsuperscript{197} This flood only encompassed vitamins A, B, and C, and as the Journal noted, “We undoubtedly have an absolute working knowledge of the prevention and cure of some of the deficiency diseases. Rickets, however, has been a puzzle.”\textsuperscript{198} Edwards A. Park, of the Yale Department of Pediatrics published a comprehensive 58-page literature review on “The Etiology of Rickets” in 1923. He provided 141 references and noted that the previous four years had been the most important in understanding the disease.\textsuperscript{199}

\textsuperscript{194} J.V. Greenbaum, as below (1926).
In her book *Vitamania*, historian Rima Apple demonstrated how scientific interests intersected with commercial and political interests in the regulation and promotion of vitamins. Apple described the medical profession’s frustration with manufacturers’ promotional techniques of vitamins. As early as 1922, an editorial by the American Medical Association pronounced the whole field of vitamin sales a “gigantic fraud.” Such pronouncements did nothing to stem the production and promotion of vitamins, particularly after the 1920s when the science was fully established.

Vitamin D had not yet been fully isolated in the mid-1920s, but this did not stop vitamin makers from promoting cod liver oil and its concentrates as their first commercial products. Advertisements for cod liver oil in *Good Housekeeping, Parents,* and *Hygiea* promoted the concepts of scientific motherhood, emphasized the importance of experts, and warned mothers that in the case of vitamin deficiency, looking and feeling healthy did not necessarily guarantee health.

A 1926 advertisement for Squibb’s Cod-Liver Oil in *Good Housekeeping* featured a physician studying a stylized X-ray. “Doctors say a startling percentage of babies’ X-ray pictures show failure of bones to grow perfectly,” the advertisement warned. The largest text continued, “Now the X-RAY shows tiny bones and teeth developing imperfectly—even in the healthiest-looking babies!” The smaller text continued, playing on mothers’ fears, “Inside, where it can’t be seen, the damage starts! A defective development of the bone structure so insidious that it is more than likely to touch even the well-cared-for babies in intelligent modern homes! This is the new knowledge which X-ray investigators are disclosing.” The ad continued, “long before its pronounced symptoms appear, long before even the most watchful mother could tell that her baby is not perfectly all right—the imperfections start.” The advertisement played on the advances in science (“For it is now scientifically established…”) and the new language of vitamins. It

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201 Apple, p. 16.
explained that cod liver was widely prescribed as the “one food which is extremely rich in the vital rickets preventing element.” An asterisk directed readers to the bottom of the page and a definition for “anti-rachitic (rickets preventing).” 202

A December 1928 ad in Hygeia for Oscodal emphasized the seasonal nature of rickets and reminded mothers to provide their children with the benefits of sunlight even in the midst of winter. “They Need it NOW,” the tagline blared. “Housed in school or home the greater part of the time after a summer spent in the sunshine, children need the vital element which scientists call vitamin D, and which is often spoken of as The ‘Sunlight’ Vitamin.” Oscodal, the advertisement continued, “protects against rickets, from which so many young children suffer, and helps to form good teeth.” 203

Squibb’s Cod Liver Oil advertisements in the 1920s and 1930s often played on this theme of poor physical development. A 1937 Squibb’s ad displayed two small children without clothes and asked rhetorically, “Straight strong legs, a fine full chest, will he grow tall?” As the advertisement promised, “Your baby too can build a well-shaped head, a fine full chest, a strong back and straight legs—if you give this special help now.” The advertisement offered scientific explanations: “You can help your baby build teeth than come in sound and even by giving him plenty of one special factor—Vitamin D. This helps transform the calcium and phosphorus he gets in his food into hard tooth structure.” It also linked Vitamin D to facial appearance, noting, “Your baby also needs Vitamin D to develop his jaw and chin fully. Sunshine, alone, does not supply him with enough Vitamin D these dreary winter months. Give him a dependable source daily--Squibb’s Cod Liver Oil.” Other ads played on mothers’ sense of guilt and responsibility: “Can you keep your child off the ‘Casualty list’? Will you help him build strong bones, sound teeth, and a sturdy body this winter?” 204 Still others advocated the importance of following

202 Advertisement reprinted in Apple, p. 23.
204 Apple, p. 27.
expert advice (assuming the expert advocated cod liver oil): “Everything turned out just as the
docor said—my baby has such a well shaped head, such a fine full chest.”

Apparently these advertising strategies, coupled with public health and medical
campaigns, worked. National cod-liver oil consumption grew from 1,921 million
gallons in 1926 to 5,790 gallons in 1937. Overall, annual U.S. vitamin sales increased
from $12 million in 1931 to more than $82 million in 1939, to more than $130 million in
1942. By 1940, manufacturers offered more than 20 different vitamins as
supplements.

Politics of Preventive Medicine

On August 19, 1908, with little fanfare, the Board of Health of the City of New York
created the nation’s first municipal body to focus on child health. The Commissioner of Health
introduced the Bureau of Child Hygiene and its new director, Dr. S. Josephine Baker, as the
leaders of “potentially one of the most important [developments] in the history of preventive
medicine.” This explicit alignment with the relatively young field of preventive medicine
demonstrates the changing goals and programs of the Board of Health. New York’s Metropolitan
Board of Health had been founded in 1866 with just two divisions: the Sanitary Bureau and the
Bureau of Vital Statistics. For the first three to four decades of its existence, the Board had
concerned itself mainly with the city’s overwhelming sanitation problems, attacking New York’s
garbage-filled streets and overflowing sewers, and supplementing that work with the enforcement

[206] In an article examining the concept of the patient as “consumer,” historian Nancy Tomes points to the rise of the
health care “industry” by the early 1930s as an important factor. Nancy Tomes, “Merchants of Health: Medicine and
[207] Apple, p. 27.
[208] Apple, p. 11.
[210] A Bureau of Child Hygiene: Co-operative Studies and Experiments by the Department of Health of the City of New
York and The Bureau of Municipal Research (New York: Bureau of Municipal Research, 1908), Foreword.
[211] Gordon Atkins, Health, Housing, and Poverty in New York City 1865-1898, Diss. Columbia University (Ann Arbor:
Edwards Brothers, 1947), p. 34.
of quarantines and other emergency containment procedures during epidemics. Both sanitation work and epidemic containment had much more political and practical currency than endemic and usually milder diseases like rickets.

The Department of Health originally created the Bureau of Child Hygiene with an administrative goal of centralizing the city’s disparate programs for children—and particularly school-aged children. Many of the scattered programs transferred to the new Bureau in 1908 were disorganized, with little coordination of work in the boroughs. As the Department noted in a clear understatement, “owing…to a division of forces, the work has not proved as constructively efficient as could be desired.”212 The Division of Contagious Diseases, for example, had begun inspecting ill school children in 1907. Responsibility for issuing youth employment certificates had fallen to the Division of Sanitary Inspection (due to the fact it had previously inspected merchants). The Assistant Sanitary Superintendent supervised the women who were nursing foundlings, while midwives had become the responsibility of the General Medical Officer. In a 38-page booklet highlighting the problems that the new Bureau would tackle, 18 pages discussed the physical examination of school children, while only three pages addressed the care of infants.

However, the leaders of the new Bureau of Child Hygiene saw an opportunity in the political landscape. The growth of the Health Department in the first 15 years of the twentieth century was unprecedented as its focus shifted firmly to preventive health. In 1900, the entire Health Department had a budget of around $1 million and fewer than 1,000 employees. Just 10 years later, it had more than doubled in size, employing 2,500 people at a budget of $2.75 million. By 1913, its estimated budget was $3.88 million.213 The Bureau (later Division) of Child Hygiene, born in 1908, employed 697 employees by the time the first infants it served had turned six. It boasted, by 1914, 56 milk stations, along with five clinics for children with nasal breathing and tonsil problems, and six dental clinics.

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213 Ibid., p. 264.
While 40 years earlier, reformers had concentrated efforts on epidemics and sanitation, now the promise of public health seemed to extend to the possibility of reforming individual behavior and of eradicating disease before it even emerged as a physical entity. Work with children was the most preventive work possible. For the first time, the Bureau of Child Hygiene focused its energies on treating and examining children before they were ill, with examinations in schools, community education programs, and, above all, expansion of the inroads on infant mortality that the milk stations had made. The Bureau of Child Hygiene worked to translate the Progressive ideals of education, expertise, and efficiency into fewer infant deaths and used the politics of preventive health to wield significant political power. Baker, its leader, used specific political strategies, including focusing on infants rather than older children and rearticulating the goals of baby-saving to fit the language of efficiency or sentimentality, depending on circumstance, while navigating the controversial minefield of physician-public health relations. With a little education and governmental help, her Bureau’s reports argued, mothers living in tenements could raise infants as successfully as mothers on Park Avenue.

Rickets was a favorite poster disease for preventive medicine. A laudatory 1927 article about advances in preventive medicine gushed, “The prevention of rickets is one of the romantic pages in preventive medicine.”214 The 1911 *Encyclopedia Britannica* echoed preventive medicine’s goals well in its definition of rickets: “The treatment of rickets is necessarily more hygienic than medicinal, and includes such preventive measures as may be exercised by strict attention to personal health and nutrition on the part of mothers....” As George Rosen has described, early preventive medicine grew out of advances in bacteriology and immunology, as the vague dangers of dirt or vapors crystallized into enemies-- germs visible and invisible--that could produce illness. In the urban setting, tuberculosis attracted the most attention because of the realities of crowding and the fear of contagion. But in this pre-antibiotic era, prevention and

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treatment of tuberculosis were both imperfect sciences. Rickets, in contrast, was a common
disease that proved tractable to inexpensive interventions, both for prophylaxis and cure. The
realization that sunlight was a specific treatment seemed to justify all of the environmental
arguments the public health and sanitation workers had been making for years.

In the first decades of the twentieth century, preventive medicine was seen as a separate
endeavor from traditional medicine, with a focus on preventing rather than curing disease. This
shift toward preventive health was reflected in organizations across the country. Just the name
changes of the American Association for the Study and Prevention of Infant Mortality
demonstrate this move toward general preventive public health. “In 1909,” a self-published
history of the organization recalled fondly, the Association was the “only organization devoted
exclusively to the health of the child.” In 1922, when Herbert Hoover was the association’s
president, it was renamed the American Child Hygiene Association before finally settling on the
moniker American Child Health Association a few years later. These shifts—from a specific
focus on infants to a general focus on children and from a specific focus on mortality to a broader
focus on hygiene and finally a general focus on health—mirror the changes in other public health
organizations. In 1935, the Association noted that (thanks in part to its own work), national health
organizations finally realized that the child is the “keystone of Preventive Measures.”

The end result of a successful rickets intervention was a child who was taller, sturdier,
and visibly healthier. In the early 1920s, this played well into the idea of health as a national
resource, as public health and preventive medicine workers successfully co-opted the language of
war. Preventive medicine generals led campaigns, fought battles, and conquered diseases,
translating the Progressive ideals of education, expertise, and efficiency into healthier infants.
“There is no disease that has attracted the attention of the clinician and the laboratory worker
during recent years more than rickets,” a 1922 article noted. “The reason is not far to seek. It is a

215 Philip Van Ingen, The Story of the American Child Health Association (New York: American Child Health
216 Ibid., p. 40.
disease that strangles a nation’s vitality at its source.\textsuperscript{217} S. Josephine Baker gave a widely publicized lecture in 1919 discussing war-related health problems in Belgium. As a reviewer wrote, she directly linked rickets and tuberculosis to images of poor health: “Dr. Josephine Baker, in a recent lecture, spoke of the tremendous need of conserving the Belgian children now, from rickets and tuberculosis, if Belgium is to be! We know, but sometimes forget, how truly the structure of the nation of tomorrow is being builded today.”\textsuperscript{218} The deprivations of war could lead to poor health in children, even indirectly. The 1921 \textit{International Compendium on Progress} argued that rickets was significantly more common in the U.S. after World War I than it had been before, and pinned the increase to “a result of defective nutrition during the war,” perhaps specifically caused by “exhaustion of the soil from excessive crop rotation” during the war.\textsuperscript{219}

By 1914, the Department concentrated its efforts on public, preventive health and created the first Bureau of Public Health Education.\textsuperscript{220} In May of that year, the commissioner announced that detecting medical conditions earlier could add three to five years to New Yorkers’ average life expectancy. He suggested expanding the school programs of the Division of Child Hygiene to the general public and advocated free physical exams for every person in the city. It never happened, but the mere proposal of such a huge undertaking suggests the heady atmosphere of accomplishment and possibility that marked the new field of preventive medicine. Proactive policies like physically examining every New Yorker, regardless of whether he or she was ill, show how far the Board had come from its purely reactive early sanitation and epidemic containment programs. The following year, the 1915 \textit{Annual Report of the Board of Health}

\textsuperscript{220} \textit{Ibid.}, p. 266.
contained the oft-quoted maxim: “The City can have as much reduction of preventable disease as it wishes to pay for.”221

Politics of Pediatrics

The twentieth century has been called the century of the child for good reason. At the most basic level, improvements in general living conditions meant that many, many more infants had a chance at childhood. A lack of complete birth and death records means that statistics are imprecise, but estimates suggest that between 15 and 20 percent of all infants born in the U.S. between 1850 and 1900—and perhaps as many as 30 percent in some large cities--died before reaching the age of one.222 Baker had previously been employed in New York’s Bureau of Municipal Research and had been shocked by the high rates of infant mortality of children under five, which reportedly accounted for nearly one in three deaths in New York City.223 But between 1900 and 1930, infant mortality in this country decreased by more than 50 percent.224 Richard Meckel calls this reduction of infant mortality “arguably the most dramatic and far-reaching” health revolution in this country since 1850 (a strong statement when the alternatives include the adoption of the germ theory).225 The story was at its most inspiring in New York City; the city that had suffered one of the highest infant mortality rates in the country had the lowest rate of any of the major American or European cities by the end of Baker’s tenure at the Bureau of Child Hygiene. The first sentence of Baker’s 1945 obituary in the New York Times focused on this aspect of her work and praised her “pioneering” in making New York “one of the safest instead of the worst cities for babies to be born in.” While she headed the Bureau of Child Hygiene, the

223 Duffy, p. 260.
obituary continued, the infant death rate had fallen from 144 in every thousand births to 66; a health department statistician credited her programs with saving 82,549 lives. Of course, it was not only the programs of the Bureau of Child Hygiene that brought about the reduction in infant mortality, as infants stood the most to gain from the general improvements in the standard of living seen across the country during this time. But the improvements in the city’s infant mortality rate stood as a crude and highly impressive marker of the Bureau’s success.

The American Pediatric Society, founded in 1888, focused attention on the diseases of children and provided a forum for discussing research. Many of the most important discoveries about rickets were first reported in this society’s meetings. Pediatrics was a brand-new specialty; in 1890, the society’s 49 members had included “practically every pediatrician in the United States and Canada” (none of whom had limited his practice exclusively to pediatrics). At the sixth annual American Pediatric Society meeting, in 1894, three of the 23 research papers focused on the disease.

The American Medical Association began publishing The American Journal of Diseases of Children in 1911, helping to validate child health as a separate discipline and emphasizing scientific innovation, perhaps even over clinical practice. Rickets did not appear significantly in the early years of this “strictly scientific magazine”; treatment and clinical practice were not the focus. In the first two years, 1911 and 1912, no full articles and only one short, two-paragraph note appeared, entitled “Rachitis, nitrogen and sulphur metabolism in rachitic dwarfism.” However, as the years brought an increasing focus on preventive public health and laboratory advances, more and more articles on rickets appeared. In 1928 alone, the American Journal of

228 Ibid. Less than 50 years later, in addition to its “100 carefully picked members, all of them devoting their lives to pediatrics,” the society had a “potential waiting list” of the nearly 4,000 pediatricians listed in the American Pediatric Directory.
229 Mention as treatment cod-liver oil in winter, hypophosphites in the summer (142). Another article, “causation of neuroses” concluded “fat starvation is the commonest cause of rickets,” (155).
Disease of Children featured 27 articles related to rickets, on subjects ranging from the antirachitic efficiency of winter sunlight in Washington, D.C.\textsuperscript{230} to a comparison of the efficacy of cod liver oil and ultraviolet light in preventing the disease.\textsuperscript{231}

**New Haven Politics**

From the beginning, the New Haven rickets study was intimately connected with the national Children’s Bureau in Washington, D.C., which had been founded in 1912. Many physicians who eventually became national and international leaders in public health and children’s health participated in this study.\textsuperscript{232} Martha Eliot, the lead physician on the study, had recently been named the first pediatrics chief resident in Yale’s new Department of Pediatrics, while Edwards A. Park, the head of the department, was the senior investigator. In the middle of the New Haven study, in 1924, Eliot was named the director of the Division of Child and Maternal Health of the national Children’s Bureau. She began commuting to Washington one week a month from New Haven and did so for the next 10 years, until being named assistant chief of the Children’s Bureau.

Eliot and her colleagues conducted the rickets study was at a time when records show the New Haven Health Center was struggling to find its niche in the new field of preventive medicine. The Health Center attempted to put the New York commissioner’s universal physical exam theory into practice. However, the goal of providing free preventive physical examinations for New Haven residents proved difficult to achieve. Between July 1920 and June 1923, the one full-time and one half-time New Haven Health Center physicians performed 20,622 physical exams. Of these, 5,905 were compulsory exams of school children (who needed an exam before

\textsuperscript{230} American Journal of Diseases of Children, 1928, 17: 582.
\textsuperscript{231} Ibid., p. 952.
\textsuperscript{232} Eliot, for example, completed medical school at Johns Hopkins trained at Peter Bent Brigham Hospital, and completed her one-year pediatrics residency at St. Louis Children’s Hospital before becoming the first pediatrics resident at Yale. Years later, Eliot was part of the U.S. delegation to the first-ever World Health Assembly, and was the only woman to sign the founding document of the World Health Organization. In 1949, she moved to Geneva as the associate director general of the WHO, and finally returned to be the chief of the Children’s Bureau. In 1947, she was the first woman to be elected president of the American Public Health Association.
returning to school if they had been absent more than three days for illness) and 1,896 were of infants at the Well Baby Conferences. This left 12,821 adult visits by 7,215 different individuals, which, as the report concluded, “may be said to be of a voluntary nature.” However, most of these adults had come to the Health Center not in health but in sickness—and, were therefore, seeking traditional curative medicine rather than the preventive medicine the clinic was designed to promote. “They came in large measure to the Health Center as to a dispensary, in distress, and they desired to be relieved not with a truer understanding of their difficulty and sound counsel and a plan for the future, but with a mysterious panacea,” the report noted, with some frustration.

Although this seems like a fairly significant number—and, in fact, the report estimated that it had provided some assistance or information to 85% of the families living in the district--the Center minced no words in expressing its dissatisfaction. “The wonder is not that many came, but that so many did not come,” it argued. “For after all, 12,821 visits in three years means an average of only 14 a day, and 862 voluntary physical examinations of adults is a very small number in a population containing 16,000 adults.” The report succinctly stated the fundamental difficulty of preventive medicine. The most important reason for under-use of the clinic was simply that, “the Health Center did not give the community what it wanted. It wanted treatment, medicine. It could not accustom itself to the idea of a physician who did not prescribe medicine.” Here was the challenge of preventive medicine in a nutshell. Some of the blame was also pinned to the residents of New Haven as the report described the “character of the population” as a secondary obstacle. “As a general rule the idea of sickness prevention is so primitive that faith in ancient superstitions, advertised nostrums and Divine interference leaves no place for modern knowledge,” it concluded. “When understanding and cooperation in the simplest principle of communicable disease control could not be obtained it is not surprising that the Health Center’s diverse appeals in behalf of annual physical examinations should bear small results.”

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233 Between July 1920 and December 1921, the service recorded 6333 visits for examination and consultation.
One area of preventive medicine that has now been ingrained into the culture proved similarly unpopular in the 1920s. “There is no doubt about the fact that the prenatal clinic can be set down as a failure,” the 1923 report concluded bluntly, noting that only 208 pregnant women had been examined over three years during which 2600 babies had been born. “To the women of the district it was an anomaly and a superfluity.” Again, the fact that the “woman physician who conducted the clinic possessed a sunny, sympathetic disposition which won the hearts of her patients” was not enough. Nor was the fact that in 1921, 56% of the New Haven children who had died under the age of one succumbed in the first month of life, calling attention to the need for “prenatal hygiene.” New Haven women in the early 1920s came to the “prenatal” clinic with two goals: “to be assured that there was no pregnancy and...for information how to prevent conception.” Ironically, though the latter goal was exactly the kind of “preventive” behavior the clinic hoped to promote, the report reminded readers that disseminating contraceptive knowledge was illegal in Connecticut.

The Health Center had not been established to provide medicine of any kind. The records note that representatives from the New Haven Health Center spoke with representatives from both the New Haven and Grace Hospital Dispensaries as it reconsidered its decision not to provide its own dispensary. As the report concluded, “It was clear that the establishment of any but the highest type of polyclinic dispensary service, which was manifestly impossible, would only be providing a partial, unscientific service that would not be for the best interests of either the population or the future of modern medicine in New Haven.”

The Health Center’s field staff of 15 nurses and community workers made home visits, but they, too, were increasingly called for “consultations” rather than preventive “examinations.” Personal relationships were not enough. As the report said, “The heart to heart talk with the sympathetic ‘health’ doctor who was willing and anxious to explain was often a revelation to the patient. Repeatedly, the patient would thank the doctor with the remark that he had never before
had a real examination like that.” However, the public’s “expectation of a pill (not to be fulfilled!) which would cure all” could not be overcome.

The Health Center staff took a range of promotional and outreach approaches. They produced an “attractive colored calendar poster urging a physician examination,” as well as “attractive certificates” for people who actually came for preventive exams. Staff visited a large New Haven factory, and gave 14 separate 6-minute talks “in either English or Italian” during the workers’ breaks, leaving appointment cards “in the foreman’s hands,” without results.

Finally, the New Haven Health Center report turned its attention to “Special Efforts Against Rickets,” the only disease singled out. Again, “The Health Center’s efforts to arouse the community to some appreciation of the importance of rickets and the means for its prevention were not very successful.” A range of promotional approaches, including newspaper articles, letters asking local physicians for cooperation, “striking colored posters, 3 feet by 5 feet,” presentations at well baby conferences, and follow-up home visits by nurses all met with “indifference and scepticism.” The Health Center promoted cod liver oil heavily and provided it “at less than half the retail price” at all stations. But practitioners found that even the 300 mothers who had purchased cod liver oil at well baby conferences in 1921 generally stopped giving it to their children in the privacy of their homes. The frustration is palpable in the report: as the nurses reported, “’The baby doesn’t like it’ was the usual and the sufficient excuse, though the many variations on this excuse took almost unbelievable forms.”

Simply providing technology for diagnosis and promoting an established, standardized treatment was not enough to guarantee clinical success or acceptance of preventive medicine tactics.

**Conclusion**

Rickets has been used to advance a wide variety of theories and agendas. In the early 20th

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century, before diagnosis or treatment had been fully standardized, and before the scientific research had been completed, many people debated the etiology of rickets. Some saw the disease as the price of urbanization, some used it to promote assimilation for immigrants, some used it in debates over eugenics, and nearly everyone emphasized the importance of race—and, in particular, targeted African-Americans and Italian-Americans. Multiple community-based studies in New York and New Haven between 1917 and 1925 worked to standardize diagnosis, treatment, and prevention regimes. With increasing dependence on the new technology of the X-ray in the context of the nascent preventive medicine movement’s politics, rickets in the northeast was redefined as a nearly universal disease. The shifting definitions and prevalence of rickets with changing diagnostic methods can help illuminate current debates over the role of race and ethnicity as risk factors for disease; the use of diagnostic technology in defining disease; and the promotion of targeted interventions for common diseases.
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