REVIEW ARTICLE

Environmental Factors in Tiny Tim's Near-Fatal Illness

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Physicians, Dickens scholars, and historians have tried to diagnose the condition that affected Tiny Tim in *A Christmas Carol*. Leading entities include tuberculosis (TB), rickets, malnutrition, cerebral palsy, spinal dysraphism, and renal tubular acidosis. This article posits that an examination of the environment of London of 1820 to 1843 (when the novella was written) can provide important clues as to his condition. The blackened skies from burning coal, the crowding of people in tenements, the limited diet of the underclass, and the filth of London resulted in a haven for infectious diseases and rickets in children. Sixty percent of children in London had rickets, and nearly 50% had signs of TB. Tiny Tim likely had a combination of both diseases. After Ebenezer Scrooge's transformation, Scrooge could have ensured an improved diet, sunshine exposure, and possibly cod liver oil for Tiny Tim, which could have led to a "cure." Dickens was familiar with both rickets and TB and wrote about cod liver oil as a possible cure for rickets and scrofula. Improved vitamin D status can result in enhanced macrophage synthesis of 1,25-dihydroxyvitamin D, which increases the synthesis of the antimicrobial peptide cathelicidin (LL-37). This component of the innate immune system has strong killing properties for *Mycobacterium tuberculosis*. The combination of rickets and TB represent a crippling condition that could be reversed by improved vitamin D status. *Arch Pediatr Adolesc Med. 2012;166(3):271-275*

Charles John Huffam Dickens was one of the greatest writers of the 19th century and a master of the English language. His descriptions of scenes and iconic characters are timeless. Whether we see him as brilliant, as sentimental, as an almost Marxist social critic, or as a remarkable chronicler of the human condition, he was a noteworthy scribe of characters, names, locations, and complex plotting. Dickens deserved his place in Poet's Corner in Westminster Abbey.[1] Because his depictions are so accurate, he has achieved an almost matchless status as a definer of medical conditions.[2,3] Joe, the fat boy in *The Pickwick Papers,* provides us with the term *pickwickian* to describe sleep apnea with obesity.[4] In *The Old Curiosity Shop,* the ultimate demise of Little Nell due to exhaustion and a broken spirit defines a psychosocial model of illness. Jenny Wren, in *Our Mutual Friend,* was a tiny woman who sewed dolls' clothes and used crutches.[2] James Gamble wrote that she was a "rickety dwarf."[5] Dickens has been called a syndrome spotter,[2] having characterized, for example, the Uriah Heep syndrome.[6] He gave us fine-tuned portraits of insanity, of neuropsychiatric states, and of dystonia and provided 3 accurate descriptions of epilepsy: (1) Monks, the devious half-brother of Oliver Twist; (2) Guster, the maid servant in *Bleak House,* who had one convulsion after another; and (3) Charles Hexham's school headmaster in *Our Mutual Friend*.[2] However, no medically based character is as famous as Tiny Tim, the crippled lad in *A Christmas Carol*.[7]

Tiny Tim Cratchit is the prototypical vulnerable child in *A Christmas Carol*, a novella of 30 000 words written by Dickens in 6 weeks.[7] It has never gone out of print. The book's sustained success has contributed to how Christmas is celebrated and how children's charities raise funds. It has been adapted to film 28 times and seen as a play, an opera, and a cartoon feature.[8] The story introduced the phrases "Merry Christmas" and "Bah! Humbug," the return to Old English Yuletide traditions, and Christmas-themed

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charities.[1,7,8] While Christmas is a component of a winter solstice holiday that is pagan (Christmas trees, Yule logs, and mistletoe) and Christian (nativity scenes and the birth of Jesus), the story helped reintroduce the notion of this holiday as a time of joy and gaiety. As Tiny Tim states in the final words of the story, "God bless us, everyone."[7]

Other influences of the novella are notable. One of Dickens' most finely drawn characters, Ebenezer Scrooge, has given his name to a skinflint or miserly person. Christmas trees and cards were introduced in 1841 and 1843, respectively. This story was published in 1843. Its main aspects are the interaction of Scrooge and the Ghosts of Christmas Past, Present, and Future. Bob Cratchit is a good-spirited but downtrodden employee of Scrooge. Tiny Tim, who appears in only a few scenes, carries a single crutch and wears iron bars on his legs. His condition is not fully described. It was fatal if untreated, as indicated in the vision offered by the Ghost of Christmas Present. He lived because of the conversion of Scrooge to a more generous path. Hence, the question: What ailed Tiny Tim?

Tiny Tim has been the subject of medical detective work to establish his medical condition.[9-13] Tiny Tim is believed to have had rickets, tuberculosis (TB), polio, and/or cerebral palsy. Lewis[12] built a logical case for renal tubular acidosis because it would affect the skeleton and could be reversed with the administration of alkaline salts. Demme[11] believed that nutritional rickets was the answer, as did others.[13] Callahan[9,10] makes a strong case for TB with Pott disease of the spine. For a child of the first half of the 19th century who required iron braces and a crutch and who had a withered hand, other possible diagnoses include myelodysplasia, including a tethered cord and a skin-covered myelomeningocele; a lower spine injury; or a paraplegic form of cerebral palsy and nutritional deficiency. It has been said that Tiny Tim was patterned after the son of a friend of Dickens who owned a cotton mill in the Ardwick section of Manchester.[14] Dickens' nephew, Henry, died of consumption and is also claimed as a model.[1,2] In a large family, the youngest may have been underfed, which is another possibility for Tim Cratchit's condition. Malcom Andrews, a Victorian-era historian, has concluded that Dickens did not spell out the clinical specifics but created a penetrating fable.[15]

The depictions of Tiny Tim and, indeed, all Dickens' memorable child characters—Oliver Twist, David Copperfield, Pip, and Little Dorrit—were also influenced by the powerful illustrators who created sketches for Dickens' publications.[16] *A Christmas Carol* was illustrated by Charles Leech, but he did not show Tiny Tim, focusing more on the ghostly images.[16] Illustrations of Tiny Tim first appeared after Dickens' death in 1870, and we are all familiar with the depiction of Tiny Tim on his father's shoulders as they go off to church for a Christmas service. The figures of him with a single crutch have led to speculation that his disorder was unilateral, but this is not reflected in the story nor is it necessarily correct.[17,18]

That Tiny Tim's illness was chronic and not acute is likely because his growth was impaired. Lewis' thesis of renal tubular acidosis is credible[12] because type I renal tubular acidosis has bone disease as a manifestation[19-22] and it is associated with impaired linear growth.[23] Tuberculosis affected approximately 50% of the British population of the time.[24] Callahan[9,10] speculated that Tiny Tim could have been treated at a sanitarium, such as the Royal Sea Bathing Infirmary in Margate, Kent. Either polio or cerebral palsy is possible, although how either would have led to death with a chronic course is unclear. A spinal dysraphism could have been fatal if urosepsis occurred or with the development of chronic kidney disease and hypertension. Had there been skin breakdown over a covered myelomeningocele, meningitis might have occurred.

Another approach is to examine the environment of Tiny Tim—its time and place. London of 1820 to 1840 was crowded with its narrow lanes and tenements, plagued with blackened skies, and filthy.[7,25] The validity of an environmental approach is that Dickens was a realistic writer. What he saw, he described. Dickens described the darkened streets encountered by Bob and Tim Cratchit. He also knew the immense gap between the wealthy (Scrooge) and the working poor (the Cratchits). Among the conditions of childhood that were common in London of that era were all infectious diseases of childhood, rickets, TB, and infantile diarrhea, the latter irrelevant in Tiny Tim's case.

Why were rickets and infectious diseases so frequent? Following the Great Fire of London in 1666, the rate of rebuilding of the city was staggering. With expansion of the population, the supplies of available wood were rapidly outstripped. Sea coal and, later, mined coal replaced wood as major fuel sources.[26-28] The Industrial Revolution, which began in the United Kingdom, was fueled by coal and involved heavy manufacturing in London, the Midlands (Manchester), and the Clyde-Firth region between Glasgow and Edinburgh.[26] The output of coal in the United Kingdom increased approximately 6-fold between 1750 and 1830.[29] Industry led to an energy consumption 5 times higher in 1836 than in 1650.[30] London was a center for the distribution of coal. By 1700, the United Kingdom produced five-sixths of the world's supply; this allowed the development of railroads and the steamship in the 1820s and 1830s.[26-29] The skies were blackened with soot and particles, which absorbed UV-A and UV-B rays.[31] The coal had a high sulfur dioxide content, which further absorbed UV-B rays. This diminution of UV-B exposure in the urban United Kingdom prevented the photocutaneous synthesis of vitamin D.[32-34] Moreover, the diet of the working classes of London and other industrial centers included starch, carbohydrates, scant protein, and limited intake of fat.[7,25] The children also had limited intake of vitamin D–containing foods and a lack of sunshine, and they frequently developed rickets, with a prevalence of 60%.[33] In a 1909 autopsy series of infants and children perishing in the environs of Dresden, which is at the same latitude as London, 92% to 98% of 4- to 18-month-old children had bone histologic evidence of rickets, with undermineralization of the osteoid and typical metaphyseal changes.[35] Before 1880, the science of bone histologic examination did not exist[33] and these observations could not be made.

Rickets was a mysterious condition that increased in frequency after 1650, when it was described by Glisson[36] and Whistler.[37] While it may have existed before

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the onset of the Industrial Revolution, it was so common in London and the southwest of England that it was termed "the English disease."[33,38] Because of its prevalence, rickets was frequently investigated, and numerous theories of its cause were espoused, as reviewed in the 1920s.[33,39] No theory of its cause was embraced as vigorously as that of an infectious or contagious cause.[40-42] Before 1880, contagion was thought to be the result of a "miasma." Children could be protected from miasma by remaining indoors and covering their entire bodies with clothing. Many children worked from before dawn to after dark; many worked in home-based textile manufacturing and were always indoors.[43] Thus, to avoid miasma, children were not exposed to sunlight. Not only did rachitic children experience severe illnesses more frequently, but often the deaths of these children were related to pneumonia or to TB, which was known as *consumption* in Dickens' times.[40,44,45] Only after Robert Koch's observations[42] were bacteria recognized as the infectious agents. Among the infectious theories prevalent were that the infectious agent could be found in the growth plate of rachitic bone,[42] that infections of the intestine caused rickets,[38,41] and that rachitic children died an infectious cause–related death.[45] In Jenner's treatise[41] on the conditions of childhood, he suggested that rickets was a component of TB.

The infectious disease theory persisted for a long time. However, in 1890, Theobald Palm, a Scottish medical missionary, realized the roles of sunshine and northern latitudes on the prevalence of rickets and recognized the urban nature of the disease.[44] Anecdotally, cod liver oil could reverse rickets,[46] and administration of cod liver oil was recommended in the mid-18th century by Thomas Percival of Manchester.[43] Nonetheless, it was nearly 1920 before nutritional deficiency and a lack of sunshine exposure were espoused as the causes of rickets.[33] Finally, the recognition of a dual source of the curative agent—vitamin D—from the diet and from skin synthesis was reconciled only in the late 1920s.[32,47] Dickens would not have understood infectious agents or UV-B rays of sunlight, but the environmental conditions leading to rickets and TB were the same between 1820 and 1843 as they were in the 1890s.

To return to Tiny Tim: his family lived in the Camden Town area of London. This area was undistinguished and was traversed by the Regents Canal, which was an artery for shipments of coal that were off-loaded in London Docklands and shipped to the midlands via a canal system.[29] Tiny Tim's diet was undoubtedly limited by the "15 bob" per week earned by Bob Cratchit. In the 1820s and 1830s, 15 shillings could purchase 4 one-pound (448-gram) loaves of bread. A Christmas goose was a rare event. Obviously, the children of the Cratchit family had to work, but Tiny Tim couldn't help. Martha was a hard-working milliner's apprentice, and Bob Cratchit thought he had found a "situation" for Master Peter for 5 and 6 pence weekly, an opportunity he announces at Christmas dinner.[7]

The blackened skies would have prevented skin synthesis of vitamin D, and Tiny Tim's chances of having rickets were substantial.[11,33] As evident for centuries, pneumonia, upper respiratory infections, and TB were far more common in vitamin D–deficient and rachitic populations.[40,44-46] I hypothesize that Tiny Tim could have had a combination of rickets and TB, both of which could have been cured (rickets) or improved (TB) by improved vitamin D status.[32,48-50]

Why would Tiny Tim have been afflicted with rickets and TB and not his older siblings? First, his siblings might well have had milder forms of rickets before the age of 3 to 4 years, when rickets peaks.[33,35] It is entirely possible that they also had TB, as most people infected with the organism do not display signs of active disease,[51] and "galloping consumption" usually occurred in the context of poverty, crowding, malnutrition, and other factors. One of the remarkable features of rickets is that it is often found in children of high or very high birth rank.[52-54] The traditional explanations for the development of rickets have been family size, birth order, and crowding.[55] These factors were evident in the Cratchit household. Tiny Tim was the fourth child, and his family of 6 lived in 4 rooms.[7]

Today, we understand that vitamin D is a prohormone and that its most biologically active form, 1,25-dihydroxyvitamin D (1,25-$[OH]_2D$), increases the nuclear synthesis of 200 to 300 proteins and peptides.[56] Among these proteins are calcium-binding proteins, which enhance intestinal calcium and phosphate absorption and permit bone mineralization and the reversal of rickets.

A nonosseous immune function has also been shown that includes stimulation of the innate immune system.[48-50] Macrophages take up 25-(OH)D, and, under the influence of the enzyme CYP27b within the mitochondria of the macrophage, 1,25-$(OH)_2D$ is synthesized. After binding of this highly active metabolite to an intracellular vitamin D receptor and heterodimerization with the RXR (retinoic acid) receptor, this complex binds to a vitamin D response element on the promoter region of genes for several antimicrobial peptides, including cathelicidin (LL-37). Cathelicidin binds to bacterial membranes and kills the cells (particularly *Mycobacterium tuberculosis*).[48,50,57,58] As TB-induced granulomas are populated by macrophages in the environment of vitamin D adequacy, there is ample killing of TB organisms.[58] This role of vitamin D in supporting the body's immune response to TB organisms explains the success of sanitariums and the sunlit porches where patients sunbathed in the late 19th and early 20th centuries.[24,40]

Another factor that darkened the skies of London was the massive 1815 eruption of the volcano Tambora near the Sunda straits in Indonesia.[59,60] This was the most massive eruption in more than 1300 years. An ejected volume of 160 km^3 of ash and magma entered the atmosphere in an eruption column that rose 43 km. Massive amounts of sulfur dioxide were also released. The next summer, 1816, was called "the year without a summer."[59,61] Snow fell in New England and Quebec during the summer months, crops failed, and thousands of animals died worldwide. The cost of food rose enormously, and the economically disadvantaged segments of society experienced malnutrition. The sunsets were wonderful for years to come because of suspended particles, as typified in the paintings of London and Chichester by J. M. W. Turner in the 1820s.[62] Because the ash

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blocked UV-B rays, it could have contributed to insufficient sunlight exposure for children in London in the time of Tiny Tim, the 1820s.

The salary earned by Bob Cratchit would have influenced the diet available to Tiny Tim.[7] The 4 one-pound loaves of bread that 15 shillings would buy may have been adulterated with alum (hydrated potassium aluminum sulfate) to whiten the bread and disguise the use of poor quality flour.[63,64] Alum is used today as an antiperspirant, in styptic pencils, in baking powder, and as a pickling agent in many cuisines[65] but was added to bread throughout the 19th century in London. It, like other aluminum salts used as antacids, can bind phosphate in the intestine and prevent its absorption. John Snow, the father of British epidemiology who was famed for his statistical studies of the cholera epidemic in Soho, London, and his deduction of a water-borne agent as the cause,[25] wrote an article in 1857 on his theory regarding the adulteration of bread as a cause of rickets.[64] If Tim ingested alum-containing bread, the phosphate-binding property of the compound would have contributed to his rickets. However, we are led to believe from the story that Mrs Cratchit cooked for her family and possibly baked her own bread. If so, Tim would not have been exposed to alum. Snow made the point that flour merchants did not adulterate flour because the penalties were too great.[64] Small bakeries could add up to 1.5 oz (42 g) of alum per loaf and not fear food inspectors, who concentrated on bigger fish. As noted previously, vitamin D deficiency is associated with enhanced pathogenicity of *M tuberculosis*. The mechanism is probably related to reduced innate cellular immunity. In addition, phosphate depletion is a stimulus for persistence of the TB organism within macrophages.[66] Had Tiny Tim developed hypophosphatemia from adulterated bread, his rickets and TB might have been intensified, especially because patients with more advanced rickets have underlying hypophosphatemia.[33]

How could Scrooge, who upon his transformation embraced being Tiny Tim's "second father," have had such an effect as to save Tim? He could have ensured that Tim received a better diet, including fish, dairy products, good quality bread, and more calories. Trips to the countryside would have increased sunshine exposure.[12] Medical specialists could have advised more contemporary and possibly effective orthopedic devices, and, with the curing of rickets, Tim's limbs may have become straighter. We are not told whether he was fully cured, but he definitely survived.

Other writers have used Tiny Tim as a model for an impoverished childhood and for the betterment of the lives of children. Francis Horner, writing for the Wesleyan-Methodist Sunday-School Union,[67] used Tim in an anti-alcohol, anticrime potboiler that emphasized the darkened skies of London slums.

Would Dickens have known rickets and TB? Most assuredly. His public craved his stories and sketches. He began to publish a daily journal in which he could emphasize his view on social reform. The journal, *All the Year Round,*[68] contains in volumes 13 and 14, dated June 24, 1865, some medical insights: "One of the worst forms of scrofula—rachitism, or rickets . . . arises under the influence of chilly dwellings and insufficient alimentation."[68(p515)] He averred that this was due to "insufficient alimentation and milk deprivation in infants." Dickens even notes that some children were dosed with cod liver oil. There are references to cod liver oil in several of his books, and he took it himself.[69] Dickens also knew that rickets led to death. These comments predate by 25 years the use of cod liver oil to cure lion cubs with rickets (the first animal model of disease) and by 50 years the same cod liver oil therapy for beagle pups in a famous experiment, both in London. The lion cubs were cured by a diet of goat bones, goat meat, milk, and cod liver oil in the London Zoological Garden in Regents Park.[46] Twenty-five to 30 years later, from 1913 to 1918, Sir Edward Mellanby, at King's College Medical School for Women, cured rachitic beagles with cod liver oil added to their cereal diet.[70] Ironically, Regents Park abuts Camden Town, and King's College for Women of that day was in Kensington and was only a few kilometers from Camden Town. Tiny Tim may have received cod liver oil as well, at least in the mind of Dickens, who frequently mentioned this therapeutic agent.[68]

Dickens knew Camden Town, where he lived from the age of 10 years until he left home in his 20s. He first resided there as a lodger. To pay for lodging and board for his sister and himself, who were away from the rest of his family, Charles Dickens worked up to 10 hours a day in Warren's Blacking Warehouse near Charing Cross. Not only did Tiny Tim reside in Camden Town but so did Wilkins Micawber in *David Copperfield.* After his father left debtor's prison, Dickens lived with his family in Camden Town. It was from these humiliating experiences that the author acquired his sense of social justice and could draw powerful descriptions of the demeaning working conditions of the time.[1,2] In this sense, Tiny Tim was a symbol of the plight of the working poor with a disabled child.

Dickens' sister, Frances Elizabeth (called "Fanny"), lodged with him in Camden Town, and they were especially close. Fanny died at age 38 years of consumption and, soon after, so did her son, Henry. Dickens described her terminal status.[71] Fanny was a model for Fan in *A Christmas Carol,* and some believe that Henry was a model for Tiny Tim.[71] *A Christmas Carol* was penned only 23 years after Dickens was a child laborer. Based on his intimate knowledge of poverty, slums, child labor, childhood death, and the medical maladies of the environment, his character lived in a milieu where a combination of rickets and TB were common. Hence, on an environmental basis, Tiny Tim had both conditions. A Christmas Day transformation of Scrooge saved him.

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