brought to the dispensaries, in towns where respectively the people buy chiefly flour or ready-made bread, would best help to decide the question.

It does not follow, if my conclusions are correct, that every child eating bread adulterated with alum ought to have rickets, or that every child fed with good bread ought to be free from the complaint; for, on the one hand, the other articles of food may often supply sufficient phosphate of lime without that of the bread, and, on the other hand, derangement of the digestive and urinary functions may prevent the phosphate of lime being assimilated when present. What we might expect, however, would be precisely what we observe—that rickets would be much more common in the children of the working classes fed almost entirely on bread than in those who have a greater variety of food. It can also be explained how the bones ultimately become hard from the gradual accumulation of the scanty supply of phosphate of lime derived from milk, potatoes, and other articles of food, whilst that which ought to be supplied in the bread is still withheld.

If the deformity in the bones of the legs does not proceed too far, it has a great tendency to diminish, and even disappear, as the children grow up; and the artificial support which is afforded by iron instruments and splints, both in the various hospitals for deformities, and under the advice of private medical men in London, diminishes very much the amount of permanent deformity which would otherwise be met with.

In my examination of bakers' bread I have been much struck with the apparent universality of the practice of using alum, and with the large quantity employed—a quantity between twenty and thirty times as great as that usually stated by authors. I have met with alum, not only in the ordinary bread sold by bakers, but also in captains' biscuits, and in the so-called farm-house bread; and I was somewhat surprised to find that the high-priced bread, sold in the fashionable neighbourhood to the west of Regent-street contained more alum than the cheap bread sold in many of the poorer districts. I found that the bread supplied to me last autumn contained 10.13 grains of alum in 500 grains—i.e. 561 grains, or more than an ounce and a quarter in the 4lb loaf; whilst some bread obtained from a very noted baker contained 11.37 grains in the 500 grains, or nearly an ounce and a half in the 4lb loaf. The following is a brief account of the analysis of the latter bread: 500 grains, being carefully dried at the temperature of 100 Fah., lost 128 grains of water, or more than one-fourth. Being carefully incinerated in a crucible, the ashes weighed 5.85 grains. The ashes yielded alumina, which, being washed, dried, and ignited, weighed 1.2 grain, representing 11.37 grains of crystallized alum; with chloride of barium, they yielded 1.4 grains of sulphate of baryta, and with the nitrate of silver, 6.7 grains of chloride of that metal, representing 2.8 grains of common salt.

Dr Hassall and some other authors have very properly pointed out that the only safe way to seek for alum is to incinerate the bread, and examine the ashes; but many writers go on repeating the statement that alum may be found by digesting the bread in distilled water, filtering, and applying tests to the water. In this way seldom more than a trace of alumina can be detected, even when the bread contains a large quantity; but it is probable that many persons take this short and easy method of examining it, and it is probably in a great measure owing to this circumstance that the bakers continue to use alum with so much impunity. An instance came under my notice not many months ago where a baker expected, with the utmost confidence, to have a satisfactory certificate to lay before the committee of a club-house respecting his bread, although it contained a great quantity of alum.

A probable way to break through what seems the universal practice of bakers to adulterate bread, would be for the committees of the public hospitals and the guardians of the poor to oblige the bakers who contract to supply their respective institutions to furnish an unadulterated article. No one pretends that alum is either nutritious or wholesome; and if the loaves without alum should cost a little more, owing to their carrying less water, no one can doubt that as much nutriment would be obtained for a given sum as under the present system.

Sackville-street, June, 1857

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Commentary: Bread and alum, syphilis and sunlight: rickets in the nineteenth century

Anne Hardy

Rickets

Rickets was one of the most important hidden diseases of 19th century Britain: hidden because it did not appear among the certified causes of death, and because, not being a killer, it attracted little attention from the public health administration, whose pre-occupation was largely with the causes of death. By 1850, medical men were variously agreed on heredity, early weaning, improper diets, dirty skin, impure air, and a northern climate as playing a part in its aetiology, and in the 1880s its relationship with syphilis was much debated.[1,2] Although not a

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cause of death, it was a concern for the nascent paediatric profession, especially in Europe, where it generated a large literature. The root of that concern lay in the way the disease physically marked those who had suffered from it in early life. As Charles West, founder of the Children's Hospital at Great Ormond Street remarked, the physical characteristics of such people were 'familiar to us all': a stunted figure, large head, misshapen chest, twisted long bones, and enlarged wrists and ankles resulted in 'a physiognomy so peculiar that the effects of rickets cannot be confounded for a moment with those produced by any other disease'.[3]

Rickets was not a new disease in the 19th century. Descriptions of its physical signs can be found in early Chinese, Greek, and Roman texts. It seems, however, first to have become common in England around 1600, at a time when atmospheric pollution by coal smoke first became severe. Already in the 1650s it was noted that keeping clothes clean was problematic in smoky London.[4] At this time, the appearance of rickets as a new disease within recent decades was noted by Daniel Whistler and Francis Glisson, who also described its uneven geographical distribution: commoner in the south and west of the country than in Scotland and the north.[5]

By the mid 19th century, rickets was widespread throughout urban Britain, as in several north European countries. Contemporaries linked the rise of the disease to the great growth of cities that followed the industrial revolution. They were overcrowded, unplanned, sunless cities, whose peoples lived in dark and overcrowded conditions, meagrely fed, in conditions of poverty. In the space of a century, Britain became an urban country: in 1801, one-fifth of her population lived in towns; by 1901, four-fifths did so. Britain's skies became overcast from smoke, both from the great new industries, and from the millions of domestic coal fires kept burning for heating and cooking purposes. Coal consumption soared. The country's coal output rose from 17.4 million tonnes in 1811–1815, to 287.4 in 1913. Consumption doubled between 1830 and 1850, and again to 1875.[6] Little sunlight penetrated the urban smoke canopy, and it also encouraged women and children to spend their time indoors, out of the constant fall of oily, smoky smuts. For many babies and small children, the physical consequences of sunlight deprivation were compounded by poor diets and misguided childcare practices. The diets of working class women and children too often consisted largely of bread and tea, with sugar and the occasional smear of jam or margarine. Babies of all social classes were generally weaned on 'pap'—bread and water or bread and milk, depending on local custom; and they were often also kept indoors throughout the winter months. The impact of urban life on the incidence and distribution of rickets was very plain. A survey undertaken by the British Medical Association in the 1880s revealed a sharp distinction between the high incidence of rickets in the great urban conglomerations, and its virtual absence from small towns with populations of less than 5000, villages, and the countryside.[7]

John Snow and medical science

John Snow seems to have been a model of the able and ambitious Victorian doctor. He was apprenticed to a practitioner in Newcastle-on-Tyne between 1827 and 1833, after which he saw practice in Burnop Field and Pateley, Yorkshire, before going to London to finish his studies. He qualified in 1838, and set up practice in the then poor district of Soho. Snow is best remembered for his contributions to the epidemiology of cholera, and the introduction of inhalation anaesthesia, but he was a man of wide-ranging interests, publishing on a variety of topics, from the resuscitation of the newborn to capillary circulation. More particularly, Snow approached medicine from the basis of *scientific principle*, as can be seen in his work both on anaesthesia,[8] and on cholera. This approach places him within the radical, modernizing wing of 19th century medicine. By using scientific methods, including statistics, microscopy, chemistry, and animal experiments, such practitioners sought to distance orthodox medicine from the irregular practitioners, to elevate its social and professional status, to extend knowledge and improve practice—to make medicine respectable.[9] In adopting this approach, medical men also sought to transform their own personal prospects within a still overcrowded, socially marginal, and financially insecure profession.[10] It is this agenda that can be seen to lie behind Snow's work on the chemistry of anaesthesia and on the statistical distribution of cholera cases: he was demonstrably using science to inform practice. This is also evident in his article on the causes of rickets.[11]

Snow's epidemiology of rickets

Snow's paper on rickets[11] should be read in the light of the modernizing enterprise of 19th century medicine. It is a curious paper none the less. Unlike his cholera work, it rests on no firm statistical foundation. It is speculative: its basis lies in Snow's observation that rickets was common in London but not in the northern towns where he had practised, and the central hypothesis, that bread adulterated with alum, when the staple diet of young children, causes rickets, remains unproven. Snow admits the need for chemical and statistical evidence, but says he never had time to make those investigations. The hand of science is, however, clearly visible. The names of Liebig and Hassall signal that Snow's theory has an irreproachable scientific context.

Justus von Liebig (1803–1873) was a name to conjure with in the 1850s. An innovative, entrepreneurial German chemist, Liebig's object was to extend the boundaries of chemistry into agriculture, medicine, pharmacy, industry—to establish it as 'the most significant fundamental science for the modern age'.[12] He was especially influential in Britain, where ambitious groups of young chemists, doctors, and engineers adopted him as an icon for their own professionalizing campaigns. Snow's invocation of Liebig's observations on alum and its action on the chemical properties of wheat was, by way of an imprimatur for his own earlier surmise, that the sulphuric acid of alum would destroy the phosphate of lime in wheat, and thus its value in bone formation. Indeed, it was by this time widely accepted that a shortage of phosphate of lime in the diet caused rickets.[1,13]

Arthur Hill Hassall (1817–1894) was London's best known contemporary microscopist and food analyst. He had recently (1851–1854) published a series of devastating analytical reports on London foodstuffs in *The Lancet*.[14] Science here was again to the fore. Not only was Hassall using the newly rigorous technology of the microscope,[15] and the skills of the chemist in making his analyses, but the popular impact of his work had been greatly heightened by sets of diagrams illustrating pure

and adulterated foodstuffs under both medium and high-powered magnifications.[16] Snow's linking of his own analytical technique to Hassall's was intended to demonstrate both the sophistication and scientific credentials of his research methods.

The scrupulousness with which Snow detailed the highest scientific authorities for his chemical and analytical evidence sits oddly with the reticence he displayed in producing statistical or witness testimony for his arguments in respect of eating habits, type of bread consumed, and the distribution of rickets. As regards the latter, he excused himself: 'as my inquiries have only been of a colloquial nature, I hesitate to mention places and persons'. He had not even attempted a correspondence survey, although this was a method employed by other contemporary investigators. He did suggest a design for a comparative statistical inquiry, but had made no attempt to implement it himself. The article thus establishes that alum destroys the bone-hardening factor in bread, and that London bread is highly adulterated with alum. The inference is that a diet composed largely of such bread causes rickets, but the link is not scientifically proven.

Why did Snow publish this incomplete piece of research? Why did he not complete the statistical analysis—a task comparable, surely, to his work on cholera and the London water company fields in 1849–1854?[17] The article explicitly states that he thought it better to publish an imperfect inquiry, so that the medical profession might be alerted and the question resolved more quickly. Was he overburdened by his anaesthetic caseload, with the business of earning a living? Or had the chronic ill health and renal disease which he suffered sapped his energy and capacity for a rigorous research inquiry?[18] Had he completed it, what would such an inquiry have demonstrated? Could he—would he—have 'proved' his case?

The geography of rickets

It may be that Snow's desire to base his rickets theory on 'scientific principles', both in its metabolic and geographical aspects, narrowed his epidemiological vision and led him astray. The geographical focus of his inquiry was very narrow: London and the three northern towns where he had seen practice in his youth. Other 19th century inquiries into the epidemiology of rickets approached the question more broadly, several making use of the correspondence survey technique to cast a wide net. The expatriate Hungarian paediatrician, A Schoepf Merei, then practising in Manchester, used this technique in the early 1850s to ascertain the prevalence of rickets across Britain. Previously an exponent of the nutrition theory of rickets, his results convinced him that air quality was the most important causative factor.[1] In respect of his own home city of Manchester, notably, he inveighed against the 'vast mass of air … impregnated with unwholesome elements', which extended its rickets causing influence up to 4 miles outside the town. The 1889 BMA survey was, similarly, conducted by questionnaire to the BMA membership, and demonstrated that the disease was common in the large conurbations and the coalfields, but that small settlements and agricultural areas were virtually exempt.[19] The great medical geographer August Hirsch, whose research was very thoroughly grounded in the published European literature, and who employed a global geographical perspective, concluded that rickets was a disease of cold, wet climates, prevalent in Holland, Britain, Germany, and northern Italy, but absent in tropical and sub-tropical climates. And he noted, too, the speed with which rickety children recovered when removed to country air, or to tropical climates; and that the geographical distribution of rickets by no means corresponded with that of syphilis.[20] In the late 1880s, Theobald Palm, an Englishman who had practised in Japan and noted the absence of rickets there, consulted medical missionaries from India to China and North Africa and beyond, in an effort to establish the global reach of the disease. His conclusion, informed by the new 'Chemistry of Light', was that sunlight was the critical factor determining the geographical distribution of rickets. Sunlight, he observed, 'is essential to the healthy nutrition of growing animals … and is the most important element in the aetiology of the disease'.[21]

Palm's analysis began with the recognition that 'rickets is essentially a form of malnutrition', and he admitted that, 'it is most natural to think first of food in studying its aetiology'. By a process of elimination, he reached the fact that countries immune to rickets enjoyed abundant sunshine and clear skies. Britain, by contrast, suffered grey skies and want of sunshine, compounded in towns by 'a perennial pall of smoke, and ... high houses cut off from narrow streets a large proportion of the rays which struggle through the gloom.' It was in the narrow alleys where the children of the poor played, he noted, that this exclusion was worst, and it was here that most victims of rickets were found. Palm urged investigation of the physiological and therapeutic actions of sunlight: although the action of light on plants had received much attention, he observed, 'physiological chemistry has yet not much to tell us as to the action of light in animal nutrition'. In the early decades of the 20th century, scientific research began to unravel the complex relationship between sunlight and dietary vitamin D in the aetiology of rickets.[22,23]

Where does Snow's observation that children fed home-baked bread were free of rickets fit with the established model of rickets causation? Was it purely fortuitous? It may have been. Merei recorded rickets in Newcastle-on-Tyne in the early 1850s. And it is possible, as Snow himself almost admitted, that those northern diets contained other elements that kept rickets at bay—if not milk, then eggs or fish or bacon. Or maybe the medical practices in which he worked lay in areas where, as yet, the pall of smoke was not too dense, and children playing outside did so in sunlight.

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Commentary: John Snow and alum-induced rickets from adulterated London bread: an overlooked contribution to metabolic bone disease

M Dunnigan

Dr John Snow (1813–1858) is remembered for his hypothesis that cholera was communicated by contaminated drinking water. *On the Mode of Communication of Cholera*, published in 1849, was validated on 2 September 1854 when he persuaded the Soho parish Board of Guardians to disconnect the handle of the Broad Street pump.[1] The number of cases of cholera in the parish plummeted and Snow's fame was assured.

In contrast, Snow's Lancet paper of 4 July 1857[2] suggesting that the adulteration of bread with alum might be a cause of rickets has been forgotten. At first sight, scepticism seems justified since infantile rickets had been endemic in Northern Europe since at least the 17th century when the first clear descriptions of the disease appeared.[3,4] To an audience uninformed by chemical insight, Snow's hypothesis must have seemed eccentric. To a 21st century eye, aware of the potential interactions of aluminium salts with calcium and phosphorus metabolism, Snow's hypothesis is astonishingly prescient.

Snow observes that rachitic deformity is prevalent in areas where baker's bread adulterated with alum (aluminium potassium phosphate) is consumed (principally London and the south of England), while children in areas where home-baked bread, made from unadulterated flour, is consumed are rarely affected. He also observes that rachitic deformity is equally prevalent in children consuming adulterated bread in villages around London (where fresh air and sunlight are unrestricted), and in urban children of the more affluent middle classes. The absence of an urban–rural and socioeconomic gradient is not typical of classical Glissonian infantile rickets determined by restricted exposure to ultra-violet radiation and adherence to a strict lactovegetarian diet (predominantly bread and milk [saps] with added sugar in the first 2 years).[5]

Snow then proposes a hypothesis to explain the link between rickets and the consumption of alum which predates modern evidence by 70 years.[6] Utilizing the findings of the distinguished German chemist Leibig (1803–1883), that aluminium salts react with phosphorus-containing compounds to form insoluble aluminium phosphate, he suggests that this reaction may inhibit the absorption of dietary phosphorus required for the formation of skeletal 'phosphate of lime'. Finally, Snow proposes a case-control study of the prevalence of rachitic deformity in children under 4 years in 'towns where respectively the

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