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REPRINTS AND REFLECTIONS

On the adulteration of bread as a cause of rickets

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On commencing, in the year 1839, to see a considerable amount of practice amongst the poor of London, chiefly the out-patients of a public hospital, I was very much struck with the great number of cases of rickets. The complaint was shown more particularly in the bones of the leg, causing an outward curvature of the tibia and fibula; in children in their second and third year, it seemed almost the rule, and might be observed in the streets and the parks, as well as amongst children brought for advice. The complaint, moreover, was not by any means confined to the poor, but affected the children of the middle classes to a considerable extent.

The usual causes to which rickets are attributed are of a somewhat general nature, such as vitiated air, want of exercise and nourishing food, and a scrofulous taint. These explanations, however, did not satisfy me, as I had previously seen a good deal of practice in some of the towns in the north of England, where the over-crowding and the other evils above mentioned were as great as in London, whilst the distortion of the legs in young children was hardly present; moreover, I noticed that the most healthy-looking and best-nourished children often suffered most from curvature of the bones of the legs, owing to their greater weight; and I afterwards found that this complaint was quite common in the villages around London as well as in the metropolis itself.

The bones owe their hardness to phosphate of lime, which exists ready formed in many articles of food, and only requires to be assimilated, whilst in rickets the phosphate of lime in the bones is known to be deficient; and therefore it seemed extremely probable that the want of this earthy salt in the food of the infants of this metropolis was the chief cause of the soft state of the bones. My attention was naturally directed to milk, which contains one chief supply of phosphate of lime, and which is somewhat scarce and dear, and not of the best quality in London; but I immediately recollected that in some of the mining and manufacturing districts in the northern counties of England milk was scarcely used at all in the families of the operatives, and yet I had hardly seen a case of curvature of the legs from rickets. On reflecting on the subject of bread, however, there seemed to be something which might explain the prevalence of this complaint in London. In the northern counties, where coals are cheap, it was the universal custom for every family to bake their own bread, and I believe still remains so; whilst in the south of England it is as much the custom to buy bread from the baker. Now, the bakers, so far as I have examined, all put alum in their bread, whilst this is never done in domestic practice, and the flour dealers rarely adulterate the flour with this substance. They are liable to a heavy penalty for adulterating flour, but the law is never enforced against the bakers. I have never examined a specimen of flour which contained alum, or a specimen of baker's bread which did not contain it.

When my attention was first turned to the subject of rickets, I thought it likely that the sulphuric acid of the alum would decompose the phosphate of lime of the wheat, and form sulphate of lime, which would not be available as nourishment for the bones; and I formed an intention to investigate the question both chemically and statistically; but this intention was long postponed, on account of other engagements and inquiries. In the meantime, and without any regard to the question of rickets, Liebig has inquired into the action of alum in bread, and his investigation will justly have more weight with the reader than any inquiry of mine. He says, 'Since phosphoric acid forms with alumina a compound hardly decomposable by alkalies or acids, this may perhaps explain the indigestibility of the London bakers' bread, which strikes all foreigners.'*

It is evident from the above passage that Liebig has ascertained that alum decomposes the phosphate of lime of wheat, and it is not likely that the bones would be able to nourish themselves with this salt out of phosphate of alumina and sulphate of lime; and where baker's bread forms the chief and almost the only article of food, as it does amongst the children of the working classes in London and many other towns, one might expect the bones to be ill-nourished, as regards their earthy and hardening material. This appears to be the actual fact, as far as I have been able to extend my inquiries. The subject is capable of being decided by an exact numerical investigation, but I have thought it better to publish my inquiry in its present imperfect state, than to wait till I should be able to make such a complete research as I could wish, more especially as, by directing the attention of the profession to the question, it may be earlier decided. I expected to be able to contrast some of the large institutions containing young children in this metropolis with each other; but, so far as I have inquired, they are all supplied alike with bakers' bread containing alum. So far as I have been able to learn, rickets are not common at present in the towns in the north and west, where home-made bread is chiefly used; and I was lately told, that in one town in Cornwall, where the people make their own bread, this complaint is almost absent; whilst in a town a few miles off, where bakers' bread is consumed, the complaint is extremely common; but as my inquiries have been only of a colloquial nature, I hesitate to mention places and persons. If it could be obtained, perhaps a return of the number of cases of rickets in the children under four years, as compared with the whole number, which are

*Letters on Chemistry, third edition, p. 443.

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

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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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people buy chiefly flour or ready made bread … to decide the question'.

This hitherto neglected paper is clearly the product of astute clinical and epidemiological observation combined with scientific insight, resulting in what appears to be the first paper in the medical literature to raise the issue of the potential toxicity of ingested aluminium compounds in man.

The unrecognized implications of Snow's observations for the public health of Londoners in the 19th century are sobering. Snow states that London bakers would add about 1½ ounces of alum per 4 lb loaf (42 g per 1800 g bread). Given that a manual labourer might consume 70% of his energy requirements as bread, he could ingest 20 g of alum daily [$AlK(SO_4)_2 + 12H_2O$], equivalent to the aluminium content of 4 g aluminium hydroxide [$Al(OH)_3$]. This is the maximum recommended daily dose of this compound as an antacid and alum in equivalent quantities may have been consumed continuously in adulterated bread by a substantial proportion of the capital's population.

Long-term intakes of aluminium salts of this order may induce hypophosphataemic osteomalacia.[7] A phosphorus depletion syndrome has been described in which prolonged intakes of aluminium-containing antacids resulted in hypophosphataemia, hypercalciuria with calcium resorption from bone, and general malaise, debility, anorexia, and muscle weakness.[8] In children subsisting on bread as a basic food following weaning, at a period of peak growth velocity, the effects of chronic aluminium ingestion may have been severe, resulting in rachitic deformity. In subjects with impaired renal function, unable to excrete the small proportion of aluminium absorbed from the gut, the potential benefits of a reduction in high serum phosphate levels may have been offset by an accumulating body burden of aluminium leading to worsening renal osteodystrophy and deteriorating cognitive function, well illustrated by aluminium-induced dialysis encephalopathy and dementia.[9]

Analogous to its present day use as a decolourant in water treatment plants, alum appears to have been added to bread as a whitener (as was chalk and bone-meal). A statute to prevent the adulteration of bread which specifically mentions alum was passed in the reign of King George II in 1757 and widely ignored, as Snow notes. I have been unable to discover when the practice was discontinued.

It would be of interest to verify Snow's hypothesis. Spectrophotometric analysis of bone samples from the skeletons of mid-19th century Londoners, presumably available in the city's many anatomy and pathology collections, would detect the presence of abnormal quantities of aluminium. This investigation might belatedly vindicate (or rebut) Snow's perception of a major toxicological hazard to which London's population were unwittingly exposed as they consumed their daily bread.

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Commentary: Snow on rickets

Nigel Paneth

John Snow's little piece on rickets,[1] written less than a year before his death, illustrates in miniature the integrative thought processes that made him a founding figure of both epidemiology and scientific anaesthesiology. Snow was one of those rare medical scientists who move effortlessly across conceptual categories usually kept distinct. In studying cholera, anaesthesia, and rickets, he investigated the distribution of molecules in solution and the distribution of diseases in populations. Snow's great contribution to epidemiology—unravelling the mode of transmission of cholera decades before germ theory—was an exercise in the blending of ideas operating at molecular, pathological, clinical, and epidemiological levels. His understanding of molecular forces in living things led him to hypothesize a minuscule, reproducing agent of disease. His view of the intestinal nature of cholera

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pathophysiology led him to hypothesize fecal–oral transmission. And his observations of the geographical and temporal features of acute outbreaks led him to hypothesize that municipal water supplies maintained urban cholera epidemics.

Snow turned the administration of anaesthesia from a parlour game, a hit-and-miss medical oddity, into a medical technology of supreme importance because of his insight into the chemistry of vaporizing gases, including the influences of temperature, humidity, and dosage. But Snow also recognized the need for systems of care to monitor anaesthetic safety (he was the first to insist that the anaesthetist not be the surgeon), and was virtually unique in compiling careful comparative records of the rate of anaesthetic accidents with different agents. His scientific work was nearly always accompanied by specific suggestions for improving clinical practice or maintaining public health.

In his paper on rickets, Snow's first observation is epidemiological, his second chemical. His chemical knowledge taught him that the underlying problem in rickets is undermineralized bones, deficient in phosphate of lime. Nearly 70 years later, the pathophysiology of rickets would be described in virtually identical terms.[2] From chemistry, Snow reasoned that deficiency of milk —which he knew to be rich in calcium phosphate—might lead to rickets. But the epidemiology was unsupportive—few cases of rickets were seen in Snow's early practice in the less-industrial corners of northern England (he was apprenticed in Newcastle, and in the Yorkshire villages of Pateley Bridge and Burnop Field), but many poor children in the north were without milk in their diets. Searching for a cultural or behavioural difference that would explain the excess of rickets in London, and focusing especially on diet, Snow hit upon bread, which northerners baked themselves, but Londoners bought from stores.

What Londoners bought to eat, as Snow's friend Arthur Hill Hassall showed repeatedly during the 1850s, was hardly what they thought it was. Hassall made a career of buying foods in London shops, testing them chemically, examining them microscopically, and publishing his usually shocking findings in the *Lancet* or in his several books. The level of deliberate adulteration of foods in London then was truly astonishing. Additives, frequently quite poisonous, were put in foods to add weight, to add colour, or to cover offensive odours. Of 42 samples of mustard tested by Hassall, not one was free of flour colored with turmeric.[3] Not only were 90% of coffee samples adulterated with chicory, but the chicory was itself adulterated with flour, corn, ground acorn, or even sawdust. Black tea was coloured with black lead, green tea with Prussian blue (ferric cyanide). Bread was, as Snow found, contaminated with alum, but you were a lucky Londoner indeed if your bread did not also contain mashed potatoes, water, or rice flour.

Hassall reports that alum (potassium aluminum phosphate) contamination of bread was a cottage industry for London bakers. The compound is most stable with water molecules attached, making it excellent for adding weight, and it makes flour look whiter. Still a component of some baking powders today, it also helps bread rise. Virtually every baker in London had a druggist who supplied him with what was called 'hards' or 'stuff' in the trade, a mixture of rock alum and salt, added, so Hassall estimates, at about half a pound per sack of flour. The loaves Snow tested, with 500–600 grains of alum per 4 lb loaf, were even more densely contaminated.

Was Snow right that alum could precipitate out dietary calcium phosphate and thereby contribute to rickets? While such a process has not been reported for alum, other aluminum salts, such as are found in antacids, have been found to interfere with intestinal phosphate absorption by competitive binding, even producing rickets on occasion.[4]

In Snow's time, rickets was just another part of the vast spectrum of disease that was the special plague of the poor. The social distribution of disease implicated the evils of the industrial world—close living quarters, bad hygiene, bad ventilation. To many public health reformers of 19th century England, distinguishing one 'miasmatic' disease from another was hardly worth the effort. Edwin Chadwick put it thus:

> The various forms of epidemic, endemic and other disease caused, or aggravated, or propagated chiefly among the laboring classes by atmospheric impurities produced by decomposing animal and vegetable substances, by damp and filth and close and overcrowded dwellings prevail amongst the population in every part of the kingdom.[5]

To the modern ear, Chadwick's failure to implicate nutrition among the list of disease-inducing evils seems a curious omission, but, as Christopher Hamlin has shown, it was not accidental.[6] To Chadwick, and to many in the sanitary reform movement, criticizing drains and housing removed the onus of ill-health away from factories and their near slave-labour conditions.[7]

Snow saw things differently. The son of a Yorkshire labourer and of a mother born out of wedlock, he knew the distinction between filth and disease, and sought the specific elements of the social environment that facilitated the development and spread of specific diseases. An experimentalist at heart, he sought out the 'natural' (but usually man-made) experiments that could test his disease hypotheses. The Thames water that supplied South London houses came from two sources—one far above, the other just below, London's sewer outlets—a circumstance which Snow exploited to show the impact of fecalized water on cholera mortality. He searched for an analogous contrast to test his bread–rickets hypothesis, but could not find comparable settings supplied with different kinds of bread.

Having concluded that cholera was transferred by the fecal–oral route, Snow thought of ways to address the public health problem. The poor suffered more from cholera, not, as many sanitarians held, because of their immoral behaviour, but, said Snow, because they had less light in their homes to notice fecal contamination, and fewer washing facilities to avert it. Miners —a special concern for Snow since his youthful experience managing a cholera epidemic in a coal mine—needed shorter shifts so they would not have to bring their meals to the running sewers in which they worked, inevitably contaminating their food. His recommendations on keeping urban water supplies free of fecal matter, not widely implemented until a decade after his death, did more to control cholera than did the liming of streets and the abolition of cesspools recommended by London sanitary authorities. Indeed, since cesspools were replaced by sewer lines that fed raw sewage directly to the Thames, and thereby to the water supply, this sanitary 'improvement' surely increased cholera mortality, as Snow quietly pointed out.[8]

In rickets too, Snow sought aetiological uniqueness within the broader theme of poverty and disease. He missed perhaps the chief culprit, the lack of sunlight which shone more on the ruddy village poor of Yorkshire than on the denizens of London's

dark alleys, but his approach to the problem—a blend of astute social observation and up-to-date chemistry—has much to recommend it to the modern epidemiologist. And in proposing that the authorities 'oblige' bakers to supply institutions for the sick and poor with unadulterated bread, he emphasized another useful message—the importance of translating science into public policy.

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