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(SO4)2 + 12H2O], 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.

References


© International Epidemiological Association 2003 Printed in Great Britain

ON THE ADULTERATION OF BREAD AS A CAUSE OF RICKETS 341

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

DOI: 10.1093/ije/dyg159

College of Human Medicine, Michigan State University, Department of Epidemiology, Suite 600, 4660 S Hagadorn Road, East Lansing, MI 48823, USA. E-mail: paneth@msu.edu

Downloaded from https://academic.oup.com/ije/article-abstract/32/3/341/637087 by guest on 10 March 2019
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 Burnnop 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.

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.

References
5 Chadwick E. Report to Her Majesty’s Principal Secretary of State for the Home Department, from the Poor Law Commissioners on an Inquiry into the Sanitary Condition of the Labouring Population of Great Britain; with Appendices. London: HMSO, 1842, p. 369.