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Scientific knowledge and clinical authority in dentistry: James Sim Wallace and dental caries

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ABSTRACT Once the germ theory had become generally accepted within medicine, the importance of experimental science to the improvement of medical practice could no longer be reasonably doubted. However, clinicians still sought to retain control of how knowledge that had originated in the laboratory was interpreted and applied within practical diagnostics and therapeutics. Thus how practitioners incorporated new scientific knowledge into their medical discourse and practice is a matter for careful empirical inquiry. James Sim Wallace, born in Renfrewshire in 1869 and a graduate in medicine from the University of Glasgow, was a leading figure in British dentistry throughout the first half of the twentieth century. Through an examination of his voluminous writings, we explore how the new 'chemico-parasitical' theory of dental caries was accommodated within dentists' understanding of oral hygiene. The paper also looks at the controversies that surrounded the application of the vitamin theory to the problems of rickets and dental caries, focusing on the contentious interaction between Sim Wallace and his colleagues, on the one hand, and the eminent physiologists May and Edward Mellanby, on the other.

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The process by which the germ theory of disease became accepted by the medical profession, at the end of the nineteenth century, has recently been accorded considerable attention by historians.[1,2] That the bacteriological revolution ushered in major innovations in medical knowledge and practice is undeniable. However, in an authoritative overview, Michael Worboys has tellingly argued that in many fields of medicine an important factor in the acceptance of bacteriology was not that it compelled radical changes in practice but that it could be accommodated at the level of theory, while leaving day-to-day routines to a great extent unchanged.[3] Public health doctors, for example, could argue that the identification of a waterborne *Vibrio* as the immediate cause of cholera merely reinforced the concern that they had long had with the proper disposal of faecal matter. How practitioners incorporated new bacteriological knowledge, or indeed any other scientific innovation, into their medical discourse and practice cannot be taken for granted and is, therefore, a matter for careful empirical inquiry.

Both Worboys and Terrie Romano have fruitfully linked their discussion of the rise of the germ theory with another issue that has, in the past two decades or so, structured much of the historiography of late nineteenth- and early twentieth-century medicine, namely the relation between laboratory science and clinical medicine.[4,5] The bacteriological revolution was, of course, a great triumph for laboratory-based workers.[6] The crucial importance of experimental science to the improvement of medical practice could no longer be reasonably doubted. However, clinicians still sought to retain control of how knowledge that had originated in the laboratory was interpreted and applied within practical diagnostics and therapeutics. Experimental results, it was argued, could not necessarily be applied to the understanding of human diseases. This attitude did not entail, as Lawrence has emphasised, an outright rejection of science.[7] Some of the leading clinicians of the late nineteenth and early twentieth centuries had scientific interests of their own, and many were happy to accommodate new diagnostic aids, for instance, into their clinical routine. The key issue was rather one of authority and control. Their crucial claim was that it should be the clinician, and only the clinician, who should decide when and how it was appropriate to apply new scientific knowledge within the consultative encounter.

It is remarkable that there has as yet been no serious study of the impact of the bacteriological revolution upon dental theory or practice. Nor has any scholarly attention been given to exploring how tensions between laboratory scientists and clinical practitioners might have been expressed within dentistry. The present paper examines aspects of the writing of James Sim Wallace, a leading British dentist (and controversialist) of the early twentieth century, with the intention of initiating the exploration of these important questions within the historiography of dentistry. We hope to show that dentistry promises to be a particularly fruitful area in this regard because its central clinical problems encompass not only bacteriology but also issues of diet and nutrition. How scientific knowledge was best applied to food and feeding has already been identified as having been a contentious matter in the

context of the relations between clinicians and laboratory workers in early twentieth-century Britain.[8] We will explore Sim Wallace's close involvement in the lively debates surrounding the role of 'accessory food factors' that followed the enunciation of the vitamin theory in the second decade of the twentieth century.

SIM WALLACE AND THE CAUSES OF DECAY

James Sim Wallace was born in 1869, in Shawlands, then part of Renfrewshire in the west of Scotland.[9] He studied at the University of Glasgow, graduating with a BSc in biology in 1889 and then with a medical degree in the following year. He attended the Glasgow Dental Hospital and probably acquired some experience of dental work in the practice that his father and elder brother ran in the city.[10] In 1893, after a period as a ship's doctor, he gained an MD, also from Glasgow University. Moving to London, he studied at the National Dental Hospital of London (the forerunner of University College Dental Hospital) and the London Dental Hospital (the forerunner of the Royal Dental Hospital), obtaining the Licentiate of Dental Surgery of the Royal College of Surgeons of England in 1895.

Sim Wallace quickly established himself in dental practice in London. By 1903, when he first chose to enter his name on the Dentists' Register, he had rooms in Wimpole Street, a fashionable location for a London practitioner. He was, or had recently been, Honorary Dental Surgeon to the West End Hospital for Nervous Diseases and the Kingston Victoria Hospital, and Assistant Dental Surgeon to the National Dental Hospital. He had already published his first book, *The Cause and Prevention of Decay in Teeth*, which appeared in 1900 and for which he was awarded a Doctorate of Science, again by Glasgow University.[11] This is said to have been the first DSc to be awarded for a dental topic by a British university.

Sim Wallace went on to be a leading figure in British dentistry for more than 40 years. He combined his successful private practice with, among several other appointments, a lectureship in preventive dentistry at King's College Hospital, the first such post in the UK, to which he was appointed in 1923. He was the President of the British Society for the Study of Orthodontics in 1910 and recipient of the Tomes Prize (1923) and the Cartwright Prize (1925) from the Royal College of Surgeons of England. Remarkably prolific, he was a frequent contributor to the dental periodical literature and the author of several books, including one on oral health for a lay readership.[12]

An active public campaigner in the field of oral health, Sim Wallace believed that it should be the duty of medical officers of health (MOH) to work toward the prevention of dental disease. He worked closely with Dr James Wheatley, MOH for Shropshire, in designing a dental health campaign and an inspection regime for that county.[13] As a member of a committee set up by the Society of Medical Officers of Health, he helped devise recommended dietaries and prepare a leaflet on oral hygiene, which was distributed widely by the People's League of Health.

In his 1900 publication, Sim Wallace acknowledged that the 'chemico-parasitic' theory of the aetiology of dental caries had now become 'universally recognised'. Dentists had, in other words, accepted that the germ theory was applicable to diseases of the teeth. This crucial extension of the new knowledge of bacteriology had first been fully articulated 15 years earlier by Willoughby Dalton Miller, an American dentist who had worked in Robert Koch's laboratory in Berlin.[14–16] Bacterial fermentation of dietary carbohydrates, on the dental surfaces and interstices, produced acid, which eroded the enamel. Sim Wallace maintained, however, that micro-organisms, and their injurious by-products, should only be regarded as the 'direct or exciting cause' of decay. He wished to concentrate upon what he saw as the underlying, 'predisposing' causes, which to him were diet and other factors fundamental to oral hygiene.

Sim Wallace continually emphasised that his opinions were based on his clinical experience, upon observations carried out on his own patients.[17] He had begun his research into the aetiology of caries by asking those of his patients whose teeth were badly affected by decay what they ate. Having made a list of suspect foodstuffs, he then carried out a series of experiments to determine what he termed 'lodgeability', the extent to which different foods adhered to the teeth after chewing. On the basis of these observations, he concluded that it was not so much the type of food that was important as how the food had been prepared. He became concerned, in particular, about the elimination by modern methods of milling and cooking of naturally occurring fibrous matter from foodstuffs. A lack of fibrous matter reduced the need for mastication and, as a consequence, food debris was removed from the teeth less effectively. It was noted that, by contrast, sugars and refined carbohydrates were increasingly abundant in the modern diet. Because of their abundance and the shorter time spent chewing, sugars accumulated in the film that naturally covered the teeth. These were broken down by micro-organisms to form the acids that initiated the first phase of caries.

Sim Wallace also considered that an irregular dentition might be an important predisposing cause of caries. He echoed a common medical opinion that the full development of organs, including those of the mouth, was dependent on functional activity in childhood. To achieve an efficient masticatory organ it was necessary that the dental arches be fully developed and that the teeth be accurately positioned within them. For this to occur spontaneously, some organising force must guide the teeth into their proper place. Normally, the tongue, together with the cheeks and lips, provided this control. Modern diet, rid of its coarse fibrous material, required little or no mastication

and led to reduced activity of the tongue and, thus, to its underdevelopment. Consequently the teeth and alveolar bone were denied the moulding effect of the tongue and the constricting forces of the lips and cheeks were unopposed. During breast-feeding, Sim Wallace argued, the lingual muscles were actively employed in suckling. By contrast, a bottle-fed infant could take in milk more easily, leading to less exercise for the tongue. If the tongue did not develop fully, the lateral forces it exerted on the dental arches were reduced, which could result in V-shaped arches, in which the teeth were crowded together. The resultant irregularity and malocclusion diminished the ability of the mouth to cleanse itself, again predisposing to the accumulation of food debris on and between the teeth.

Sim Wallace dismissed the widely held view that caries was an inevitable result of civilisation and/or hereditary degenerative changes in human dentition.[18] He maintained, on the contrary, that decay could be prevented by proper attention to diet and oral hygiene. He recommended foods of what he termed a 'detergent nature' and provided examples of good and bad diets. One particular *bête noir*, frequently castigated in his writings, was milk pudding. Food of this sort was dismissed as 'pap'. On the other hand, he endorsed the consumption of fish, meat, poultry, bread, vegetables and fruit. He doubted the utility of antiseptic mouthwashes, which had been recommended by some authorities on the basis of the germ theory of caries. Sim Wallace pointed out that neither the mouths of animals, nor of human beings who were free from caries, were aseptic.

In 1902, a second edition of *The Cause and Prevention of Decay in Teeth* was published.[19] The only substantial new material was contained in an appendix, which described an attempt to induce decay experimentally in a monkey. To simulate the modern human diet, Sim Wallace fed his solitary experimental subject biscuits, milk, chocolate and cheese, all foodstuffs containing little fibrous material. However, once the monkey had consumed the food provided, it proceeded to eat its bed of hay, obviously rich in fibre, thus spoiling the experiment. Sim Wallace removed the hay and lined the box with cork. The monkey turned to chewing the cork and was then transferred to an unlined wooden box, the walls of which it once again promptly started to gnaw. Sim Wallace then rubbed the exposed surfaces of the box with 'foul tasting' quinine, until eventually the frustrated monkey gave up.

The original experimental diet continued, occasionally supplemented by treacle and the much-maligned 'milk pudding'. After four months the monkey's teeth were examined. Sim Wallace was suspicious that caries had developed but decided to wait another six months. However, a few days later, the experiment was brought to a premature end, an outbreak of fire having resulted in the suffocation of the monkey. Sim Wallace was, however, able to recover the teeth and section them. Caries was definitely present, he concluded.

Sim Wallace's contentions about the importance of the 'predisposing causes' of caries, as against the 'immediate cause', have a close structural similarity to the arguments that Warboys has described as being central to the response of public health doctors to the germ theory in the late nineteenth century. As John Simon put it:

> Whatever be the explanation… we know that… the pestilence rages only when there are definite sanitary evils. The knowledge remains unchanged and unchanged remain also our practical means of applying it… Excrement-sodden earth, excrement-reeking air, excrement-tainted water, these are for us the causes of cholera.[20]

In other words, as the scientific evidence for the germ theory accumulated, the public health doctors argued that whatever the immediate cause of cholera might be, it was still their efforts in keeping the cities clean that would ensure the protection of the population from an outbreak. In other words, the measures upon which public health doctors had long based both their own professional reputations and the reputation of their discipline had been effective in the past and would continue to be effective. The advent of a new bacteriological explanation for the transmission of cholera should not lead to the public losing confidence in the advice that they had previously received from the medical profession.

The germ theory received a great boost in 1882 when the German scientist Robert Koch identified the bacillus of tuberculosis. The British medical profession quite quickly accepted that the immediate cause of tuberculosis was a micro-organism. However, once the search for the tubercle bacillus was undertaken systematically, it was soon realised that it could be found everywhere. A large number of healthy people were exposed to the germ everyday, yet did not succumb to infection. The preferred explanation for this observation was often couched in the form of a 'seed and soil' metaphor. Some people were susceptible – their bodies provided suitable 'soil' for the 'seed' of tuberculosis, the bacillus, to grow in – and others were not. In other words, the germ theory of tuberculosis could be readily accommodated within the older view that the essential predisposing causes of the disease were defects in environment, lifestyle or genetic inheritance.[21]

In a similar fashion, Sim Wallace was willing to accept the germ theory of dental caries but continued to emphasise, in a series of publications, that the matters that had traditionally been the central concern of dentists – namely diet, efficient mastication and oral hygiene – remained crucial to its successful prevention:

> … nature keeps the mouth and other parts clean and in a hygienic state by preventing the undue lodgment or stagnation of food. So long as food and the accompanying parasites or microbes are kept 'on the

J R Coll Physicians Edinb 2009; 39:64–72

> move', the hygiene of the mouth and alimentary canal is generally well provided for, but let food stagnate too long in the crevices of the teeth or in kinks elsewhere and trouble is very likely to ensue.[22]

Food debris was, almost literally, the seedbed in which the germs of dental caries would grow. To ensure protection from disease, patients could, therefore, continue to rely on the advice on oral hygiene that dentists had long provided them with. Indeed, Sim Wallace argued that the fundamentals of an adequate preventive regime had first appeared in the dental literature as early as 1530, thus anticipating the germ theory by several centuries.[18]

While Sim Wallace based his arguments firmly and primarily on his clinical experience, he did not reject experimentation as a means of providing further support for his views. The laboratory method, in the shape of feeding experiments upon a solitary monkey, had served an auxiliary, confirmatory function in the buttressing of his argument. Nor did he reject the use of instruments of precision. Indeed he endeavoured to improve the application of such instruments within his own discipline. In 1909, Sim Wallace published his design for an improved prosopometer, an instrument for measuring the face and the jaws.[23] A modification of an instrument originally devised for anthropological work, Wallace's prosopometer, was an effective clinical tool for taking standardised measurements at different ages, producing a composite picture of the changes that had occurred consequent to growth and orthodontic treatment.

RICKETS, DENTAL CARIES AND ORAL HEALTH

By 1912, Sim Wallace had extended his dietary hypothesis to consider the aetiology and development of some major diseases, in particular tuberculosis and rickets. He argued, for instance, that adequate fibre in the diet, resulting in the proper development of the buccal cavity and its associated structures, coupled with prompt and effective treatment of tonsillar and adenoidal pathology, would reduce the chances of the tubercle bacilli gaining entry to the body through the mouth.[24] But it was principally his views on rickets that were to lead him into serious controversy in the 1920s and 1930s.

The aetiology of rickets had been a contentious issue for some time. In 1908, Leonard Findlay, a Glasgow paediatrician, had reported the results of experiments in which he had tried to induce rickets in pups, concluding that:

> [I]t is doubtful… if feeding plays any part in the aetiology of rickets. Examination of the conditions under which rachitic children are reared reveals one constant and invariable factor in their lives, namely confinement. Alike, then, on clinical and experimental grounds I accordingly conclude that confinement, with consequent lack of exercise, is the main factor in causing the disease.[25]

Glasgow University was a major centre for rickets research in the 1910s and 1920s. Findlay and his colleague, Noël Paton, Regius Professor of Physiology, led a series of investigations into the incidence and cause of the disease. In 1915, Findlay argued that the data that had been generated supported the exercise and hygiene theory, as it was taught in Glasgow, against the dietary hypothesis.[26] However, in 1917, Edward Mellanby claimed to have shown that rickets could be produced or prevented in pups by the manipulation of their diet.[27] In 1918, Mellanby explicitly linked his work on rickets to the newly emerging vitamin theory, stating categorically that rickets was 'a condition primarily due to the lack of an accessory food factor in the diet'.[28]

This assertion was emphatically rejected by Paton, Findlay and their colleagues, and a major scientific controversy ensued between the 'Glasgow School', as they were known, and those who supported Mellanby and the vitamin theory of rickets. Prominent in Mellanby's camp was Frederick Gowland Hopkins, Professor of Biochemistry in Cambridge and the doyen of the discipline in Britain. Sim Wallace, who was roughly of the same generation as Findlay and who had been taught the exercise and hygiene theory of the aetiology of rickets as an undergraduate in Glasgow University, chose to participate actively in this controversy.

In the early 1920s, May Mellanby, Edward's wife, received funding from the Medical Research Council (MRC) to conduct a large-scale study of the effect on the teeth of experimental manipulation of the diet.[29] Mellanby used pups as her experimental animals and sought, in particular, to assess the role of the 'fat-soluble accessory food factor' in tooth formation and in susceptibility to dental caries. She concluded that the absence of the fat-soluble accessory food factor in the diet produced atrophy and metaplasia of the enamel-forming organ and, subsequently, atrophy and irregular functioning of odontoblasts. The result was hypoplasia of the teeth and serious abnormalities of both enamel and dentine. All of which, Mellanby argued, increased susceptibility to caries. In effect, she was associating her work on caries with her husband's work on rickets. Both, it was argued, were deficiency diseases, resulting from disorders of calcium metabolism, consequent upon the absence from the diet of an accessory food factor.

Following on from her initial studies using dogs, May Mellanby collected more than 1,000 human deciduous teeth.[30] She claimed that only about 14 per cent of the total sample were fully developed. About one-quarter were slightly underdeveloped, but nearly two-thirds were moderately or grossly hypoplastic. She then examined 266 adult teeth, removed during orthodontic treatment, and concluded that not one was sound. Accordingly, she recommended dietary supplementation, of both adults and children, with cod liver oil as a preventative against the development of caries.

Mellanby's observations were based upon the careful sectioning and microscopical examination of teeth. She

argued that information about the structure of teeth obtained macroscopically, as in the clinical situation, was necessarily fallacious. She therefore regarded the practising dentist, as JD Manson puts it, as 'an imperfect witness' on the matter of dental health.[31] Moreover, she asserted that her work had shown Miller's chemico-parasitic theory to be 'comparatively unimportant'[32] and she described the theory of 'defective hygiene' as 'hopeless'.[33] The incursion of May Mellanby, a physiologist, into the sphere of human oral health understandably antagonised several leading members of the dental profession. The distinguished dental surgeon Sir Frank Colyer described her as a 'harmful intruder',[31] but Sim Wallace led the counterattack.

Not only was Mellanby associating the cause of dental caries with the still controversial vitamin theory and casting doubt, at least implicitly, on the efficacy of the advice on oral hygiene routinely given by dentists, she also directly challenged the healthiness of the fibre-rich diet that Sim Wallace recommended. Edward Mellanby had claimed, in the course of his research into rickets in dogs, that 'phytic acid', present in cereals and legumes, blocked the calcifying action of the fat-soluble accessory food factor. May Mellanby extended this assertion to cover the formation and maintenance of teeth. She asserted that as cereals increase in the diet, the consumption of protective foodstuffs, such as milk, butter or cod liver oil, must also be increased to offset their anticalcifying effects.

Of all the pulse and grain foodstuffs, Edward Mellanby particularly condemned oatmeal. He had the courage to express his disapproval of Scotland's national cereal while addressing the 1922 British Medical Association meeting in Glasgow. His comments were met with derision. When Mellanby's second MRC report[34] was published, three years later, there was a fresh wave of ridicule. *The Scotsman* newspaper asked under the headline 'Ban on Porridge':

> Are Scotsmen to forswear oatmeal because a Sassenach Professor has pronounced it to be deficient in 'anti rachitic vitamin'? … The suggestion that the consumption of oatmeal is a fertile cause of rickets, and that animals that eat oats must be 'exposed to some sort of ultra violet radiations', if their bones are to grow straight and of the right shape, is enough to fill the national bosom, if not with wrath, with derisive laughter.[35]

Mellanby had also antagonised Scottish medical opinion. Douglas Chalmers Watson, physician to both the Royal Infirmary of Edinburgh and the Royal Hospital for Sick Children, and the author of *Food and Feeding in Health and Disease: a Manual of Practical Dietetics*,[36] wrote a strong letter to *The Scotsman*, reassuring its readers of the value and safety of oatmeal.[37] Dr William Robertson, MOH for Edinburgh, likewise criticised Mellanby in the *Manchester Guardian*.[38]

Although a Scot, Sim Wallace stood a little apart from his medical compatriots on the issue of the intrinsic healthiness of the national cereal. He did express concern that if oatmeal was milled too finely, the resulting porridge would not need much mastication and thus the cleansing effect normally associated with fibrous foodstuffs might be lost.[17] He was particularly antipathetic to the practice of adding sugar to porridge. But any danger lay in the artificial refinement of the oatmeal and sugar, not, as the Mellanbys maintained, in a cereal-based diet per se.

In a long series of publications, Sim Wallace expressed strong criticism of both May Mellanby's methods and her conclusions. While he conceded that vitamin deficiency might produce tooth hypoplasia under experimental conditions, he contended that such results could only be achieved with 'freak diets which are seldom even simulated in human beings or wild animals'.[39] Here Sim Wallace was echoing one of the key arguments of the 'Glasgow School'. Edward Cathcart, the third principal member of the Glasgow group and Paton's successor as Regius Professor of Physiology, regularly argued that the overall importance of vitamins in practical dietetics was greatly exaggerated.[40]

Cathcart warned that laboratory scientists were trading in abstractions of little relevance to clinical actualities and that extrapolating from artificial feeding experiments to human circumstances was inherently problematic. Similarly, Sim Wallace contended that:

> The possible association of dental hypoplasia, rickets and dental caries was investigated years ago by reliable observers in the medical and dental professions – not with regard to what happens in dogs, but with regard to children, which is more important, for it is this that we want to know about.[41]

Sim Wallace argued that May Mellanby had failed to demonstrate any increased susceptibility to caries, even in hypoplastic teeth, that could be attributed to vitamin deficiencies. Mellanby had found fewer hypoplastic teeth in the mouths of children attending private schools, compared with those attending state schools, and had claimed on this basis that a qualitatively adequate diet protected against dental disease. But Sim Wallace pointed out that the social distribution of caries was precisely the reverse:

> In recent years, dental examinations of multitudes of school children have been made, and one of the generalisations resulting from such examinations is that 'the better the school, the worse the teeth', which rather confirms the view, long held by dentists, that dental caries is not a deficiency disease; rather it is a luxury disease, depending upon the stagnation of easily fermentable and generally expensive carbohydrates.[22]

Sim Wallace was not the only leading dentist who took issue with May Mellanby's claims. Wilfred Fish, a lecturer at London's Royal Dental Hospital and a West End practitioner, also criticised her work:

J R Coll Physicians Edinb 2009; 39:64–72

> … it is claimed [… Mellanby, 1934] that the exhibition of therapeutic doses of the vitamin will both prevent and arrest dental caries. This conclusion is based on a series of controlled clinical experiments, but does not coincide with general clinical experience.[42]

Fish conducted a feeding test with five dogs (three experimental animals and two controls), which showed, to his satisfaction, that it was 'impossible to affect the calcium content of the dentine in any way whatever' by manipulation of the diet. He came to a similar conclusion with respect to the enamel. Fish was a more sophisticated experimentalist and clinical scientist than Sim Wallace but, in this instance at least, his argument is structurally identical to that of his older colleague. Like Sim Wallace with his solitary monkey, Fish undertook a relatively small-scale experiment that provided data to confirm a prior finding based on clinical observation. Fish also implicated the lack of fibre, not the lack of accessory food factors, in the diet of Western civilisation as the cause of dental disease.

In the 1920s, Sim Wallace began to place more emphasis on the role played by saliva in oral health.[17] He pointed out that salts dissolved in saliva acted as buffers, conferring chemical protection to the oral tissues when acidic foods were consumed, and stressed the significance of recent reports that saliva was capable of 're-hardening' superficial layers of enamel that had been softened by the acids produced by carbohydrate fermentation. Sim Wallace accorded great importance to the role of mucin in saliva, which, he believed, facilitated the collection of food particles from the teeth and aided their transference within the buccal cavity prior to swallowing. Being alkaline, the mucous coat that formed on teeth also conferred direct protection against fermented or dietary acids. This action was particularly beneficial, he asserted, in those parts of the dentition less readily cleansed by the detergent fibres in the food.

Sim Wallace's firm statement of his views on the action of saliva constituted another significant polemical engagement with his laboratory-based colleagues. He was vigorously challenging the then conventional physiological teaching as to the purpose of the salivary secretions. Received scientific opinion was that saliva had two main functions: It lubricated the bolus preparatory to swallowing, and its enzymes began the process of digestion. Sim Wallace argued to the contrary, again on the basis of his clinical observations, that the primary function of the glands opening into the mouth was the maintenance of oral hygiene. He maintained that the amount of ptyalin produced by the salivary glands was small and therefore could be of little importance in digestion.

His view was that saliva was produced in a quantity and quality proportionate to the amount of food residue to be cleared from the mouth. He also asserted that the 'salivary corpuscles', amoeboid phagocytic cells found in the oral mucus, acted as scavengers and thus were also part of the oral hygiene mechanism. Sim Wallace was here daring to dispute with the highest scientific authorities, since the digestive role of saliva had been championed by many eminent physiologists, including Walter Bradford Cannon, Professor of Physiology at Harvard[43] and one of the leading experts on the function of the digestive system.[44] It is instructive, moreover, that Sim Wallance nominated Noël Paton, a colleague and close collaborator of Findlay and Cathcart, as one of the very few physiologists whose views on the function of saliva could be given any credence.[18]

The misgivings voiced by Sim Wallace and Fish regarding the application of the vitamin theory within dentistry seem to have had little general impact at this time. Apart from rickets, dental caries was the disease most commonly linked to vitamin deficiency in the advisory literature of the Department of Health, throughout the late 1920s and 1930s. In 1931, at the urging of Edward Mellanby, the Department of Health set up an Advisory Committee on Nutrition.[45] Mellanby saw this body as a means of disseminating the 'newer knowledge of nutrition', a favourite phrase of his by which he meant the vitamin theory.[46] He claimed, erroneously as we have seen, that: 'All scepticism as regards the importance of these substances in the maintenance of good health has now disappeared (except probably in Glasgow).'[45]

Major Greenwood, Professor of Epidemiology and Medical Statistics at the London School of Hygiene and Tropical Medicine, was appointed chair of the Committee, the membership of which included Cathcart. In 1932 and 1933, despite the fact that its membership did not include a single dentist, the Committee discussed the relation between diet and dental disease. It was decided that Greenwood would prepare a memorandum for the Minister of Health advising that 'increased intake of calcium and vitamin D … would reduce the incidence of rickets and dental disease':

> The experimental evidence supporting the conclusion that rickets and dental disease can be mitigated or prevented by an adequate supply of Vitamin D and calcium rich foods… is so cogent that it would be proper…to call the attention of the Local Authorities to the results.[45]

Despite the outright opposition of Cathcart, who found the evidence far from 'irresistible and conclusive', this recommendation of the Advisory Committee was embodied in a Ministry of Health circular, which was issued soon afterwards to local authorities.

CHANGING VIEWS OF VITAMIN D

Official backing for vitamin supplementation as a prophylactic against caries did not silence Sim Wallace. In 1932 the International Dental Federation sponsored a competition, the purpose of which was to encourage experimental work into the influence of diet on the

production of caries in dogs. This provoked the International Humanitarian Bureau of Geneva into organising a counter-competition, in which a prize was offered for the best demonstration of the unnecessary cruelty, as they saw it, that such experiments would cause.

Sim Wallace accepted the challenge, producing an essay with the provocative title *Dental decay in Man: its best means of prevention, and the uselessness and cruelty of the projected experiments of the International Dental Federation*.[47] He maintained that the enamel of dogs' teeth was markedly different from that of humans. Moreover, dogs were virtually immune from dental caries. Hence experimentation on dogs had little clinical value. Dogs had been used extensively by May Mellanby, and Sim Wallace took the opportunity once again to make trenchant criticism of her research. He concluded that erroneous laboratory work had resulted in a great deal of gratuitous and needless canine suffering and its incorrect conclusions had wasted public money, while the rise in the incidence of caries had not been stemmed. He pointed to the purely clinical finding that shortages of refined carbohydrates during the First World War had led to a dramatic decrease in dental decay in children.

As the 1920s progressed, a broad consensus emerged as to the general validity of the vitamin theory and the correctness of the deficiency aetiology of rickets. By the 1930s, the members of the Glasgow School, including even Findlay, who was now practising in London, thought it tactical to withdraw from further controversy over the causation and prevention of rickets, although Cathcart continued to maintain that the importance of vitamins in practical dietetics was greatly exaggerated. Sim Wallace, however, was not so inhibited. In his pronouncements, to both lay and professional audiences, he continually repeated his core message that it was not the minute nutritional composition of foodstuffs that protected the teeth from caries but rather the food's physical quality. He regularly stressed the importance of adequate dietary fibre, both for its mechanical cleansing value and to stimulate mastication. Echoing Cathcart, he emphasised 'the unlikelihood, or rather the impossibility, of any otherwise at all reasonable diet in this country, producing diseases that might be induced by deficiencies in these substances [vitamins]'.[48]

By the late 1930s, moreover, it was becoming apparent that vitamin D was unlike the other accessory food factors. It could be obtained, for instance, by means other than ingestion. It had been established in the early 1920s that exposure to ultraviolet light had a curative effect upon rachitic patients. Initially, Mellanby and some other proponents of the vitamin theory maintained that light merely activated the vitamin in the skin, but it was soon recognised that human beings could manufacture sufficient vitamin D to maintain health if they received enough exposure to sunlight, even if the substance was effectively absent from their diet. Moreover, the biochemical action of vitamin D was, it was discovered, more like a hormone than an accessory food factor.[49]

Sim Wallace interpreted this shift in the scientific understanding of vitamin D as a confirmation of his own views and those of the Glasgow School:

> The Medical Research Council may continue to try to uphold the Hopkins-Mellanby food deficiency idea with regard to the causation of rickets, but by those who can weigh evidence we can surely say that it has been discarded, as also have the Mellanby ideas with regard to caries, periodontal disease, irregularities and hypoplasia of the teeth, and this well nigh unanimously by the leaders of thought in the dental profession.[48]

Sim Wallace took evident delight in continuing his polemic engagement with the Mellanbys, by proclaiming the rehabilitation (in his view) of the Glasgow hygiene and exercise theory of rickets and the vindication (as he saw it) of his own dietary theory of the aetiology of dental caries. Provocatively and cleverly, he described the two theories together as 'the newer knowledge' of diet and nutrition, thus assuming ownership, for his own purposes, of Edward Mellanby's favoured sobriquet for the vitamin theory.[50]

It is important to note, however, that Sim Wallace's quarrel was not only with Edward and May Mellanby. Rather he indicted physiologists generally for what he saw as a long history of counterproductive involvement in practical dietetics. Returning to the absence of vegetable fibre in the modern diet, he asserted that, at the beginning of the twentieth century, lab scientists had been misguided in their preoccupation with the calorific content of food:

> … physiologists *advocated* [emphasis in original] the refinement of diet, and were themselves, to a considerable extent, responsible for the changes that were taking place. They also took a share in recommending the kinds of foods which should be given more especially to children, notably the advocacy of foods which were highly nutritious and the banning of foods such as fruits and vegetables.[48]

Such advice might have been, Sim Wallace contended, 'the cause of many of the diseases which they were … diligently seeking to cure'.

On the other hand, Sim Wallace should not be regarded as being essentially antagonistic to basic research as it applied to either dentistry or dietetics. He followed the scientific literature and was capable of changing his views in response to new research findings. On one occasion, for instance, he stated that new evidence had led to the conclusion that enlarged adenoids and mouth breathing had little effect upon production of jaw anomalies, which was a direct negation of his earlier view on the matter.[51] More significantly, his pioneering work in orthodontics was firmly

J R Coll Physicians Edinb 2009; 39:64–72

based on his own in-depth study of oro-facial variation and development and his endeavours to elucidate the underlying causes of malocclusion.[51] In marked contrast, the contemporary textbooks by the leading US orthodontists were full of orthodontic technique but contained little investigation of the fundamental scientific basis of the subject. Sim Wallace's book was unique in its commitment to developing a detailed biological understanding of orthodontic problems and their treatment.

CONCLUSION

The key issue for Sim Wallace was not, thus, the rejection of science. In many respects, he had better scientific credentials than many of his clinical contemporaries. What mattered was the establishment of a proper hierarchy of authority, on matters relating to human health, between the laboratory scientist and the clinician. Only the experienced clinician had the intimate knowledge of the lives of patients that was essential if diseases were to be effectively treated or prevented.

To an extent, both his general adherence to this point of view and his specific antagonism to the vitamin theory of rickets can be seen as a product of Sim Wallace having been trained at Glasgow University and Glasgow Dental Hospital. Despite his long sojourn in London, he still regarded the Glasgow Dental Hospital as the centre in which he had received his formative training.[48] In Scotland, as one of us has argued in an earlier paper, strong commitments to medical education and clinical practice within the medical faculties discouraged the physiologists and physiological chemists from pursuing research projects that would lead them far away from immediate clinical concerns.[8] Laboratory science had a hierarchical, service relationship to clinical teaching and practice, which contrasted sharply with the research freedom enjoyed by Hopkins at the University of Cambridge, which did not have a clinical school.[52] Sim Wallace would have understood and appreciated the co-operative relationship that pertained in the Glasgow Medical Faculty between Findlay, a paediatrician, on the one hand, and Paton and Cathcart, physiologists, on the other.

It is also the case that the British Society for the Study of Orthodontics, in general, and Sim Wallace, a founder member and early President, in particular, shared something of the ethos of London patrician medicine.[53] It is telling that, when Sim Wallace gave his Presidential Address, in 1910, he chose as his title, 'Specialism in relation to the study of orthodontics'.[54] He expressed a concern that specialisation 'confined the practitioner into a narrow rut, distorted their sense of proportion and limited the large and liberal outlook, which should be characteristic of a learned profession'. This was, he asserted, the 'curse of specialism'. Christopher Lawrence has identified the championing of generalism as a characteristic feature of the discourse of elite London medicine at this time.[7] Lawrence has also argued that this antipathy toward specialisation was related to a wish to emphasise the authority of the clinician over the laboratory scientist. The experience of the scientist was too restricted and specialised, it was argued, to allow him or her to pronounce authoritatively within the clinical sphere.

It is perhaps relevant here to note that the elite London dentists seem also to have been particularly concerned with status at this time. As we have documented in our earlier paper, those dentists also qualified in medicine, such as Fish and Sim Wallace, formed the most influential grouping with the profession.[53] Not only were they the leaders in achieving political recognition for dentistry, they tended to dominate both Harley Street practice and the dental staffing of the London teaching hospitals.

Their professional and social position was not secure, however. Sim Wallace took particular exception to the fact that the MRC's Caries Committee was dominated by medical practitioners, endorsing the view, held by several of his colleagues, that this was an 'unmitigated insult' to the dental profession.[18] The passage of the 1921 Dental Act was opposed by some doctors, who resented the full granting to dentists of the monopolistic privileges enjoyed by the medical profession.[55] In the medical periodicals, unfavourable comparisons were made between the professional status of dentistry and that of medicine. These issues of relative social and professional standing undoubtedly coloured the debate surrounding dental caries.

The dentists were also sensitive to negative attitudes on behalf of the laboratory scientists. Fish's biographer, JD Manson, considers that both the Mellanbys had 'a great disdain for dentists and their research'.[57] Lady (as she became) Mellanby was said to have cast aspersions on Fish's social background and the quality of his education. May and Edward Mellanby were public schooled and were both graduates of Cambridge University.[56] Fish, on the other hand, seems to have suffered from a social and professional insecurity that even his enormous financial success and a knighthood did not wholly assuage.

Sim Wallace's relentless combativeness, in print and on the conference podium, hints at a similar attitude, at least to an extent. The leading British dentists found themselves in a position in which their claim to professional status and their claim to distinctive therapeutic and preventive expertise were both under intense scrutiny. It is in this context that the adoption of a form of discourse that emphasised their possession of a special sort of authority, primarily clinical but secondarily scientific, was particularly advantageous.

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J R Coll Physicians Edinb 2009; 39:64–72