Colonial Australia: A dental perspective

Introduction

The Colonial Period of Australia from 1788-1901, was grounded on imperialistic imperatives which led to the British colonisation of Australia, and devastating impacts on native Indigenous communities and the wider population, particularly in the areas of general and oral health.

Australia was a “virgin soil” entity, unscathed by the many communicable diseases bourgeoning in Europe. With the arrival of the First Fleet in 1788, British settlers introduced outbreaks of infectious diseases which had devastating impacts on population health, particularly for native indigenous groups. Poor water schemes, poor sanitation and improper sewerage and waste disposal regimes enabled diseases to thrive and increase mortality rates. The Europeans introduced “civilised” customs of agriculture and pastoralism to Australia. They cultivated tobacco, sugar cane, and various grains for alcohol production, which were all important commodities of commerce, contributing to a thriving Australian economy. From a health and dental perspective sugar, tobacco and alcohol are aetiologically associated with diseases of the oral cavity ranging from Dental Caries (Tooth Decay) and Periodontal Disease (Gum Disease) to Oral cancers. This essay is in interdisciplinary exploration into the Colonial Period, exploring Art History, History and Dentistry in the context of overall and oral health.
Diet in Colonial Australia and oral health

James Alfred Turner, *(The kangaroo hunt)*, 1873. Oil on canvas. The University of Melbourne Art Collection. Purchased 1994, the Russell and Mab Grimwade Miegunyah Fund
PART 1

Indigenous traditional diet

Traditionally Indigenous Australians were hunter gatherer peoples, who foraged the lands to source food. They were sustained on a primitive diet of natural seeds, roots and wild foods including plants and native fauna.2, 3, 10

Women hunted small marsupials, shellfish, reptiles, insects, honey, eggs and plant foods such as bush vegetables (yams), seeds and fruits (wild orange truffles, bloodwood apples, fig, and bush tomatoes). Men hunted larger animals including kangaroos, emus, Tasmanian tigers, and other mammals, birds, reptiles and fish.2, 10

Their primitive diet allowed the Indigenous Australians to maintain some degree of dental health, since their diet was free from “added dietary sugars” a major aetiological agent in tooth decay.7 Nonetheless their teeth were worn down or flattened, due to the coarse and granular nature of their foods, known as “dietary abrasion”. 3, 9 Furthermore, the indigenous population used their teeth culturally as tools for various practices, including preparation of foods, leather, fibre, woodwork, which also contributed to tooth wear. 3, 9

With European Colonisation, diet evolved to include westernised processed foods including flour, sugar, and domesticated animal meats. Aboriginals were placed on cattle stations or government settlements (missions) relying on European food rations, with minimal opportunity to forage the land. Diet therefore evolved to a nutrient deficient diet, and protein, vitamin and mineral deficiencies were common.4, 7 The transition to refined carbohydrates, increased fats, and sedentary (non-active) lifestyle led to an increase in “lifestyle related diseases” such as obesity, Non-Insulin Dependent or Type 2 Diabetes Mellitus and cardiovascular diseases.4 “Major chronic diseases of Industrialised society are related to the typical Western diet” 6

In 1950 an Australian Orthodontist named P.R. Begg discovered that aboriginal groups had an ideal functional bite relationship (Occlusion). He studied both living groups, and skull remains of deceased groups, and found they possessed wide broad dental arches (of upper and lower jaws), flattened molar and premolar teeth (occlusal surfaces), narrow crowns with flattened sides, and incisors that were “edge to edge”. These teeth had very little crowding; teeth were aligned well so he believed this was an anatomically correct relationship for the teeth. 1, 5

He hypothesised that wear of the teeth (particularly between adjacent teeth; proximal wear), making crowns of teeth narrower, is essential to provide space in the order of half an inch in the lower and upper jaws to better accommodate and align the teeth, and prevent crowding of teeth or impaction of wisdom teeth. He hypothesised that the primitive diet of aboriginals is responsible for this ideal bite relationship. He also maintains that modern
techniques of straightening teeth by extracting teeth to compensate for limited space in the dental arch, is due to a modern “soft” diet of processed foods which prevents tooth wear.1,5,8

Corrucini (1990) suggested that the lack of space in the jaw to accommodate teeth, leading to crowding, impaction and orthodontic intervention, can be explained by the “disuse theory” based on the principle “if you don’t use it you lose it”. Chewing stresses from a primitive diet of the Aboriginals, led to greater forces on the jaw which were used robustly, stimulating growth. The modern soft processed diet, which not produce the same large chewing forces required, therefore reduces growth of the jaw and results in poor bite relationships (Malocclusion); namely small dental arches in jaws and overcrowded teeth. 5

The most common liquid ingested by indigenous Australians was water. Erosion, loss of tooth structure from acids of non-bacterial origin, has never been identified in the remains of Australian aboriginals, in contrast to prehistoric European populations. Nonetheless, they would have consumed acidic foods in diet (e.g from seasonal fruits) so effects were probably transient.8

References
PART 2

The Introduction of Sugar Cane in Australia

In 1788, sugar cane originating from South Africa was first introduced into Australia on the ships of the First Fleet. The first efforts to grow sugar cane occurred in 1821 in Port Macquarie, by a West Indian Convict by the name of James Williams. In 1823 T.A Scott disembarked on this “growing centre” to expand the sugar enterprise. He manufactured 20 tons of sugar by 1828. Nonetheless the venture in Port Macquarie was unsuccessful and abandoned in 1831. T.A Scott, moved to Point Claire in the Hastings Valley where he persevered in the cultivation of cane and supported the sugar enterprise. A commercial sugar mill opened in Hastings in 1867.

In 1859 the colony of Queensland separated from New South Wales. Commerce was frantically encouraged to stimulate economic growth and develop resources for the colony. Entrepreneurs were preoccupied with “finding cheap and reliable labour to clear and develop its coastal land”

In 1861 the first “white squatters” travelled to the Northern Queensland to establish a chain of sugar ports on the coastline. The father of Australia’s sugar industry, Captain Louis Hope, established the first viable sugar cane plantations near Brisbane in 1862, using sugar cane obtained from T.A Scott. Hope opened the first commercial sugar mill in Ormiston in 1864.

The plantation system would become the chief structure of Queensland’s early sugar industry in 1850-1900. It was an era where sugar was a lucrative commercial prospect, climate was fitting for viable growth of cane, and Britain had dominance over the Pacific.

The common perception during this time was that white men lacked the physical stamina to work on the sugar plantations in the harsh tropical climate. The abolishment of slavery, led plantation owners’ to indenture coloured labour from the South Pacific Ocean to expand cane growing into a viable and economical industry. The early growth of the sugar industry was “based firmly on the broad backs of the many Pacific Islanders, who were employed to toil in the fields”. Previously convicts, ticket-of leave holders, emancipists and indentured servants had worked on the sugar plantations, generating further demand for cheap and reliable labour once convict transportation ceased. With the passing of the Coolie Act in Queensland in 1862, provisions for people of Asiatic descent to be indentured to work were established. An estimated 55 000 to 62 500 Islanders were bought to Australia from 1863-1904. They were predominantly from Melanesia and Vanuatu, the Solomon Islands and New Hebrides. Small groups from the Polynesian and Micronesian Islands also worked in the sugar cane plantations. Islanders were voluntarily contracted to work, and transported from their mother land at the expense of sugar entrepreneurs.
Nonetheless, reports of kidnapping and illegal transmigration due to corruption and difficulty in regulation, known as “Blackbirding”, occurred. Deception and coercion were also used to encourage islanders to work the canes in Australia. Pacific Islanders worked on plantations in Cleveland, Caboolture and Maryborough. Peoples of South Sea Islander decent, known as “Kanakas”, also reportedly worked cane plantations in Caboolture in 1865. Captain Louis Hope also indentured Pacific Islanders in 1867. Despite their significant contribution to the Australian sugar industry Pacific Islanders were later deported due to institutionalised prejudice and a political movement towards a White Australia. Previously, “economic expediency triumphed over racial antipathy”. The impetus for Federation was partially to prevent “corruption of the colony by an inferior black race”. Pressure from unions, which aimed to protect white labour from competition by Islanders, who received very low wages, also fuelled their deportation. Australian Aboriginals from the Cape York Peninsula particularly, were also reportedly kidnapped and coerced to work the sugar plantations.

To foster further expansion the Sugar and Coffee Regulations, 1864 was introduced in Queensland. This allowed “large slabs of land” to be released for the purpose of cultivating sugar cane. By 1867 sugar production was pioneered. Sugar cane covered 1,995 acres in 1867 to 28,026 acres in 1881. The volume of sugar manufactured from 1867 to 1881 increased from 168 tons to 19,051 tons respectively. This was able to meet domestic consumption demands of the colony, in addition to consumption demands of Victoria and New South Wales where it was exported.

The Queensland sugar industry transitioned during the late 1800's and early 1900's. “Vertically integrated” plantations were divided, into smaller parcels of land, which were owned or leased. This movement corresponded with extradition of Islander labour. By 1885 102 Mills were established in NSW and 166 in Queensland. Between 1892 and 1902 the area of cane in “North Queensland” increased from 23, 623 acres to 46, 291 and sugar production from 31, 052 tons to 69, 486 tons. Sugar enterprises progressed from early small-scale milling ventures to large-scale central milling modes, where individual farmers were contracted under larger companies.

The monopoly Colonial Sugar Refining Company established in 1855, was one such model. By 1903, CSR had 15 mills across Australia and others in Fiji. It produced 1.3 million tons of sugar in 1954.

References

1. Andrew, C. and Cook, P. (editors), 2000, An oral history of descendants of the South Sea Islanders (Kanakas), Maurice Caudrey Productions, Brisbane. p 1
2. Barker, T. and Byford, I. (compilers), 1988, Harvests and Heartaches. Images and Stories of Queensland’s Agricultural Past, Department of Primary Industries, Queensland Government. P. 74
4. Fox, MJ, Fox, MT. The History of Queensland; it’s people and industries, an historical and commercial review, descriptive and biographical facts, figures and illustrations, an epitome of progress. Adelaide States Publishing Co. 1919-1923; Sugar industry chapter pg 678-691
6. Irvine, HJ, Sweet and sour: Accounting for South Sea Islanders labour at a North Queensland sugar mill in the late 1800s, Proceedings of 10th World Congress of Accounting Historians, St Louis, Missouri and Oxford, Mississippi, USA, 1-5 August 2004.
8. Kerr J, Pioneer Pageant; a history of Pioneer Shire Mackay, Pioneer Shire 1980 p 89.3
10. Noel Butlin Archives Centre, The Australian National University (NBAC). CSR Limited, Z303/Box 39/Box file D1.0/Folder 1/Document 13, Return showing the progress of the sugar and gold mining industries (Qld Parl.Paper), 1882.
The impact of sugar on oral health

The role of sugar in the pathogenesis of Dental Caries (Decay) was demonstrated in the Vipeholm study of 1945-1952, which investigated the relationship between sugar consumption and caries activity. 2, 3

The study was performed due to the rise in dental decay in Sweden, which encompassed 83% of children’s deciduous (baby) teeth and 99% of army conscripts’ teeth by 1930. Sugar consumption was hypothesised as a factor in the pathogenesis of caries in animal models and observational studies, however human trials or Gold Standard Evidence did not exist to confirm this association. 2, 3

The Vipeholm study was highly unethical by modern standards. Participants in the study were intellectually disabled patients within a mental hospital in Lund, Sweden, who lacked the aptitude to provide informed consent. The rationale for subject selection was due to the individuals controlled diet and permanent housing allowing subjects to be investigated and followed-up over a long time-frame. 2, 3

In these experiments subjects were divided into various groups which received differing physical forms of sugar (non-sticky or sticky), quantities of sugar (less than, equal to or double the standard sugar intake), and frequencies of sugar intake (during meal times and or between meals) each day. Sugar was added to a standard diet of 436 patients in various controlled forms: sucrose dissolved in beverages, sugar baked into bread, chocolate, or sticky toffees or caramels over the day. The control group was provided with fats instead of sugars to compensate for calorie consumption, and placed on a low sugar diet consumed at meal times. Saliva samples were taken every 15 minutes during the day and analysed for sugar and acid levels. The study was rigorously controlled for bias. 4

Sugars consumed during meal times in “non-sticky” formats, ranging from 30-300g, had little influence on decay rates; approximately 0.3-0.5 new surfaces of decay developed per year. In contrast sugar consumed in “sticky” physical forms, in chocolate, caramels or toffees, significantly increased decay rates, particularly when consumed in between meals, as “snacks”. Subjects who consumed 24 sticky toffees a day between meals had 4.02 new surfaces of decay per year compared to controls. When sticky sugars were withdrawn, caries activity also declined. 4

The study definitively established that frequent sugar consumption leads to increased risk of caries. Importantly the study demonstrated that sticky sugars consumed “between” meals had the greatest potential to cause decay. 4

2125 new carious lesions were developed by the end of the study period. 4 Unfortunately legislation protecting the ethical right of humans in experimentation and right to informed
consent free from coercion were later introduced; Nuremberg Code (1947) and Declaration of Helsinki(1964). 2, 3

By the 1960's the pathogenesis of Dental Caries was believed to have 3 pre-requisites; tooth, diet high in fermentable carbohydrates (dietary sugars) and dental plaque. Since then a more complex picture has evolved. It is currently recognised as a multifactorial disease which is based on an interplay of many factors in the oral environment and dental hard tissues. Oral hygiene, saliva quality (buffering ability and pH), saliva quantity, immune factors, time, use of fluorides and many other factors are also involved. 5

The role of diet high in Dietary Sugars or “fermentable carbohydrates”; glucose, fructose, sucrose and maltose, in the development of decay is reviewed below. 6

Dental plaque must be present for decay to form. Bacteria living within dental plaque use fermentable carbohydrates in food as a source of energy, and produce acids (e.g lactic, acetic, formic acids) as metabolic waste products. Acid metabolites accumulate within plaque, to lower plaque and/or salivary pH (measure of acidity or basicity). 5, 6

Frequent exposure to fermentable carbohydrates in diet, causes the pH in plaque and saliva fluid to fall (increase in acidity) beneath the critical pH extended periods of time. Critical pH is the level where saliva and plaque fluid are no longer saturated with calcium and phosphate ions, driving hydroxyapatite $[Ca_5(PO_4)_3(OH)]$ mineral in enamel or dentine to dissolve, releasing these ions. The critical pH for demineralisation is 5.5 in enamel and 6.5 for dentine. 1, 5

Increased frequency of intake of sugars may also change the dynamic of the oral environment, and create favourable conditions for ecological selection of acid tolerant species of bacteria, namely Streptococcus Mutans, Lactobacillus Casei, and Streptococcus Sobrinus. These species of bacteria are considered cariogenic, highly associated with decay. 5

The result is “tooth demineralisation”, the net loss of ions within the tooth structure and destruction of the hard tissues of the teeth. 1, 5, 6 Demineralisation of enamel, the outermost layer of the crown, leads to formation of “white spot” lesions. They are intact initially but progression of demineralisation leads to cavitation and visible loss of structure or holes in addition to discolouration of the teeth. If untreated it deepens into the dentine where progression is more rapid. This due to differences in structure, dentine crystals are smaller vs enamel crystals and have an increased surface area to volume ratio leading to more rapid acid attack. Furthermore dentine is tubular in so bacteria can penetrate these tubules deeply and degrade mineral and collagen content to destroy structural integrity. Bacteria and demineralisation can continue to the level of the pulp, containing the blood and nerve supply, leading to a condition known as pulpitis, which is a painful condition that can lead to infection of the root canal system. 5
PART 3

Water supply in the gold fields

The gold mining boom of the 1850’s led to influx of immigrants in inland Victoria. Water was imperative for almost all facets of gold mining, where the predominant method in the 1850’s was digging shallow trenches, and using the tin-dish washing or panning technique to carefully sift soil away with water and harvest the gold, often alluvial gold. Hence the term “diggers”, reflecting the technique to acquire gold-bearing gravel.

This served as the impetus for development of sophisticated water supply schemes.

Early occupants of inland towns lacked ordered water schemes, fresh water, suitable drainage and sewerage disposal. Initially miners acquired water through primitive means such as harvesting rivers, creeks, and water holes. Access to water was safeguarded, and “mis-use” of water holes, particularly by the Chinese immigrants, was reported when there was inadequate policing of supplies. Goldfield Commissioners were given a duty to safeguard water supply holes for miners and public use. In Bendigo early government camps were situated in the vicinity of watering holes in Sheepwash and Bullock Creek, during dry summers of the Bendigo gold rush.

Water was later sourced from dug wells, tunnels and water races tapping natural streams and spring water within the mountains. These systems were inefficient, with intersecting networks of races and wide spread distribution of water reducing flow to “a trickle” in various domains. It was unproductive with no attempt to collect storm and rain water.

In 1860, population growth necessitated a larger water supply, since occupants could not be sustained on stream flows and wells. Effects of large populations and mining led to increased polluted surface water. Furthermore the drought of 1864-65 worsened the already deficient water supply.
The introduction of the Waterworks Act in 1865 led to the development of local waterworks, and schemes in Creswick (1865), Daylesford (1867) and Clunes (1870). The largest water supply in the gold-mining district was the Coliban system, in 1864 which comprised a large reservoir at Malmsbury completed in 1870, and other reservoirs and channels. Gravity fed systems of pipes supplied water to Bendigo, Castlemaine, Harcourt and Cewton. In the 1870s reticulated (piped) water was extended to mining towns such as Castlemaine, Maryborough, Stawell, Talbot, and Beechworth. The Stawell system had a long section of iron fluming on dry stone that carried water to the town. 1

References

Water fluoridation and oral health
During the colonial period, the concept of water fluoridation and benefits of fluoride on oral health were largely unknown.

Water fluoridation is the treatment of water with safe levels of the naturally occurring compound, fluoride, to benefit oral health. 1, 3, 8

In the early 1900s, Dr Frederick McKay along with Dr G.V. Black noticed mottled (blotched with different shades or colours) but caries (decay) free enamel in a population of Colorado Springs, USA. This was later linked to the presence of natural fluoride in the town’s water supply.9

In 1945, Grand Rapids, Michigan, was the first town to have artificially fluoridated water, to the level of 1mg/L. A study conducted over 15 years assessing over 30 000 children revealed that decay rates had declined by 60% as a result of fluoride. The cornerstone of dental caries prevention of was born. 9

Australia followed suit with the fluoridation of Beaconsfield, Tasmania in 1953, and Bachus Marsh Victoria in 1962 8. Water fluoridation in Melbourne occurred in 1977. 5 Currently in Victoria, the optimum fluoride level in water is 1mg/L, in accordance with World Health Organisation recommendations. 5

Prior to this, communities living in the vicinity of naturally occurring fluoride in drinking water enjoyed its benefits. In Victoria these towns included Portland, Nhill, Port Fairy, Barnawartha and Kaniva. 8
Dental caries (tooth decay) is amongst the most prevalent diseases affecting the Australian population, with a myriad of consequences ranging from pain and discomfort, loss of teeth and function (e.g., eating, speaking), aesthetic, economic, to decreased quality of life and general health.  

Fluoride has a significant role in the prevention and treatment of dental caries. Fluoride ions accumulate within plaque or carious lesion fluids to promote remineralisation (rehardening) of demineralised (dissolved) enamel following acid-challenge from plaque bacteria. Fluoride ions are incorporated into the tooth to form fluoride containing apatite (Fluorapatite or fluorhydroxyapatite), which is more resistant to acid attack than the carbonated hydroxyapatite mineral form normally found within tooth enamel. This reduces the effects of future acid challenge from dental decay causing bacteria.  

It is most effective in children with developing teeth when ingested (systemic effects) however studies also shown that fluoride in water has a topical protective effect on teeth.  

The proportion of Australians with access to fluoridated community drinking water has risen dramatically from almost zero in 1953 to approximately two thirds of the population by 1977. Currently approximately 90% of Australians have access to fluoridated drinking water. This rise in access to fluoridated water is recognised as the chief cause of decreasing caries levels in Australia.  

The Australian fluoridated generation experienced substantially less, approximately half, the dental decay compared to their parent’s.  

In 2008, the Australian Research Centre for Population Oral Health (ARCPOH) presented a study examining over 16 800 children across Australia to examine the efficacy of water fluoridation. The study showed that children aged 5-6 years, who had resided for more than half of their lives in a fluoridated community had 50% less tooth decay in their primary teeth than those who had not experienced water fluoridation. Children aged 12-13 years, who lived in fluoridated areas, had 38% less tooth decay in their permanent teeth.  

Evidence supporting the effectiveness of water fluoridation in the reduction of dental caries is supported by a 2007 systemic review from the National Health and Medical Research Centre. The NHS systemic review, concluded from numerous high quality studies that with the implementation of water fluoridation, there was a significant increase in the number of caries-free children as well as a reduction in the mean DMFT (Decay Missing Filled Teeth) scores compared to regions of no water fluoridation. The 2007 review affirms that ‘water fluoridation remains the most effective and socially equitable means of achieving community-wide exposure to the caries prevention effects of fluoride’.  

According to the U.S. Centre for Disease Control, ‘Water fluoridation is one of the ten great public health achievements of the 20th century’.  

13
Community water fluoridation continues to be the most cost-effective, equitable and safe means to provide protection from tooth decay and has been successfully utilised in Australia for over 50 years. 8

References

1. Armfield JM, Roberts-Thomson KF, Spencer AJ. The Child Dental Health Survey; Trends across the 1990s. Adelaide (AUST): The University of Adelaide 2003
Pub Culture and Oral Health in Colonial Australia


**Alcohol in Colonial Australia**

Pub culture played a major role during life in Colonial Australia. In the 19th century, particularly during the gold rush era, many public houses and hotels were established across Australia. The Australian beer drinking culture stemmed from Northern European traditions, which was dominated by grain derived alcoholic beverages, distinct from Southern European traditions embracing wine culture. Between 1800 and 1950 alcohol production and consumption in Australia was dominated by beer and spirits, with many bars synonymously serving ice-cold Pilsner beer. Beer was the largest selling alcoholic beverage during this time.  

Large quantities of alcohol were consumed in Victoria, Australia. More closely, in 1839 in Port Phillip Melbourne, 20 pubs were established in addition to breweries. Tobacco was added to alcohol during production to augment the taste of the weak brew. This form of Alcohol was referred to as “swipes” and was commonly consumed quickly “in one gulp” to help evade the poor taste. In 1850, more than 3 million litres of beer, wine and spirits were transported to Port Phillip.
Public drunkenness was a common concern. In 1842, 1500 individuals, approximately 15% of the population of Melbourne, were charged with public drunkenness. The widespread prevalence of public drunkenness was captured by a local newspaper, Port Phillip Patriot, 30 December 1841: “There was not a single charge of drunkenness at the police office yesterday. Such a fact is worthy of record for its singularity”.  

It is noteworthy that Policemen were afforded 50% of every drunkard’s fine, which may have beguiled corruption and contributed to the issue as “an inducement to take sober men into custody” (*New South Wales Police Act, Section 6, 1836*)

In the 1940s Melbourne entered a time of financial depression. Drinking increased rendering a substantial number of men unfit to work to support their families. Instances of domestic violence also reportedly increased.

This led to establishment of the temperance movement, which involved women who united to encourage abstinence and generate awareness of the “evils of alcohol”. Nonetheless alcohol remained an important part of the city’s culture.

References


The “evils” of alcohol a dental perspective: What are the impacts of alcohol on oral health?

Heavy alcohol consumption, defined as a daily intake of ≥50 g of ethanol (approximately 3.5 or more drinks per day) and or frequent binge drinking behaviour, is associated with harmful impacts upon oral and overall health.

Alcohol has been identified as an important modifiable risk factor in the development of oral cancers for approximately half a century. It is also associated with greater clinical risk of developing cancers of the head and neck (pharyngeal and laryngeal cancer). The World Health Organisation (2005) has stated oral cancer is eleventh most prevalent cancer in the world.

The exact role of alcohol in the development of cancer is not completely understood. This understanding is partly hindered because approximately 75% of all oral cancers and 65% of all head and neck cancers are linked with combined alcohol and tobacco use.
Impact of these individual risk factors are therefore obscured through this synergistic relationship. 3, 9, 10

Ethanol can act locally and systemically to increase the risk of developing oral cancers. 8

On a local scale, alcohol may increase the penetration of carcinogens (cancer causing agents) across oral tissues which line the cavity (oral mucosa) by dissolving a barrier of protective lipids (fat). Tissues which are commonly affected are thin non-keratinised tissues of the buccal mucosa (lining of cheeks and inner lip), lateral (side) border of the tongue and floor of the mouth, which are sites commonly associated with oral cancer. Thick keratinised tissues e.g the hard palate are less commonly affected. 4, 16

Animal studies have demonstrated that chronic alcohol intake can cause epithelial cells (cells which line cavities and surfaces of bodily structures) to die (atrophy) and or generate changes in basal cell size. 5 One study investigating the long-term effects of alcohol, revealed that 12 months exposure to alcohol in rats led to pre-cancerous changes in oesophageal cells known as dysplasia (Characterised by increased mitotic figures, dense basal cell layer). 6

Ethanol is converted into Acetaldehyde by cells which line the oral tissues (mucosal cells) and bacteria (microflora) in the mouth. This substance is harmful and is considered carcinogenic/mutagenic. It interferes with DNA repair and synthesis, causes gene mutations, inhibits an enzyme responsible for repair of DNA (O6-methylguanittransferase), and causes proteins to attach to DNA resulting in damage. 4

Alcohol has been shown to potentiate the effects of other mutagens or carcinogens in the mouth. 4

Chronic alcohol abuse is linked with parotid gland enlargement (the largest salivary gland) and Sialosis (diffuse, non-inflammatory, non-neoplastic recurrent enlargement of the major salivary glands). Ethanol leads to peripheral autonomic neuropathy (symptoms arising from nerve disruption) and disruption of salivary gland metabolism. It may also lead to cell death (atrophy) or fatty infiltration in salivary glands. These factors lead to impaired parotid salivary flow, production of viscous saliva, and rise in local carcinogens, which are normally “flushed” by salvia from the surfaces of oral mucosa, increasing the risk of cancer. 4, 14

On a systemic level, alcohol can be linked with abnormal liver (hepatic) metabolism of “toxic or potentially carcinogenic compounds” which will accumulate in the body. It can also be linked with immunosuppression, leading to increased susceptibility to infection and certain neoplasms (tumours), e.g alcohol can supress activity of immune cells named Natural Killer cells which are involved in surveillance of tumour cells. 11
Alcohol may cause forms of Alcoholic Liver Disease (ALD); Alcoholic hepatitis (AH) or Liver Cirrhosis. Alcoholic hepatitis (AH); inflammation of the liver from harmful by-products of ethanol metabolism (reactive oxygen species and neo-antigens), affects 10-35% of heavy drinkers. Cirrhosis is a condition affecting 8-20% of heavy drinkers, where cells of the liver (hepatocytes) are destroyed, leading to changes in liver architecture, decreased liver function and portal hypertension. Both conditions can cause liver failure episodes or End Stage Alcoholic Liver Disease. 20

The liver is responsible for producing Vitamin K-dependent clotting factors (II, VII, IX and X). Hence, Liver Damage may lead to decreased production of the above factors, followed by factor V. These factors are important in coagulation (clotting) and haemostasis, the process of blood clot formation and bleeding cessation. Alcohol may also directly suppress production of platelets in the liver. Therefore haemorrhage or substantial blood loss may occur in patients after tooth extraction or soft tissue injury due to alcohol induced liver damage. 12

Dental erosion, defined as the chemical loss of tooth structure due to acids of non-bacterial origin or chelators, is also linked with increased alcohol consumption. Alcohol has acidic components which dissolve the hard tissues of the teeth. Vomiting or gastro-oesophageal reflux disease (GERD) due to alcohol use, leads to strong intrinsic acids from the stomach eroding the teeth. 21

Furthermore dental decay can also arise. Sugars contained within alcohol are broken down in the mouth and converted by plaque bacteria into acidic by-products which can demineralise tooth tissue, leading to loss of hard tissues and cavities or decay. 21

Finally, Alcohol can also cause growth deficiencies. Foetal Alcohol Spectrum Disorders (FASD) can occur due to alcohol intake during pregnancy which in addition to physical and mental retardation, may lead to impaired growth of the upper (maxilla) or lower (mandible) jaws, and disturbed development of the teeth (odontogenesis) in the developing foetus. 13, 15

References

Prior to permanent European occupation of Australia in 1788, Indigenous groups “chewed” native tobacco plants, including leaves of the *Duboisia hopwoodi*. 3 It is reported that “smoking” of tobacco was introduced to indigenous groups by “white men”. 2,3 Others, including Anthropologist, Josephine Flood, claim that indigenous Australians adopted smoking behaviour prior to colonisation from the Maccassans, Indonesian fishermen, who exchanged wooden pipes used for tobacco smoking to north-dwelling tribes to harvest their waters for a seafood delicacy known as Trepang. 5, 8, 11

British Settlers transported tobacco to Australia in 1788 with the arrival of the First Fleet. The first commercial tobacco farms in Australia were established in 1803 in Emu Plains in New South Wales and by the 1820s tobacco was cultivated by farmers in the Hunter Valley. During the 1850s growing extended to Victoria and Queensland. 13 It is likely that some proportion of the tobacco plants were used to produce pesticides to kill parasites inhabiting sheep. 7

Sir John Jamieson saw the plant as “a commodity of commerce” 13 and indeed it was as home-grown tobacco was outlawed.13

Colonisers used tobacco to foster positive relations with indigenous groups. An early encounter of settlers providing Indigenous peoples with tobacco occurred in 1804, where
Governor Phillip Gidley King presented Aboriginal men who travelled by boat from Newcastle to Sydney, with several gifts including tobacco.10

Tobacco was also a form of reimbursement to engage the services of Indigenous peoples. They were awarded rations of tobacco amongst other things in exchange for their labour on cattle stations or missions in which they were confined until 1967 with the grant of citizenship. Aboriginal peoples were also provided with tobacco by miners, fishermen, and anthropologists for labour. 9 “Blacks bring fish and oysters for which they receive flour and tobacco in return” 9 During the 1830's Aboriginal groups in the Lake Macquarie region had a widespread addiction to tobacco.9

Tobacco was a “subversive” and subjugating agent during colonisation. Although Aboriginals traditionally used narcotics its use was scrutinised by tribal kinship and trading networks. 4 Use of European tobacco use was un压制, and widespread addiction saw aboriginals demand bonuses of tobacco during disputes for increased payment by turn of the 19th century. 12 A disparity between the healths of indigenous groups with non-indigenous groups appeared which continues today. Aboriginal and Torres Strait Islander peoples, smoke 2 x more than non-indigenous Australians, according to the Australian Bureau of statistics which is of large concern to their health as a population. 1

Tobacco smoking was also common amongst free settlers, officers, and convicts. 3, 14, 15 In the early 1800s, tobacco was provided to servants, prisoners and ticket of leave men (conditionally released convicts) to engage their services, or was rescinded in reprimand. 3 Tobacco was produced and consumed across Australia in large quantities. In 1819, it was alleged that some 80 or 90% of male labourers were smokers. 15 In the mid 1820’s a reported 60 000 pounds of tobacco was consumed annually, as an addictive substance in the colony. 6 More closely, in Hunter Valley and Port Stephens, domestic productivity of tobacco was so substantial that importation was prohibited, to enable protection and growth of local industry.6

References
Effects of Tobacco on Oral Health

During the Colonial period the harmful risks associated with smoking behaviour upon health were largely unknown.

During the Pre-colonial time of 1602, it was first recognised that soot was linked with illnesses in chimney sweepers, and thought that tobacco may have similar potential. It was during the post-colonial period of 1920-1960 where the first conclusive reports transpired linking smoking to lung cancer and a range of other diseases.2

Tobacco is an aetiological factor in pathology of oral cancers. Carcinogens are present within mainstream tobacco smoke.14, 15 Tobacco-specific N-nitrosamines, aromatic amines, and polycyclic aromatic hydrocarbons are the chief components in tobacco smoke which contribute to oral cancer risk. In smokeless tobacco, nitrosamines produced after fermentation, are thought to contribute to cancer.8

Studies demonstrate an exposure-response relationship, between the number of cigarettes smoked per day and duration of smoking in years (pack years), and the risk of tobacco associated cancers. This includes oral cancer, which is 0.3% of all cancers. 1 Tobacco smoking is linked with 75% of all oral cancers 1, 17 The risk is synergistic when alcohol and smoking are combined. Over 90% of oral cancers diagnosed present as squamous cell carcinomas 1 10% of all oral cancers occur on the gingiva (gums). Metastasis is common and has a poor prognosis, particularly because of the delay in diagnosis or misdiagnosis.1
Cigarette smokers have 2-5 times the risk of developing oral cancer than non-smokers as shown by a population based controlled study. 1, 19 This risk was elevated with the number of cigarettes smoked per day and the duration of smoking.1, 19

Cigar smokers are 7-10 times more likely to develop oral cancer than non-smokers. 12

Studies have demonstrated that pipe smokers were 2-3.5 times more likely to develop oral cancer compared to non-smokers.20

Smokeless tobacco users (chew or snuff) had 4-6 times the risk of acquiring oral and pharyngeal cancers than non-users.3,19 A dose-response relationships was found in a study, with risk of cancer increasing to 50 fold after > 50 years of snuff use. 18

A study involving 17,027 adolescents in the United States identified mucosal lesions in 27% of individuals who sniffed tobacco (snuff), in contrast to 0.4% in individuals who never used smokeless tobacco from 1986-87. Lesions manifested as “slightly wrinkled” areas overlying oral mucosa (oral cavity lining) to “thickened, furrowed white or gray”. 16

Smoking is also associated with oral leukoplakia and oral conditions such as nicotinic stomatitis and hairy black tongue.10, 11

Leukoplakia is a whitish plaque or patch that cannot be removed or characterized clinically or pathologically as any other disease. Most cases of leukoplakia cause no symptoms, but infrequently discomfort or pain has been reported from patients. 10 It is of concern because 20-40% of leukoplakia’s are precancerous (dysplastic) at the time of biopsy, 4 with potential to form malignant cancer and spread. 10 Homogenous leukoplaikias are smooth white plaques, have a long term malignant cancer transformation rate of 3%. 10 Non-homogenous leukoplakias which are irregular surfaces, with white patches over red backgrounds, have 7 times greater risk of becoming a malignant cancer. 7 The location of leukoplakia in the mouth is important, because those that appear on the floor of the mouth or and underside of the tongue, (ventral) are more likely to develop into malignant cancer. The longer the duration of alcohol and tobacco use the greater the risk of malignant transformation. 10 Some forms are aggressive, Proliferative Verrucous Leukoplakia has a 60-100% rate of turning into cancer over time. 4

Black hairy tongue is a condition where filiform papillae on the tongue elongate, creating hair-like projections. Pigment from exogenous materials (food, drink, tobacco smoking etc) and bacteria which are chromogenic (pigment producing) stain the papillae, leading to black ad also brown or yellow discoloration. Increased accumulation of plaque on the surface of the tongue also occurs. The appearance is concerning to patients, however it is essentially harmless. Predisposing factors include smoking.10
Nicotine stomatitis, is a condition which manifests in palate in the mouth due to the effects of chronic heat. Palate appears white or grey, with nodules containing elevated red dots, which are ducts of minor salivary glands. This condition is not premalignant. If reverse smoking (smoke from the lit end) behaviour occurs the condition will become malignant, known as Reverse smoker’s keratosis. 11

Smoking is linked with increased risk of developing gum disease (Periodontitis or Periodontal Disease). This condition affects the supporting tissues of the teeth (Periodontium). It is inflammation that leads to destruction of gum tissues (gingivae), the periodontal ligament connecting the teeth to bone, cementum a dental hard tissue that covers the root surface of teeth, and alveolar bone. Damage is irreversible compared to gingivitis, and causes loss of tooth support, progressive bone loss and ultimately loss of teeth. It is characterised by pocket formation between teeth and gum tissues which deepen with increased severity of disease. Bacteria, which are mainly gram-negative anaerobes (non-oxygen dependent) thrive within these deep pockets and produce products (e.g LPS, toxins) that stimulate the inflammatory response. P. gingivalis, T. Denticola, and B. Forsythia are strains of bacteria associated with gum disease. It is also characterised by gingival recession (Root exposure). It is often associated with halitosis (bad breath), and in advanced cases teeth become mobile or loose and begin to drift causing spaces between teeth. If left untreated periodontitis may cause abscesses and tooth loss. 10

Smoking is the biggest preventable risk factor for gum disease. There is a dose response relationship between the severity of gum disease and smoking in pack years which is the number of cigarettes smoked per day and duration in years. Light smokers defined in study by Grossi et al 1994 as smoking 5.3-15 pack years had approximately double the risk of acquiring periodontal disease while heavy smokers defined as approximately 30-150 pack years had approximately 5 times the risk of developing periodontal disease. 5

In Haffajee and Socransky (2001) Smokers had significantly more attachment loss, missing teeth, deep pockets and fewer sites that bled (bleeding after pocket is examined for depth with a instrument) than non-smokers. 6

Finally tobacco smoking is closely linked with an increased risk of acquiring cardiovascular disease, diabetes, hypertension, emphysema lung cancer and increased morbidity 3
References


General Health during the Colonial Period

Prior to the arrival of Europeans in Australia in 1788, Australia was a “virgin soil” territory where little infectious disease was prevalent amongst Indigenous communities. This was attributed to the indigenous lifestyle of hunter gathering, where food was sourced from foraging the lands, in contrast to the European sedentary lifestyle grounded on agriculture and intimate living with domesticated herd animals that transferred microbes and disease to humans. The comparatively small indigenous population also provided an inadequate “ecological niche” for infectious diseases.1

In the Indigenous Kulin nation, of Central Victoria, disease was scarce. Indigenous tribes with a semi-sedentary lifestyle in Western Victoria and Murray region, experienced diarrhoeal disease compared to active nomadic tribes. The only infectious disease to thrive in indigenous communities were bacterial in origin, including Non-venereal (non-sexually transmitted) syphilis a chronic skin and tissue disease and Yaws a tropical infection of the skin, bones and joints, presenting as round, hard lumps on the skin which may become ulcerated. 1

Prior to European settlement instances of smallpox were introduced into Northern Australia by Macassan trepang (sea cucumber) fisherman from Indonesia. Early colonisers recognised pock-marks on indigenous peoples’ skin, which they judged as “native pox”. They also misjudged Yaws (non-venereal) for Syphilis a venereal (sexually transmitted) infection, which they condemned was “loathsome disease”, despite their role in later infecting the aboriginal people with this condition.1
European invasion led to a Virgin Soil Epidemic, where indigenous populations with no previous contact with diseases, were highly susceptible and immunologically virtually defenceless. Old-world diseases were able to flourish in their new environment. Many Europeans were immune from small pox, due to vaccination or early contact with the disease. Smallpox was a problem for Europeans from infected ships and Melbourne's most significant outbreak was 56 cases in 1885-86. In addition to small pox carried by European settlers, indigenous populations were afflicted with other outbreaks of disease including measles, influenza, syphilis and more which devastatingly conquered many aboriginal camps leading to death. “The main conqueror of aboriginals was disease and its ally demoralisation”. This was coupled with competition over resources, and occupation and destruction of their lands leading to starvation.

Diarrhoeal disease (acute watery, acute bloody/Dysentery or persistent) of viral, bacterial or parasitic origin, was a chief cause of death in early colonial Melbourne. Diarrhoea is an infection of the intestine that results in formation of loose stools or liquid bowel movements which may contain blood or mucous. It leads to fever, abdominal pain, and rectal tenesmus (a feeling of incomplete defecation). It also leads to loss of essential water and salts from the body, and may cause severe dehydration and fluid loss. The omnipresent fly in Australia aided in the transmission of diseases across Australia. Infant mortality from gastroenteritis and dysentery was high during the 1800s, and exceeded London until the end of the 19th century.

During the late 1830s diseases of epidemic magnitude occurred in Sydney, with a high death toll anticipated amongst those born in Australia.

Disease flourished due to poor sanitation, overflowing drains, contaminated water supplies (e.g. rivers, creeks), and open sewerage systems. The ubiquitous smell of human excrement overshadowed Melbourne. Rising death tolls due to typhoid and improved understanding of hygiene, transmission of diseases, and microbes (germ theory) spurred development of efficient sewerage systems in Melbourne after 1897.

“Colonial fever”, later diagnosed as Typhoid in the 1870’s, afflicted the early population. The condition was life-threatening, caused by the bacterium Salmonella Typhi. It is transmitted by ingesting food or water contaminated with the faeces of an infected person, and caused symptoms of fever, lethargy, decreased apatite, diarrhoea, malaise and aches and pains.

In 1850, Measles from was introduced to Australia from the Persia. In 1853-1854 it had epidemic magnitudes leading to a death toll of 50-200 out of every 100 000 peoples. From 1874-1875 the worst of its effects were seen.
An outbreak of Scarlet fever occurred in 1875-1876. Tuberculosis struck Australians over the next 10 years, leading to many deaths. In Victoria, death from Tuberculosis rose till mid-1890, but declined abruptly thereafter congruent to trends in other developed countries.1

Urbanisation of Melbourne, provided a large “ecological niche” for microbes, and infectious diseases were rife. Mortality rates from measles declined after the 1900s. Scarlett fever also declined.1 Influenza first broke out in Victoria in 1860-61, but was later an epidemic in 1885. In 1890-91 there was an outbreak of “Russian Influenza”, and widespread drop in morale across Melbourne.1

In the early colonial period, in Victoria, well trained dental professionals were scarce and was reportedly performed by doctors and chemists to blacksmiths and jewellers. Extraction, or removal of teeth was the most common mode of treatment for tooth aches. Anaesthetics used to remove the sensation of pain during such procedures were ordinarily unavailable, rendering such procedures agonising for patients. The establishment of the Odontological Society of Victoria in 1884 paved the way towards the introduction of dental act, hospitals accessible to impoverished groups, and formal educational training institutions for aspiring dentists. 2

The Bubonic Plague, hit Sydney in 1900, which led to the widespread clean-up of the city, particularly the infested slums, and pest (namely rats) extermination programs. Melbourne experienced 10 cases of the disease. This led to establishment of a public health movement, where the city health officials encouraged hygienic practices such as the boiling of water and milk, due an evolving understanding of germ transmission. 1

References
3. Crosby Alfred (1976), Virgin Soil Epidemics as a Factor in the Aboriginal Depopulation in America, The William and Mary Quarterly (Omohundro Institute of Early American History and Culture) 33(2): 289-299, JSTOR 1922166
Health on the goldfields


“17 days I have not been able to hold a pen…I was seized with a violent attack of dysentery. I believe the place was an unhealthy one. “During the winter it is almost entirely under water” “Thousands have been struck down, many of them are still lying on their back, from the effects of change of climate...more from the change of living, they roll themselves in a rug at night often soaked with rain, or chilled with cold of night, which is often very penetrating, especially after a day’s march under a hot sun”. Individuals were “seized with dysenteries, fevers, and rheumatism which will cripple many for life and already carried many out of it” William Howitt diary.

In the early 1850’s miners suffered from widespread disease, predominantly due to poor sanitation. Wastes and faeces contaminated rivers where drinking water was sourced for miners on the diggings. Sewerage systems were improper, allowing microbes in excrement to be transmitted to the wide community. Dysentery, typhus, and other infectious diseases were prevalent. Poor diet particularly during the early years of the gold rush, which was mainly consisted of mutton and damper, led to nutritional deficiencies. Common cold was deadly and quickly aggravated to pneumonia. 2, 3

“Dysentery and diarrhoea are a frequent occurrence, particularly in the spring, but are seldom fatal. At the diggings ill health is certainly to be more frequent”. John Sherer 1853. Dysentery arose due to improperly prepared food, poor water quality and supply, and a fibre deficient diet (low vegetable intake). 3 “Weakness of sight” due to hot winds and soil also was reported by diggers. 3

Excessive drinking was a common health problem on the goldfields, liquor was used to cope with psychological problems and loneliness. Drinking increased the risk of liver disease, and cardiovascular diseases such as stroke or fatal collapses. 2

Mental health of individuals living on the diggings was also of concern. Those considered harmless to the community were taken care of by family or friends and occasionally by members of the church. Individuals were incarcerated when considered a danger to others or themselves, or when they performed criminal acts. They were placed in gaol hospital until they were transported to the Yarra Bend or Kew Asylums near Melbourne. Mental health was not well understood in this time. Treatment slowly evolved in the early to mid 19th century from physical restraint (chained to walls, leg irons, straight jackets) to working with patient mind’s and strengthening their moral and ethical principles. Colonial mental asylums were overcrowded and patients received poor care. 2

Few doctors worked in mining towns or on the goldfields. Access to health was restricted to individuals who could afford their costly services. Markedly, during the early days of the goldrush, medical resources were lacking and patients with serious illnesses were sent to return to their tents rather than admitted into hospital. Children and elderly were most susceptible to disease, and childbirth was dangerous. 2, 3
Chemists were established in gold-mining districts. Most medicinal remedies were harmful. Bendigo’s chemist shop carried leeches, morphine, opium, hemlock, and fly poison. Patented therapies included “Garsed’s worm cakes for children”, Jayne’s Carminative Balsam for bowel complaints” and “W. Harris’s anti-bilious mixtures”. An anti-dysenteric blend derived from India and South America was promoted as a cure for diarrhoea. 1

Dr Eadie’s Sarsaparilla pills and ointment were promoted as a panacea (cure-all) for many illnesses ranging from diarrhoea, sore eyes, ulcers to cancers, tumours, piles and ringworm. “Safe and invaluable remedy for bilious complaints and pains in the stomach and bowels, remedy for indigestion, loss of appetite, headache and a blood purifier”. Sarsaparilla pills and ointments, containing the herb, were sold at the Bendigo Apothecary Hall, High Street. 1

The root of the Sarsaparilla plant, was traditionally used to make medications. It has anti-inflammatory properties and may help reduce pain, including joint pain. It also reduces itching, has antibacterial effects, and protects the liver against toxins. It is used today to treat psoriasis and other skin disorders, rheumatoid arthritis, leprosy and syphilis in combination therapy. It also increases urination to reduce fluid retention ( diuretic) and increases sweating. 4, 5

References

Conclusion

The Colonial Period of Australia was a significant period in the History of Australia, particularly from a general and oral health perspective.

With the introduction of Europeans to Australia, came the introduction of a “Virgin Soil Epidemic”, where indigenous populations with no previous contact with European diseases, were conquered by Small Pox, Measles, Scarlett Fever, Venereal Syphilis, Influenza, Pneumonia and other communicable diseases. Poor water schemes, water supply, sanitation and improper sewerage and waste disposal regimes enabled diseases to thrive and increase mortality rates. Faeces transmitted diseases such as Diarrheal Disease, Dysentery and Typhoid, were therefore very common, particularly in the Gold Fields where water supplies were frequently contaminated.

Europeans introduced customs of agriculture and pastoralism, cultivating tobacco, sugar cane, and various grains for alcohol production. The Australian economy was wrought by these commodities of commerce, which led to disparities in health amongst the population, particularly amongst Indigenous Australians. Sugar is recognised as an aetiological agent in Dental Caries (tooth decay), amongst the most prevalent diseases affecting the Australian population. Without the ubiquitous availability of Fluorides present today, particularly via fluoridated reticulated community water supplies, individuals were more susceptible to decay rates. This is due to Fluorides remineralisation and prevention of demineralisation capabilities, and formation of Fluorapatite mineral in dental hard tissues (teeth) which is highly resistant to acid challenge. Tobacco and Alcohol are synergistic aetiological agents in the pathogenesis of oral and head and neck cancers. Furthermore they are linked with other mucosal lesions in the oral cavity such as Leukoplakia which is a precancerous condition that can transform into malignant cancers. Heavy Alcohol consumption is also likened with impaired haemostasis, due to impaired liver function and clotting factor production, causing increased bleeding following extractions. It is also linked with decay, erosion, and Foetal Alcohol Syndrome leading to developmental problems in the craniofacial complex (skeletal structures of the head) and teeth. Smoking is also linked with increased risk of developed Periodontal (Gum) Disease which leads to loss of tooth support, progressive bone loss and ultimately loss of teeth. It also associated with Hairy Tongue and Nicotinic Stomatitis.

In addition to health disparities, the sugar cane, tobacco and alcohol industry also led to many social injustices amongst the South Pacific islander and Aboriginal communities. Aboriginal communities were provided rations of tobacco, sugar, and alcohol which were subversive agents, used to engage their services. This is instrumental to the current disparity in health between indigenous and non-indigenous Australians. Although many Pacific Islanders were indentured to work on sugar plantations legally, reports of kidnapping and coercion are apparent in the literature, known historically as Blackbirding, a form of slavery.