

Grace Suckling: Talk to NZ branch of ANZSPD, November 2013

Recently, I was asked when I first became interested in enamel defects. I can give you the exact place and date when that occurred. It was quite late in my dental career close to my 50th birthday in a shearing shed at a farm not far from Kaikohe in Northland in June 1970. I was selecting sheep for a trial and out of a group of 48 sheep, 17 had enamel defects of their incisors and the question was why. How did I land there? A little bit of background information is needed to answer that question.

I started at Dental School in Dundee, Scotland the day after war was declared in Sept 1939 and my first day was spent filling sandbags to protect a stained glass window which is still at the school. The war was a dominant feature during my student days.

I then had 2 years in the School Dental Service and 5 years in a busy practice in England. I wanted to learn more and prepared for the FDS exam. This required at least 6 months experience at an approved hospital and I became a registrar at the London Hospital Dental School. After a year, I moved to the teaching staff, my subject was Dentistry for Children, then in its infancy being pushed along by Prof Max Horsnell, later Dean at Adelaide Dental School.

I met my husband, a NZ orthodontist at the school and came with him to NZ in 1956. 13 years and 5 children later I joined the Dental Research Unit part time. The unit was involved in research into dental problems of sheep of economic importance at that time. The association of tooth wear and soil ingestion had just been established and research was then into a different problem of increased tooth length.

Sheep have no upper incisors and the lower incisors bite into a hard fibrous pad. On affected farms the lower incisors moved forward in front of the pad, became long, loose and fell out at an early age. They could not eat properly and so had to be culled. There were no ear tags and so the age of a culled sheep was not known. I was on my first visit to the affected and control farms to inspect and photograph the sheep teeth. I needed to find out the cause of the hypoplasia in case it was related to the tooth loss. Later, we found out that a severe drought during the period of tooth formation was the cause. Then, I was struggling to learn about sheep teeth and farming language. What was a 2 tooth, a rising 2 tooth, a full mouth and what did condition mean? At that time little was known about the development of sheep teeth and the information available about enamel defects in text books was limited.

This interest in sheep teeth led to an involvement in the preparation required before the Bluff Aluminium Smelter began its operation in the 1970s. Experience with smelters elsewhere in the world had shown that there was a danger of skeletal and dental fluorosis in districts around a smelter. The effect decreased as the distance from the smelter increased and was related to the amount of F in the effluent coming out of the stack. There was extensive research done around Bluff into the F levels on land and sea. The smelter was situated so that the effluent was blown out to sea. Monitoring farms were placed around the smelter and one was placed downwind. I was asked to look at the teeth of sheep on the monitoring farms. Fluorosis was seen in teeth of sheep grazing around a fertiliser works and we found that superphosphate contains 1% F. Later, I made visits to the farm downwind and saw the odd band of pitting that connected to an effluent problem.

The Medical Research Council, our funding body, decided that sheep teeth were an agricultural problem so I transferred my interest to defects in human teeth. I was in an ideal place to do this as the Unit was situated on the top floor of the Dental Clinic in Willis Street. Over 7,000 children attended the Clinic twice a year. The staff, especially Dr Betty de Liefde, were very cooperative and alerted me when any interesting teeth were seen and there were many.

The difficulties associated with malformation of the sixes were well known, with the increased risk of caries, the retention of fillings, incomplete anaesthesia, whether to extract and cause spacing problems later. It has been interesting to see how many papers have been written on this subject recently. I had access to medical and dental histories but was only rarely able to find out the cause. The exceptions were defects caused by the drug, tetracycline, when given during the period of tooth formation and those caused by trauma to the deciduous predecessor.

It was only in 1971 that evidence was provided by Andreasen and co that defects could occur even after x rays showed that crown formation was complete and the realization that there was a longer maturation phase of enamel development.

At that time most of the basic and epidemiological research worldwide centred on the role of F. That was to be expected in view of the widespread and recent interest in methods of controlling dental decay. Fluoride had been introduced into the water supply in Lower Hutt in 1959 and in Wellington in 1965. Fluorosis related defects were not seen or not recognised as such. There was a great need to be able to distinguish between fluoride defects and those caused by other factors. Apart from Dean's Index, the terminology was hopeless. Hypoplasia could mean missing enamel or all other defects, mottling and white spots were also used. Comparison between papers or even to know what the authors meant was impossible.

We conducted a survey of all defects in the Wellington area using the terminology of Andreasen. It was not suitable. This study published in 1976 had one major benefit. Figures were available for the number of tetracycline cases and gave support for the removal of the drug from the subsidised drug list during the period of tooth development. Defective 6s were also recorded and the still unanswered question asked---why sometimes 1, 2, 3 or 4 teeth malformed?

By around 1973-1974, I had realised that all defects could be recorded into one of 3 groups, not on their cause, but on the basis of their appearance. My long term aim was to establish a classification system to determine the prevalence and when possible the etiology of defects in NZ and later to help in the development of a descriptive classification with worldwide acceptance and use. Dr Cutress was at that time a member and later chairman of the FDI Commission on Oral Research and Epidemiology. He was able to form a working group in 1977 with just such an objective. Clinic staff helped by testing different proposals.

The original DDE index was presented at an FDI conference in 1981 with definitions of hypoplasia and opacities and the introduction of the terms demarcated and diffuse. Coloured photographs were provided. I used the Index in 3 studies in NZ and 1 in Japan.

Thanks to help from Harvey Brown I became involved in the Dunedin MDHDS inspecting the children when aged 9 y (now in their forties) and having access to their records. One memory is seeing diffuse opacities on a single upper incisor with a history of trauma to the deciduous upper incisor--- most unusual.

I was in Japan for a conference. I visited a school near Nagoya. There were 16 pupils who had been subjected for a period of time to drinking water containing 7.8 ppm fluoride. I inspected their teeth and subsequently worked with the Japanese researchers who had documented the problem. A new water supply had been introduced and it was about 5 years later that changes in the appearance of the teeth were noticed and the high F level in the drinking water discovered. A new water supply was quickly installed. There were 3 groups among the affected children, some exposed early, throughout or late in tooth development. The severity of the changes and the position of the defects on the tooth surface varied between the groups. The 16 students were in the group exposed to the high F water early in tooth development. The photograph is an example of one exposed throughout tooth development. I have never seen such severe F elsewhere. Pits occur in human teeth but we were able to support earlier research and show from serial photos that the pits were post-eruptive.

The Auckland survey in 1984 followed claims of disfiguring fluorosis in F water districts and was given great publicity as the mayor was a known anti-F. Great care had to be taken so that I as examiner of the 1758 pupils did not know the F background of each child. There are 2 memories about that survey and they are not about defects. One was the dental age. All were 9 years old yet one child had only centrals present while others, mainly Polynesian, had 24 permanent teeth erupted. Last week while preparing this talk, I came across a paper on just that subject. The other was the very poor state of oral hygiene—extremely poor through all decile schools. Some had not seen a toothbrush let alone F toothpaste for months at a time when it is needed.

Several criticisms of the Index resulted. Technology had improved. A proposal to alter the Index but not the definitions or the terminology was made at the DDE symposium in Rotorua in 1988; a second working Party was established and the amended Index was published in 1992.

Concurrently with this activity, I was producing defects in sheep incisors using methods known to result in defects in children and which I had already seen in sheep—trauma, systemic illness and F at different levels. We needed to study the normal development of the incisor teeth. An important finding was that age was not a reliable indicator of tooth development although tooth length was.

I was lucky in choosing sheep. The crowns are long (about 32 mm) and the 4 zones, secretory, early mat, late mat and fully min are easily recognised and can all be present in a nearly mature tooth. We managed to produce the same 3 types of defects. The changes in the ameloblasts at different times of development were seen using conventional histology and the defects were examined by physical methods. Some extracted, damaged human teeth were also assessed and examined. Human and sheep findings were integrated in my thesis submitted in 1994.

In both, the main factors that influence the appearance of defects seem to be

- 1 the stage of tooth development at the time of injury, that is the Sec, EM or LM stage. In the experimental sheep this could be determined from a tetracycline band but is not accurately known for humans for ethical reasons.

- 2 the severity of the insult, that is does it cause death of the cells, temporary inactivity and recovery or just going slow

- 3 the duration of the insult that is short say a few days, longer say a few months or for prolonged periods.

The ameloblasts have a finite period in which to complete the mineralisation before eruption

starts and so the marks seen on the tooth reflect an earlier stage of development.

Let's look at a few of my early photographs

- 10 Hypo secretory severe short local upper pm ?trauma
- 11 Hypo secretory severe short many teeth, systemic illness
- 12 Opacity yellow early mat severe short lateral trauma 2.5-3y
- 13 Hypo secretory severe short trauma intrusion of tooth 3.5 year
Opacity maturation recovery of some cells
- 14 Opacities maturation severe centrals severe burns long recovery period
- 15 Diffuse opacities ?FI long time low level going slow
- 16 Lip line teeth dry
- 17 Hypoplasia genetic child, mother 18
- 19 Opacities genetic Polynesian yellow teeth

I must acknowledge the assistance given by other members of the DRU and my many collaborators.