

THE MICROBIOLOGY OF THE CAMÁN

Editor,

Camogie is a popular sport amongst women and involves an estimated 100,000 players, administrators, referees and coaches through 573 clubs across Ireland, as well as clubs in Europe, Australia and North America (www.camogie.ie). Fifteen-a-side camogie is a stickhandling, high velocity, multidirectional field sport,¹ played with a hurl or *camán*, which is usually crafted from ash (*Fraxinus excelsior*) plants. The hurl is not allowed to be greater than 13cm in width at its base, thus combined with the high velocity nature of the game, sports injuries in camogie have been reported, which have mainly consisted of hand, facial and laceration injuries.² Given the history and potential for lacerations and open wounds from hurl-related injuries, we wished to examine the microbiological flora of these, with particular attention to the types of bacteria that may be potentially introduced from the hurl into an open wound, from a laceration-related injury.

Hurls (n=24) were sampled anonymously from active amateur camogie players in the Ulster provincial game during active training sessions. A 2cm x 2cm area of each hurl was swabbed using a sterile pre-moistened transport swab (Sterilin, UK) and was examined microbiologically by inoculating the swab onto Standard Plate Count agar (Oxoid CM0463, Basingstoke, UK), followed by incubation at 37°C for 48h, as previously described.³ Resulting colonies, which were phenotypically different, were purified and identified using matrix-assisted laser desorption/ionization – time-of-flight (MALDI-TOF) mass spectrometry technology. The taxonomy of bacteria identified is shown in Table 1.

Seven bacterial species were identified from the surface of the hurls, including four Gram-positive bacteria and three Gram-negative bacteria, from six taxonomic genera. The natural niche of these bacteria is the environment, including soil, so it is most likely that the wooden hurls became contaminated when they were in contact with the soil on the grass pitch. Whilst some of the bacterial species isolated have previously shown some degree of pathogenicity in infection and therefore may be considered as opportunistic pathogens, they are not normally considered to be an infection risk to immunocompetent hosts, such as healthy camogie players.

Laceration injuries in camogie which are inflicted from a hurl should seek medical advice and appropriate wound management. GPs and Accident & Emergency clinicians should be aware of the spectrum of endogenous organisms from the player's skin, as well as those detailed above originating from the hurl, in any related wound complication from such injuries. Additionally, whilst our study did not isolate any *Clostridium tetani* organisms, given the origin of the organisms we identified as being from soil, camogie players should ensure that they have a complete and up-to-date tetanus vaccination record and should seek re-vaccination in accordance with the Green Book,⁴ where there is an incomplete vaccination history or where deficits exist.

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REFERENCES

1. Buckley CS, Blake C. The incidence of injury in elite camogie, an in-season prospective study. *BMJ Open Sport Exerc Med.* 2018;4(1): e000315.
2. Crowley PJ, Condon KC. Analysis of hurling and camogie injuries. *Br J Sports Med* 1989;23(3):183-5.
3. Furukawa M, McCaughan J, Stirling J, Millar BC, Addy C, Caskey S, *et al.* Who's at the door? - surface contamination of door frames in a single-bedded in-patient adult cystic fibrosis (CF) unit. *Ulster Med J.* 2020 89(1):17-20.
4. Public Health England. Immunisation against infectious disease. Chapter 30 Tetanus. In *The Green Book*. London: Gov.UK. [cited 2020 Apr 23]. Available from <https://www.gov.uk/government/collections/immunisation-against-infectious-disease-the-green-book#part-2:-the-diseases,-vaccinations-and-vaccines>

DEATH FROM DIABETES IN IRELAND/HISTORY

Editor,

In your journal in 1987 Crawford reported on the history of deaths from diabetes in Ireland.¹ His report showed that in the second half of the nineteenth century death rates associated with diabetes rose exponentially and he hypothesized that this was due to increases in the intake of carbohydrate and fat. For the last thirty years I have always opened my lectures on type 2 diabetes with this report. However, due to recent information from epidemiological studies of the consequences of famines, I believe that the original interpretation of this study is incorrect.

China's Great Famine (1959-1961) showed that adults born between 1960 and 1961 had a 23% increased risk of developing diabetes and if born in a particularly affected area



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there was a 40% increase. This suggests that fetal exposure during the famine increased the risk of diabetes in adulthood.²

In the Ukraine Famine, individuals born between 1930 and 1939 had in 2001 an increased risk of developing diabetes. The prevalence of diabetes increased by 47% in those born in regions with severe famine compared with those born in areas where a famine did not occur.³

The Dutch Winter Famine occurred during the final six months of the second World War. 702 subjects born in Amsterdam between November 1, 1943 and February 20, 1947 were shown at age 50 to be more likely to have glucose intolerance and insulin resistance.⁴

The Irish Potato Famine began in 1845 and ended in 1852. From Crawford's paper it can be seen that the greatest increase in death from diabetes occurred between 1880 and 1911 when those born during the famine would be between 30 and 60 years old.

The reason that starvation in utero is associated with a higher risk of type 2 diabetes in later life is that the fetus prepares for its likely adult environment which is not encountered (thrifty phenotype). These epigenetic changes are due to increased gene activity and expression rather than by starvation induced changes in the DNA sequence.⁵

Therefore, after 33 years, I believe it is time to reinterpret Crawford's data and conclude that the large increases in death from diabetes during nineteenth century in Ireland was due to the in utero effects of starvation during the Irish Potato Famine and not due to increases in the intake of fat and sugar.

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REFERENCES:

1. Crawford EM. Death rates from diabetes mellitus in Ireland 1833-1983: a historical commentary. *Ulster Med J.* 1987; 56(2): 109-15.
2. Zhang Y, Song C, Wang M, Gong W, Ma Y, Chen Z, *et al.* Exposure to chinese famine in fetal life and the risk of dysglycemia in adulthood. *Int J Environ Res Public Health.* 2020; 17(7): 2210.
3. Lumey LH, Khalangot MD, Vaiserman AM. Association between type 2 diabetes and prenatal exposure to the Ukraine famine of 1932-33: a retrospective cohort study. *Lancet Diabetes Endocrinol.* 2015; 3(10): 787-94.
4. Ravelli AC, van der Meulen JH, Michels RP, Osmond C, Barker DJ, Hales CN, Bleker OP. Glucose tolerance in adults after prenatal exposure to famine. *Lancet.* 1998; 351(9097): 173-7.
5. Zimmet PZ. Diabetes and its drivers: the largest epidemic in human history? *Clin Diabetes Endocrinol.* 2017; 3:1. eCollection.

SEPTIC CAVERNOUS SINUS THROMBOSIS; A RARE CAUSE OF UNILATERAL EXOPHTHALMOS

Editor

We wish to present an interesting case of Septic Cavernous sinus thrombosis (CST), which is fatal yet infrequent condition associated with high mortality and debilitating morbidity.¹ A 28-year-old man presented with cold like symptoms for 7 days which progressed into worsening headache, fever and nausea. His past medical history was unremarkable with no recent travel history. His examination revealed temperature of 38.1 °C and facial puffiness with no other systemic findings. His investigations only showed raised C-reactive protein 288 mg/L and white cell count 15.0 x 10⁹/L. In light of headache and visual symptoms he had Computerized tomography scan (CT) Head done without contrast which only revealed sphenoid & posterior ethmoid sinusitis. He remained febrile for four days despite being on broad spectrum antibiotics, and on day 5 he developed double vision, Cranial nerve VI palsy and unilateral exophthalmos, confirmed by formal ophthalmological/orthoptic assessment with no papilledema or any retinal disease. Repeat CT imaging of head/orbits failed to identify any cause of unilateral exophthalmos. Both initial blood cultures from the admission of day grew *Proteus mirabilis* sensitive to piperacillin/tazobactam while subsequent multiple blood cultures did not grow any organisms. A CT Head venogram was performed which confirmed a filling defect consistent with CST and inflammatory changes in sphenoid & ethmoid sinuses (figure 1 and 2). Other investigations such as chest X-ray, viral serology, abdominal ultrasound, echocardiogram, lumbar puncture, urine and stool cultures were all negative for any alternate source of infection.

He was initially treated with ceftriaxone/amoxicillin for



Figure 1 CT Venogram with arrow showing the cavernous sinus thrombosis on the right