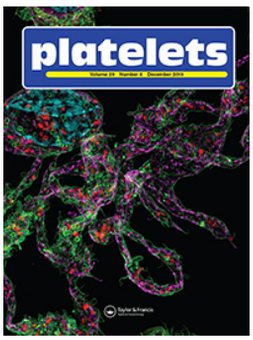

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Gus Born: Scientific Reflections of Later Years, Inspiring the Next Generation of Blood Researchers

Valerie B. O'Donnell

Systems Immunity Research Institute, School of Medicine, Cardiff University, Cardiff, Wales, UK

ABSTRACT

I am delighted to have been be asked by Steve Watson to write a short piece for this series in Platelets, on my friendship with Prof Gus Born which began in 2011, when he came to Cardiff to present at our UK Platelets meeting. Gus was an insipiration, his story resonates today and it is an honour to have the opportunity to record a summary of our meetings here.

KEYWORDS

Platelets, Cara

Back in 2011, the UK Platelet Group meeting was to be in Cardiff, and as it was the fiftieth anniversary of the invention of the Born Aggregometer, Steve Watson suggested I might invite Gus, to deliver our plenary talk. I knew nothing of Gus or the history of this instrument, despite having used one for about 11 years and was intrigued to find out more.

Gus gratefully (and delightfully) accepted and from then on an unlikely friendship based on shared science started. Our emails ran from early 2011 to late 2012, then after that he preferred to write letters of which a few I can find. Always on headed card (his London address usually) and with his warm greetings always included.

Leading up to the meeting, he wrote:

Dear Valerie,

Many thanks for telling us how to get to University Hall by taxi and for the full program. I look forward to it tremendously, so does my Welsh wife.

Best wishes, Gus

Dear Valerie,

Greatly looking forward to seeing you. What about dress? May I not bring a suit? I will, of course, be in jacket and tie. Of course, if you want a suit I will bring one but I would rather not.

Best wishes, Gus

In the end, he came without Faith, braving the train alone, and in my stress with trying to keep the meeting running did not manage to give him much time or attention. This did not matter; he had great fun catching up with friends and talking science, as the pictures here show (Figure 1). His lecture, charting the (his) discovery of ADP as a platelet activator was a typically him (Figure 2) – an understated, charming, and modest presentation, but you could not miss the huge importance of his contribution to platelet biology, nor his unending interest in platelet research . . . the reason we stayed in touch from then until 2017, the last time I met him at his home in Highgate.

After Cardiff, we met in London a few times a year. When I was up for work, we would go for afternoon tea, usually at the Royal Society of Medicine where he was a Member and always so that he could quiz me about the latest platelet research, what were the latest discoveries, what was I working on, how amazing platelets were. . ..

In 2013 after the UK Platelet Group Meeting in Birmingham, he wrote:

Lyneham, Oxfordshire

Dear Valerie,

It was particularly nice seeing you yesterday in Birmingham. So much to talk about. So I look forward to seeing you next when you are in London. Let me know please in good time – if you have time. We are over the weekend in our country paradise where one has time to think. Did I tell you that I am busy with a general think piece about something my father wrote? It is nice to get away from the little blood cells and think about something else. Hope you enjoyed the meeting, and that we will meet again soon.

Love to you and family – Gus(tav)

In 2015, he wrote:

Highgate, London

Dear Valerie,

Just a note to ask how you and your family are: totally well, we hope! Are you still busy with research, as well as teaching and admin?

Faith and I are well, though I am – hard to believe – 94! She is the best looker-after imaginable. Three of our "children" with their families are nearby, which is a blessing. I have written reminiscences – really for family of course. But if you of any interest, you are one of the few non-members who might enjoy them.

All best wishes and love – Gus (Born)

He stayed fully engaged in research, and had a fundamental question he was working on: why does cancer never metastasize to muscle? As far as I know, he is right, it is an intriguing question, one that might lead us to new ways to prevent cancer spread if we knew the answer.

We talked about his life, his history, where he came from. Each time a new and more jaw dropping anecdote about his famous relatives (singers, actors, comedians we all know), Nobel Laureate father and post doc fellow/friend (Vane), his first impression of Hiroshima, how he saw radiation sickness caused bleeding and this inspired him as a young army medic to research platelets, his family friends (Einstein, Marie Curie, famous composers), his childhood friends (Marie Curie's

Correspondence: Valerie B. O'Donnell, Systems Immunity Research Institute, School of Medicine, Cardiff University, Cardiff, CF14 4XN, UK. E–mail: o-donnellvb@cardiff.ac.uk

Figure 1. Panel A. Gus delivering his plenary lecture at the UK Platelet meeting in Cardiff in 2011. Panel B. Gus with Prof. Alastair Poole, University of Bristol, in Cardiff, thank you to Prof. Ingeborg Hers (University of Bristol) for the photographs.

children), and on we went. Many invitations to visit his country home with my family came, but we never made it. Each time, he would leave and go back to Highgate by tube, much to my worry, until the last time we met (I had convinced him he did not need to bother with the tube and I would come visit). So, I came to his flat and met Faith. We sat at the window, in the heart of London, high up with a huge garden of trees outside, giving the impression of deep countryside. Books everywhere, and coffee and cake. As always, talking about platelets.

His phenomenal family story is covered in two books he wrote on the topic, of which I have signed copies. There are some things I need to mention for reasons that will be clear later. **In Defence of Learning** charts his migration from Nazi Germany as an 11-year old, with his father, the Nobel Laureate Max Born in 1933 [1]. His traveling to Cambridge with his mother and dog, and his father's new job as Stokes Lecturer. Many times he said to me how grateful he and his family were to the people of Britain, he never forgot the generosity he received at that time. This is something I will never forget; and in this book he says "The overriding fact was the welcome and warmth with which we refuges were received by the British people." Later on he charts the huge legacy made by refugee scientists that came to Britain at that time: eighteen Nobel Laureates, seventy-one Fellows or Foreign members of the Royal Society, fifty Fellows of the British Academy, and sixteen Knights." At the end he writes: "Scientists of the second generation of immigrants, of which I am one, mostly remain well aware of the facts and effects of the change of country. But such is our generous acceptance by society in Britain, that our children, the third generation, already wonder what all the fuss is about: they are entirely assimilated and integrated. It is probably just as well that they are no longer concerned with historical predicaments of the kind that gave rise to Cara."

But, on that last point sadly, time tells us otherwise. We have to be concerned with these predicaments, and even more so now.

The first time I heard of Cara was two weeks after Gus passed through, an email from our International Dean at Cardiff arrived supporting an initiative to help Syrian academic refugees in Turkey continue their academic engagement whilst in exile. The briefing note said:

Annex 1. Cara: organizational background

Origins The Council for At-Risk Academics is a UK-registered charity founded in 1933 under the leadership of William Beveridge, to rescue academics suffering persecution under the rise of Nazism and facilitate their continued work in safety. Sixteen Cara Fellows from the 1930s and 1940s became Nobel Laureates, and many more innovators in their fields, including Ernst Chain, Nikolaus Pevsner, Lise Meitner, Max Born, Hans Krebs, Karl Popper, and Sigmund Freud with scientific legacies still in evidence today. Cara has been a lifeline to academics at risk for over 80 years, as and when world events have placed them in the line of fire: Hungarian Uprising, Cold War, Apartheid South Africa, Latin American Junta, Vietnam, Kosovo, Rwanda, Sudan, Zimbabwe, etc. and, more recently Iraq, Yemen, Iran, and Syria. Cara support is framed as temporary sanctuary offered at times of heightened risk.

Of course, I had to sign up when I saw this was the organization that brought Gus to the UK. Kate Robertson from Cara knew Gus and Faith, they were great supporters of Cara through the years she said, and she identified a placement who she thought would work well for us. Mr Ziad AlIbrahim, a displaced hematology clinical scientist from Syria, visited in July and August 2018. In a twist of fate, Ziad has now been to visit several platelet and hematology colleagues in the UK, including some who also personally knew Gus.

Gus and I were friends for such a short time, but the deep impression he has left on me continues far beyond. His inspiring story resonates strongly today, and it is our duty to carry on his legacy, both as platelet researchers, and as humanitarians. I feel honored to have been able to link with Cara, and do something small in Gus's name. I hope he would feel proud that we can continue his inspiring work in this way.

Why ADP? An evolutionary explanation.

G.V.R BORN

William Harvey Research Institute, Charterhouse Square, London, EC1M 6BQ

Abstract (max 350 words).

Adenosine diphosphate (ADP) was the first platelet-aggregating agent to be discovered (Gaarder et al, 1962; Born, 1962). ADP induces haemostatic platelet aggregation in man and other mammals, but not in the functionally corresponding cells of other vertebrates including birds and reptiles, nor in those of other animal phyla (Macfarlane, 1970; Levin, 2006). Other agents which activate cells to prevent loss of body fluid, viz. collagen and thromboxane A_2 (TXA_2) possess this function much earlier in evolution.

So why ADP, rather than any of the other ubiquitous extracellular transmitters, including ATP (Burnstock, 2006)? The answer is suggested by comparing haemostasis in man and other mammals with the corresponding process in other animals. In mammals the blood pressure is high, so that blood loss from an injury is very rapid. Therefore, haemostasis must be very rapid, a requirement met by platelet aggregation: the activation time of human platelets at 37°C is certainly less than 100 msec (Born and Richardson, 1980). The clotting of plasma does not become effective for many seconds. With ATP leaking from damaged cells, the dephosphorylating enzymes on cell surfaces ensure very rapid production of ADP. The further dephosphorylation to ineffective AMP is significantly slower, ensuring a temporary increase in local ADP concentration.

In vivo evidence indicates that ADP is responsible for at least half of haemostatic platelet aggregation (Born et al, 1981; Begent et al, 1982). As TXA_2 and collagen are also effective, the addition of the ADP mechanism in mammals is presumably in the nature of a fail-safe system employed by engineers. One curious question persists; why not in birds?

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Figure 2. Abstract submitted by Gus to the UK Platelet Group meeting in Cardiff, 2011.

Disclosure Statement

The author has no conflicts of interest.

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