The power of simplicity

Inaugural lecture Prof. José Borghans

June 21, 2023

UMC Utrecht

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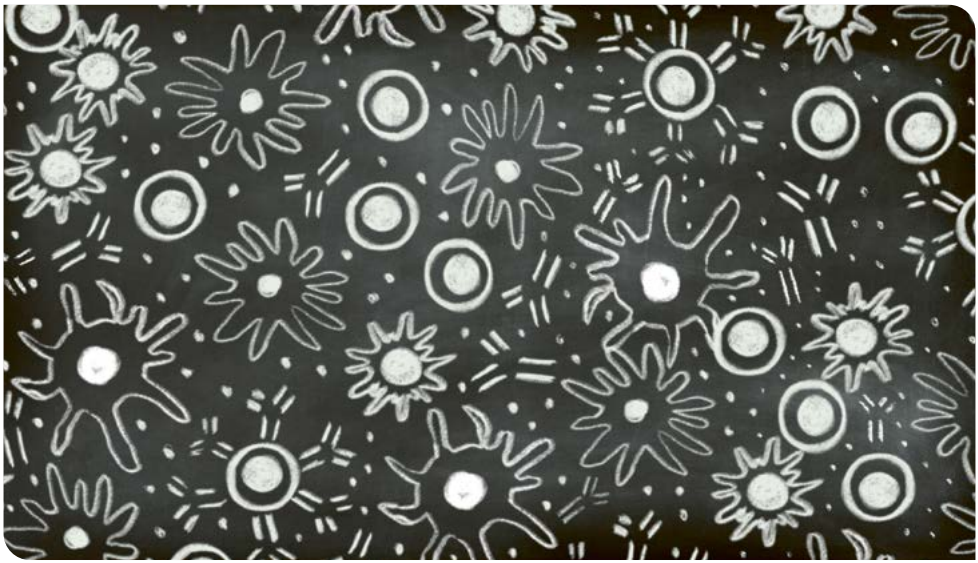
Inaugural lecture delivered at the acceptance of the chair of Quantitative Immunology at the Faculty of Medicine of Utrecht University on Wednesday June 21, 2023 by Prof. José Borghans

Rector Magnificus, dear colleagues, friends and family,

Exciting... I certainly thought so. I had prepared myself very well, tried out my story on many, and had spent a lot of time making beautiful figures. I was also very much looking forward. I was invited to present my first research findings as a student at a conference in San Diego. I was very enthusiastic about our results. We had calculated – after about half a year of work – how specific our immune system needs to be to provide maximal protection. A very fundamental question that had kept us quite busy. Quite a few highly respected immunologists presented their work at the conference. People whom I only knew by name, and from whose textbooks and articles I had learned immunology. I was also well-prepared for all possible questions. But for this one, asked by one of the most famous immunologists at the conference, I was totally unprepared. She had studied my poster in great detail, looked at me and asked: "Why don't you just go and do experiments?" She added some very heartfelt advice. If I still wanted to save my career, I had better quit that maths right away and just go back to the lab. Immunology really did not need maths.

I was flabbergasted. At the same time, I felt an urge to convince her of the added value of mathematics in immunology. I suggested to have dinner together, thinking that I would convince her then. But it did not get any better. At dinner, she delicately added that her team typically calculated the things we did over lunch, on the back of an envelope. It made very clear to me that the field I had fallen so much for was not everybody's favourite.
A lot still had to be done before the field of immunology would embrace mathematical modelling.

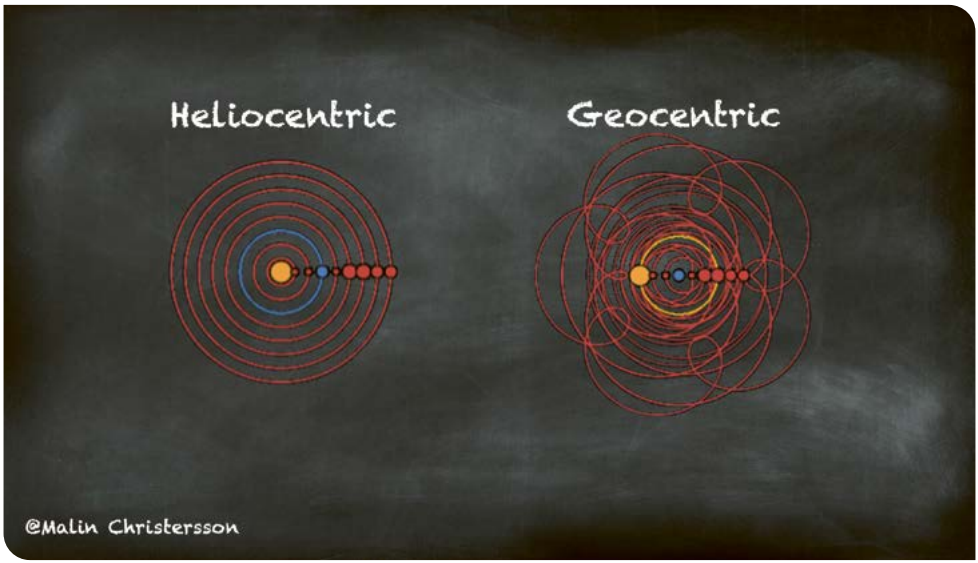
Now you may also be wondering: what is the link between mathematics and immunology? I would almost say: everything! Our immune system is an insanely complex system of many billions of cells and molecules that together need to make decisions about life and death. It is exposed to all kinds of triggers all day long and constantly needs to decide: do I react to it or better not? While it does have to react to the coronavirus, even though our body had never encountered it before 2020, it should definitely not react to the body's own molecules. And while it does need to react to bacteria like Salmonella in spoiled food, we do not want it to react to the food itself, even if we have never eaten it before. These are tough decisions, which are made thanks to an intriguing communication between the many cells and molecules of our immune system. How do you study such a complex system? That is exactly where mathematics comes in. With your intuition alone, you simply won't get there.



Over the next 40 minutes, I would like to show you, through some examples, how mathematics can contribute to our understanding of the immune system. And while not everyone will immediately think of something simple when they think of maths, I hope to show you how mathematical models provide simplicity and insight into the incredibly complex system that our immune system forms.

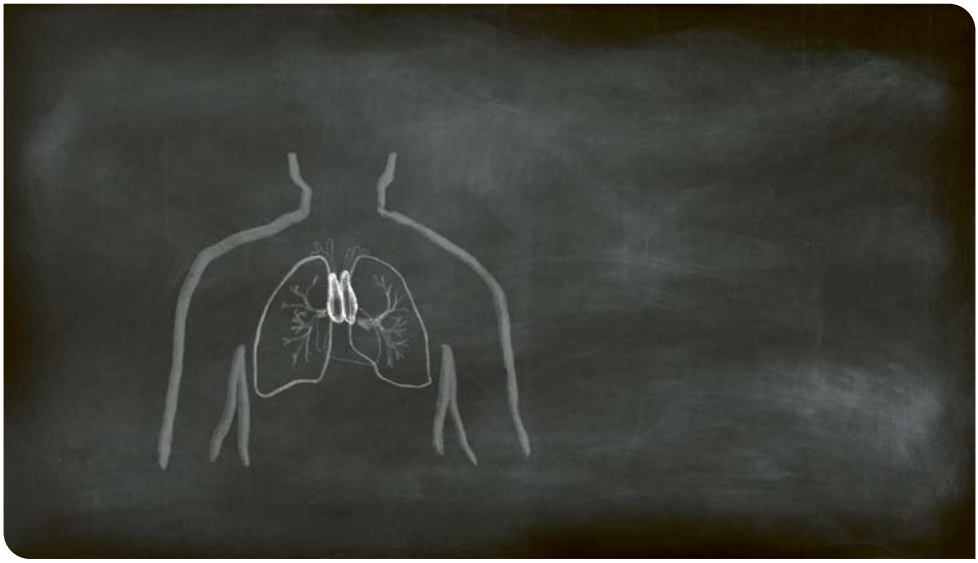
The use of mathematics has long been the norm within physics and chemistry. No quantum mechanics or theory of relativity without mathematics. Within biology, and within immunology in particular, mathematics has only recently gained a foothold. It was definitely not the norm yet at that immunology conference in San Diego. An important contribution was made by Alan Perelson. He showed, using a – very simple – mathematical model, that the life cycle of the virus HIV covers less than three days. Within three days, each virus

particle has multiplied. That allows the virus to increase exponentially from a single viral particle on day 1, to a thousand in a month, and a million in two months. Because the virus makes a lot of errors during this multiplication, this leads to a huge variation of new viral particles in a matter of weeks. This gives HIV the chance to escape from our immune system or from the drugs a patient takes. By now we know no better, but it is important to realize that before this finding, our view of HIV was completely different. HIV seemed to be a latent virus. A virus that keeps quiet in our body for a long time, only to break out after about 10 years and cause AIDS. It could not have been further from the truth. HIV turned out to be a virus that is anything but latent. Today's standard treatment for people with HIV involves giving multiple drugs at the same time, as early as possible, to prevent the virus from escaping. We owe that standard in part to this very basic finding, which was made thanks to researchers who looked at existing data with a mathematical eye. For myself, this study also stands out for a very different reason. This study demonstrated so powerfully how great the value of mathematics can be within immunology, that it became a real game changer: it formed the start of a new era in which mathematical models were slowly getting more accepted in immunology.

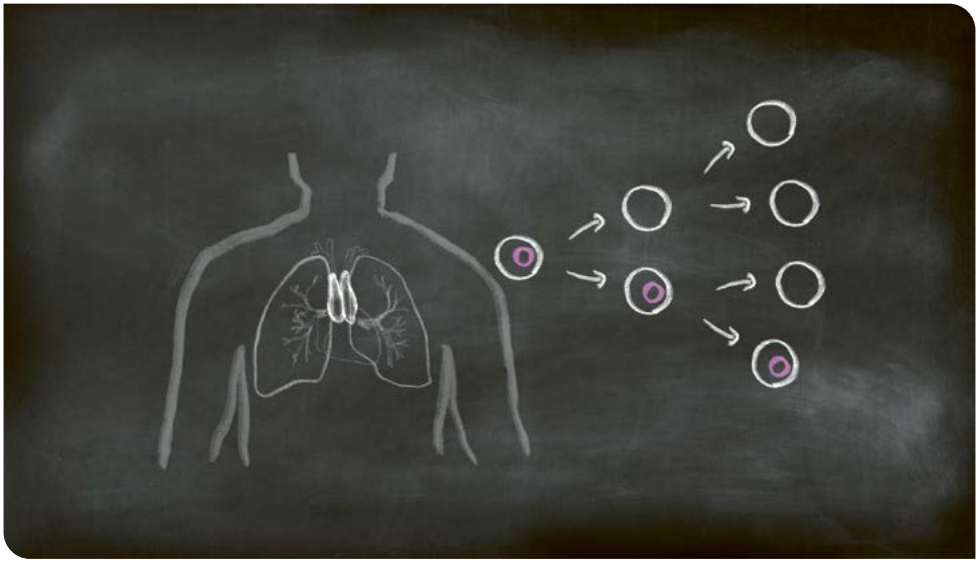


One of the most important contributions of mathematical models is that they provide simplicity and insight. To illustrate this, I will use an example from astronomy. The astronomer Nicolaus Copernicus, who, by the way, was also a mathematician, formulated a new model of the universe in 1543. Until then, the Geocentric model prevailed, which assumed that the earth is central and that the sun, moon, stars and planets orbit the earth. In fact, this is not a strange idea because, viewed from the earth, the earth appears to be stationary and everything around it seems to be rotating. The complexity with which

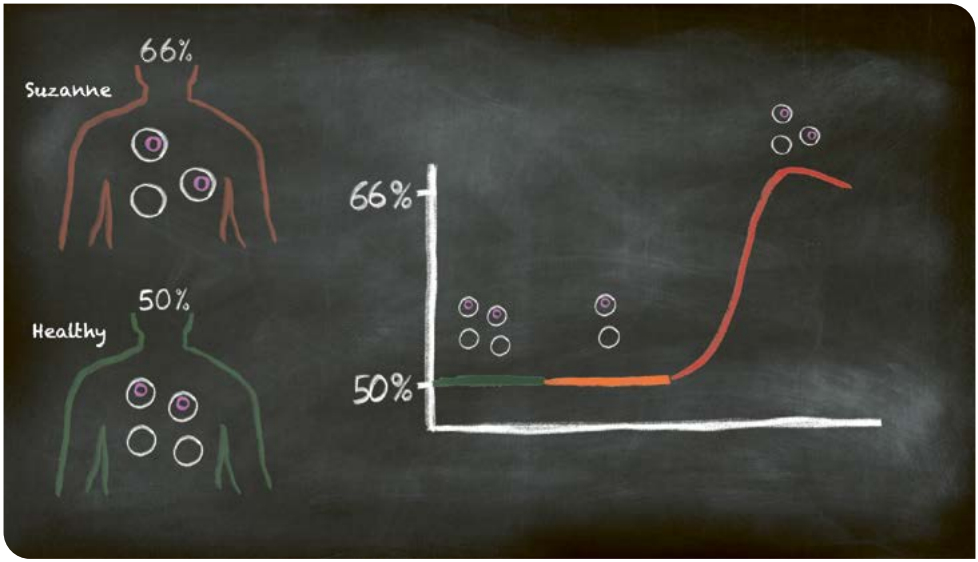
the stars and planets seem to orbit around the earth resembles the complexity of our immune system. Copernicus proposed a revolutionary, different model by placing the sun rather than the earth at the centre. In his Heliocentric model, there appeared to be an unprecedented simplicity in the orbits of the stars and planets revolving around the sun. The apparent complexity of the universe was suddenly reduced to a clear and simple insight.



Mathematical models similarly provide simplicity and insight into the complexity of the immune system. To demonstrate this, I first need to tell you a little more about the immune system. Within immunology, there is a lot of discussion about the role of the thymus. This is a fairly unknown organ, close to the heart, that most people know only from the veal thymus, a piece of meat you can eat in classy restaurants. The thymus is a fascinating organ. It is the only organ that does not grow but gets smaller with age. In the thymus, our T cells grow up. T cells play an important role in our defence against bacteria, viruses and cancer cells. Thanks to an ingenious system of genetic recombination, it is estimated that over a billion different T cells, each with their own very specific receptor, are produced in the thymus. As a result, we have 100,000 times more different T cells in our body than we have genes in our DNA! In the thymus, T cells learn what they should and should not respond to. This is needed to prevent them from starting immune reactions against our own body. Although everyone agrees that a young child's thymus works better than an adult's, there is much debate about its role in various diseases. For example, some researchers think that impairment of the thymus causes problems in HIV infection. Other researchers on the other hand, are convinced that the thymus does not play any role in adults at all.



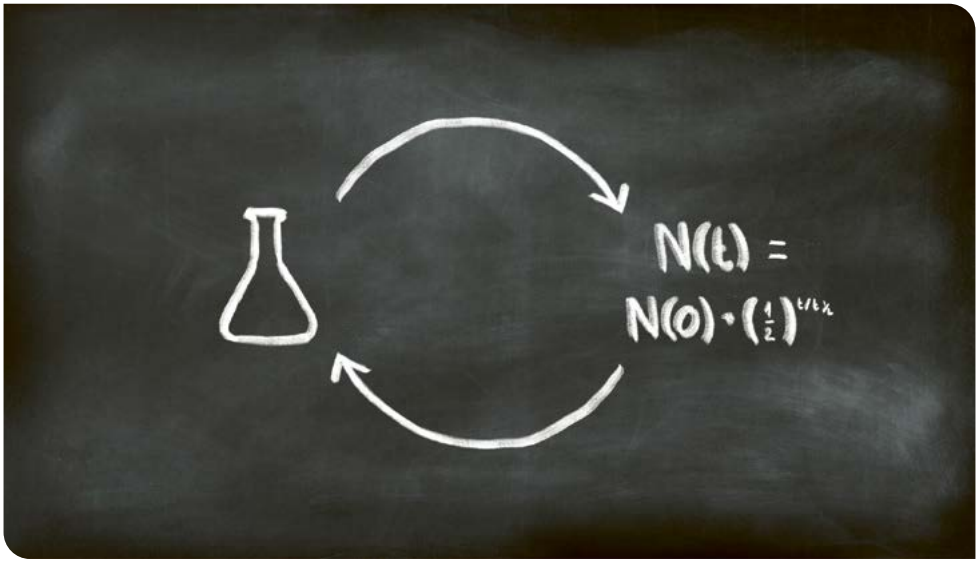
How do you study the role of the thymus? To determine how many T cells are made by the thymus, we often measure TRECs. TRECs are small chunks of DNA that end up in the cell during genetic recombination. They have no function; they are simply a residual product of T cells made in the thymus. The beauty of TRECs is that they can only be formed in the thymus. If a T cell divides outside the thymus, the TRECs are not copied and the existing TRECs will be randomly distributed over the two daughter cells. Cells that have divided a lot therefore have a lower TREC content than cells that just emerged from the thymus. Because TRECs can only be made in the thymus, they are often used as a measure of thymus activity. They have been measured in numerous studies: for example, T cells from people with HIV have been found to contain fewer TRECs, while T cells from people who have undergone a stem cell transplantation contain more TRECs than T cells of healthy people. Without a mathematical model, you then quickly draw the wrong conclusions.



To illustrate this, let us look at a specific example. Suzanne is a 25-year-old woman who was admitted to hospital with leukaemia. Fortunately, a suitable stem-cell donor was quickly found. She was treated with severe chemotherapy to fight the leukaemia and to prepare her body for the stem-cell transplant. After the transplantation, her immune system had to recover. When her T cells were examined two months after the transplantation, they were found to have a higher TREC content than you would expect based on Suzanne's age. The researcher who made these findings enthusiastically concluded that Suzanne's thymus had started to work harder, in an attempt to quickly restore her T-cell count. Using a simple mathematical model, you immediately see that this conclusion is wrong. Even if the thymus continues to work as hard as it normally does, the model predicts that the TREC content of Suzanne's T cells will shoot up to supranormal levels. The increased TREC content is a direct result of her low T-cell numbers. Because there are so few T cells, after transplantation the new T cells that are just coming out of the thymus become over-represented, and these still have a lot of TRECs. So increased TRECs are no evidence at all that the thymus works harder after a stem-cell transplantation! For me, examples like these are the gems of mathematical immunology. Isn't it beautiful that a simple model can give you such a different view of the data? The model – much like Copernicus' model – helps you gain insight; to interpret the data in the right way.

What I like about these types of models is that they excel in simplicity. They are an oversimplified representation of reality, and that is precisely why you learn so much from them. Afterwards you often get a response like: "I could have done that without a mathematical model." And that is indeed true. The problem is that without a model you would not have thought of it, and without a model you have way too much freedom of

reasoning. As far as I am concerned, the best models are even the ones that you did not need with hindsight. Those are the cases where you have really gained a new insight. And once you have seen it, it is difficult not to see it anymore. In the words of Sir Robert May and Martin Nowak: “Mathematics is no more, but also no less, than a way of thinking clearly.” Or as Lee Segel so eloquently put it: “Mathematical biologists? They are people who make oversimplified models and do not even feel embarrassed.”



As a researcher in training, I discovered that I found modelling even more interesting when performed in close collaboration with experimentalists. I dreamed of working in a lab where mathematical modelling and experiments would go hand in hand. That allows you to create the full research cycle: you can test model predictions in an experiment, interpret the acquired results using mathematical models, and then form new hypotheses, which you can test again in the lab. That dream has come true. I am proud that in the research group that I lead with Kiki Tesselaar, experimental immunologists and mathematicians work together on a daily basis. We learn to understand each other’s language, each looking at the same problem from a different angle and based on our own expertise. This is interesting, refreshing and sometimes quite challenging. It requires flexibility and understanding of each other’s work. But if you make the effort, you often gain valuable new insights.

I have already highlighted how mathematics can help bring simplicity and understanding to the complexities of the immune system and how it helps us to think clearly. Another important contribution of mathematical models is that they make immunology more quantitative; that they give a sense of numbers, such as production rates and lifespans.

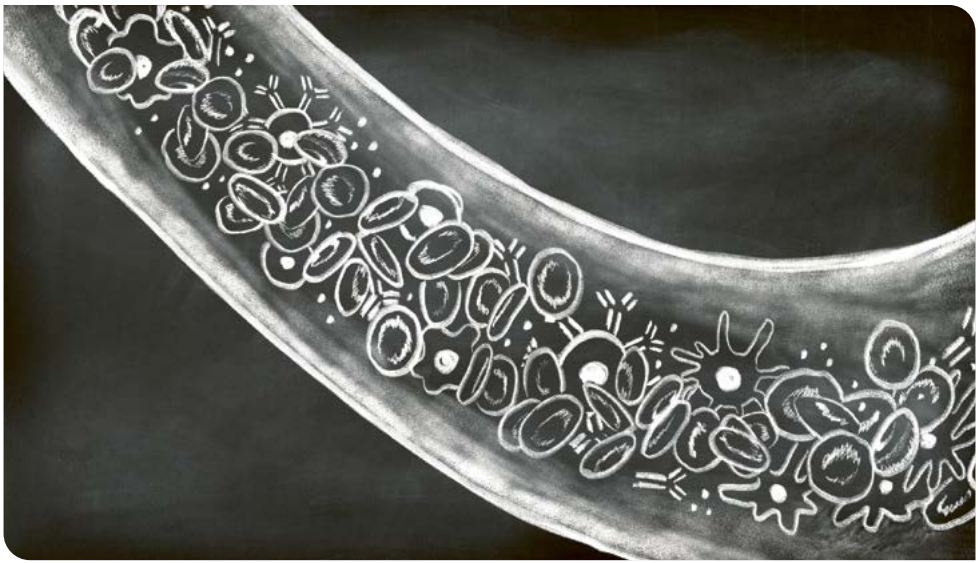
Those numbers are desperately needed in immunology. I already addressed it in the example about HIV. Once it had become clear how fast the virus replicates, the consequences for the treatment of patients were immediate. But those numbers are very often still missing in immunology. It is truly amazing what technical *tours de force* we can perform in immunology, while even the most fundamental quantitative knowledge is often lacking. For example, for one of our models, we needed to know how long a T cell lives. It did not have to be very precise, but we did need to know whether we should think in terms of days, months or years. When I asked that question to some respected immunologists, I was baffled by their responses. Whereas one immunologist confidently stated that a T cell lives for about 2 weeks on average, another was able to tell me with equal confidence that a T cell lives for about 20 years. How could it be that we had no answer to such a basic question, while at the same time we were trying to understand how these cells get disturbed during infections? Or why the immune system recovers so slowly after a stem-cell transplantation? This made very clear to me how much work still had to be done.

And although the question sounds very simple – how long does a T cell live? – answering that question turned out to be far from trivial. One might be able to follow a cell that stays in one place in our body, from the time it was created to the time it dies. But T cells make life a lot harder for us. From the thymus, a T cell travels throughout the body, looking for pathogens that need to be eliminated. It moves through the blood and lymph, divides in the lymph nodes when it recognizes a virus or bacteria, and lodges itself in all kinds of organs, from the skin and intestine to the liver. It is not trivial to then try and keep track of when that one cell that was born in the thymus dies in any place in the body.

In our research group, we study these questions using *in vivo* deuterium labelling. Healthy people or patients drink heavy water, which is a form of water that contains deuterium, which allows us to measure how much deuterium is built into the DNA of the cells. By analysing those data with mathematical models, we can calculate how often cells divide and how long they live. This has already yielded important fundamental insights. For example, we have shown that the T cells responsible for our immunological memory live for only a few months, while they often protect us throughout our lives. Before they die, they pass on what they have learned to their daughter cells, to ensure the information is not lost. Naive T cells – those are the cells that have never recognized anything in our body – on the other hand, appear to live for a very long time, on average for about six to nine years. We also discovered that the way naive T cells are maintained differs fundamentally between mice and humans. While naive T cells in the mouse are almost all produced by the thymus, in adult humans only about 10% of these cells appear to come from the thymus. The other 90% are all formed by cell division. This is quite disturbing, to say the least, for a field that is largely based on findings in mice. It shows how terribly important it is that we shift our focus and perform our studies – whenever possible – in humans. Unfortunately, we are still far away from that dream. This became very clear to me when I attended a thymus conference several years ago. There were three full

days of presentations about the thymus, without exception in the mouse. Among the one hundred posters, there were only two – those from our lab – about the thymus of humans. Even though we already knew by then that the role of the thymus is so different in mice and humans!

Using a similar combination of deuterium labelling and mathematical modelling, we investigated why T cells recover so slowly after stem-cell transplantation. Suzanne, the patient with leukaemia, also suffered for months after her stem-cell transplantation from the slow recovery of her immune system. She was extremely susceptible to fungi, viruses and bacteria that would normally cause no problems. People speculate a lot about the cause of the slow recovery of the immune system. It could be that the thymus is affected by the intense chemotherapy. Or that T cells no longer undergo enough cell divisions. But perhaps the slow recovery is to be expected, because T cells in a healthy person are not replaced very often either. We found that the rate at which T cells are produced is significantly increased after stem-cell transplantation. We saw no indication that the thymus was affected. However, we did see that the increased production of T cells was accompanied by increased loss of cells from the blood. The cause of that accelerated loss remains unclear. If it is due to increased cell death, that is obviously not good news; in that case, the cells that you have just painstakingly created would be lost again through cell death. But perhaps we see an accelerated loss of cells from the blood because they go into the tissues. In that case, the slow recovery of T cells in the blood would paint a too pessimistic picture, because the immune system in the tissues is recovering in the meantime.



I already emphasized the importance of more research in humans. The questions arising from our research on stem-cell transplantation also illustrate the great need for research on cells in tissues. It is important to realize that almost all research on the human immune system is based on cells isolated from the blood, while only 2% of our T cells are circulating through the blood. The vast majority of our T cells are in our tissues: in the bone marrow, the lymph nodes, but also for example in the skin and the intestine. We have no idea how those cells preserve our immunological memory. And yet those very T cells in tissues have a crucial role in immune responses against viruses and cancer, and they are responsible for undesired immune responses, such as in MS and rheumatoid arthritis. Thanks to a Vici grant from the Dutch Research Council (NWO), we are currently studying the dynamics of T cells in tissues. We recently discovered that memory T cells in the skin are much shorter-lived than previously thought. Memory T cells in the bone marrow also live for only a few months, but they live longer than memory T cells taken from the blood or skin. These insights provide essential fundamental knowledge if we are to understand how we should inhibit these cells when they cause undesired immune responses. Should we inhibit their migration to the tissue? Or perhaps their cell division? I look forward to taking these fundamental findings a step further towards clinical applications in the coming years.

I hope these examples have given you a glimpse into how mathematical models can contribute to our understanding of the immune system. How they provide simplicity and insight, a tool for logical thinking, and how they can provide the numbers that are so important to gain a better understanding of the immune system.

I began my story telling you about my first experience as a mathematical biologist at a conference in San Diego. It is fascinating to see how much has changed in the meantime. When I started in this field, mathematical models of the immune system were published almost exclusively in journals that no immunologist would ever read. And, according to some, you could only build a decent career in immunology by doing experiments. Nowadays, the situation is completely different. There is almost no lab that does not produce Big Data, and with that, the demand for mathematical knowledge has grown tremendously and mathematical analyses have become much more common in the immunological literature. I would like to ask the lady in San Diego how she looks at it now. Whether she still thinks we do not need more than some scribbles on an envelope during lunch time.

I myself am convinced that we can make great steps forward by combining experimental work and mathematical modelling on a much larger scale. It will require very close collaboration between researchers from different disciplines, and we must have the courage to stimulate that. What is needed is Team Science. The prevailing norm within academia is still that there is one principal investigator per research group. To solve complex issues, that is often no longer sufficient. We cannot expect that all the expertise needed can always be provided by one person. It is by working together, by sharing expertise and by looking at a problem from different angles that we make progress. Unfortunately, that very collaboration is coming under pressure as science is increasingly turning into a competition. Prizes can be won, and the main sources of money to perform fundamental scientific research are literally called “competitions.” Moreover, such grants place excessive emphasis on the individual. A major drawback is that researchers start to compete with each other. Discussions about authorships become overheated because one’s career prospects and chances of getting grants are directly dependent on them. The fact that within the Biomedical Sciences the greatest value is attached to first and last authorships gets in the way of Team Science. And even though we pretend that there is nothing we can do about it, that, of course, is nonsense. Within other fields, things are often quite different. Can’t we make it simpler here too? Why not always put the list of authors in alphabetical order? A short description of everyone’s contribution can do justice to the differences that undoubtedly exist between authors and that are allowed to exist. That way everyone gets the credits he or she deserves, and teams can work together much better and more pleasantly.

The fact that there is competition between different research groups probably feels more natural than competition within groups. Each team, of course, would like to make that big breakthrough. But I think even that competition is undesirable in science. As a result of competition between groups, researchers become cautious about sharing their latest findings at conferences, afraid of being scooped. I am convinced that science thrives not on competition but on connectedness and on knowledge sharing. Ultimately, we want to solve complex problems, and the chances of succeeding will only increase if we share our knowledge and work together. After all, it does not matter whether you or I invent the new effective treatment against cancer, what matters is that it gets there as soon as possible.

With the exaggerated emphasis on individual success, we are paying another high price. Everyone benefits from painting as rosy a picture of themselves as possible, because that increases the chances of success. In doing so, we create a make-believe world that is miles away from reality. For example, the probability of a grant application being awarded is on average around 15%. That means that every researcher submits an average of 6 applications that are not awarded, only to have one successful application. Nevertheless, you rarely hear about those failed applications. As a result, if you read all the success stories on LinkedIn, Twitter, or in the UMC Utrecht newsletters, you would almost lose heart as a young researcher. As long as we only share our successes, we keep up a fake image, much like a slick Instagram profile. That is neither fair nor encouraging to young researchers. It suggests you are to be blamed if something fails. That pressure is starting to become too high for many young scientists. My research career was full of failures too. I realize very well that at those moments, I was not inspired by slick CVs and stories of the supposedly most successful researchers. It was thanks to the researchers who had the courage to share with me their failures that I had the courage to move on. I am very enthusiastic about the recent movement of scientists sharing their real CVs. Instead of the standard Instagram-like CV, they published a sort of BeReal CV, with some successes but mostly a lot of failures. I find this a very healthy and important development, which I fully embrace. The power of simplicity also applies here. Let's not increase the pressure by always presenting the perfect picture. Let's make sure that science remains sustainable and inclusive, that it is a place where we can learn from each other in all transparency, where failures are accepted, and where diversity is embraced.

For above all, science remains a wonderful profession! It is enormously inspiring to work year after year with people who are so intrinsically motivated, who are creative and curious, who want to know how things work, and who are so much driven by content. I look forward to working with them to make immunology even more quantitative, and to discover new simplicity in the complexity of the immune system. There are exciting times ahead. The data sets produced in immunology are getting bigger and more complex. Expectations around Big Data are high and huge amounts of money are being spent on it. To be more precise, a lot of money is being spent on creating all that data. Its analysis is still too often not given the priority it should. Without investment in highly skilled bioinformaticians and mathematicians, the production of all that data is in fact a waste of money. We need the people who can reduce all that complexity to simple, conceptual insights. I therefore see an important task for myself not only in the field of research, but also in the field of education. I look forward to contributing to the training of the researchers of the future. Some of them will hopefully later specialize in the mathematical analysis of immunological data. But I find it equally important to train researchers who are much more interested in the experimental side of the work. To give them the necessary background to be able to work together in multidisciplinary teams and to be able to appreciate the literature in which complex data analysis is forming an increasingly important part. I hope to let them all experience the power and beauty of mathematical modelling in immunology.

I would like to close with a word of thanks. I owe the fact that I am standing here today to a lot of people. More even than I can name right now. First and foremost, I would like to thank all the PhD students and post-docs who have enriched our group over the years. The current team: Elena, Carina, Lyanne, Anna, Shiva, Erdem, Arpit, Joukje, Terry and Jingwen, but also all previous PhD students: The research would not be there without your tremendous commitment and motivation. Being able to work together with you at the limits of what is possible is the best part of my job. I am extremely happy that almost all of you are here today to celebrate this day together, and that some of you even travelled all the way from abroad.

Many thanks to Joukje Willemsen, for the beautiful slides she created for this inaugural lecture. I was amazed how you managed to incorporate the theme of simplicity so naturally in all the slides. I would also like to thank Simone Schut, for her valuable feedback.

I would like to thank Kiki Tesselaar, with whom I have been leading our research group for almost 20 years. I know very few people who are as smart and think as out of the box as you. It is amazing how you always come up with totally unexpected, creative ideas. The fact that our research group can be multi-disciplinary is thanks to you. Thanks to Sigrid Otto, who has strengthened our research group for as many years, and to all the students who have worked in our group over the years. All members of the Computational Immunology Core, in particular Julia Drylewicz, Alex Yermanos, Bram Gerritsen and former colleague Aridaman Pandit, thank you very much for the inspiring collaboration.

I would like to thank the Boards of Directors of UMC Utrecht and Utrecht University for the trust they have placed in me. I am proud to hold the Chair of Quantitative Immunology at a university that was investing in mathematics in biology long before the Big Data era.

I can still hardly believe how lucky I was to find someone working on models of the immune system within Paulien Hogeweg's Theoretical Biology Group. And not just anyone. Rob de Boer first supervised my internship, then my PhD research, and we have kept on working together ever since. You are a source of inspiration for me as a scientist, as a team leader and as a person. I hope to work with you for many years to come. Within the same group, I was fortunate to work with Can Kesmir, with whom I share not only a love for theoretical immunology, but also a long-standing close friendship. I owe my move into

experimental labs to Antonio Freitas and Frank Miedema, both of whom were open to using mathematical models within their experimental groups at a time when that was not at all popular yet. Frank, your enthusiasm is contagious. I have learned an awful lot from you. Without the many opportunities you have given me, I would not be standing here today.

It is great to work in the Centre for Translational Immunology, the CTI, originally founded by Erik Hack, and now managed for many years by Linde Meyaard and Margje Jansen. Your personal and committed way of leadership makes the CTI a fruitful and pleasant place to work. I am glad that within the CTI, the whole range of research is nurtured, from very basic to applied, and that there is such an important place for mathematical work. The CTI is enthusiastically supported by Anne Marie Tazelaar, Saskia ter Braak and Anne van Aalst. Anne Marie, your support is worth its weight in gold. Not only for the organization of this day, but also in the hustle and bustle of normal times. It is such a pleasure to work with you!

The fact that we can be so proud of our education, we owe in large part to the tremendous drive of Kristin Denzer and Edward Knol. Many thanks also to all the people with whom I have been working for many years, especially Debbie van Baarle, Louis Bont, Leo Koenderman, Nienke Vrisekoop, Monique Nijhuis, Anne Wensing, Ad Koets and Lars Ravesloot, and of course to all the other members of the CTI. A special word of thanks to Femke van Wijk, my inspiring roommate, who always knows how to encourage me, when things are going well and when the going gets tough. Not only professionally but also personally it is always inspiring to talk with you.

My close friends and family. Carla and Joris, Can and Ludo, Hans Jaap and Sigrid, and Gert Jan, I am so lucky to have such close friendships and to always feel so much at home with you. My sister Ine and brother Lex and their partners Wim and Barbara and their children and grandchildren. I cherish our close relationship. I dedicate the title of my inaugural lecture to my father who was convinced at heart of the power of simplicity. I think I owe my love of simplicity to him. I am grateful to both my parents for the loving basis they gave me. I am so glad I grew up in a family in which content, friendship and caring for others were far more important than your looks, money or status. My dear mother-in-law Betty who, along with her husband John who is sadly no longer with us, has always been giving me such a warm second home in England. I am so lucky to have you all around me.

And finally, my own family. I am so happy to have you. Dear Hanna, I am proud of your independence, your creativity and how you enjoy life. Dear Ellen, I enjoy your empathy, and how you fill our home with beautiful piano music and African dance. Dear Laura, our young scientist, with whom I can talk for hours and who cares so much about others. I am so happy to see all three of you – each in your own way – develop into loving, independent and critical women. Dear Philip, let me keep it simple: together with you, everything is more beautiful.

I have spoken.

José Borghans, together with Kiki Tesselaar, leads an interdisciplinary research group focused on the quantification of leukocyte dynamics in health and disease. José was originally trained as a mathematical immunologist at Utrecht University and obtained her PhD in 2000 on studies of diversity in the immune system. After her PhD, she worked in various immunology laboratories, aiming to bridge the gap between laboratory experiments and mathematical modelling. She worked as a postdoc at the *Institut Pasteur* in Paris and then, with a Veni grant from the Dutch Research Council (NWO), at Sanquin Research in Amsterdam, where she studied lymphocyte dynamics in HIV infection.

Foto: Ed van Rijswijk

José has been working at the University Medical Centre Utrecht since 2004, where she was appointed Associate Professor in 2009. With a Vidi grant from NWO, she started the interdisciplinary Leukocyte Dynamics Group in 2009, in which experimental immunologists and mathematicians work together on a daily basis. Thanks to a recent Vici grant (NWO), the group is currently investigating the dynamics of leukocytes in tissues. In 2021, José was appointed Professor of Quantitative Immunology. She also leads the Computational Immunology Core of the Centre for Translational Immunology, which brings together all researchers who use mathematics or bioinformatics to study the immune system.

Together with her husband Philip Davies, José has three daughters: Hanna, Ellen and Laura.

N.B.: This summary gives a distorted version of reality, as it only lists the successes and not the many failures on the path towards them.

The inaugural lecture (in Dutch) can be viewed back at: https://youtu.be/bwHb9mccABY

All illustrations belonging to this inaugural lecture were created by Joukje Willemsen.

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