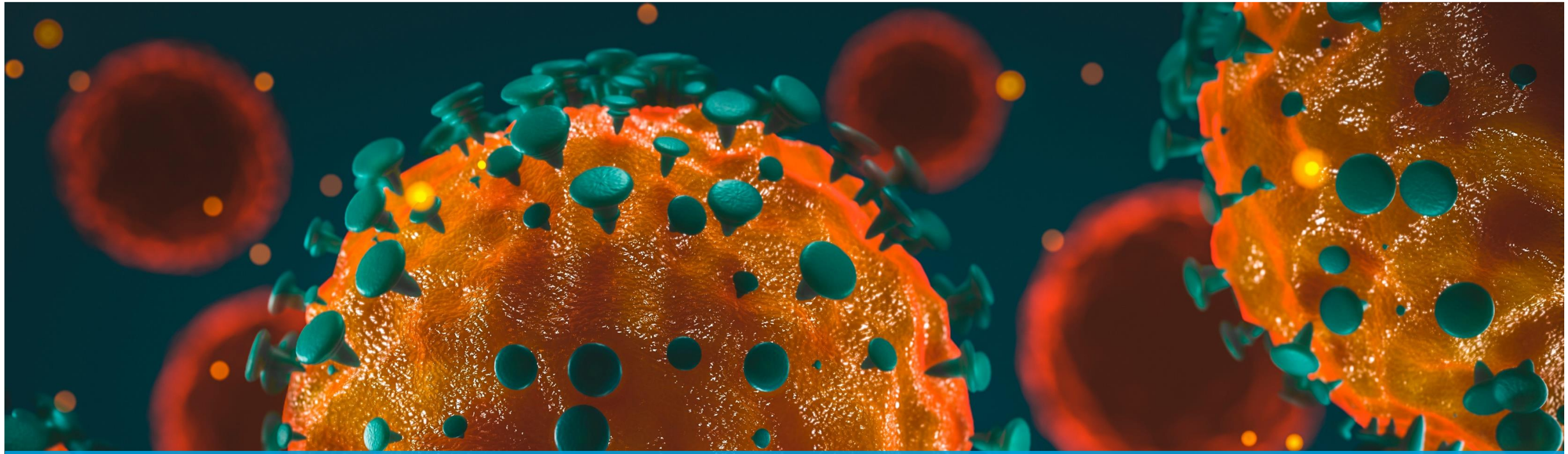

Janssen Perspective on Clinical and Non-clinical Aspects of VITT/TTS MoA

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Pictured: a representation of a coronavirus

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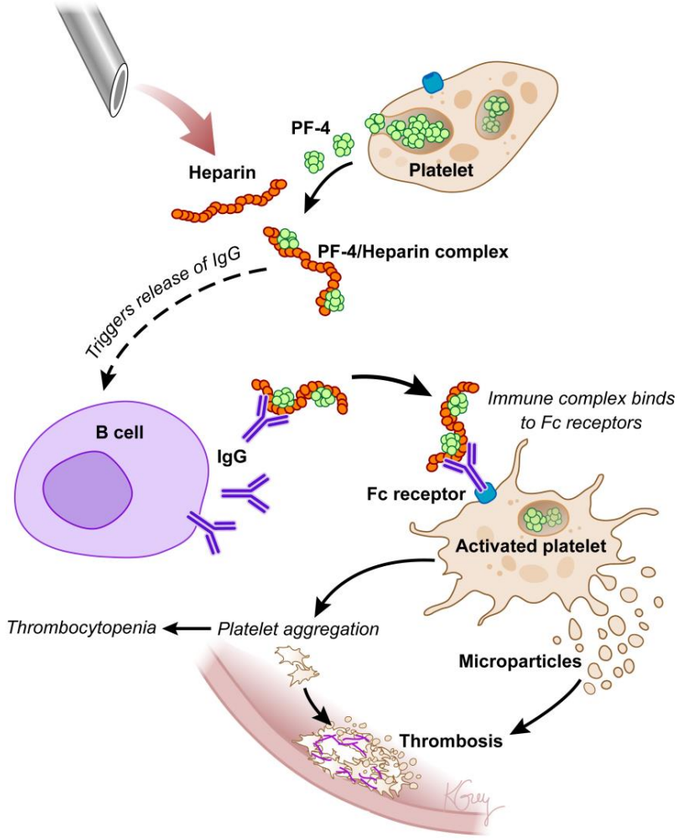
Summary of studies to evaluate pathogenesis of VITT/TTS

Executive Summary:

- Investigation of VITT/TTS pathogenesis and identification of risk factors is high priority in Janssen
- Pathogenic mechanism not yet identified
- Some original hypotheses likely to be rejected and new ones being developed based on emerging data

Original hypothesis: VITT considered 'HIT-like’

Heparin induced Thrombosis with Thrombocytopenia (HIT) (1)



Pathogenesis of HIT

- Binding of PF4 to high molecular weight heparin exposes neo-epitope on PF4
- Antibodies against heparin/PF4 neo-epitope are formed
- Together with heparin/PF4 complexes antibodies activate platelets via their FcyRIIa
- Triggering thromboembolic events and thrombocytopenia

Vaccine Induced Thrombosis with Thrombocytopenia (VITT) = vaccine-induced TTS

Binding of VITT-associated PF4-antibodies to PF4 and platelet activation are both heparin-independent(2)

1. Roberts M.K, et al. (2018). Heparin-induced Thrombocytopenia. The Journal for Nurse Practitioners.
2. Huynh A, et al (2021) Antibody epitopes in vaccine-induced immune thrombotic thrombocytopaenia. Nature

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Mechanistic studies explore multiple factors that could lead to VITT

A. Possible Host and Cofactors

- Genetic polymorphism: e.g. HLA, FcγRIIa and more
- Clinical background: chronic infection, tissue injury
- Pre-existing immune responses to e.g. PF4, Spike, Ad26 or others
- Pre-existing hypercoagulable state
- Coronavirus infection (SARS-CoV-2 or other Coronaviruses)

B. Possible Vaccine factors (or combination thereof)

- Ad26 vector
- Spike protein
- Inflammatory signature
- Expression profile/biodistribution (including accidental IV vaccine administration)
- Host Cell (HC)-Protein/HC-DNA
- Process- and product-related impurities/excipients

Host and Cofactors:

- Probably define the rarity of VITT
- Challenging to identify, since access to VITT cases and appropriate samples is required but challenging

VITT

- In a small number of vaccinees
- Multifactorial/multistep pathogenesis

Earlier hypotheses on underlying cause of VITT that may likely be rejected

Based on data from Janssen and others

Hypothesis 1: Negatively charged Adeno-vector could mimic role of heparin by binding to PF4[(1)]

- Baker et al.[(2)] : Adeno-vectors and wild-type Ad26 can bind to PF4 in vitro
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1. Greinacher A et al, Insights in ChAdOx1 nCoV-19 vaccine-induced immune thrombotic thrombocytopenia. Blood, 2021a; 2. Baker AT et al, ChAdOx1 interacts with CAR and PF4 with implications for thrombosis with thrombocytopenia syndrome. Science Advances, 2021. 3. Michalik S, et al, Comparative analysis of ChAdOx1 nCoV-19 and Ad26.COV2.S SARS-CoV-2 vector vaccines. Haematologica, 2022. 4. Kowarz E et al. Vaccine-induced COVID-19 mimicry syndrome. ELife, 2022. 5. Krutzke L et al, Process-related impurities in the ChAdOx1 nCov-19 vaccine. Research Square, 2021. 6. Nicolai et al, Thrombocytopenia and splenic platelet directed immune responses after intravenous ChAdOx1 nCov-19 BioRxiv, 2021.

Hypothesis 1: Negatively charged Adeno-vector mimics role of heparin by binding to PF4

Janssen data: Binding of Ad26.COV2.S to PF4 based on experimental artefact (Biacore)

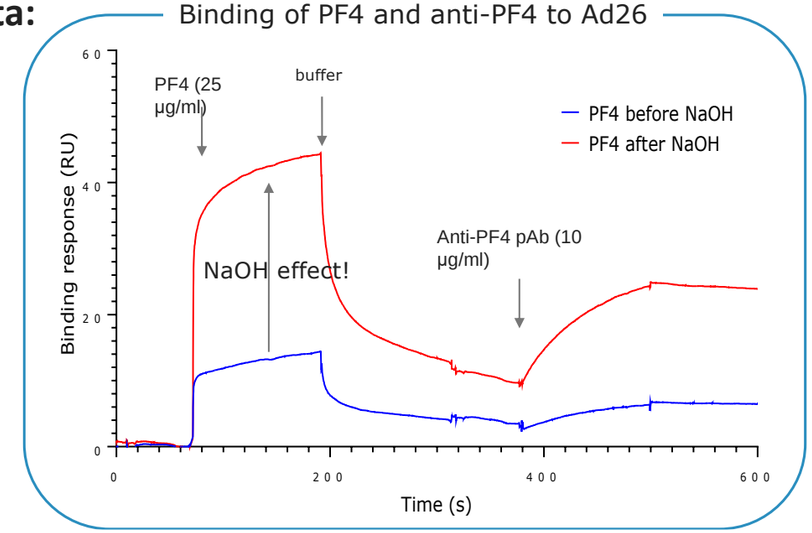
Background:

- It was hypothesized that anti-PF4 antibodies could be induced by a complex of PF4 with viral vaccine vectors
- **Baker et al.[1] observed PF4 binding to ChAdOx1 nCoV-19 vaccine, Ad5 and wild type Ad26**

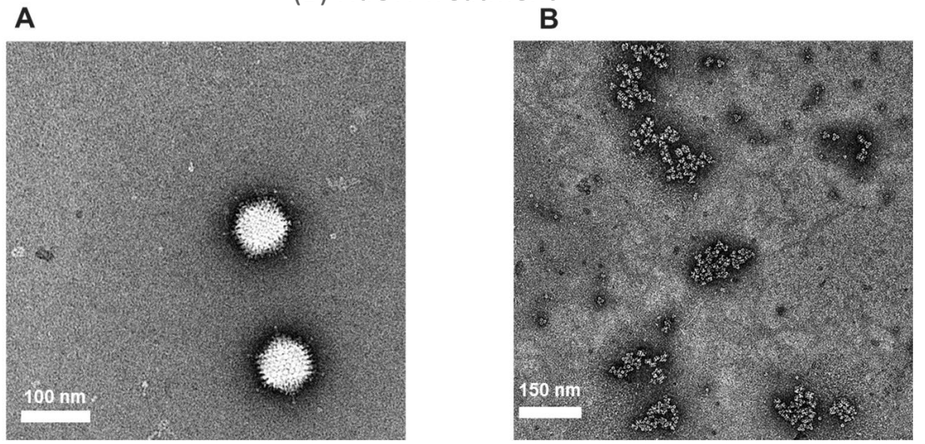
Aim: To assess the interaction between PF4 and Ad26.COV2.S

The same Biacore surface plasmon resonance (SPR) experimental set up as described by Baker et al. was used besides other conditions

Janssen data:



Electron Microscopy of Ad26.COV2.S Before (A) and After (B) NaOH Treatment



- **Very low binding of PF4 observed in first run (blue line)**, likely induced by method (uncontrolled immobilization and long exposure to acetate buffer)
- Chip-regeneration with **NaOH treatment results in strong binding of PF4 (red line)** and anti-dsDNA (data not shown)
- Electron microscopy analysis: NaOH treatment severely damages Ad26

➢ **Janssen data suggest that PF4 is binding to genomic Adeno-DNA or newly exposed sites on viral particle after NaOH treatment**

1. Baker et al. ChAdOx1 interacts with CAR and PF4 with implications for thrombosis with thrombocytopenia syndrome. Science Advances. 2021;7(49).

Hypothesis 1: Negatively charged Adeno-vector mimics role of heparin by binding to PF4

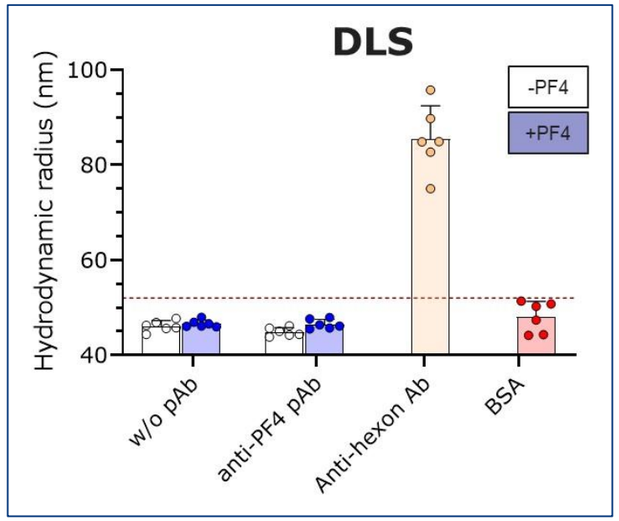
Janssen data: No binding of Ad26.COV2.S to PF4 demonstrated using DLS and Octet

Background:

- It was hypothesized that anti-PF4 antibodies could be induced by a complex of PF4 with viral vaccine vectors
- Baker et al.[1] observed PF4 binding to ChAdOx1 nCoV-19 vaccine, Ad5 and wild type Ad26
- **Michalik et al[2]: No complex formation of PF4 with Ad26.COV2.S (DLS)**

Aim: To assess the interaction between PF4 and Ad26.COV2.S using alternative methods

Janssen data:



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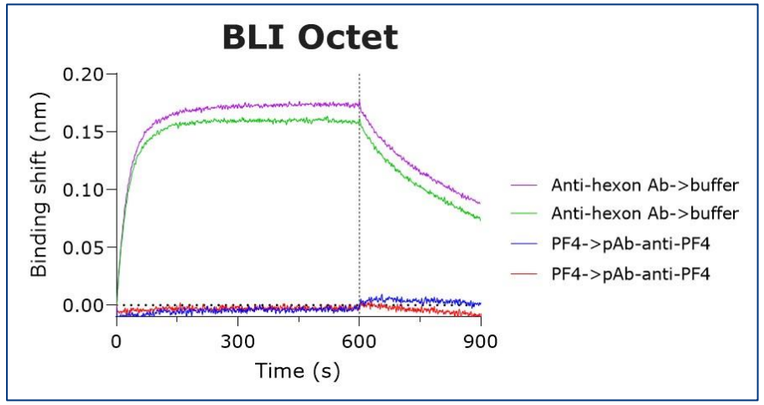

Data summary:

		Complex with PF4?
	Method	Ad26.COV2.S
Baker[1]	SPR Biacore	ND (wtAd26 Yes)
Janssen[3]	SPR Biacore	Very low (**only after NaOH regeneration** Yes)
Michalik[2]	DLS	No
Janssen[3]	DLS	No
Janssen[3]	Octet	No

- **No binding of Ad26.COV2.S and PF4 demonstrated using Octet and DLS**
- **Ad26.COV2.S binding as demonstrated with Biacore is likely due to an experimental artefact damaging the Ad26**

1. Baker AT et al, (2021) ChAdOx1 interacts with CAR and PF4 with implications for thrombosis with thrombocytopenia syndrome. Science Advances.
2. Michalik S et al, (2022) Comparative analysis of ChAdOx1 nCoV-19 and Ad26.COV2.S SARS-CoV-2 vector vaccines. Haematologica.
3. Manuscript in preparation

ND: not determined

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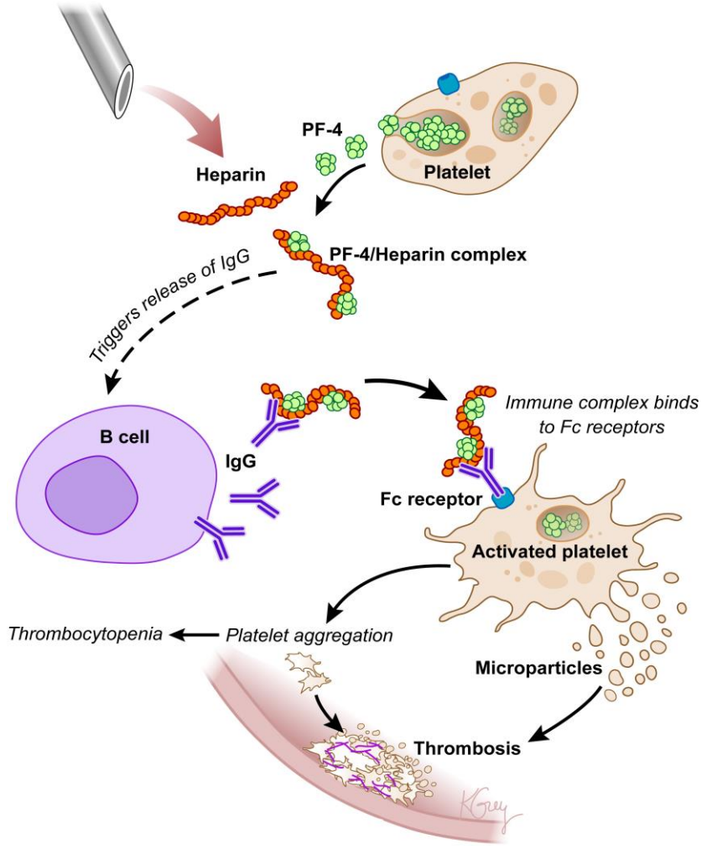
VITT considered 'HIT-like' but underlying mechanism is distinctively different

HIT: Heparin induced Thrombosis with Thrombocytopenia (1)



VITT: Vaccine Induced Thrombosis with Thrombocytopenia

- Binding of VITT-associated PF4-antibodies to PF4 and platelet activation are both **heparin-independent**(2) *
- **Epitope for VITT-associated PF4-antibodies** overlaps with heparin binding site on PF4 and is **different from epitope for HIT-associated PF4-antibodies***
- PF4 antibodies in HIT are polyclonal, PF4 antibodies in autoimmune HIT as well as VITT/TTS are **monoclonal/oligoclonal**(3)§
- VITT antibodies comprised of **highly stereotyped clonotype**, with identical CDR3 length and homologous sequences(4)*

Hypothesis that Ad-based vaccine component in VITT mimics role of heparin in HIT likely not valid

* Data reported for ChAdOx1 nCoV-19
§ Data reported for ChAdOx1 nCoV-19 and Ad26.COV2.S

1. Roberts M.K, et al. (2018). Heparin-induced Thrombocytopenia. The Journal for Nurse Practitioners. 2. Huynh A, et al (2021) Antibody epitopes in vaccine-induced immune thrombotic thrombocytopaenia. Nature. 3. Singh et al, (2021) Anti-PF4 VITT antibodies are oligoclonal and variably inhibited by heparin. medRxiv, 4. Wang et al (2022) Vaccine-induced immune thrombotic thrombocytopenia (VITT) is mediated by a sterotyped clonotypic antibody . medRxiv

Towards alternative hypotheses for VITT

VITT after Ad-based COVID-19 vaccines but also in severe COVID-19 patients and sometimes after mRNA vaccines

➢ **VITT due to a combination of Spike protein - inflammatory milieu - predisposition of the patient?**

Role of Spike?

- Severe COVID-19 cases with 'HIT' like syndrome identified even before VITT was known(1)
- Incidence of suspected "HIT" cases with high anti-PF4 positivity in severe COVID-19 cases 10-fold greater than in non-COVID cases in the ICU (8% vs 0.89%)(2)
- Increased frequency of CVST in severe COVID-19 cases requiring ICU care(3)
  - ➢ Collaborations initiated to further characterize PF4 antibodies in sera from these COVID-19 patients to identify potential similarities with VITT PF4 antibodies
- How is different "delivery" of Spike by SARS-CoV-2, Adenoviruses or mRNAs influencing its role?
- Potential involvement of Spike via binding ACE2
  - on platelets, inducing platelet activation and release of PF4(4)
  - on endothelial cells, reducing anti-inflammatory signal, increasing inflammation(5, 6)

1. Nazy I et al. Platelet-activating immune complexes identified in critically ill COVID-19 patients suspected of heparin-induced thrombocytopenia. J Thromb Haemost. 2021 May;19(5):1342-1347. Epub 2021 Mar 14
2. Daviet et al. Heparin-induced thrombocytopenia in severe COVID-19. Circulation, 2020
3. Bikdeli et al. Cerebral Venous Sinus Thrombosis in the US population, After Adenovirus-Based SARS-CoV-2 Vaccination and After COVID-19. J AmColl Cardiol, 2021.
4. Zhang et al. SARS-CoV-2 binds platelet ACE2 to enhance thrombosis in COVID-19. J Hematology & Oncology, 2020
5. Lei et al. SARS-CoV-2 Spike Protein Impairs Endothelial Function via Downregulation of ACE 2. Circulation Research, 2021
6. Perico et al. SARS-CoV-2 Spike Protein 1 Activates Microvascular Endothelial Cells and Complement System Leading to Platelet Aggregation. Front Immunology, 2022

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➢ **VITT due to a combination of Spike protein - inflammatory milieu - predisposition of the patient?**

Role of inflammatory milieu and Ad26 as a cofactor?

- Ad26.COV2.S gives strong pro-inflammatory response
- Inflammatory response seems to be less strong post dose 2, in line with lower VITT incidence; Potentially related to
  - presence of Adeno vector-neutralizing antibodies and/or
  - presence of Spike-neutralizing antibodies or
  - different quality of inflammatory response during a memory response

Towards alternative hypotheses for VITT

VITT after Ad-based COVID-19 vaccines but also in severe COVID-19 patients and sometimes after mRNA vaccines

- **VITT due to a combination of Spike protein - inflammatory milieu - predisposition of the patient?**

Predisposition of the patient?

- Host genetics - genome-wide analysis including e.g. polymorphisms of ACE2, Fc receptors, PF4, etc.
- Pre-existing PF4-specific autoimmune B cells? Is a component of the vaccine responsible for a (re-)activation? Which component? How were those B cells triggered in the first place?
  - Sequences of VITT PF4 antibodies recently identified[1]
    - Antibodies can potentially be generated and used to investigate possible mimicry between PF4 and vaccine components

- We will continue to monitor the developments in the field, interact with experts and investigate the pathogenesis of VITT/TTS
- We will continue our monitoring of VITT

1. Wang et al (2022) Vaccine-induced immune thrombotic thrombocytopenia (VITT) is mediated by a stereotyped clonotypic antibody . medRxiv

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Thank you

Skin cells at 20x magnification

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