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P227

Complement Activation and Cell Adhesion by Hydroxylated Model Surfaces

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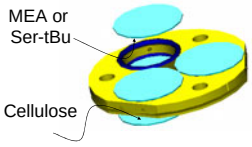
Background

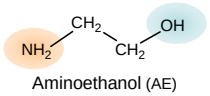
Leukocyte activation during extracorporeal blood purification mainly can be attributed to complement activation on surfaces bearing hydroxyl-groups.
The responsible adhesion ligands - integrins or complement receptors - are not yet determined.

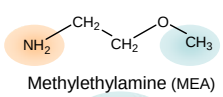
Method

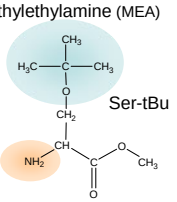
- Selected molecules containing OH-groups (AE, Ser-OH, reference: cellulose) or corresponding ethers (MEA, Ser-tBu)
- Immobilization onto polyethylene maleic acid copolymer substrates.
- Hemocompatibility in vitro assay using whole human blood
- Hemocompatibility parameters:
  - activation of blood coagulation (TAT)
  - complement activation on surface (C3b) and in plasma (C5a)
  - leukocyte activation (expression of activation marker integrin CD11b), leukocyte adhesion
  - fibrin /fibrinogen deposition on the surfaces

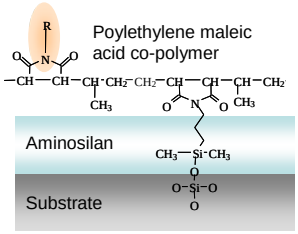


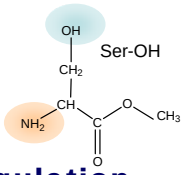
Incubation chamber with top and bottom samples.
Combination of two equal or two different surfaces on top / bottom for *in vitro* assay.







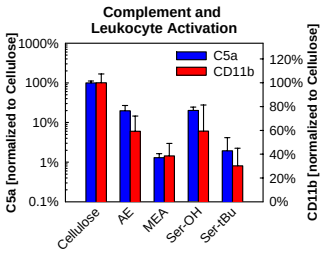


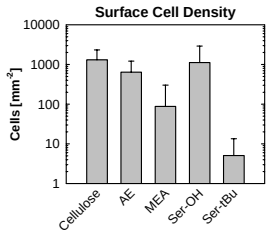


Results

Immune Response

Top / Bottom equal

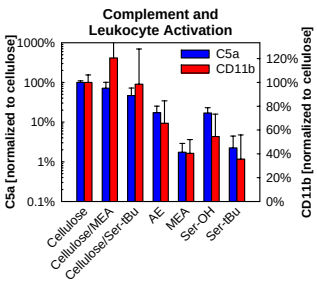


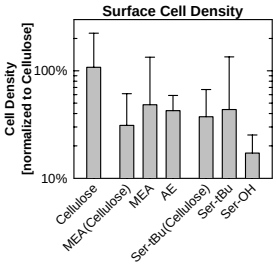


OH group bearing surfaces induce more complement and leukocyte activation than the corresponding ethers.
The number of surface adherent cells corresponds with the activation of the cells.

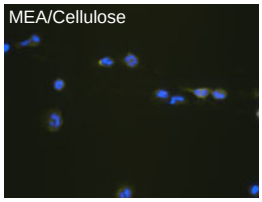
Immune Response

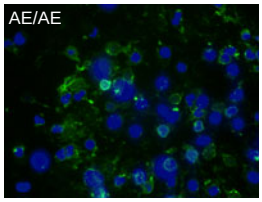
Top / Bottom different





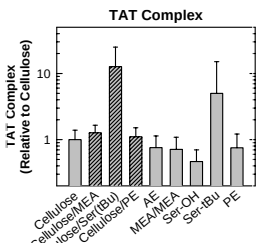
Co-Incubation of blood with an activating and a nonactivating surface:
- Complement and cell activation are predominated by the activating surface (cellulose)
- The cell adherence is mainly independent of their activation.

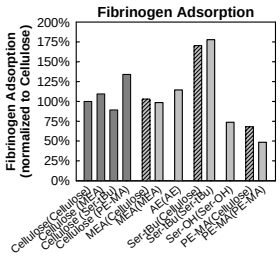




Fluorescent microphotographs of adherent cells
blue: Cell nuclei; green: Cd11b

Coagulation





- Coagulation and fibrin(ogen) deposition was mainly independent of hydroxyl or ether presenting surface.
- Ser-tBu caused highest coagulation activation

Conclusion

Activated leukocytes preferentially adhere to surfaces via their complement receptor.

Integrin-type receptor binding to fibrin(ogen) plays a minor role.

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