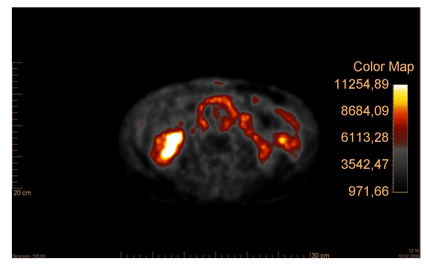
Maximum peak wall stress is associated to increased glucose metabolism in aortic aneurysm wall assessed by FDG-PET-CT



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Background

Interactions of biomechanical forces and subsequent tissue reactions in the wall of aortic aneurysm (AAA) are postulated frequently for AA pathogenesis and rupture but so far have never been demonstrated to be existent in vivo. Meantime, individually acting forces can be calculated precisely by computational finite element analyses (FEA) and inflammatory metabolic activity of AA wall can be visualized in vivo by 18F-fluorodeoxyglucose positron emission tomography/ angiography CT (FDG-PET/aCT)[1] **(Fig. 1a)**. For better insights in stress-tissue interactions in AAA in vivo we therefore analyzed the correlation of computational biomechanics with metabolic activity.

Patients and Methods

FDG-PET/CT data sets of 6 AA patients with notably increased FDG uptake in AA wall were studied. For further analyses detailed 3-D geometry of each AAA including thrombus was reconstructed from angio-CT in 3mm-slices. Later sophisticated non-linear-FEA-simulations considering thrombus, hyperelastic material behavior and pre-stress state of AA geometry were performed as described in [2]. Peak wall stresses (PWS), strains and their distributions were obtained and visualized as shown in **Fig.3b** and **4b.**

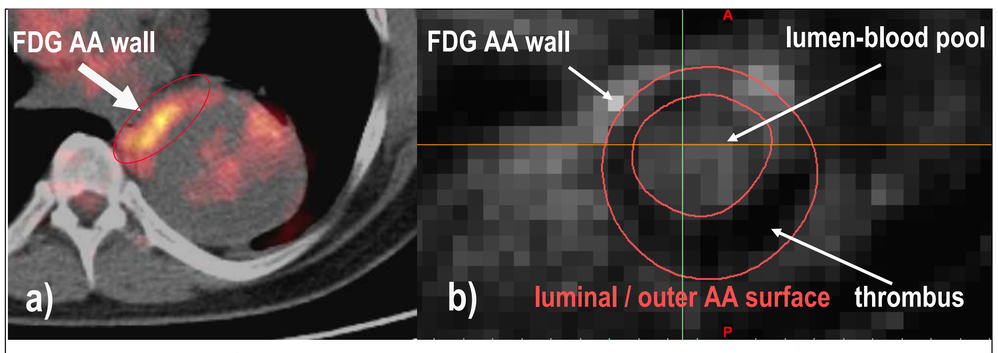


Fig.1 TAA 61J ♂ **a) conventional FDG-PET/CT** transversal fusion (ROI: SUV_{max}: 4.8 SUV_{mean}: 3.2)
b) multiplanar alignement of segmented and reconstructed AA geometry with outer AA wall, thrombus and lumen to FDG-PET cloud signal data set.

Further FDG-PET clouds were anatomically fitted **(Fig.1)**, reduced to FDG activity in AA wall, mathematically processed (Gauß-filter, 85% Rank-order-filter), superimposed to the 3-D AA geometry **(Fig.2)**. Moreover, the maximum standard uptake values (SUV_{max}) of metabolic activity were acquired by analyses of conventional fusion images. Subsequently PWS and strain values and their local distributions were correlated to corresponding FDG-uptake in AAA wall.

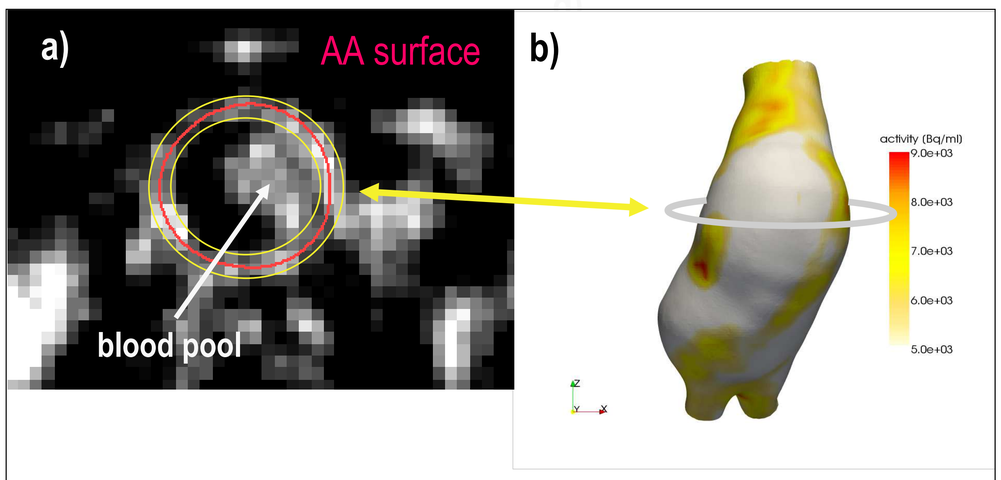
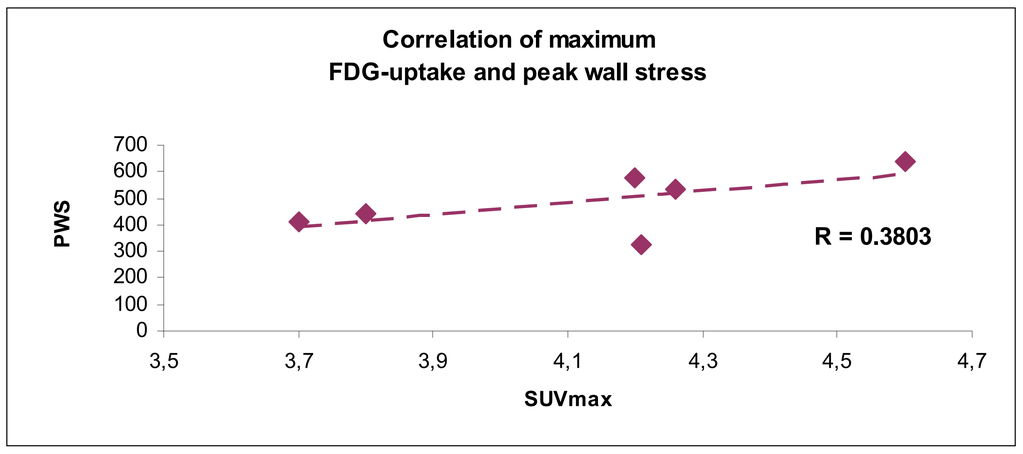


Fig.2: AAA 75J ♂ **a)** shema: mathematical restriction (Gauß / 85% Rank order filter) of PET information to FDG signal according to AA wall using surface geometry obtained from CT. **b)** resulting FDG signal intensity (Bq/ml) from a) mapped on 3-D AAA surface reconstruction of AA wall

Results

SUV_{max} of AAA wall varied from 3.7-4.6 (mean 4.1±0.33) and computational PWS and strains ranged from 29.0 N/cm² to 64.0 N/cm² (mean 48.3±12.7 N/cm²) and from 0.20 to 0.26 (mean 0.236±0.021) respectively.



Maximum PWS levels showed a trend to correlate with SUV_{max} (R= 0.38). In all but one patient, areas with increased FDG uptake showed well visible correlation to areas with increased computational PWS and strains while areas with low PWS and strains showed no or negligible metabolic activity.

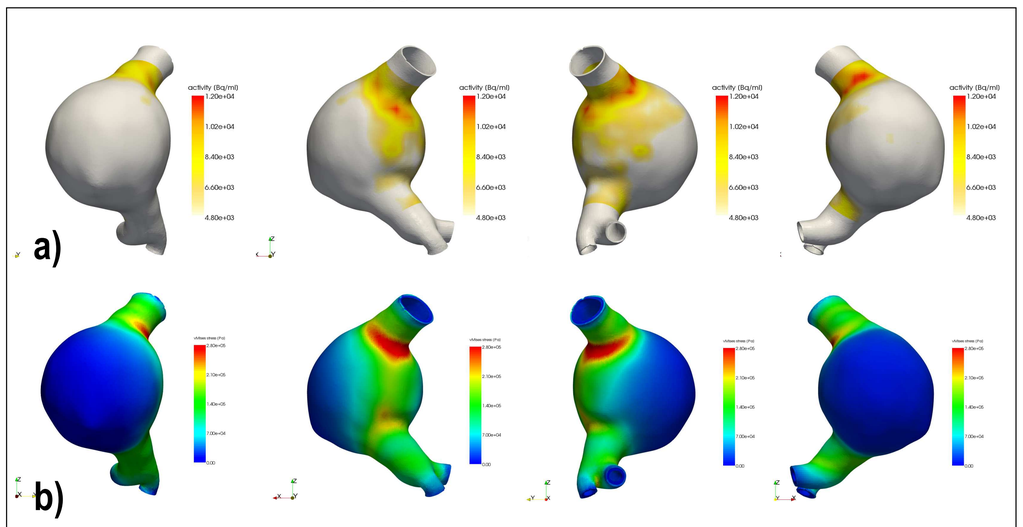


Fig. 3 Abdominal AA in anterior, left lateral, posterior and right lateral view with high spatial correlation of **a)** regional metabolic activity (FDG-uptake; Bq/ml, Gauß-filter) and **b)** computed peak wall stress distribution in AA wall (von Mises stress; kPa); Colors indicate FDG-uptake and wall stress levels

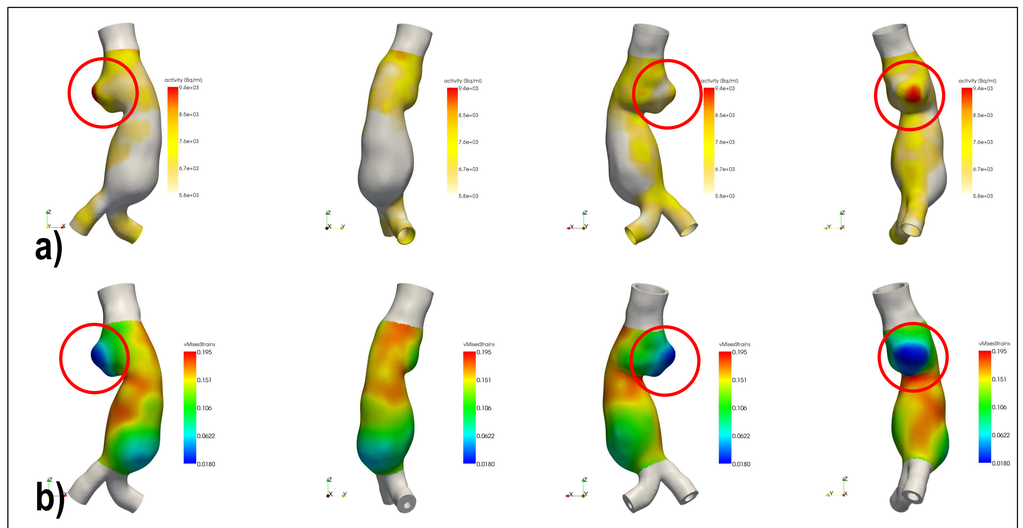


Fig. 4 Abdominal AA in anterior, left lateral, posterior and right lateral view with correlation of **a)** regional metabolic activity (FDG-uptake; Bq/ml, Gauß-filter) and **b)** peak wall stress distribution in AA wall (von Mises stress; kPa);
○ Stress independent metabolic activity

Conclusion

Our findings demonstrate and visualize the complex interactions between AA biomechanics and tissue reactions in vivo for the first time. Results indicate that biomechanical forces may be causative for regionally increased FDG uptake and therefore inflammatory activity in AAA wall. Thereby, inflammatory reaction seems to correlate quantitatively to level of PWS. Our results strongly support the often postulated but never proven hypothesis that biomechanical forces are highly relevant for pathogenesis and formation of AAA. However, stress independent metabolic activity was found also indicating additional autochthon inflammatory biological activity. Larger studies will be performed to confirm these results.

References:

[1] Reeps C., Essler M, Pelisek J, Seidl S, Eckstein HH, Krause BJ. Increased 18F-fluorodeoxyglucose uptake in abdominal aortic aneurysms in positron emission/computed tomography is associated with inflammation, aortic wall instability, and acute symptoms. *J Vasc Surg. 2008* ;48(2):417-23;
[2] Gee MW, Reeps C, Eckstein HH, Wall WA. Prestressing in finite deformation abdominal aortic aneurysm simulation.J Biomech. 2009; 42(11):1732-9.

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