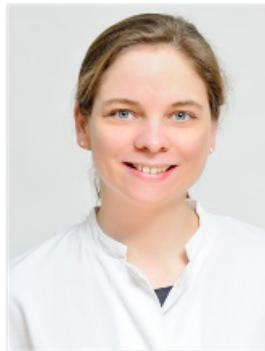


Prof. Sibylle von Vietinghoff, MD

Internal Medicine I



Rheinische Friedrich-Wilhelms-University Bonn
Head of Nephrology Section

E-Mail: Sibylle.von_Viectinghoff@ukbonn.de

Research Expertise

In recent years, my group has addressed mechanisms of vascular inflammation in atherosclerosis and renal impairment, inflammatory mechanisms in peritoneal dialysis and in the kidney. Human cohort and sample (specifically renal biopsies, urine and peritoneal dialysate and tissue) collection have been central in both long-term (atherosclerosis, peritoneal dialysis) and acute clinical (EHEC-HUS) projects.

Education / Training

Consultant Immunologist (Deutsche Gesellschaft für Immunologie), 2016

Habilitation Internal Medicine, 2014

Board certification Nephrology and Internal Medicine, 2013

University of Würzburg, Medical Thesis, Institute of Pharmacology, 2003

Appointments / Positions Held

since 2021

Head, Nephrology within First Medical Clinic, University Hospital Bonn, Germany

2015 - 2020

Consultant physician, Nephrology and Hypertension section, MHH, Hannover, Germany

2010 - 2015

Nephrology fellow and nephrologist, Nephrology and Hypertension Section, MHH, Hannover, Germany

2008 - 2010

Postdoctoral Fellow (DFG-funded), Division of Inflammation Biology, La Jolla Institute for Immunology, CA, USA

2003 - 2008

Nephrology intern and fellow, Clinic for Nephrology and Hypertension, Charité Campus, Berlin, Germany

10 Most Relevant Publications for Prof. Sibylle von Vietinghoff

1. Helmke A, Hüsing AM, Gaedcke S, Brauns N, Balzer MS, Reinhardt M, Hiss M, Shushakova N, de Luca D, Prinz I, Haller H, **von Vietinghoff S**. Peritoneal dialysate-range hypertonic glucose promotes T cell IL-17 production that induces mesothelial inflammation. *Eur J Immunol*. 2020 Sep 14. doi: 10.1002/eji.202048733.
2. Helmke A*, Nordlohne J*, Balzer MS, Dong L, Rong S, Hiss M, Shushakova N, Haller H, **von Vietinghoff, S.** CX-3CL1-CX3CR1 interaction mediates macrophage-mesothelial crosstalk that promotes peritoneal fibrosis in vivo. *Kidney international* 2019 Jun;95(6):1405-1417.
3. Casper J*, Schmitz J*, Bräsen JH, Khalifa A, Schmidt BMW, Einecke G, Haller H, **von Vietinghoff S**. Renal transplant recipients receiving loop diuretic therapy have increased urinary tract infection rate and altered medullary macrophage polarization marker expression. *Kidney International* 2018 Nov;94(5):993-1001. *shared first authorship
4. Bräsen JH, Khalifa A, Schmitz J, Dai W, Einecke G, Schwarz A, Hallensleben M, Schmidt BMW, Kreipe HH, Haller H, **von Vietinghoff S**. Macrophage density in early surveillance biopsies predicts future renal transplant function. *Kidney international* 2017 Aug;92(2):479-489.
5. Dong L*, Nordlohne J*, Ge S, Hertel B, Melk A, Rong S, Haller H, **von Vietinghoff S**. T Cell CX3CR1 Mediates Excess Atherosclerotic Inflammation in Renal Impairment. *J Am Soc Nephrol*. 2016 Jun;27(6):1753-64. *shared first authorship
6. Ge S, Hertel B, Koltsova EK, Sørensen-Zender I, Kielstein JT, Ley K, Haller H, **von Vietinghoff S**. Increased atherosclerotic lesion formation and vascular leukocyte accumulation in renal impairment are mediated by Interleukin 17A. *Circ Res*. 2013 Sep 27;113(8):965-74.
7. Koltsova EK, Garcia Z, Chodaczek G, Landau M, McArdele S, Scott SR, **von Vietinghoff S**, Galkina E, Miller YI, Acton ST, Ley K. Dynamic T cell-APC interactions sustain chronic inflammation in atherosclerosis. *J Clin Invest*. 2012 Sep 4;122(9):3114-26.
8. **von Vietinghoff S**, Koltsova EK, Mestas J, Diehl CJ, Witztum JL, Ley K. Mycophenolate mofetil decreases atherosclerotic lesion size by depression of aortic T lymphocyte and IL-17-mediated macrophage accumulation. *J Am Coll Cardiology* 2011, May; 57: 2194-204.
9. **von Vietinghoff S**, Ley K. Interleukin 17A controls Interleukin 17F production and maintains blood neutrophil counts in mice. *J Immunol*. 2009 Jul 15;183(2):865-73.
10. **von Vietinghoff S**, Tünemann G, Eulenberg C, Wellner M, Cardoso MC, Luft FC, Kettritz R. NB1 mediates surface expression of the ANCA antigen **proteinase 3** on human neutrophils. *Blood*, 2007 May 15;109(10):4487-93.