

Soline Bourgeois

Independent Scientific Advisory & Consultancy

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Summary

With more than 30 years of internationally recognized expertise in renal physiology and translational nephrology, I provide independent scientific advisory and consultancy services to Academia, biotechnology and pharmaceutical companies developing therapies for kidney, cardiovascular and metabolic diseases. My research on genetic defects in rare monogenic kidney diseases and the adverse effects of acidosis in chronic kidney disease and aging has provided me with deep field knowledge and a strong foundation for more clinical role. I am eager to contribute to clinical development efforts aimed at advancing therapies and solutions for kidney-related disorder

Key Competencies

*Translational Disease
Renal & Cardiorenal Physiology Expertise
Scientific Advisory and Consultancy in CVR*

*Molecular Mechanism Studies
Bioanalytical Development for Biologics
Project & Team Management*

Work Experience

Founder | Independent Scientific Advisor, *BTNS| Zürich, Switzerland* **August 2026 - Present**
Bourgeois Translational Nephrology Solutions provides strategic scientific advisory services to pharmaceutical, biotechnology, and academic organizations. Specialized in renal physiology, nephrology, translational medicine, and preclinical research, with expertise in study design, target validation, biomarker strategies, scientific due diligence, data interpretation, and scientific communication. Supporting clients in advancing robust, reproducible, and clinically relevant research programs.

Visiting Lecturer, *University Paris Sorbonne| Paris, France* **Nov 2025 - Present**
Teaching Renal Physiology and Physiopathology, MSc Program in Biology and Integrative Physiology
Deliver a Master's course on acid-base homeostasis, focusing on metabolic disorders, renal physiology, and chronic kidney disease.

Senior Clinical Scientist, *University Hospital Zurich (USZ) | Zurich, Switzerland* **May 2025 - Nov 2025**
Exploring the Link Between Hypokalemia and Primary Aldosteronism in hypertensive Patients
My clinical research aims to enhance diagnostic accuracy and clarify hormonal and cardiovascular mechanisms, ultimately improving care pathways in endocrine hypertension.

Full Membership, *Zurich Kidney Center (UZH) | Zurich, Switzerland* **Sep 2023 - Present**
Member of the Zurich-based scientific network for experimental and clinical nephrology, specializing in biochemical profiling and phenotyping of rodent models (<https://www.zurich-kidney.uzh.ch>)

Senior Scientist, *University of Zurich (UZH) | Institut of Physiology | Zurich, Switzerland* **Apr 2013 - Dec 2024**
Led research on rare kidney diseases, metabolic disorders, and aging, resulting in 7 peer-reviewed publications and 3 press reviews. Supervised students and taught physiology to medical students.

Postdoctoral Researcher, *UZH | Institut of Physiology | Zurich, Switzerland* **Apr 2007 - Apr 2013**
Led research investigating renal compensatory and maladaptive responses to a western diet, culminating in 9 peer-reviewed articles and 3 review publications - Supervised and mentored undergraduate, master's, and PhD students throughout the project lifecycle.

Engineer, *University Paris 6 | Paris, France* **Jan 1996 - Apr 2007**
Led research on acid-base metabolism and transport in the renal nephron - 10 peer reviewed publications, 1 review. Developed and taught the microperfusion technique for isolated renal tubules along with microdissection and nanoliter sampling.

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Education

Privatdozentin Renal Physiology and Physiopathology <i>University of Zurich Institut of Physiology Zurich, Switzerland</i>	Jan 2020 - Jan 2021
Doctorat of Phylosophy PhD Renal Physiology - GPA: A <i>University Paris 6 Paris, France</i>	Sep 2001 - Jan 2005
Master Cellular differentiation and Integrative function - GPA: A <i>University Paris 6 Paris, France</i>	Sept 2000 - Jul 2001

Languages

English (Full Proficiency)
French (Native)
German (Working Proficiency)

Core Skills

Transferable Skills

- Leadership
- Critical Thinking
- Scientific communication
- Project and Time Management
- Project solving
- Team Working
- Adaptability
- Creativity
- Data-Driven Decision Making

Hard Skills

- Grant and animal Licence writing
- Scientific expertise in Physiology (spe. in CVR)
- Statistical Analysis of big clinical dataset
- Rodents Phenotyping
- *Ex vivo* live microscopy
- RNAScope, Confocal and light microscopy
- Molecular and cellular Biology
- Cell culture techniques
- Prism, SPSS, ImageJ, Endnote, MS Office R (notions)

Certifications, Recent Grants and Awards

• Medical devices regulations (BNF, National Qualification program, online course)	2025	
• Good Clinical Practice II (Sponsor-Investigator) (TRREE and DCR certifications)	2025	
• Felasa level B (Université Paris 5)		2006
• Grant from the “Jubiläumsstiftung Swisslife”	2022	
• Best Poster presentation at the Annual meeting of the German society of physiology (DPG)		2021

Personnal information

- Birthdate 31.05.1973 /Swiss and French Nationalities /Married /2 children (2014 - 2016)
- Soprano singer in vocal ensembles (Orléans, Paris, Basel) and large choirs (Gemischter Chor, Tonhalle, Zürich). Participation in performances and festivals has strengthened my creativity, adaptability. Performing in ensembles has honed my flexibility and teamwork skills.
- Mountain hiking and cross-country ski: Promotes perseverance, determination and the ability of resilience and risk management.

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Publications list

First authorship, # Last authorship

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Peer reviewed publications selection

- # Auwerx H, Busch-Dohr M, Li X, Wagner CA, **Bourgeois S**. WDR72 Is Required for Urinary Acidification and Normal H⁺-ATPase Activity in Intercalated Cells in Mice. *Acta Physiol (Oxf)*. 2026 Mar;242(3):e70165. doi: 10.1111/apha.70165. *Biallelic inactivating variants in WDR72 are associated with renal and enamel defects in Human, yet the precise function of WDR72 in the kidney remained undefined. We demonstrate that WDR72 is highly expressed at the apical membrane of intercalated cells, where in type A, it regulates urinary acidification through modulation of H⁺-ATPase activity. In Wdr72 knockout mice, loss of WDR72 impairs H⁺-ATPase function and induces a pronounced, female-specific metabolic acidosis under dietary acid challenge. These findings identify WDR72 as a critical regulator of renal acid-base homeostasis and reveal a novel mechanism underlying sex-specific differences in renal acid handling.*
- # **Bourgeois S**, Kovacikova J, Bugarski M, Bettoni C, Gehring N, Hall A, Wagner CA. The B1 H⁺-ATPase (Atp6v1b1) Subunit in Non-Type A Intercalated Cells is Required for Driving Pendrin Activity and Renal Defense Against Alkalosis. *J Am Soc Nephrol*. 2024 Jan 1;35(1):7-21. doi: 10.1681/ASN.0000000000000259.
 - *This study reveals that the B1 subunit of the vacuolar H⁺-ATPase (Atp6v1b1), previously linked to renal type A intercalated cells and the rare human disorder distal renal tubular acidosis, also plays a vital role in non-type A intercalated cells. Its loss impairs pendrin function, leading to reduced bicarbonate excretion and compromising the kidney's ability to counteract metabolic alkalosis.*
- Stettner P, **Bourgeois S**, Marsching C, Traykova-Brauch M, Porubsky S, Nordström V, Hopf C, Koesters R, Sandhoff R, Wiegandt H, Wagner CA, Gröne HJ, Jennemann R. Sulfatides are required for renal adaptation to chronic metabolic acidosis. *Proc Natl Acad Sci U S A*. 2013 Jun 11;110(24):9998-10003. doi: 10.1073/pnas.1217775110.
 - *We introduced the novel concept that sulfatides, a class of glycosphingolipids involved in cell structure, are crucial for the kidney's adaptation to chronic metabolic acidosis. In sulfatide-deficient mice, acid excretion was impaired, acid-base transporter expression was disrupted, and intercalated cells showed structural abnormalities, underscoring the essential role of sulfatides in preserving acid-base balance.*
- # Biver S*, Belge H*, **Bourgeois S***, Van Vooren P, Nowik M, Scohy S, Houillier P, Szpirer J, Szpirer C, Wagner CA, Devuyst O, Marini AM. A role for Rhesus factor Rhcg in renal ammonium excretion and male fertility. *Nature*. 2008 Nov 20;456(7220):339-43. doi: 10.1038/nature07518. (*shared-authorship)
 - *This study identifies the Rhesus factor Rhcg as a crucial ammonia channel, addressing the longstanding question of how ammonia is transported into the urine. Rhcg-deficient mice showed impaired urinary ammonia excretion and decreased male fertility, underscoring its dual function in maintaining acid-base balance and supporting reproductive health. These major findings were discussed in a comment by M. Knepper Nature. 2008 Nov 20;456(7220):336-7. doi: 10.1038/456336a.*

Review Articles selection

- # **Bourgeois S**, Houillier P. State of knowledge on ammonia handling by the kidney. *Pflugers Arch*. 2024 Mar 7. doi: 10.1007/s00424-024-02940-1.
 - *This review provides a comprehensive update on current knowledge of renal ammonia handling. It details the molecular transporters, cellular mechanisms, and physiological significance of ammonia metabolism in the kidney, emphasizing its central role in acid-base homeostasis and highlighting recent advances and remaining gaps in the field.*

Complete Bibliography <https://pubmed.ncbi.nlm.nih.gov/?term=soline+Bourgeois>