

POSTDOCTORAL FELLOW AT THE LETTRE LAI

Montreal Heart Institute

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Research and scientific interests: Human genetics, Single cell OMICS, Heart development, Bioinformatics, Open source software

Experience	
Montreal Heart Institute	Montrea
Postdoc	Aug.2018 - ongoing
Lettre group	
Universite de Sherbrooke	Sherbrook
Professionnel recherche niv. II	Jan. 2019 - ongoin
• GenAP team	
IEB, University of Münster	Münster, German
Student Research assistant	Jan.2012 - Jul.201
Acquisti group	
IEB, University of Münster	Münster, German
Student assistant	Sep.2011 - Dec.201
Acquisti group	
IEB, University of Münster	Münster, German
Student assistant	Mar.2011 - May 201
Bornberg-Bauer group	
Education	
University of Münster / CHU Sainte Justine Research Center	Münster, Germany / Montrea
	Canad
PH.D (DR.RER.NAT) The role of genetic factors in pathogenesis and progression of cardiac malformations"	Apr.2014 - Apr.201
University of Münster	Münster, Germar
MASTER OF SCIENCE (MSC)	Oct.2011 - Feb.201
Functional and genetic characterization of a novel arrhythmic syndrome	
University of Münster	Münster, German
BACHELOR OF SCIENCE (BSC)	Oct.2008 - Sep.201
• Impact of nutrient limitation in insects: Comparative genomics of the pea aphid and the human body louse	
Funding History	
Postdoctoral Training (Canadian citizens and permanent residents) scholarship, Fonds de recherche Québec santé (FRQS)	2019 - 202
Achievements and Awards	
Montreal, Canada	
	20.

2017

PRIX D'EXCELLENCE BY THE FONDATION DU RECHERCHE DU QUÉBEC (FRQS) FOR THE BEST PRESENTATION, 32E CONGRÈS DE

LA RECHERCHE DES ÉTUDIANTES DES CYCLES SUPÉRIEURS ET DES POST-DOCTORANTS EN RECHERCHE AU CHU SAINTE-JUSTINE

2016

Presentations __

American Society of Human Genetics (ASHG) Meeting 2019

PRIORITIZATION OF GENOMIC LOCI FOR CORONARY ARTERY DISEASE USING TARGETED CRISPR SCREENS FOR ENDOTHELIAL DYSFUNCTION

XXIIe Journée de la recherche ICM

VALIDATION OF GENOME-WIDE POLYGENIC RISK SCORES FOR CORONARY ARTERY DISEASE IN FRENCH CANADIANS

American Society of Human Genetics (ASHG) Meeting 2017

IDENTIFICATION OF A NOVEL MARKER FOR VALVE MATURATION: LOSS OF ADAMTS19 FUNCTION CAUSES PROGRESSIVE VALVE DISEASE IN MICE AND MEN

Congrès de la recherche des étudiantes des cycles supérieurs et des post-doctorants en recherche au CHU Sainte-Justine

HEART VALVE DYSFUNCTION IN MEN AND MICE IS CAUSED BY LOSS OF FUNCTION MUTATIONS IN ADAMTS19, A NOVEL MARKER FOR VALVULAR INTERSTITIAL CELLS

Weinstein Cardiovascular Development and Regeneration Conference 2016

LOSS OF ADAMTS19, A NOVEL MARKER FOR VALVULAR INTERSTITIAL CELL POPULATIONS DURING VALVE MATURATION, CAUSES AORTIC VALVE DYSFUNCTION

Evolgen, collaborative meeting on genome evolution

ACTRANSDB: AN ONLINE DATABASE FOR ACANTHAMOEBA CASTELLANI TRANSCRIPTS,

2nd Muenster graduate school evolution symposium

BIOGEOCHEMISTRY MEETS MOLECULAR EVOLUTION VIA METAGENOMICS: TRACING NITROGEN FLUXES FROM ECOSYSTEMS TO GENOMES IN MICROBIAL COMMUNITIES

Houston, Texas, USA

15.10.2019 - 19.10.2019

Montroal Canada

Montreal, Canada

06.06.2019 - 06.06.2019

Orlando, Florida, USA

18.10.2017 - 18.10.2017

Montreal, Canada

26.05.2017 - 26.05.2017

Durham, North Carolina, USA

18.05.2016 - 21.05.2016

Cigżeń, Poland

27.06.2012 - 28.06.2012

University of Münster, Germany

18.06.2012 - 19.06.2012

Poster presentations

Weinstein Cardiovascular Development and Regeneration Conference

A SINGLE-CELL PERSPECTIVE ON GROWTH AND MATURATION PATHWAYS IN THE MOUSE HEART.

American Society of Human Genetics (ASHG) 2014

DE NOVO MUTATION IN SOX18 CAUSES A NOVEL FORM OF HYPOTRICHOSIS-LYMPHEDEMA-TELANGIECTASIA WITH SEVERE

42nd Annual Meeting of the Ecological Society of Germany, Austria and Switzerland 2012

SOIL METAGENOMICS TO UNRAVEL THE SIGNATURE OF FERTILIZERS ON THE MOLECULAR COMPOSITION OF THE BACTERIAL RIBOSOME

Nara, Japan

16.05.2018 - 18.05.2018

San Diego, California, USA

18.10.2014 - 22.10.2014

Lueneburg, Germany

10.09.2012 - 14.09.2012

Publications

- 1. Wünnemann, F., Tadjo, T. F., Beaudoin, M., Lalonde, S., Lo, K. S., & Lettre, G. (2021). CRISPR perturbations at many coronary artery disease loci impair vascular endothelial cell functions. *bioRxiv*.
- 2. Wünnemann, F., Ta-Shma, A., Preuss, C., Leclerc, S., Vliet, P. P. van, Oneglia, A., Thibeault, M., Nordquist, E., Lincoln, J., Scharfenberg, F., & others. (2020). Loss of ADAMTS19 causes progressive non-syndromic heart valve disease. *Nature Genetics*, *52*(1), 40–47.
- 3. Audain, E., Wilsdon, A., Breckpot, J., Izarzugaza, J. M., Fitzgerald, T. W., Kahlert, A., Sifrim, A., Wuennemann, F., Perez-Riverol, Y., Abdul-Khaliq, H., & others. (2020). Integrative analysis of genomic variants reveals new associations of candidate haploinsufficient genes with congenital heart disease. *bioRxiv*.
- 4. Churakov, G., Kuritzin, A., Chukharev, K., Zhang, F., Wünnemann, F., Ulyantsev, V., & Schmitz, J. (2020). A 4-lineage statistical suite to evaluate the support of large-scale retrotransposon insertion data to reconstruct evolutionary trees. *bioRxiv*.

- 5. Gould, R. A., Aziz, H., Woods, C. E., Seman-Senderos, M. A., Sparks, E., Preuss, C., Wünnemann, F., Bedja, D., Moats, C. R., McClymont, S. A., & others. (2019). ROBO4 variants predispose individuals to bicuspid aortic valve and thoracic aortic aneurysm. *Nature Genetics*, *51*(1), 42–50.
- 6. Luyckx, I., Kumar, A. A., Reyniers, E., Dekeyser, E., Vanderstraeten, K., Vandeweyer, G., Wünnemann, F., Preuss, C., Mazzella, J.-M., Goudot, G., & others. (2019). Copy number variation analysis in bicuspid aortic valve-related aortopathy identifies TBX20 as a contributing gene. *European Journal of Human Genetics*, 27(7), 1033–1043.
- 7. Wünnemann, F., Sin Lo, K., Langford-Avelar, A., Busseuil, D., Dubé, M.-P., Tardif, J.-C., & Lettre, G. (2019). Validation of genome-wide polygenic risk scores for coronary artery disease in french canadians. *Circulation: Genomic and Precision Medicine*, 12(6), e002481.
- 8. Gould, R., Aziz, H., Woods, C., Seman-Senderos, M., Sparks, E., Preuss, C., Wünnemann, F., Bedja, D., Moats, C., McClymont, S., & others. (2019). Baylor-hopkins center for mendelian genomics; MIBAVA leducq consortium. ROBO4 variants predispose individuals to bicuspid aortic valve and thoracic aortic aneurysm. *Nat Genet*, *51*(01), 42–50.
- 9. Preuss, C., Wünnemann, F., & Andelfinger, G. (2017). At the heart of a complex disease "molecular genetics of congenital heart disease." *eLS*, 1–9.
- 10. Gillis, E., Kumar, A. A., Luyckx, I., Preuss, C., Cannaerts, E., Van De Beek, G., Wieschendorf, B., Alaerts, M., Bolar, N., Vandeweyer, G., & others. (2017). Candidate gene resequencing in a large bicuspid aortic valve-associated thoracic aortic aneurysm cohort: SMAD6 as an important contributor. *Frontiers in Physiology*, 8, 400.
- 11. Gillis, E., Kumar, A. A., Luyckx, I., Preuss, C., Cannaerts, E., Beek, G. van de, Wieschendorf, B., Alaerts, M., Bolar, N., Vandeweyer, G., & others. (2017). Corrigendum: Candidate gene resequencing in a large bicuspid aortic valve-associated thoracic aortic aneurysm cohort: SMAD6 as an important contributor. *Frontiers in Physiology*, 8, 730.
- 12. Wünnemann, F., Kokta, V., Leclerc, S., Thibeault, M., McCuaig, C., Hatami, A., Stheneur, C., Grenier, J.-C., Awadalla, P., Mitchell, G. A., & others. (2016). Aortic dilatation associated with a de novo mutation in the SOX18 gene: Expanding the clinical spectrum of hypotrichosis-lymphedema-telangiectasia syndrome. *Canadian Journal of Cardiology*, 32(1), 135–e1.
- 13. Preuss, C., Capredon, M., Wünnemann, F., Chetaille, P., Prince, A., Godard, B., Leclerc, S., Sobreira, N., Ling, H., Awadalla, P., & others. (2016). Family based whole exome sequencing reveals the multifaceted role of notch signaling in congenital heart disease. *PLoS Genetics*, *12*(10), e1006335.
- 14. Wünnemann, F., & Andelfinger, G. U. (2016). Molecular pathways and animal models of hypoplastic left heart syndrome. In *Congenital heart diseases: The broken heart* (pp. 649–664). Springer, Vienna.
- 15. Chetaille, P., Preuss, C., Burkhard, S., Côté, J.-M., Houde, C., Castilloux, J., Piché, J., Gosset, N., Leclerc, S., Wünnemann, F., & others. (2014). Mutations in SGOL1 cause a novel cohesinopathy affecting heart and gut rhythm. *Nature Genetics*, 46(11), 1245.

Languages _

- German (mother-language)
- English (fluent)
- French (fluent)