Avatud juurdepääsu nõuded publikatsioonidele ning nende järgimine H2020 projektides

Elena Sipria-Mironov

TÜ sisekoolitus 03.04.2019

- AJ nõuded publikatsioonidele
- *Green OA* ehk isearhiveerimine samm-sammult
- Avaldamine TÜ digitaalarhiiv DSpace-is

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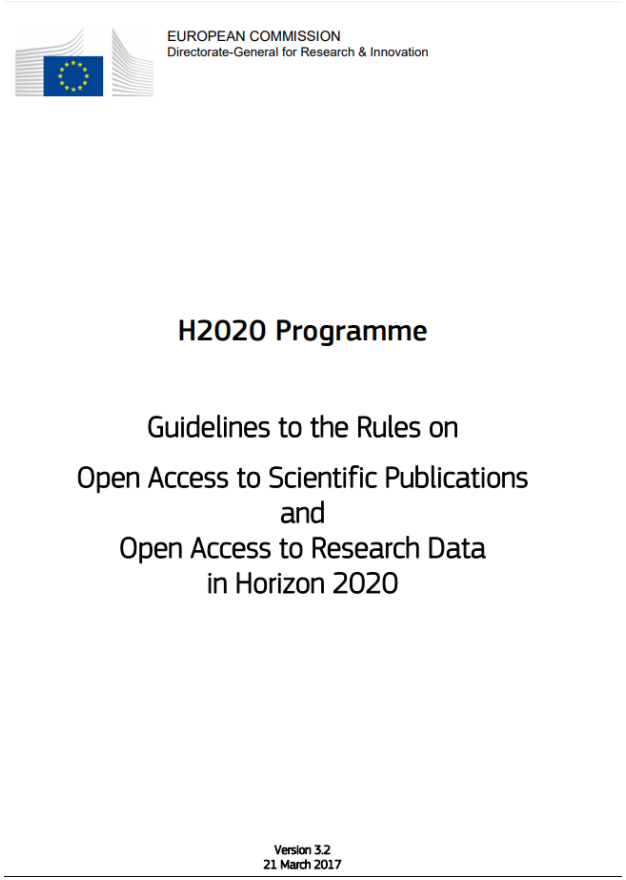

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H2020 Programme

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Version 3.2
21 March 2017

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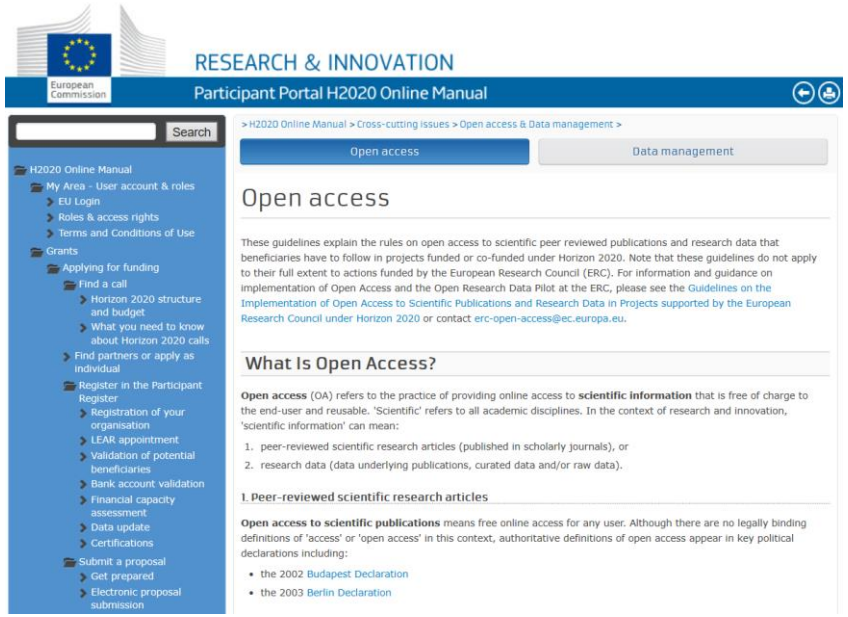

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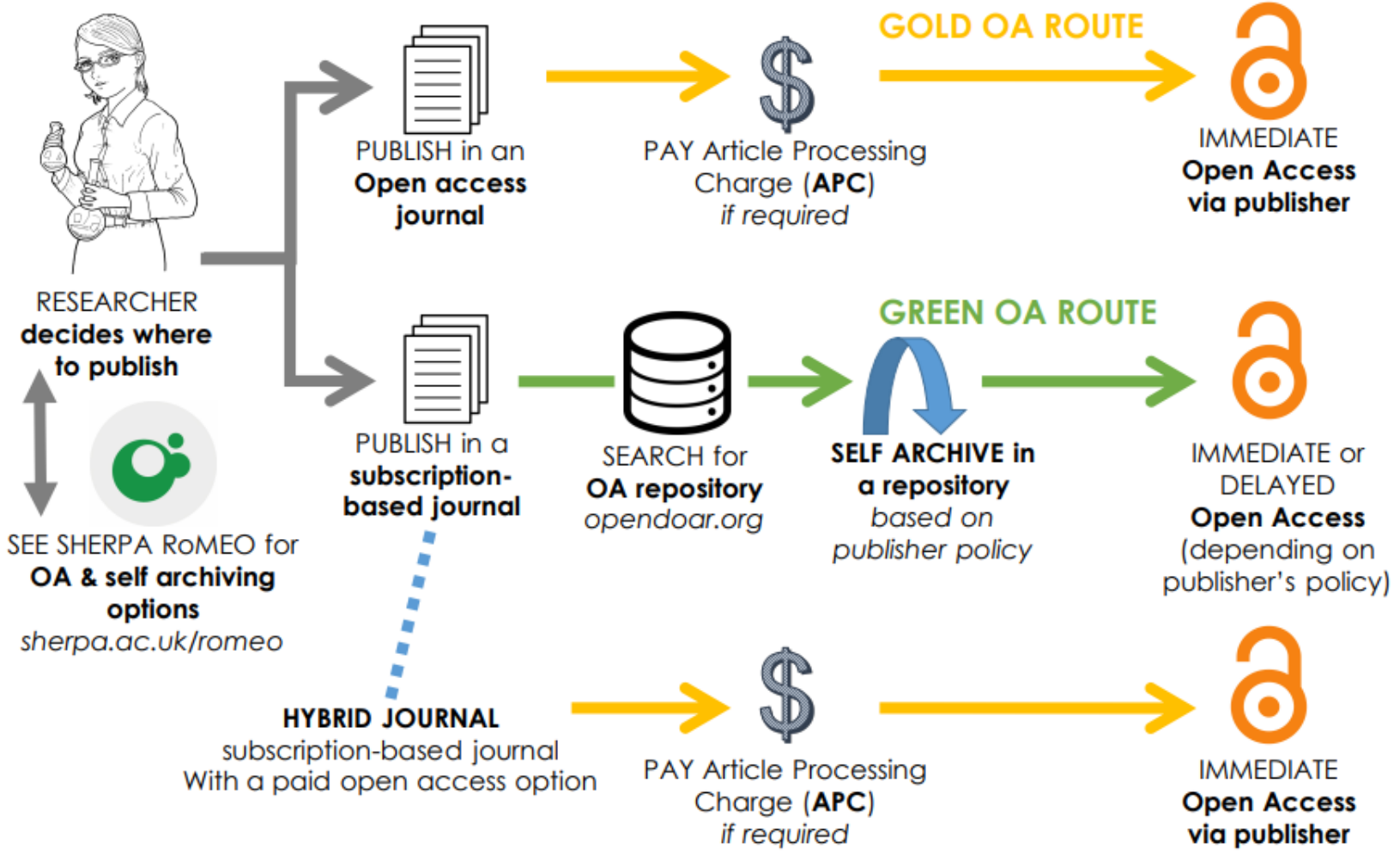

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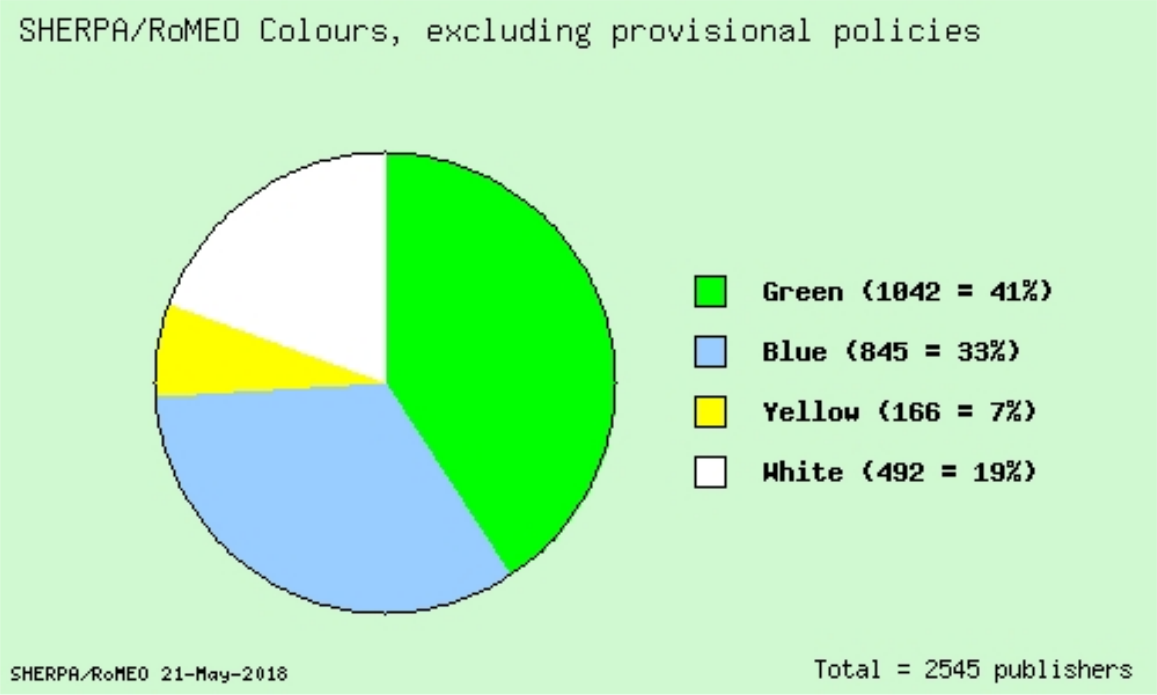

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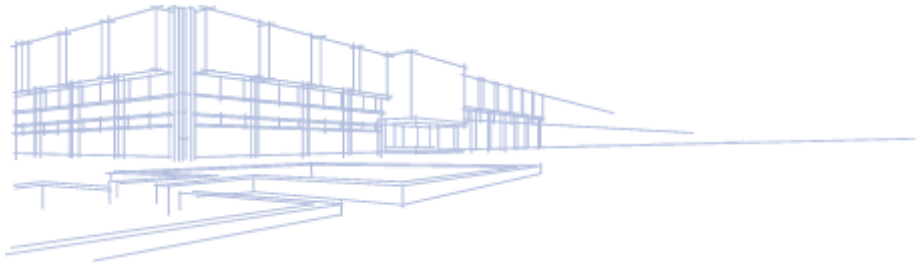

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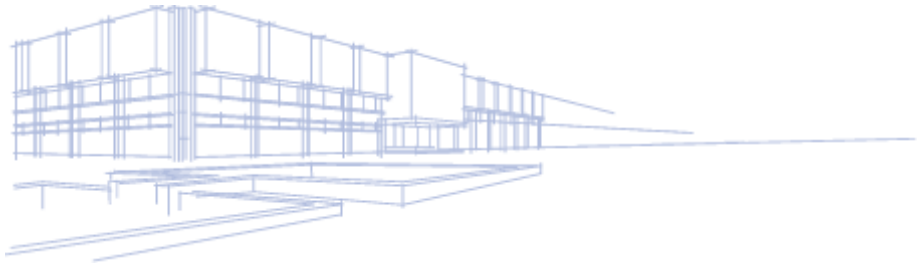
Done in two originals, in English

History of changes		
Version	**Publication date**	**Change**
1.0	20.03.2017	• Initial version

[1] Choose 12 months for publications in the social sciences and humanities and 6 months for publications in other domains.



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Definition	Manuscript before peer-review	Version of manuscript, improved and corrected by the peer-reviewing. Publisher´s layout and page numbers excluded.	Published article, final publisher´s version with the layout.

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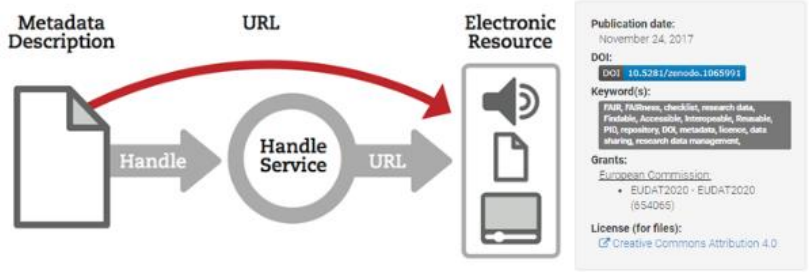

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dc.date.issued	2018	
dc.identifier.uri	http://hdl.handle.net/10062/63389	
dc.description.abstract	This article reviews the policy positions of Estonia, Latvia, and Lithuania with respect to the Ukraine crisis – the biggest foreign policy challenge for the Baltic states since they regained independence. Ukraine dominated the Baltic foreign policy agenda from the outbreak of the crisis, because it touched upon a dimension of existential threat for the Baltic countries. While giving an overview of the main policy domains where the effect of the Ukraine crisis could be observed, this article demonstrates that the three Baltic countries adopted a comprehensive approach to security and foreign policymaking, underlining cooperation both at a national and European level. In light of this, the Ukraine crisis can be seen as a maturity test for postindependence Baltic foreign policy.	et
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Kus on teie H2020 projekti publikatsioonide nimekiri?

H2020 grant WIDENLIFE
Widening the Scientific Excellence for Studies on Women's and Fetal Health and Wellbeing

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Publications

Widening the Scientific Excellence for Studies on Women's and Fetal Health and Wellbeing - WIDENLIFE, Universitas Tartuensis DSpace

1. Saare, Merli; Modhukur, Vijayachitra; Suhorutshenko, Marina; Rajashekar, Balaji; Rekker, Kadri; Sõritsa, Deniss; Karro, Helle; Soplepmann, Pille; Sõritsa, Andrei; Lindgren, Cecilia M; Rahmioglu, Nilufer; Drong, Alexander; Becker, Christian M; Zondervan, Krina T; Salumets, Andres; Peters, Maire. The influence of menstrual cycle and endometriosis on endometrial methylome. Clin Epigenetics. 2016 Jan 12;8:2. doi: 10.1186/s13148-015-0168-z (JP)

2. Pervjakova, Natalia; Kasela, Silva; Morris, Andrew P; Kals, Mart; Metspalu, Andres; Lindgren, Cecilia M; Salumets, Andres; Mägi, Reedik. Imprinted genes and imprinting control regions show predominants intermediate methylation in adult somatic tissues. Epigenomics, 8(6), 789-799, 23.03.2016. doi: 10.2217/epi.16.8 (JP)

3. Triin Laisk-Podar, Cecilia M. Lindgren, Maire Peters, Juha S. Tapanainen, Cornelis B. Lambalk, Andres Salumets, Reedik Mägi. Ovarian Physiology and GWAS: Biobanks, Biology and Beyond. Trends in endocrinology and metabolism: TEM. 27 (7), 516–528, doi:10.1016/j.tem.2016.04.011, 21.05.2016. (JP)

4. Tšuiko, O.; Nõukas, M.; Žilina, O.; Hensen, K.; Tapanainen, J.; Mägi, R.; Kals, M.; Kivistik, PA.; Haller-Kikkatalo, K.; Salumets, A.; Kurg, A. (2016). Copy number variation analysis detects novel candidate genes involved in follicular growth and oocyte maturation in a cohort of premature ovarian failure cases. Human Reproduction, 31 (8). 2016 Jun 14. doi: 10.1093/humrep/dew142

5. Tiirats, Airi; Viltrop, Triin; Nõukas, Margit; Reimann, Ene; Salumets, Andres; Kõks, Sulev. C14orf132 gene is possibly related to extremely low birth weight. BMC genetics. 2016, 17 (1), 132-132, doi: 10.1186/s12863-016-0439-5

6. Boggavarapu, Nageswara Rao; Lalitkumar, Sujata; Joshua, Vijay; Kasvandik, Sergo; Salumets, Andres; Lalitkumar, Parameswaran Grace; Gemzell-Danielsson, Kristina. Compartmentalized gene expression profiling of receptive endometrium reveals progesterone regulated ENPP3 is differentially expressed and secreted in glycosylated form. Scientific reports. 2016, 6, 33811–33811, doi: 10.1038/srep33811

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Project Manager
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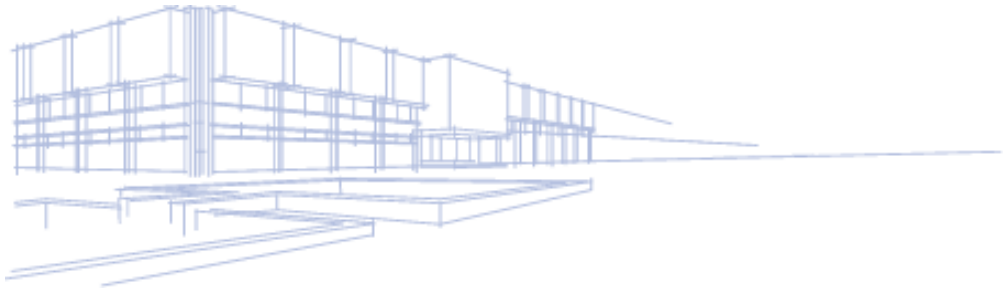

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- Isearhiveerimine on üks võimalus vastata rahastaja **AJ nõuetele**;
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Given that one in six couples face infertility, often caused by female factors, women's reproductive health is a significant medical and socio-economical challenge. Herein, while addressing this issue, the Estonian research on female reproductive health and medicine has rapidly developed with the lead of University of Tartu (UT). Nevertheless, the UT's capacity and expertise still falls short of the leading institutions. At the same time, also the most competitive research groups face significant barriers to perform world-class science due to substantial networking gaps that still exist between previously non-linked research teams. TWINNING funding scheme is designed to overcome these aforementioned shortcomings. Thus UT has formed a WIDENLIFE consortium with its world-renowned partners: University of Oxford and Katholieke Universiteit Leuven, and altogether we have set two ambitious objectives to address. Firstly, for the UT: to become one of the leading research and teaching centres for reproductive and fetal medicine in Eastern and Northern Europe. Secondly, for all members of consortia: to intensify trilateral synergies between the research groups in the areas of female reproductive health and medicine. Our specific goals are to highlight the associations between female metabolic health and infertility, provide deeper understating for embryonal development, and offer new tools for infertility treatment and prenatal diagnostics. In order to resolve these objectives, exchange of know-how, ideas and information between the partners will be enhanced, creating the novel clinically valuable information through pooling the expertise and synergy of resources, interests and commitments by universities from Estonia, UK and Belgium. This could also mean a significant contribution to the scientific capacity of the Estonian research community as well as the health technology industry, which is one of the main focus areas for Estonian Smart Specialisation Strategy.

Recent Submissions

GWAS Identifies Risk Locus for Erectile Dysfunction and Implicates Hypothalamic Neurobiology and Diabetes in Etiology

Bovijn, J; Jackson, L; Censin, J; Chen, CY; Laisk, T; Laber, S; Ferreira, T; Pulit, SL; Glastonbury, CA; Smoller, JW; Harrison, JW; Ruth, KS; Beaumont, RN; Jones, SE; Tyrrell, J; Wood, AR; Weedon, MN; Mägi, R; Neale, B; Lindgren, CM; Murray, A; Holmes, MV (2019)

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Peters, Maire (9)

Rekker, Kadri (6)

Mägi, Reedik (5)

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Tšuiko, Olga (5)

Kollektsioon TÜ DSpace-is „LT Euroopa Liidu rahastatud projektid“

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GWAS Identifies Risk Locus for Erectile Dysfunction and Implicates Hypothalamic Neurobiology and Diabetes in Etiology

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dc.identifier.uri	http://hdl.handle.net/10062/63435	
dc.language.iso	eng	et
dc.relation	info:eu-repo/grantAgreement/EC/H2020/692065///WIDENLIFE	et
dc.relation.ispartofseries	Am J Hum Genet. 2019 Jan 3;104(1):157-163.	

GWAS Identifies Risk Locus for Erectile Dysfunction and Implicates Hypothalamic Neurobiology and Diabetes in Etiology

Jonas Bovijn,[1,2,14,*] Leigh Jackson,[3,14] Jenny Censin,[1,2,14] Chia-Yen Chen,[4,5,14] Triin Laisk,[6,7,14] Samantha Laber,[1,2,14] Teresa Ferreira,[1] Sara L. Pulit,[1,8,9] Craig A. Glastonbury,[1] Jordan W. Smoller,[5] Jamie W. Harrison,[10] Katherine S. Ruth,[10] Robin N. Beaumont,[10] Samuel E. Jones,[10] Jessica Tyrrell,[10] Andrew R. Wood,[10] Michael N. Weedon,[10,14] Reedik Mägi,[6,14] Benjamin Neale,[4,5,14] Cecilia M. Lindgren,[1,2,8,14] Anna Murray,[10,14,*] and Michael V. Holmes[11,12,13,14]

Erectile dysfunction (ED) is a common condition affecting more than 20% of men over 60 years, yet little is known about its genetic architecture. We performed a genome-wide association study of ED in 6,175 case subjects among 223,805 European men and identified one locus at 6q16.3 (lead variant rs57989773, OR 1.20 per C-allele; $p = 5.71 \times 10^{-14}$), located between *MCHR2* and *SIM1*. *In silico* analysis suggests *SIM1* to confer ED risk through hypothalamic dysregulation. Mendelian randomization provides evidence that genetic risk of type 2 diabetes mellitus is a cause of ED (OR 1.11 per 1-log unit higher risk of type 2 diabetes). These findings provide insights into the biological underpinnings and the causes of ED and may help prioritize the development of future therapies for this common disorder.

Erectile dysfunction (ED) is the inability to develop or maintain a penile erection adequate for sexual intercourse.[1] ED has an age-dependent prevalence, with 20%–40% of men aged 60–69 years affected.[1] The genetic architecture of ED remains poorly understood, owing in part to a paucity of well-powered genetic association studies. Discovery of such genetic associations can be valuable for elucidating the etiology of ED and can provide genetic support for potential new therapies.

We conducted a genome-wide association study (GWAS) in the population-based UK Biobank (UKBB) and the Estonian Genome Center of the University of Tartu (EGCUT) cohorts and hospital-recruited Partners HealthCare Biobank (PHB) cohort. Subjects in UKBB were of self-reported white ethnicity, with subjects in EGCUT and PHB of European ancestry, as per principal components analyses (Supplemental Material and Methods).

ED was defined as self-reported or physician-reported ED using ICD10 codes N48.4 and F52.2, or use of oral ED medication (sildenafil/Viagra, tadalafil/Cialis, or vardenafil/Levitra), or a history of surgical intervention for ED (using OPCS-4 codes L97.1 and N32.6) (Supplemental Material and Methods). The prevalence of ED in the cohorts was 1.53% (3,050/199,352) in UKBB, 7.04% (1,182/16,787) in EGCUT, and 25.35% (1,943/7,666) in PHB (Table S1). Demographic characteristics of the subjects in each cohort are shown in Table S2. The reasons for the different prevalence rates in the three cohorts may include a higher median cohort age for men in PHB (65 years, compared to 59 years in UKBB and 42 years in EGCUT; Table S2), "healthy volunteer" selection bias in UKBB,[2] a lack of primary care data availability in UKBB, and intercultural differences, including "social desirability" bias.[3,4] Importantly, we note that the assessment of exposure-outcome relationships remains valid, despite the prevalence likely not being representative of the general population prevalence.

GWASs in UKBB revealed a single genome-wide significant ($p < 5 \times 10^{-8}$) locus at 6q16.3 (lead variant rs57989773, EAF_{UKBB} [C-allele] = 0.24; OR 1.23; $p = 3.0 \times 10^{-11}$). Meta-analysis with estimates from PHB (OR 1.20; $p = 9.84 \times 10^{-5}$) and EGCUT (OR 1.08; $p = 0.16$) yielded a pooled meta-analysis OR 1.20; $p = 5.71 \times 10^{-14}$ (heterogeneity p value = 0.17; Figures 1A–1C). Meta-analysis of all variants yielded no further genome-wide loci. Meta-analysis of our results with previously suggested ED-associated variants also did not result in any further significant loci (Supplemental Material and Methods; Table S3), nor did X chromosome analysis in UKBB.

[1]Big Data Institute at the Li Ka Shing Centre for Health Information and Discovery, University of Oxford, Oxford OX3 7LF, UK; [2]Wellcome Centre for Human Genetics, Nuffield Department of Medicine, University of Oxford, Oxford OX3 7BN, UK; [3]Institute of Biomedical and Clinical Science, University of Exeter Medical School, University of Exeter, Exeter EX2 5DW, UK; [4]Analytic and Translational Genetics Unit, Massachusetts General Hospital, Boston, MA 02114, USA; [5]Psychiatric & Neurodevelopmental Genetics Unit, Massachusetts General Hospital, Boston, MA 02114, USA; [6]Estonian Genome Center, Institute of Genomics, University of Tartu, Tartu 51010, Estonia; [7]Department of Obstetrics and Gynecology, Institute of Clinical Medicine, University of Tartu, Tartu 50406, Estonia; [8]Program in Medical and Population Genetics, Broad Institute, Cambridge, MA 02142, USA; [9]Department of Genetics, University Medical Center Utrecht, Utrecht, the Netherlands; [10]Genetics of Complex Traits, University of Exeter Medical School, University of Exeter, Exeter EX2 5DW, UK; [11]National Institute for Health Research Oxford Biomedical Research Centre, Oxford University Hospital, Old Road, Oxford OX3 7LE, UK; [12]Clinical Trial Service Unit & Epidemiological Studies Unit (CTSU), Nuffield Department of Population Health, Big Data Institute Building, Roosevelt Drive, University of Oxford, Oxford OX3 7LF, UK; [13]Medical Research Council Population Health Research Unit at the University of Oxford, Nuffield Department of Population Health, University of Oxford, Oxford, UK
[14]These authors contributed equally to this work
*Correspondence: jbovijn@well.ox.ac.uk (J.B.), a.murray@exeter.ac.uk (A.M.)
https://doi.org/10.1016/j.ajhg.2018.11.004.

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Challenges in endometriosis miRNA studies - from tissue heterogeneity to disease specific miRNAs

Merli Saare, Kadri Rekker, Triin Laisk-Podar, Nilufer Rahmioglu, Krina Zondervan, Andres Salumets, Martin Götte, Maire Peters

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Challenges in endometriosis miRNA studies - from tissue heterogeneity to disease specific miRNAs

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Key words: endometriosis, endometrium, microarray, microRNA, stroma, tissue heterogeneity

Abbreviations

EcSC - endometriotic stromal cells

EMT - epithelial-mesenchymal transition

EnSC - endometrial stromal cells

FACS - fluorescence-activated cell sorting

FC - fold change

LCM - laser capture microdissection

UTR – untranslated region

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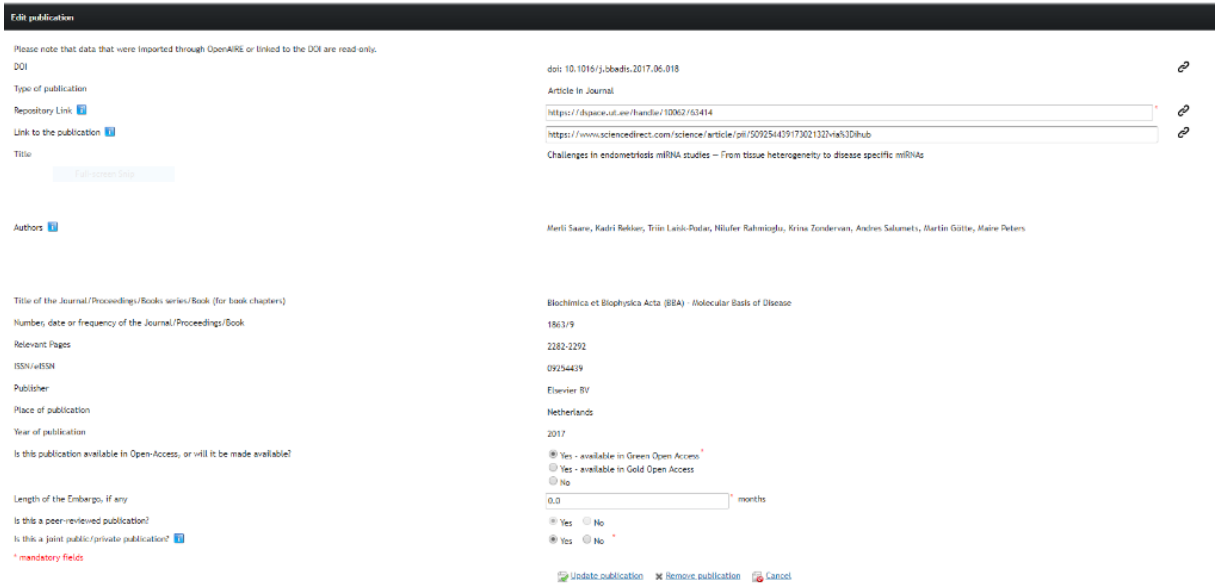

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