



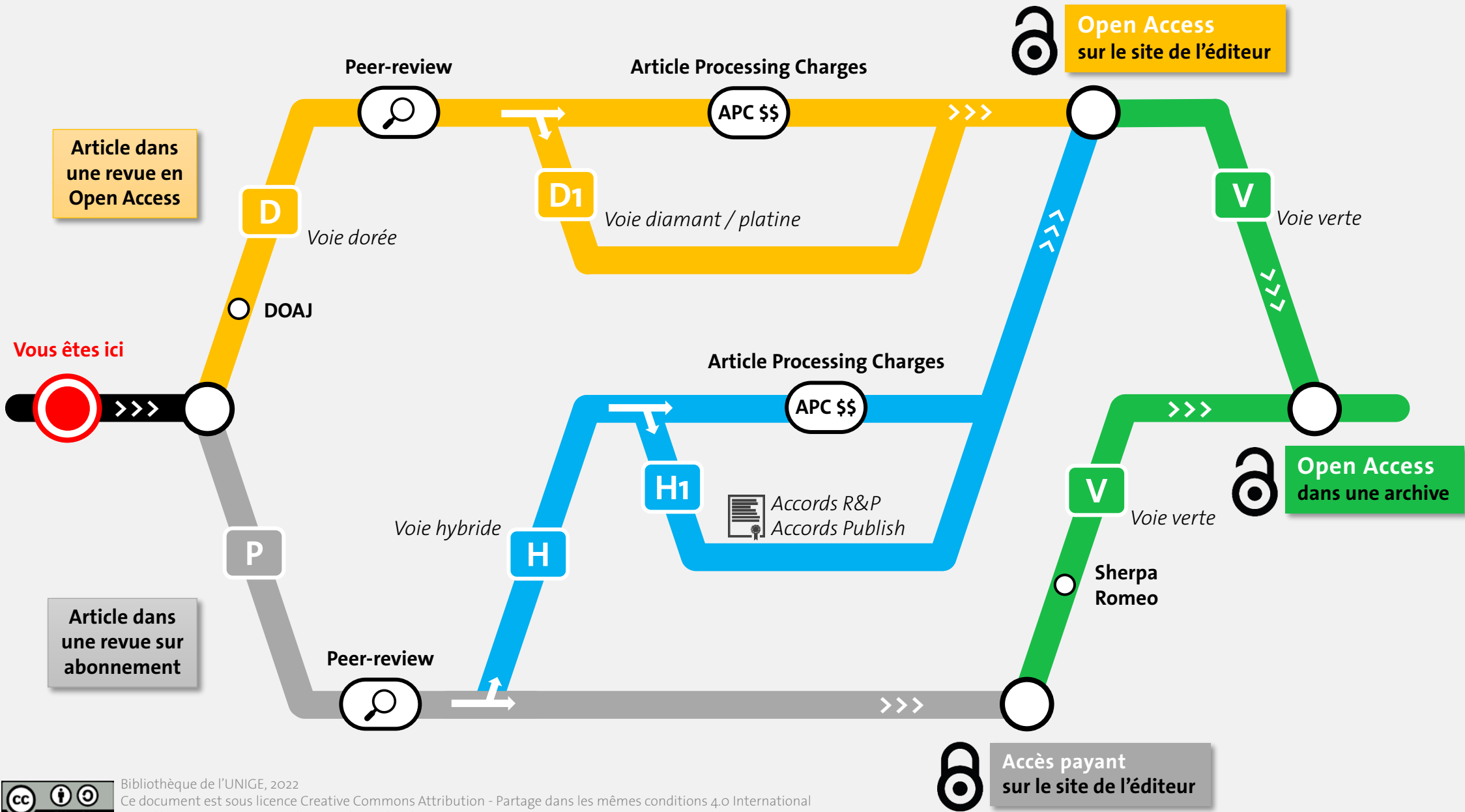
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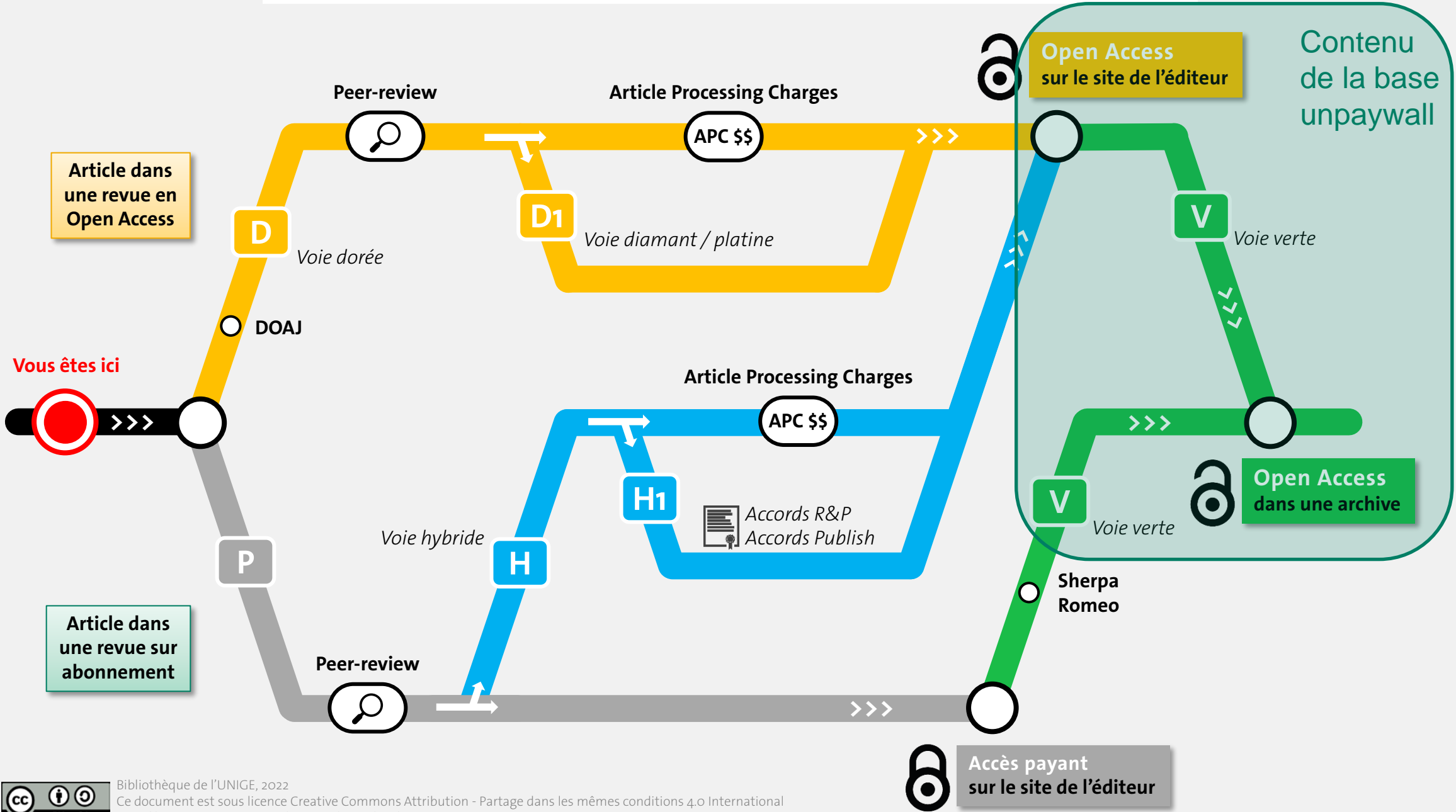
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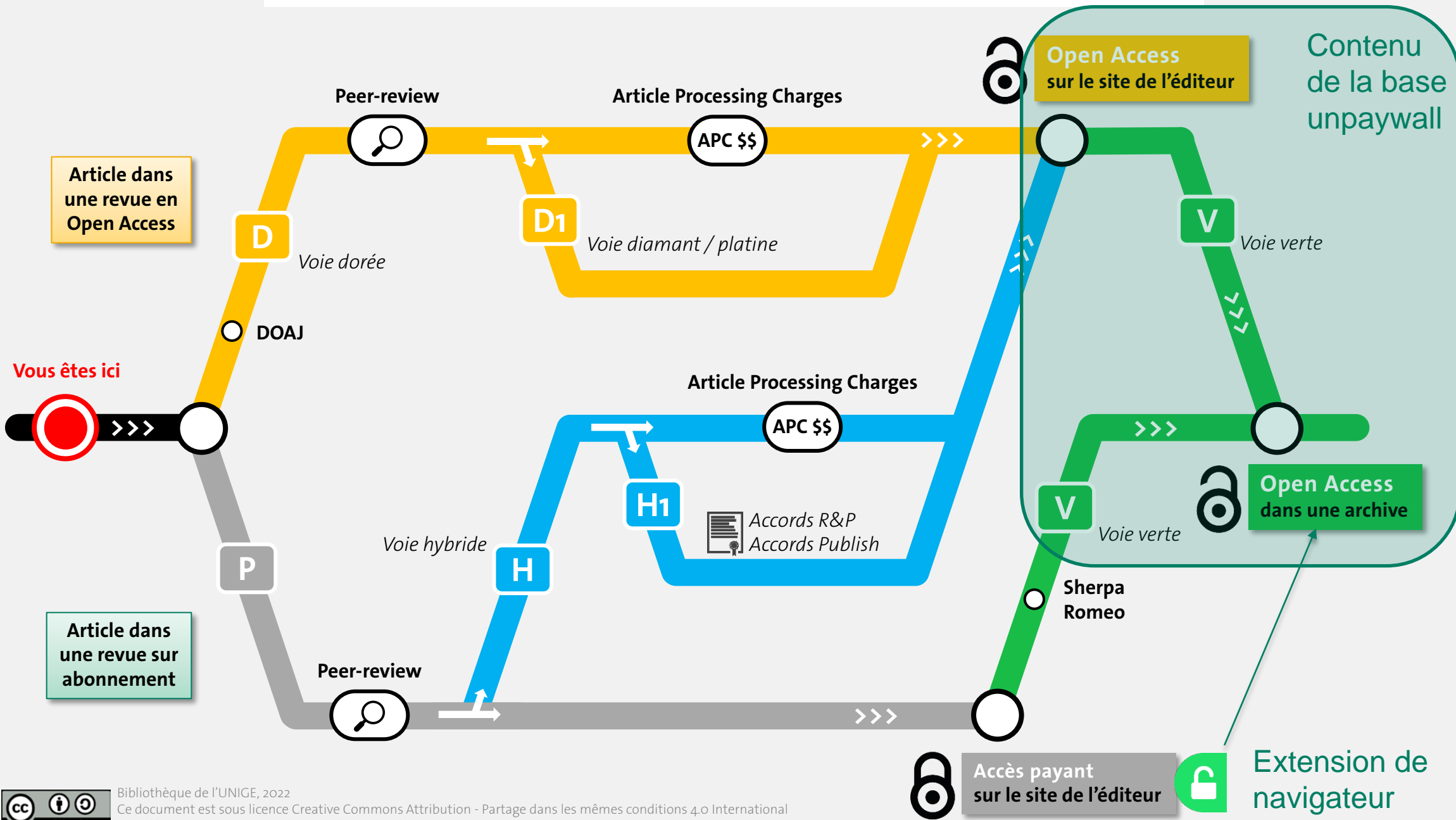
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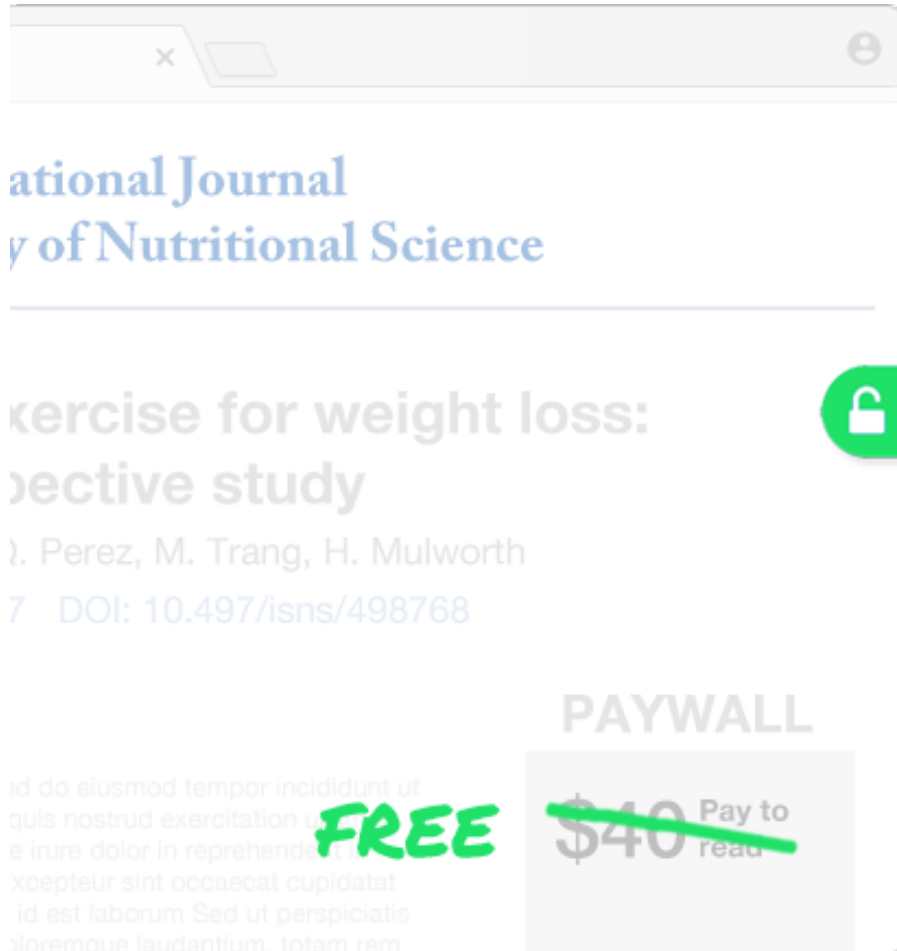
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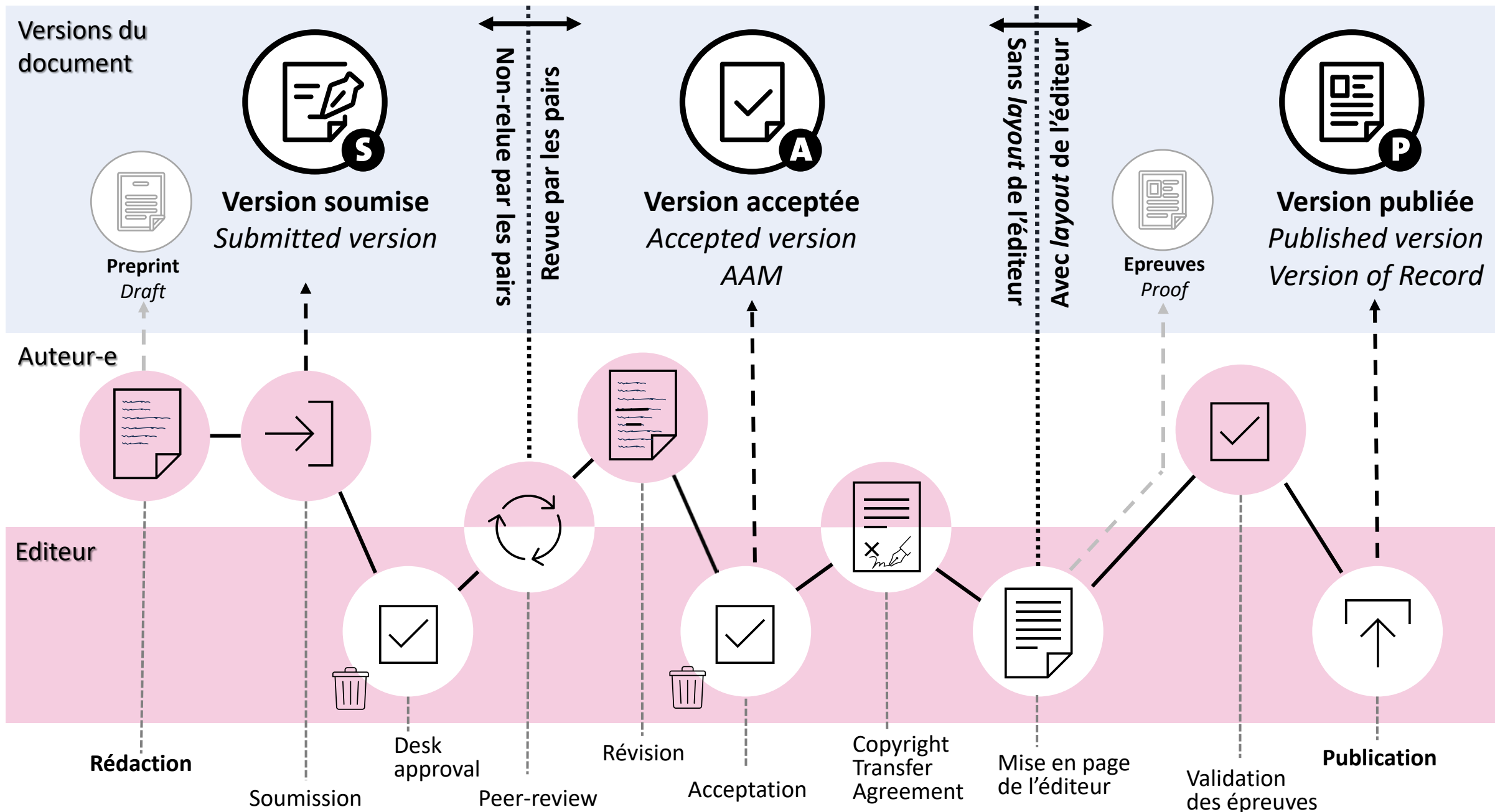
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Published: 21 November 2017

New insights into the pharmacogenomics of antidepressant response from the GENDEP and STAR*D studies: rare variant analysis and high-density imputation

C Fabbri, K E Tansey, [...] C M Lewis

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Abstract

Genome-wide association studies have generally failed to identify

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New insights on the pharmacogenomics of antidepressant response from the GENDEP and STAR*D studies: rare variant analysis and high-density imputation

Chiara Fabbri^{1,2}, Katherine E. Tansey³, Roy H. Perlis⁴, Joanna Hauser⁵, Neven Henigsberg⁶, Wolfgang Maier⁷, Ole Mors⁸, Anna Placentino⁹, Marcella Rietschel¹⁰, Daniel Souery¹¹, Gerome Breen¹², Charles Curtis¹³, Lee Sang-Hyuk¹⁴, Stephen Newhouse¹⁵, Hamel Patel¹⁶, Michel Craipon¹⁷, Nader Perroud¹⁸, Guido Bondolfi¹⁹, Michael O'Donovan²⁰, Glyn Lewis²¹, Joanna M. Biernacka^{22,23}, Richard M. Weinshilboum²⁴, Aune Farmer²⁵, Katherine J. Aitchison²⁶, Ian Craig²⁷, Peter McGuffin²⁸, Rudolf Uher²⁹, Cathryn M. Lewis³⁰

- 1 Department of Biomedical and Neurotoner Sciences, University of Bologna, Bologna, Italy
- 2 Institute of Psychiatry, Psychology and Neuroscience, King's College London, United Kingdom
- 3 College of Biomedical and Life Sciences, Cardiff University, Cardiff, United Kingdom
- 4 Department of Psychiatry, Center for Experimental Drugs and Diagnostics, Massachusetts General Hospital, Boston, USA
- 5 Laboratory of Psychiatric Genetics, Department of Psychiatry, Poznan University of Medical Sciences, Poznan, Poland
- 6 Croatian Institute for Brain Research, Medical School, University of Zagreb, Zagreb, Croatia
- 7 Department of Psychiatry, University of Bonn, Bonn, Germany
- 8 Centre for Psychiatric Research, Aarhus University Hospital, Risskov, Denmark
- 9 Biological Psychiatry Unit and Dual Diagnosis Ward, Istituto Di Ricovero e Cura a Carattere Scientifico, Centro San Giovanni di Dio, Fatebenefratelli, Brescia, Italy
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Abstract

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The Pharmacogenomics Journal (2018) 18, 413–421
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ORIGINAL ARTICLE

New insights into the pharmacogenomics of antidepressant response from the GENDEP and STAR*D studies: rare variant analysis and high-density imputation

C Fabbri^{1,2}, K E Tansey³, R H Perlis⁴, J Hauser⁵, N Henigsberg⁶, W Maier⁷, O Mors⁸, A Placentino⁹, M Rietschel¹⁰, D Souery¹¹, G Breen¹², C Curtis¹³, L Sang-Hyuk¹⁴, S Newhouse¹⁵, H Patel¹⁶, M Guipponi¹⁷, N Perroud¹⁸, G Bondolfi¹⁹, G Lewis²¹, J M Biernacka^{22,23}, R M Weinshilboum²⁴, A Farmer²⁵, K J Aitchison²⁶, I Craig²⁷, P McGuffin²⁸, R Uher²⁹ and CM Lewis³⁰

Genome-wide association studies have generally failed to identify polymorphisms associated with antidepressant response. Possible reasons include limited coverage of genetic variants that this study tried to address by using genotype- and dense imputation. A meta-analysis of Genome-Based Therapeutic Drugs for Depression (GENDEP) and Sequenced Treatment Alternatives to Relieve Depression (STAR*D) studies was performed at the single-nucleotide polymorphism (SNP) gene and pathway levels. Coverage of genetic variants was increased compared with previous studies by adding more genotypes to previously available genome-wide data and using the Haplotype Reference Consortium panel for imputation. Standard quality control was applied. Phenotypes were symptom improvement and remission after 12 weeks of antidepressant treatment. Significant findings were investigated in NEURIMIS consortium samples and Pharmacogenomic Research Network Antidepressant Medication Pharmacogenomic Study (PGIN AMP) for replication. A total of 7062 950 SNPs were analyzed in GENDEP (n = 738) and STAR*D (n = 1409), n116692768 (P = 1.30e−08, ITGAP (rs16927768) and rs78191705 (P = 2.39e−08, ANKRD1 (rs16927768) were significantly associated with symptom improvement during citalopram/escitalopram treatment. At the gene level, no consistent effect was found. At the pathway level, the Gene Ontology (GO) terms GO:0005694 (chromosome) and GO:0044427 (chromosomal part) were associated with improvement (corrected P = 0.007 and 0.045, respectively). The association between rs116692768 and symptom improvement was replicated in PGIN AMP (P = 0.047), whereas rs78191705 was not. The two SNPs did not replicate in NEURIMIS. ITGAP codes for a membrane receptor for neurotransmitters and ANKRD1 is a transmembrane neuronal adhesion receptor involved in synaptic differentiation. Despite their meaningful biological rationale for being involved in antidepressant effect, replication was partial. Further studies may help in clarifying their role.

The Pharmacogenomics Journal (2018) 18, 413–421; doi:10.1038/s41374.017.0144 published online 21 November 2017

INTRODUCTION

Major depressive disorder (MDD) became one of the five leading diseases contributing to disability-adjusted life years in 2010 in the United States. MDD is associated with a huge increase in suicide risk, poor quality of life (comparable to that observed in severe physical disorders such as arthritis and heart disease) and health expenditure (direct costs alone amount to 42 billion dollars per year in Europe).

Despite the availability of antidepressant drugs belonging to different classes, high interindividual variability is observed in response. The lack of reliable and reproducible markers of treatment outcome contributes to unsatisfactory response and remission rates as well as to side-effect burden, poor treatment adherence and early treatment discontinuation^{1,2}. Following the observation that antidepressant response clusters in families, genetic variants were considered promising biomarkers to tailor antidepressant treatments and improve the prognosis of MDD^{3,4}. Genome-wide association studies (GWAS) were a promising tool to identify the polymorphisms involved in antidepressant response after the overall contradictory and nonsignificant findings of candidate gene studies⁵. However, GWAS results fell below expectations, with no genome-wide significant signal

Via abonnements UNIGE

New insights on the pharmacogenomics of antidepressant response from the GENDEP and STAR*D studies: rare variant analysis and high-density imputation

Chiara Fabbri^{1,2}, Katherine E. Tansey³, Roy H. Perlis⁴, Joanna Hauser⁵, Neven Henigsberg⁶, Wolfgang Maier⁷, Ole Mors⁸, Anna Placentino⁹, Marcella Rietschel¹⁰, Daniel Souery¹¹, Jerome Breen¹², Charles Curtis¹³, Lee Sang-Hyuk¹⁴, Stephen Newhouse¹⁵, Hamel Patel¹⁶, Michel Guipponi¹², Nader Perroud¹³, Guido Bondolfi¹³, Michael O'Donovan¹⁴, Glyn Lewis¹⁵, Joanna M. Biernacka^{16,17}, Richard M. Weinshilboum¹⁸, Anne Farmer², Katherine J. Aitchison¹⁹, Ian Craig², Peter McGuffin², Rudolf Uher²⁰, Cathryn M. Lewis²

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17 Department of Health Sciences Research, Mayo Clinic, Rochester, MN, United States

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New insights on the pharmacogenomics of antidepressant response from the GENDEP and STAR*D studies: rare variant analysis and high-density imputation

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Abstract

Genome-wide association studies have generally failed to identify polymorphisms associated with antidepressant response. Possible reasons include limited coverage of genetic variants that this study tried to address by exome genotyping and dense imputation. A meta-analysis of Genome-Based Therapeutic Drugs for Depression (GENDEP) and Sequenced Treatment Alternatives to Relieve Depression (STAR*D) studies was performed at SNP, gene and pathway level. Coverage of genetic variants was increased compared to previous studies by adding exome genotypes to previously available genome-wide data and using the Haplotype Reference Consortium panel for imputation. Standard quality control was applied. Phenotypes were symptom improvement and remission after 12 weeks of antidepressant treatment. Significant findings were investigated in NEWMEDS consortium samples and Pharmacogenomic Research Network Antidepressant Medication Pharmacogenomic Study (PGRN-AMPS) for replication. 7,062,950 SNPs were analysed in GENDEP (n=738) and STAR*D (n=1409). rs116692768 (p=1.80e-08, ITGA9 (integrin alpha 9)) and rs76191705 (p=2.59e-08, NRXN3 (neurexin 3)) were significantly associated with symptom improvement during citalopram/escitalopram treatment. At gene level, no consistent effect was found. At pathway level, the Gene Ontology terms GO:0005694 (chromosome) and GO:0044427 (chromosomal part) were associated with improvement (corrected p=0.007 and 0.045, respectively). The association between rs116692768 and symptom improvement was replicated in PGRN-AMPS (p=0.047), while rs76191705 was not. The two SNPs did not replicate in NEWMEDS. ITGA9 codes for a membrane receptor for neurotrophins and NRXN3 is a transmembrane neuronal adhesion receptor involved in synaptic differentiation. Despite their meaningful biological rationale for being involved in antidepressant effect, replication was partial. Further studies may help in clarifying their role.

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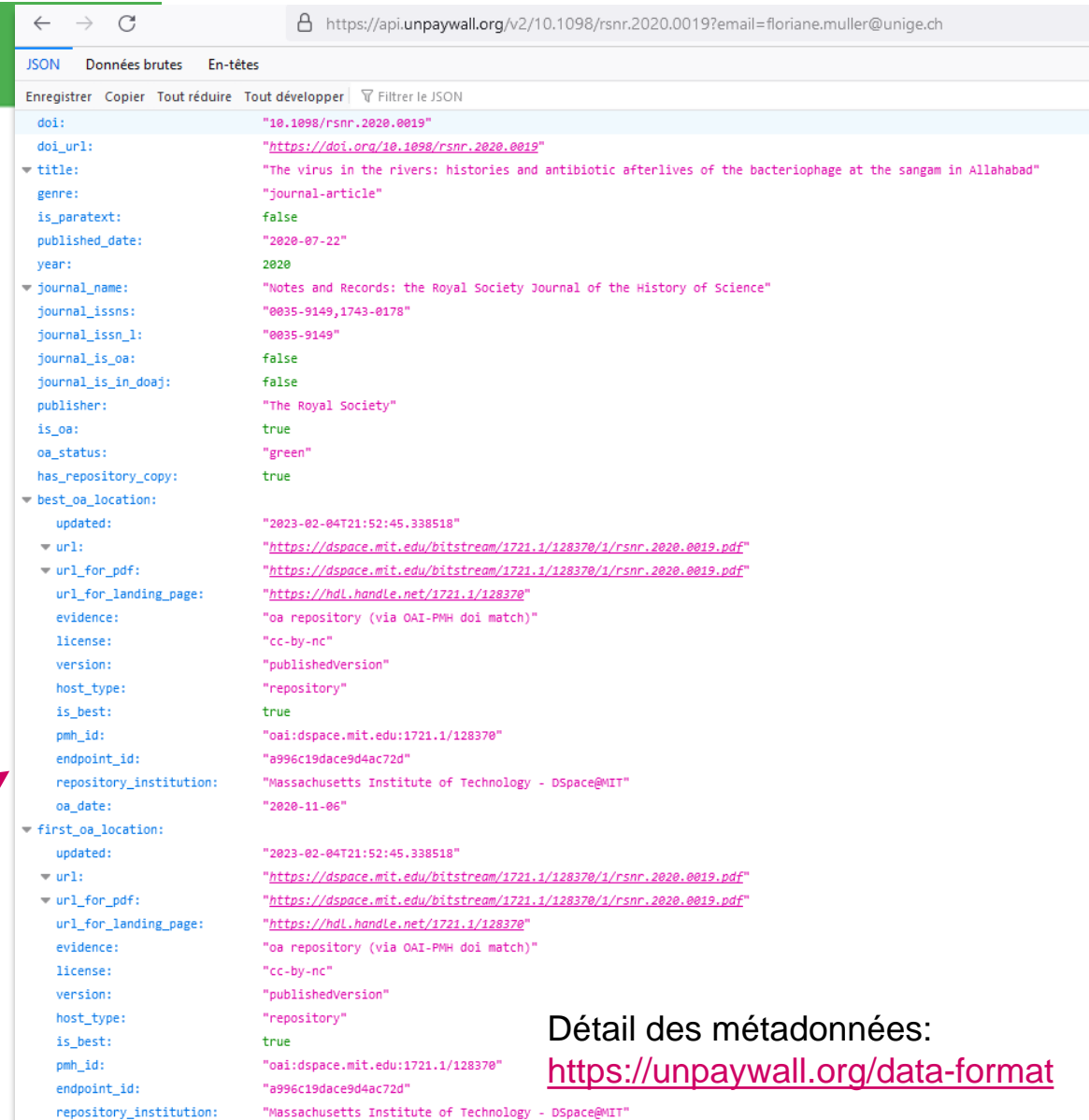
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Simple Query Tool

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- A [JSON Lines](#) file that shows what you'd get if you called our API once for each DOI.
- An Excel spreadsheet.

Don't forget to check the [schema documentation](#), which includes definitions that will help you interpret the the result files.

Submit DOIs

Paste DOIs here, one per line

Select Result Formats

CSV JSONL Excel (.xlsx)

Your email

[EXTERNAL] Your Unpaywall results

Unpaywall Team <support@unpaywall.org>
À Floriane Sophie Muller

Cet expéditeur support@unpaywall.org est externe à votre organisation.

results.xlsx 10 KB

results.csv 11 KB

results.jsonl 133 KB

13:57

Quelques applications possibles

- Les mêmes qu'avec l'API
- Vérifier le niveau d'Open Access des publications d'une personne
- Corriger le niveau d'accès ou la licence de contenus présents dans son archive institutionnelle
- Retrouver une série de manuscrits auteur-es disséminés sur le web par les co-auteur-es d'une personne
- Trouver des exemples pour une formation
- ...

<https://unpaywall.org/products/simple-query-tool>

INTÉGRÉ DANS DE NOMBREUX OUTILS

- Web of Science
- Scopus
- Dimensions
- theLens
- Swiscovery
- Zotero
- Les outils de monitoring de l'OA, comme celui du FNS par ex: <https://www.snsf-oa-check.ch/>
- ...
- Liste complète: <https://unpaywall.org/integrations>

UN EXEMPLE DANS SWISSCOVERY



The screenshot shows a search result for an article. On the left is a vertical navigation menu with options: Haut, Citations, Consulter en ligne, Détails, Liens, and Envoyer vers. The main content area displays the article title, authors (Holmes, S ; Ryan, T ; Young, D ; Harries, M), and publication information (British journal of dermatology (1951), 2016-07, Vol.175 (1), p.203-207; England: Blackwell Publishing Ltd). A quote from the article is visible: "... using the Frontal Fibrosing Alopecia Severity Index. Table S2. Kendall's coefficients of concordance W for intraobserver clinical assessments...". Below the quote are icons for 'PEER-REVIEWED' and 'OPEN ACCESS', and a green link 'Disponible en ligne >'. There are sections for 'Citations' and 'Consulter en ligne', each with a search bar and options to 'Rechercher les références', 'qui citent celle-ci', 'ou les références', and 'citées dans'. At the bottom, there is a section for 'Disponibilité du texte intégral' with two entries: 'Wiley Online Library Database Model 2018' (available since 1997) and 'Version Open Access du texte intégral trouvée grâce à :Unpaywall'. Both entries have an 'Afficher la licence' button with a checkmark icon.

ARTICLE

Frontal Fibrosing Alopecia Severity Index (FFASI): a validated scoring system for assessing frontal fibrosing alopecia

Holmes, S ; Ryan, T ; Young, D ; Harries, M

British journal of dermatology (1951), 2016-07, Vol.175 (1), p.203-207; England: Blackwell Publishing Ltd

“ (...) using the Frontal Fibrosing Alopecia Severity Index. Table S2. Kendall's coefficients of concordance W for intraobserver clinical assessments... ”

PEER-REVIEWED OPEN ACCESS

Disponible en ligne >

Haut

Citations

Consulter en ligne

Détails

Liens

Envoyer vers

Citations

Rechercher les références qui citent celle-ci ou les références citées dans

Consulter en ligne

Disponibilité du texte intégral

Wiley Online Library Database Model 2018
Disponible depuis 1997 volume: 136 fascicule : 1. Afficher la licence

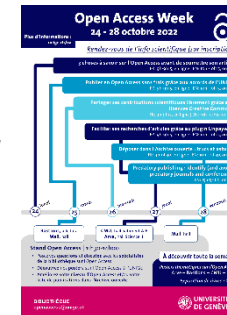
Version Open Access du texte intégral trouvée grâce à :Unpaywall Afficher la licence

https://unige.swisscovery.slsp.ch/permalink/41SLSP_UGE/177j9fn/cdi_proquest_miscellaneous_1809048606

QUELQUES SUGGESTIONS

- Un ppt de cours librement réutilisable: <https://doi.org/10.5281/zenodo.7856993> (anglais) et <https://doi.org/10.5281/zenodo.8304684> (français)
- Quand vous présentez l'outil aux usagers pour sa partie «accès» il vaut la peine de
 - Bien expliquer les versions de publication
 - Les inviter à déposer leurs manuscrits auteur-es dans une archive indexée

«Vous aussi, rendez vos articles disponibles en les ajoutant dans l'Archive ouverte UNIGE: <https://archive-ouverte.unige.ch/>»



- Contacter unpaywall si votre archive n'est pas bien indexée dans l'outil
- Trouver des exemples clés: Identifier quelques articles (idéalement par des auteur-es de votre institution) absents de vos abonnements pour lesquels une version librement accessible existe dans unpaywall/votre archive



QUESTIONS ?

Floriane.Muller@unige.ch



Floriane Muller, Bibliothèque de l'UNIGE, 2023

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(basé sur une présentation de Floriane Muller, Bibliothèque de l'UNIGE, 2021 révisée en 2022 avec Anouk Santos, puis revue et complétée par Floriane Muller en 2023)