repiSalud
Repositorio Institucional en Salud

THE INSTITUTIONAL REPOSITORY

HOW TO COMPLY WITH OPEN ACCESS MANDATES

Irene Maseda

October 2018

GOBIERNO DE ESPAÑA
MINISTERIO DE CIENCIA, INNOVACIÓN Y UNIVERSIDADES
Instituto de Salud Carlos III
Fundaciónprocnic
EXCELENCIA SEVERO OCHOA
cnic

"Open Access is about making research findings freely available to anyone"

What is OA and why it is important

How it works

Repisalud

OA REMOVES BARRIERS

WHEN WE PUBLISH

FINANCIAL
LEGAL
TECHNOLOGICAL

WHEN WE READ

FINANCIAL
LEGAL
TECHNOLOGICAL

FREE (GRATIS) ≠ FREE (LIBRE)

OA Mandates

SPAIN

LEY DE LA CIENCIA (Art. 37)

Archive a copy of all scientific publication in a repository, within 12 months

PLAN ESTATAL 2017-2020

All peer-reviwed scientific publications MUST be archived in an institutional or/and international repository

OA publications must be taken into account in the project evaluation

EUROPE-H2020

WHAT?

all peer-reviewed scientific publications

Optional:

- books
- conference proceedings
- grey literature

WHEN?

within 6 months

GOAL 2020:

100% Research Open Access

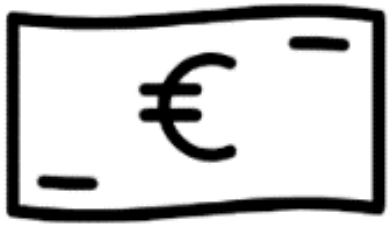
What is OA and why it is important

How it works

Repisalud

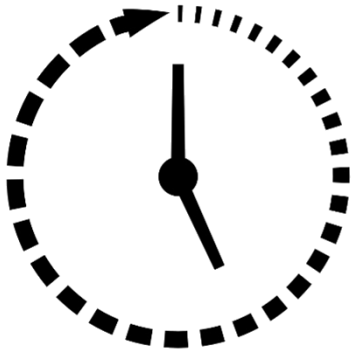
OA JOURNAL

HYBRID JOURNAL



APC

COPYRIGHT LICENCE MODELS



EMBARGO PERIODS

REPOSITORIES

How it Works: Benefits of OA

Make exposure for you work

Higher citation rates

Rapid dissemination of findings

Keep control of your autor's rights

Compilant with funder's mandates

RESEARCHERS

More institutional visibility

Gather all scientific production

Compilant with funder's mandates

INSTITUTIONS

Researchers in developing countries can see your work

The public can access your findigs

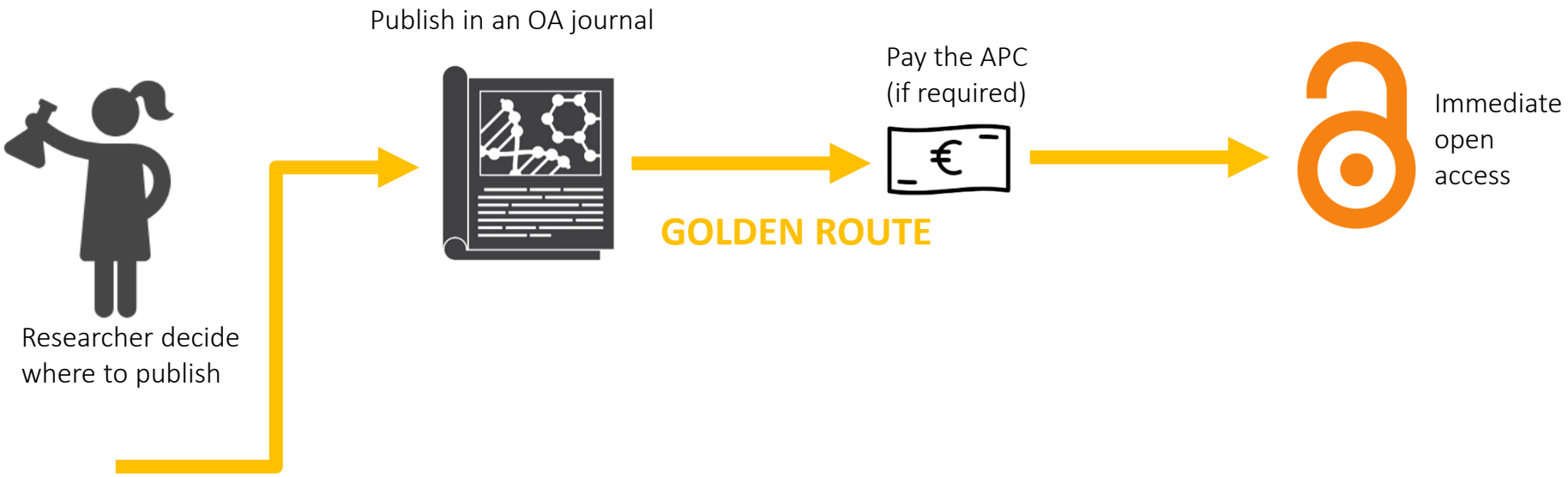
Taxpayers get value for money

THE SOCIETY

Get more readers and citations

... EVEN FOR JOURNALS

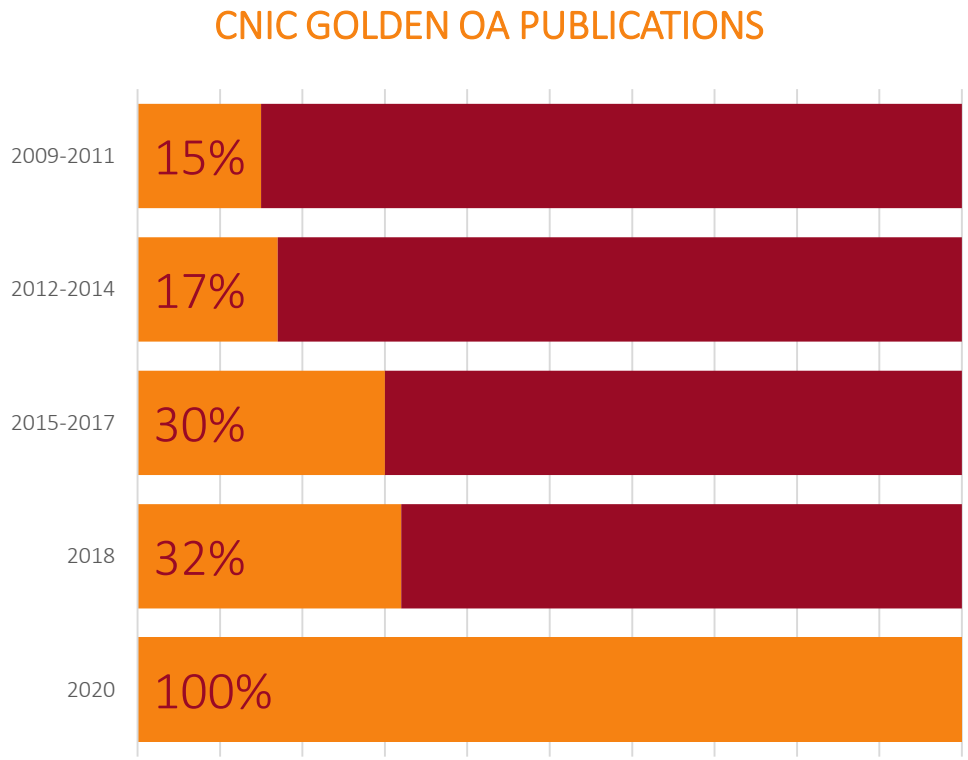
How it Works: Open Access Routes



SOURCE: www.fosteropenscience.eu

Objective 2020

TO HAVE ALL SCIENTIFIC PUBLICATIONS IN OPEN ACCESS

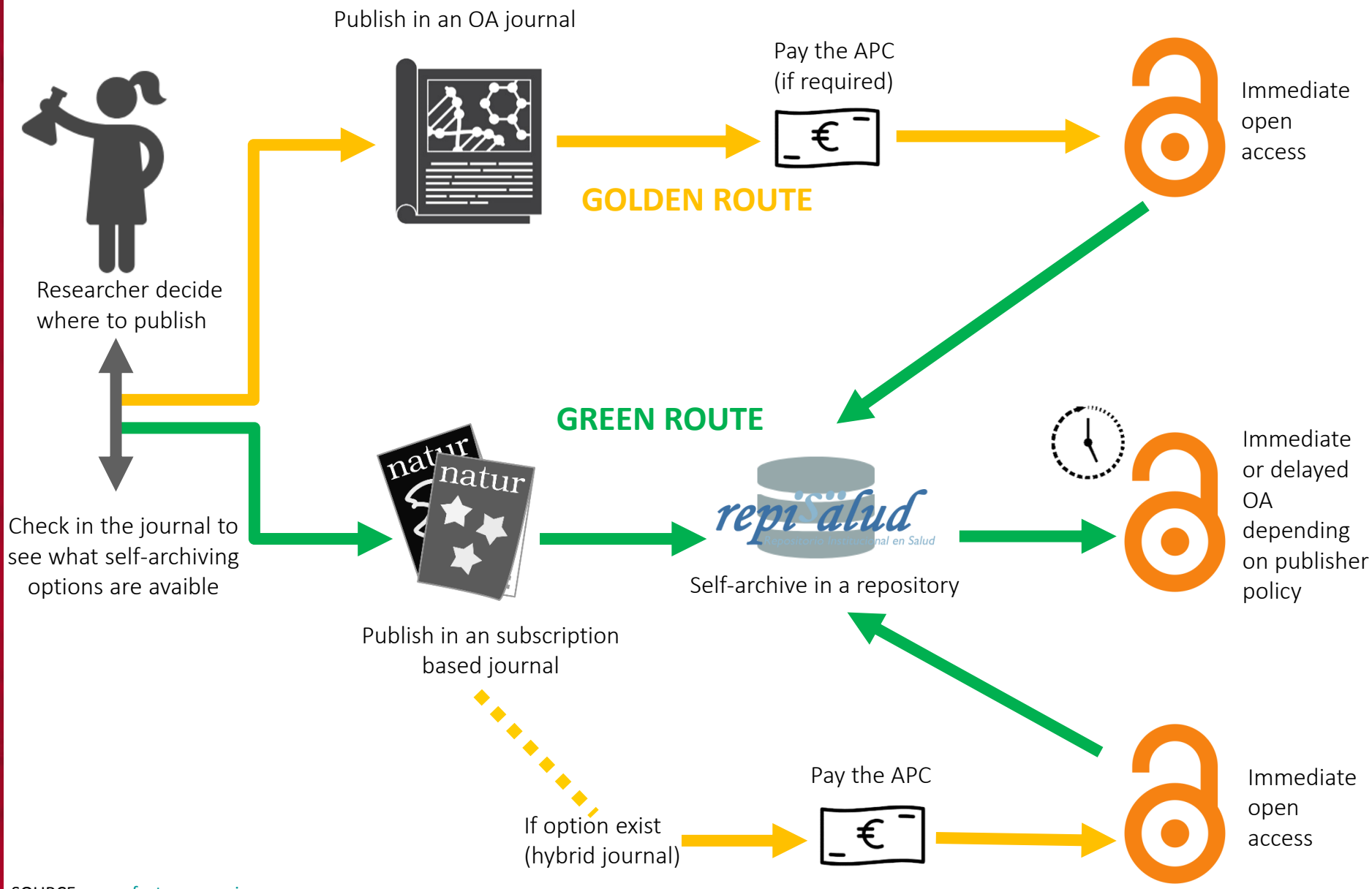


WHAT IS THE COST?

SOLUTION:

TO ARCHIVE IN INSTITUTIONAL REPOSITORIES

How it Works: Open Access Routes



SOURCE: www.fosteropenscience.eu

How it Works: Benefits of OA

Make exposure for you work

Higher citation rates

Rapid dissemination of findings

Keep control of your autor's rights

Compilant with funder's mandates

RESEARCHERS

More institutional visibility

Gather all scientific production

Compilant with funder's mandates

INSTITU

Researchers in developing countries can see your work

The public can access your findigs

Taxpayers get value for money

THE SOCIETY

Get more readers and citations

… EVEN FOR JOURNALS

What is OA and why it is important

How it works

Repisalud

Before depositing…

1	CHECK THE JOURNAL AUTO-ARCHIVE POLICIES
2	KNOW THE MANUSCRIPT VERSION YOU CAN USE

Journal self-archive options

SHERPA/RoMEO

. . . opening access to research

Home • Search • Journals • Publishers • FAQ • Suggest • About

Search - Publisher copyright policies & self-archiving

English | Español | Magyar | Nederlands | Português

One journal found when searched for: **curr opin cell biol**

Journal:	Current Opinion in Cell Biology (ISSN: 0955-0674, ESSN: 1879-0410)
RoMEO:	This is a RoMEO **green** journal
Paid OA:	A paid open access option is **available** for this journal.
Author's Pre-print:	✓ author **can** archive pre-print (ie pre-refereeing)
Author's Post-print:	✓ author **can** archive post-print (ie final draft post-refereeing)
Publisher's Version/PDF:	✗ author **cannot** archive publisher's version/PDF
General Conditions:	• Authors pre-print on any website, including arXiv and RePEC • Author's post-print on author's personal website immediately • Author's post-print on open access repository after an embargo period of 12 months • Permitted deposit due to Funding Body, Institutional and Governmental policy or mandate, may be required to comply with emb • Author's post-print may be used to update arXiv and RepEC • Publisher's version/PDF cannot be used • Must link to publisher version with DOI • Author's post-print must be released with a Creative Commons Attribution Non-Commercial No Derivatives License
Mandated OA:	(*Awaiting information*)
Paid Open Access:	Open Access
Copyright:	Unleashing the power of academic sharing - Sharing Policy - Sharing and Hosting Policy FAQ - Green open access - Journal Embarg List for UK Authors - Attaching a User License (pdf) - Funding Body Agreements
Updated:	01-Jul-2016 - Suggest an update for this record
Link to this page:	http://www.sherpa.ac.uk/romeo/issn/0955-0674/
Published by:	Elsevier: **12 months** [Commercial Publisher] - *Green Policies in RoMEO*
For:	**Current Opinion** [Imprint]
Guidance:	*Please see the list of Publisher Categories in RoMEO for guidance on interpreting the priority of multiple publishers.*

These summaries are for the journal's *default* policies, and changes or exceptions can often be negotiated by autho

All information is correct to the best of our knowledge but should not be relied upon for legal advice.

SHERPA/ROMEO http://www.sherpa.ac.uk/romeo

PUBLISHER WEBSITE

Elsevier supports responsible sharing

Find out how you can share your research published in Elsevier journals.

Green Open Access (e.g. self-archiving)

Authors can share their research in a variety of different ways and Elsevier has a number of green open access options available. We recommend authors see our green open access page for further information. An author can also self-archive their author manuscript immediately and enable public access from their institution's repository after an embargo period. This is the version that has been accepted for publication and which typically includes author-incorporated changes suggested during submission, peer review and in editor-author communications.

An author is entitled to post the accepted manuscript in their institution's repository and make this public after an embargo period (known as green Open Access). The embargo period for this journal can be found below.

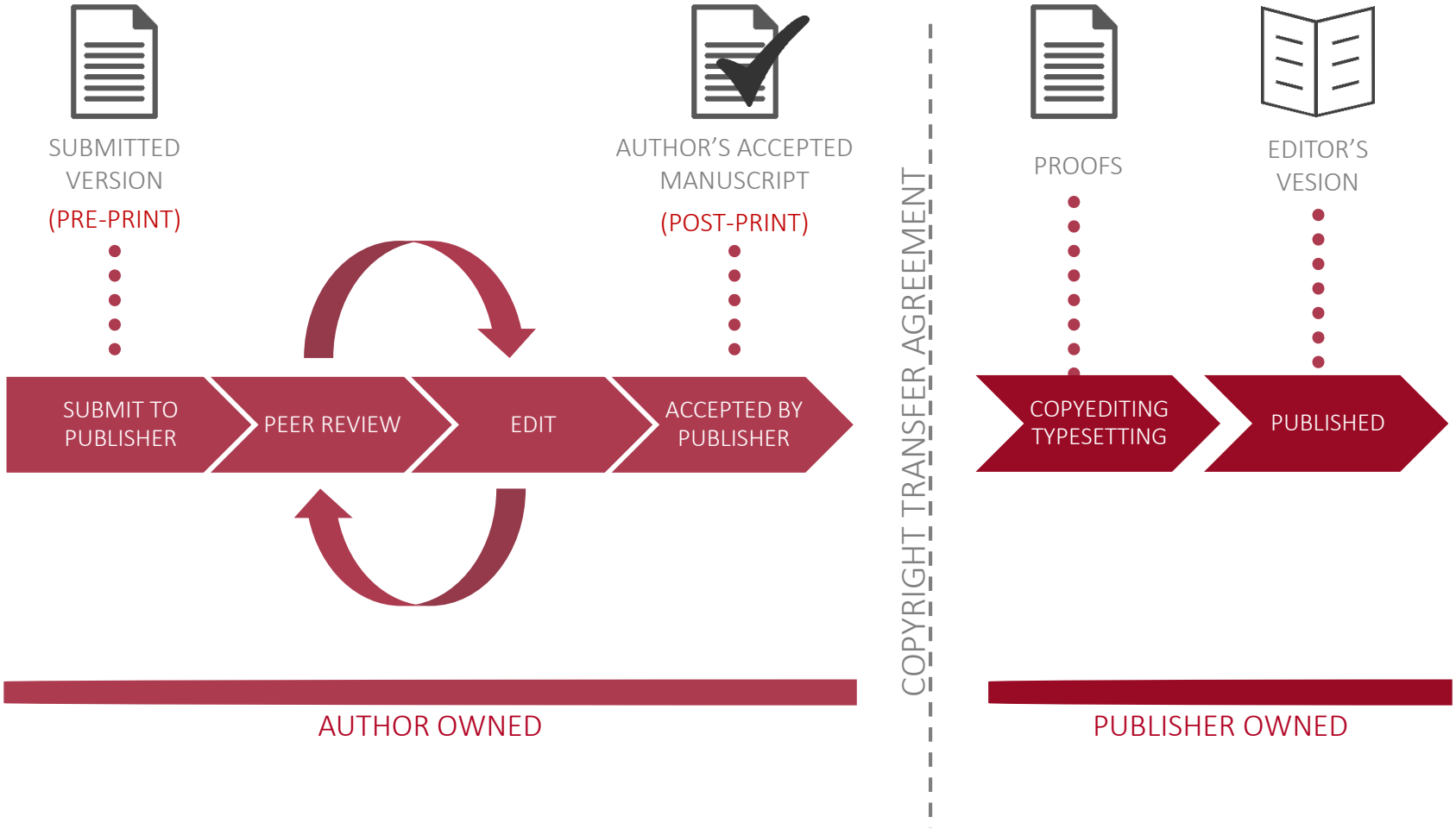
The published journal article cannot be shared publicly, for example on ResearchGate or Academia.edu, to ensure the sustainability of peer-reviewed research in journal publications.

Embargo Period

For subscription articles, an appropriate amount of time is needed for journals to deliver value to subscribing customers before a manuscript becomes available for free to the public. This is called an embargo period and it begins from the date the article is formally published online in its final and fully citable form. Find out more

This journal has an embargo period of 12 months.

Version Matters!!

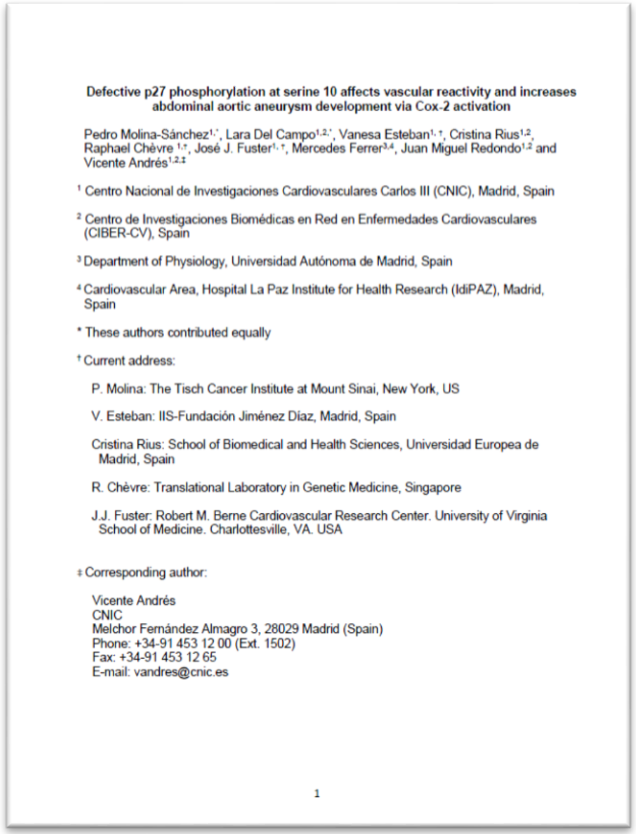


SOURCE: Claire Sewell

AUTHOR'S ACCEPTED MANUSCRIPT

EDITOR'S VERSION

SAME CONTENT



Defective p27 phosphorylation at serine 10 affects vascular reactivity and increases abdominal aortic aneurysm development via Cox-2 activation

Pedro Molina-Sánchez[1,*], Lara Del Campo[1,2,*], Vanesa Esteban[1,†], Cristina Rius[1,2], Raphael Chèvre [1,†], José J. Fuster[1,†], Mercedes Ferrer[3,4], Juan Miguel Redondo[1,2] and Vicente Andrés[1,2,‡]

[1] Centro Nacional de Investigaciones Cardiovasculares Carlos III (CNIC), Madrid, Spain

[2] Centro de Investigaciones Biomédicas en Red en Enfermedades Cardiovasculares (CIBER-CV), Spain

[3] Department of Physiology, Universidad Autónoma de Madrid, Spain

[4] Cardiovascular Area, Hospital La Paz Institute for Health Research (IdiPAZ), Madrid, Spain

* These authors contributed equally

† Current address:

P. Molina: The Tisch Cancer Institute at Mount Sinai, New York, US

V. Esteban: IIS-Fundación Jiménez Díaz, Madrid, Spain

Cristina Rius: School of Biomedical and Health Sciences, Universidad Europea de Madrid, Spain

R. Chèvre: Translational Laboratory in Genetic Medicine, Singapore

J.J. Fuster: Robert M. Berne Cardiovascular Research Center. University of Virginia School of Medicine. Charlottesville, VA. USA

‡ Corresponding author:

Vicente Andrés
CNIC
Melchor Fernández Almagro 3, 28029 Madrid (Spain)
Phone: +34-91 453 12 00 (Ext. 1502)
Fax: +34-91 453 12 65
E-mail: vandres@cnic.es

Journal of Molecular and Cellular Cardiology 116 (2018) 5–15

Contents lists available at ScienceDirect

Journal of Molecular and Cellular Cardiology

journal homepage: www.elsevier.com/locate/yjmcc

Original article

Defective p27 phosphorylation at serine 10 affects vascular reactivity and increases abdominal aortic aneurysm development via Cox-2 activation

Pedro Molina-Sánchez[a,1,2], Lara Del Campo[a,b,1], Vanesa Esteban[a,3], Cristina Rius[a,b,4], Raphael Chèvre[a,5], José J. Fuster[a,6], Mercedes Ferrer[c,d], Juan Miguel Redondo[a,b], Vicente Andrés[a,b,*]

[a] Centro Nacional de Investigaciones Cardiovasculares Carlos III (CNIC), Madrid, Spain
[b] Centro de Investigación Biomédica en Red en Enfermedades Cardiovasculares (CIBER-CV), Spain
[c] Department of Physiology, Universidad Autónoma de Madrid, Spain
[d] Cardiovascular Area, Hospital La Paz Institute for Health Research (IdiPAZ), Madrid, Spain

ARTICLE INFO

Keywords:
p27
p27 phosphorylation at serine 10
Cox-2
Endothelial cell
Aneurysm
Vascular contractility

ABSTRACT

Phosphorylation at serine 10 (S10) is the major posttranslational modification of the tumor suppressor p27, and is reduced in both human and mouse atherosclerosis. Moreover, a lack of p27 phospho S10 in apolipoprotein E null mice (apoE−/−) leads to increased high-fat diet-induced atherosclerosis associated with endothelial dysfunction and augmented leukocyte recruitment. In this study, we analyzed whether p27 phospho S10 modulates additional endothelial functions and associated pathologies. Defective p27 phospho S10 increases COX-2 activity in mouse aortic endothelial cells without affecting other key regulators of vascular reactivity, reduces endothelium-dependent dilation, and increases arterial contractility. Lack of p27 phospho S10 also elevates aortic COX-2 expression and thromboxane A_2 production, increases aortic lumen diameter, and aggravates angiotensin II-induced abdominal aortic aneurysm development in apoE−/− mice. All these abnormal responses linked to defective p27 phospho S10 are blunted by pharmacological inhibition of COX-2. These results demonstrate that defective p27 phospho S10 modifies endothelial behavior and promotes aneurysm formation via COX-2 activation.

1. Introduction

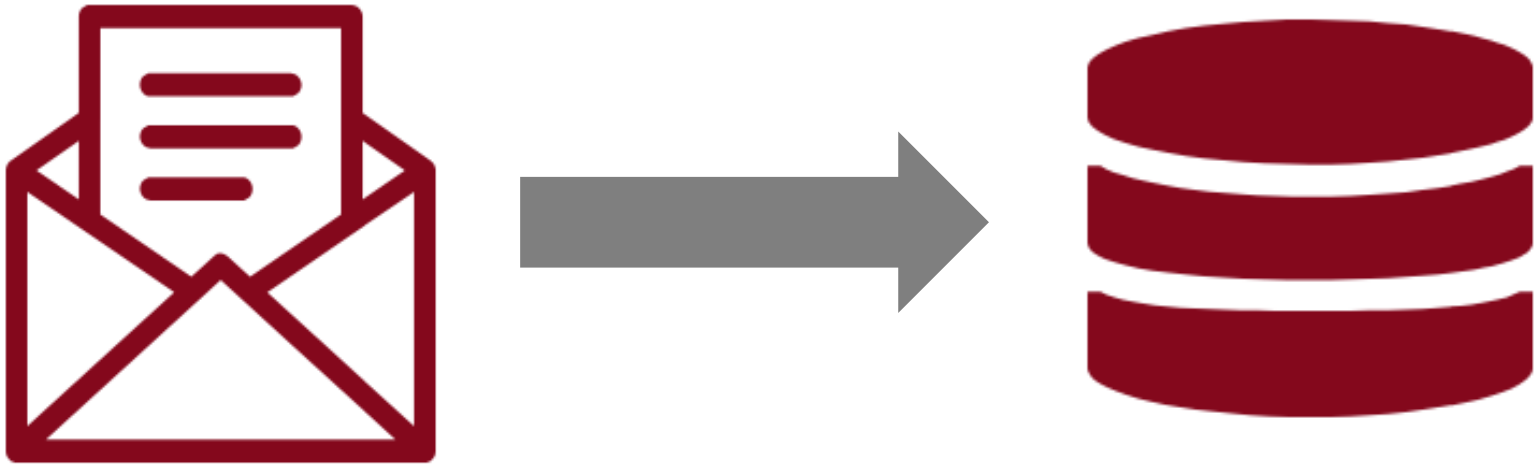
The endothelium is a key player in the maintenance of vascular homeostasis. Among other functions, endothelial cells (ECs) regulate leukocyte trafficking [1], angiogenesis [2], coagulation [3], vascular tone and arterial blood pressure [4]. Endothelial dysfunction leads to local and systemic alterations that contribute to cardiovascular diseases, and is strongly associated with hypertension [5,6], a common cardiovascular risk factor. ECs modulate the behavior of vascular smooth muscle cells (VSMCs), affecting their contractile capacity through the release of a wide variety of vasoactive factors, such as nitric oxide (NO), prostaglandins (PGI_2, PGE_2, etc.), and thromboxane (TX). An imbalance in the synthesis of these agents, caused by dysfunction of their main enzymatic producers (NO synthases, cyclooxygenases, and PG and TX synthases) can lead to hypertension [8] and generate or aggravate vascular pathological manifestations. Endothelial dysfunction also promotes other vascular disorders, such as atherosclerosis or some types of aneurysm, at least in part through overexpression of adhesion molecules that promote leukocyte extravasation and accumulation within the inflamed arterial wall [7]. These events can alter the blood flow and induce medial degeneration [8], an early event in both atherosclerosis and aneurysm.

Cell cycle inhibitors have emerged as important protective agents against vascular disease and cardiovascular risk. $p27^{Kip1}$ is a member of the Cip/Kip family of cyclin-dependent kinase inhibitors (CKIs) that inhibits VSMC proliferation and migration in vitro [9,10]. Studies in the

* Corresponding author at: CNIC, Melchor Fernández Almagro 3, Madrid 28029, Spain.
E-mail address: vandres@cnic.es (V. Andrés).
[1] These authors contributed equally.
[2] Current address: The Tisch Cancer Institute at Mount Sinai, New York, USA.
[3] Current address: IIS-Fundación Jiménez Díaz, Madrid, Spain.
[4] Current address: School of Biomedical and Health Sciences, Universidad Europea de Madrid, Spain.
[5] Current address: Translational Laboratory in Genetic Medicine, Singapore.
[6] Current address: Robert M. Berne Cardiovascular Research Center, University of Virginia School of Medicine, Charlottesville, VA, USA.

http://doi.org/10.1016/j.yjmcc.2018.01.010
Received 25 August 2017; Received in revised form 10 January 2018; Accepted 15 January 2018

When should I deposit?



DEPOSIT AT THE TIME OF ACCEPTANCE

SET THE EMBARGO PERIOD

- ✓ Permanent link
- ✓ Metadata
- ✓ Indexed in OAI harvesters

https://repisalud.isciii.es/

Communities in REPISALUD

Research

Scientific papers, congress contributions, research data, books and book chapters, patents and theses.

Institutional

Scientific reports, outreach material (brochures, posters, et.) and working papers.

Teaching

Teaching material from courses given at these institutions or by their staff, such as guides and tutorials.

Scientific events

Conferences, seminars, congresses organized by one of these institutions.

Institutional publisher

Books and journals published by one of these institutions.

EACH COMMUNITY IS DIVIDED IN THREE SUB-COMMUNITIES:

- ISCIII
- CNIC
- CNIO

Tweets by @REPISALUDisciii

Repisalud Retweeted

dadun
@DadunUnav

Repository finder, un buscador de repositorios que conocemos a través de @jalonsoarevalo repositoryfinder.datacite.org

Oct 8, 2018

Repisalud Retweeted

Concepcion Campos
@biblioGetafe

To improve search quality, NLM have applied advanced techniques, including machine learning and natural language processing, to

Embed View on Twitter

CNIC - Artículos : [155]

Browse

Issue Date | Author | Title | Document type | Institution | Subject

Subscribe to this collection to receive daily e-mail notification of new additions Subscribe

RSS 1.0 RSS 2.0 RSS

Collection's Items (Sorted by Submit Date in Descending order): 1 to 20 of 155

next >

Rights	Preview	Issue Date	Title	Type	Author(s)
		Mar-2018	Defective p27 phosphorylation at serine 10 affects vascular reactivity and increases abdominal aortic aneurysm development via Cox-2 activation	Artículo	Molina-Sanchez, Pedro; del Campo, Lara; Esteba, Vanesa, et al
		2014	Multiple evidence strands suggest that there may be as few as 19 000 human protein-coding genes	Artículo	Ezkurdia, Iakes; Juan, David; Manuel Rodriguez, Jose, et al
		2014	Noninvasive Molecular Imaging of Cell Death in Myocardial Infarction using In-111-GSAO	Artículo	Tahara, Nobuhiro; Zandbergen, H. Reinier; de Haas, Hans J., et al
		2014	Defective sarcoplasmic reticulum-mitochondria calcium exchange in aged mouse myocardium	Artículo	Fernandez-Sanz, C.; Ruiz-Meana, M.; Miro-Casas, E., et al

Refine

Author
Vazquez, Jesús (28)
Fuster, Valentin (16)
Ibanez, Borja (14)
Lopez, Juan Antonio (14)
Enriquez, José Antonio (10)
Ordovas, Jose M. (9)
Bueno, Héctor (8)
Fernandez-Jimenez, Rodrigo (8)
Sanchez-Madrid, Francisco (8)
Calvo, Enrique (7)
next >

Date issued
2018 (1)
2017 (35)
2016 (62)
2015 (45)
2014 (12)

Type
Artículo (155)

Institution
CNIC (155)
CNIO (2)

Repisalud: item

Por favor, use este identificador para citar o enlazar este Item: http://hdl.handle.net/20.500.12105/5186

Autor:	Marina-Zarate, Ester CNIC Perez-Garcia, Arantxa CNIC Ramiro, Almudena R CNIC
Título:	**CCCTC-Binding Factor Locks Premature IgH Germline Transcription and Restrains Class Switch Recombination**
Fecha de publicación:	2017
Editor:	FRONTIERS MEDIA SA
Referencia/citación:	Front Immunol. 2017; 8:1076
Identificador ISSN:	1664-3224
Identificador DOI:	10.3389/fimmu.2017.01076
Tipo de documento:	Artículo
Idioma:	Inglés
Palabras clave:	class switch recombination CCCTC-binding factor germline transcription activation-induced deaminase somatic hypermutation ACTIVATION-INDUCED DEAMINASE
Resumen:	In response to antigenic stimulation B cells undergo class switch recombination (CSR) at the immunoglobulin heavy chain (IgH) to replace the primary IgM/IgD isotypes by IgG, IgE, or IgA. CSR is initiated by activation-induced cytidine deaminase (AID) through the deamination of cytosine residues at the switch (S) regions of IgH. B cell stimulation promotes germline transcription (GLT) of specific S regions, a necessary event prior to CSR because it facilitates AID access to S regions. Here, we show that CCCTC-binding factor (CTCF)-deficient mice are severely impaired in the generation of germinal center B cells and plasma cells after immunization in vivo, most likely due to impaired cell survival. Importantly, we find that CTCF-deficient B cells have an increased rate of CSR under various stimulation conditions in vitro. This effect is not secondary to altered cell proliferation or AID expression in CTCF-deficient cells. Instead, we find that CTCF-deficient B cells harbor an increased mutation frequency at switch regions, probably reflecting an increased accessibility of AID to IgH in the absence of CTCF. Moreover, CTCF deficiency triggers premature GLT of S regions in naive B cells. Our results indicate that CTCF restricts CSR by enforcing GLT silencing and limiting AID access to IgH.

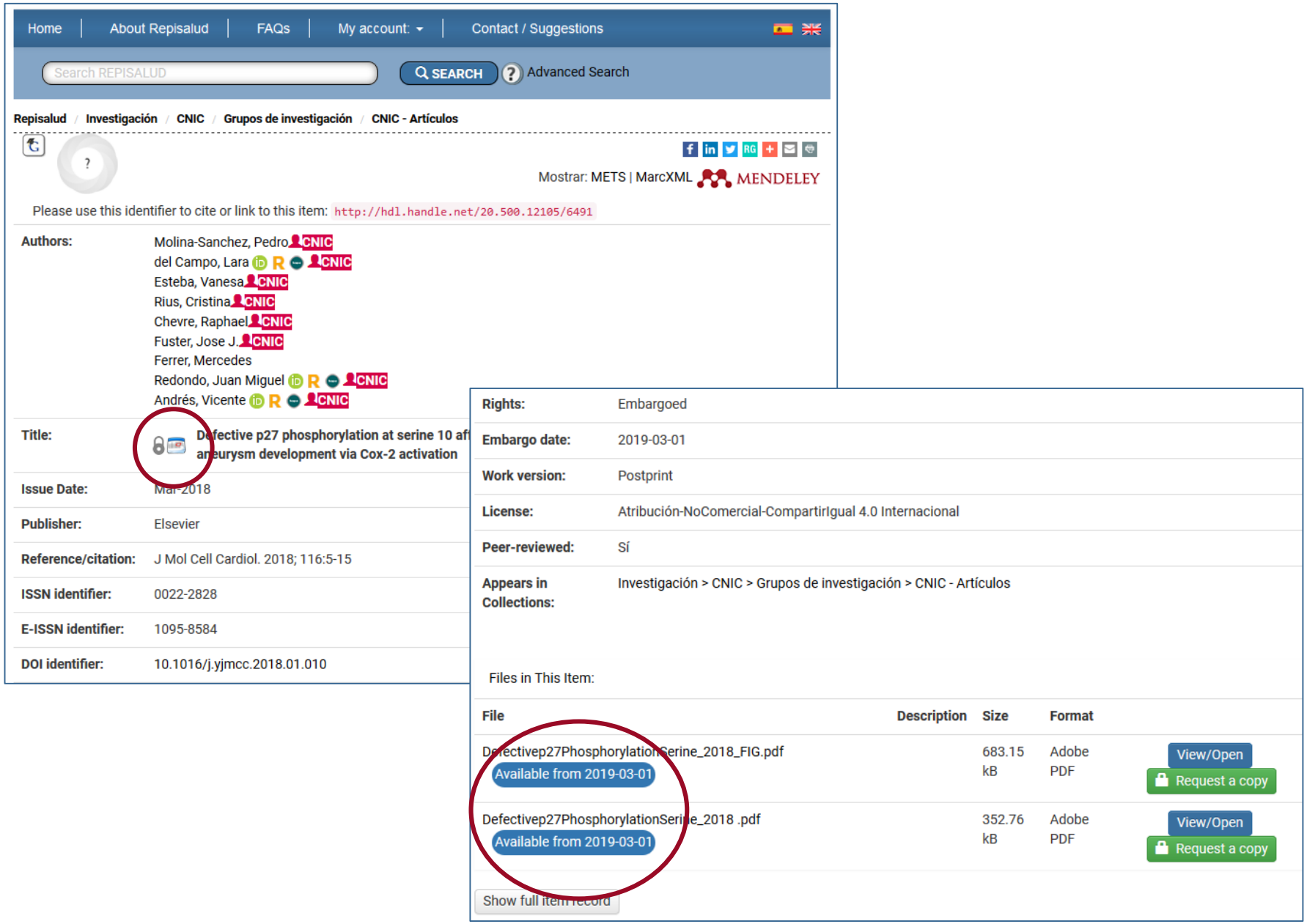
Patrocinadores:	The authors thank all members of the B Cell Biology Laboratory, J Mendez and VG de Yebenes for critical reading of the manuscript, F Alvarez-Prado for help with sequence analysis, F Sanchez-Cabo for advise on statistics analysis, and N Galjart and K Rajewsky for kindly providing the CTCF^{fl/+} and the CD19-Cre^{ki/+} mice, respectively. AP-G was a fellow of the research training program (FPU-AP2009-1732) funded by the Ministerio de Educacion, Cultura y Deporte; EM-Z is a fellow of the research training program (FPI) funded by the Ministerio de Economia y Competitividad (BES-2014-069525); AR is supported by Centro Nacional de Investigaciones Cardiovaculares (CNIC). This work was funded with the following grants to AR: SAF2013-42767-R and SAF2016-75511-R (Plan Estatal de Investigacion Cientifica y Tecnica y de Innovacion 2013-2016 Programa Estatal de I+D+i Orientada a los Retos de la Sociedad Retos Investigacion: Proyectos I+D+i 2016, Ministerio de Economia, Industria y Competitividad) and co-funding by Fondo Europeo de Desarrollo Regional (FEDER) and the European Research Council Starting Grant program (BCLYM-207844). The CNIC is supported by the Ministry of Economy, Industry and Competitiveness (MEIC) and the Pro CNIC Foundation and is a Severo Ochoa Centre of Excellence (MEIC award SEV-2015-0505).
Agencia financiadora:	Ministerio de Educación, Cultura y Deporte (España) Ministerio de Economía y Competitividad (España) Fundación ProCNIC European Regional Development Fund (ERDF/FEDER) European Research Council Ministerio de Economía, Industria y Competitividad (España)
Versión del Editor:	https://doi.org/10.3389/fimmu.2017.01076
Código de financiación:	MINECO/ICTI2013-2016/SEV-2015-0505 MINECO/ICTI2013-2016/BES-2014-069525 MINECO/ICTI2013-2016/SAF2016-75511-R MINECO/ICTI2013-2016/SAF2013-42767-R info:eu-repo/grantAgreement/EC/FP7/207844
Derechos:	Acceso abierto
Versión del trabajo:	Publisher's version
Licencia de uso:	Atribución 4.0 Internacional
Peer-reviewed:	Sí
Aparece en las colecciones:	Investigación > CNIC > Grupos de investigación > CNIC - Artículos

Ficheros en este Item:

Fichero	Descripción	Tamaño	Formato	
CCCTC-BindingFactorLocks_2017		3.16 MB	Adobe PDF	Ver

Mostrar el registro Dublin Core completo del Item

Repisalud: embargoed item



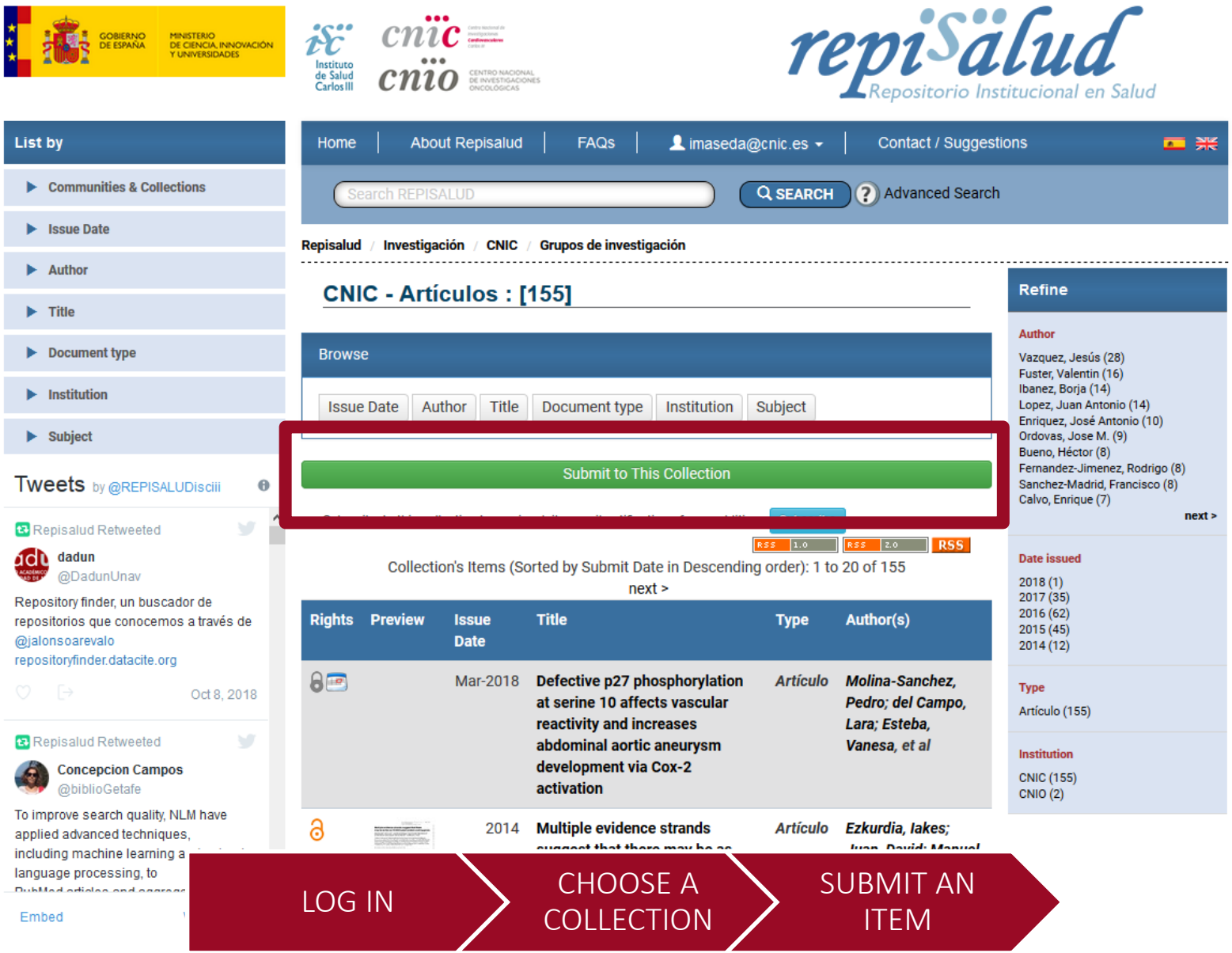
Repisalud: My account

NOTE: After you login for the first time you should send an email to repisalud@cnic.es to get privileges to archive

Repisalud: My account

To log in in Repisalud you have to use your institutional email and password

Repisalud: How do we deposit?



Repisalud: Three ways to send an item

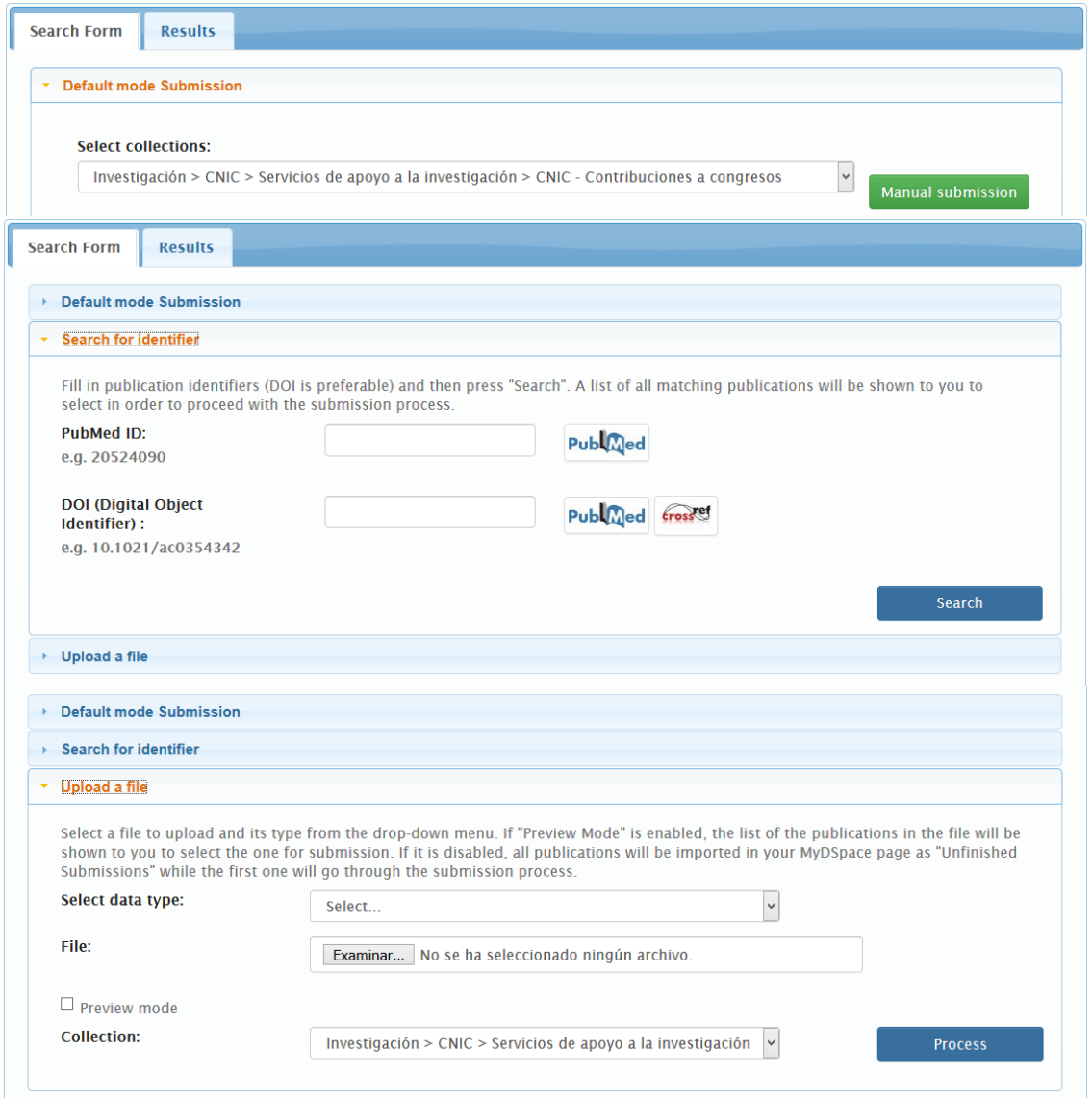
MANUAL SUBMISSION

SEARCH FOR IDENTIFIER

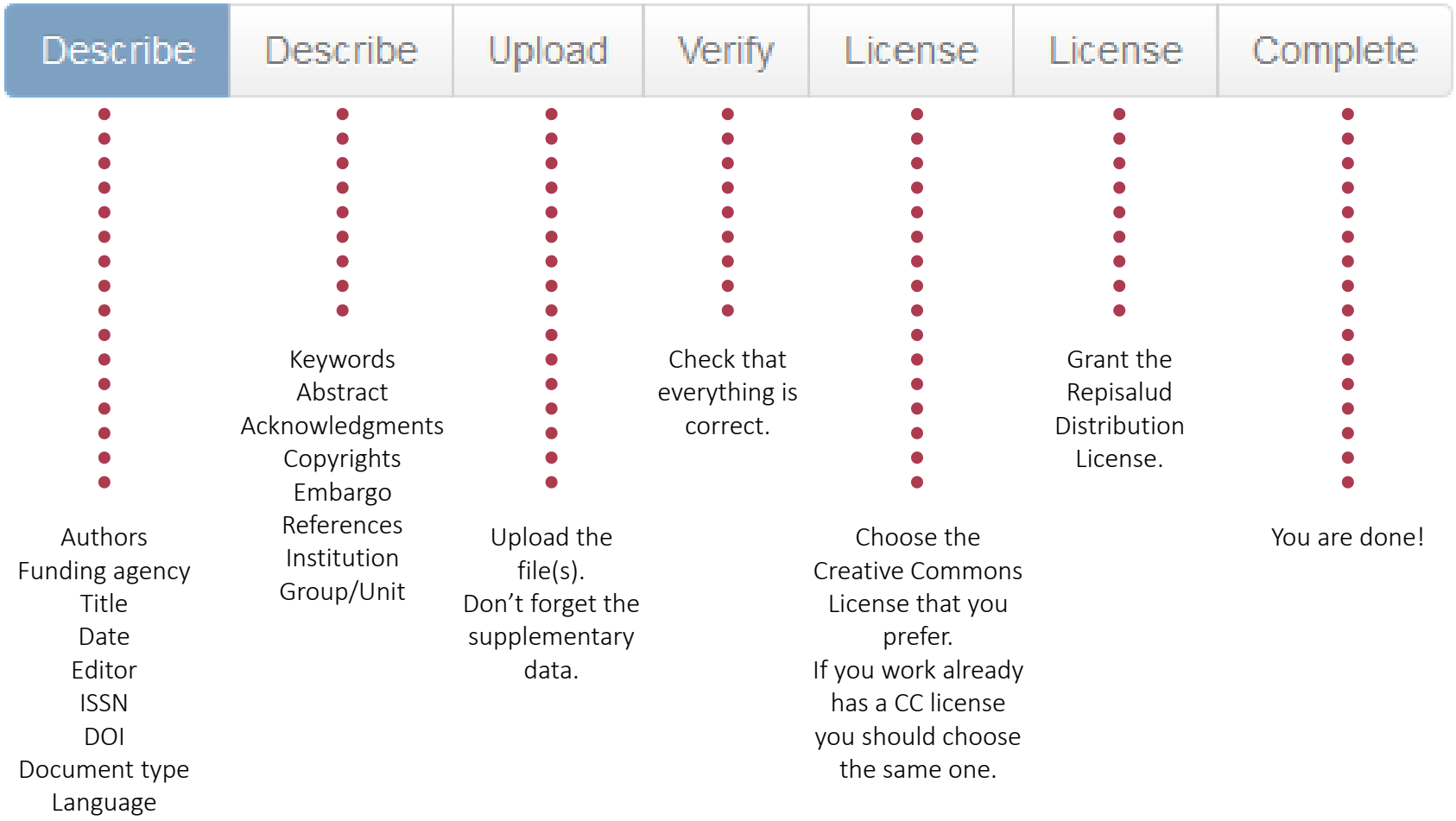
SOME FIELDS WILL BE AUTOMATICALLY FILLED IN

UPLOAD A FILE

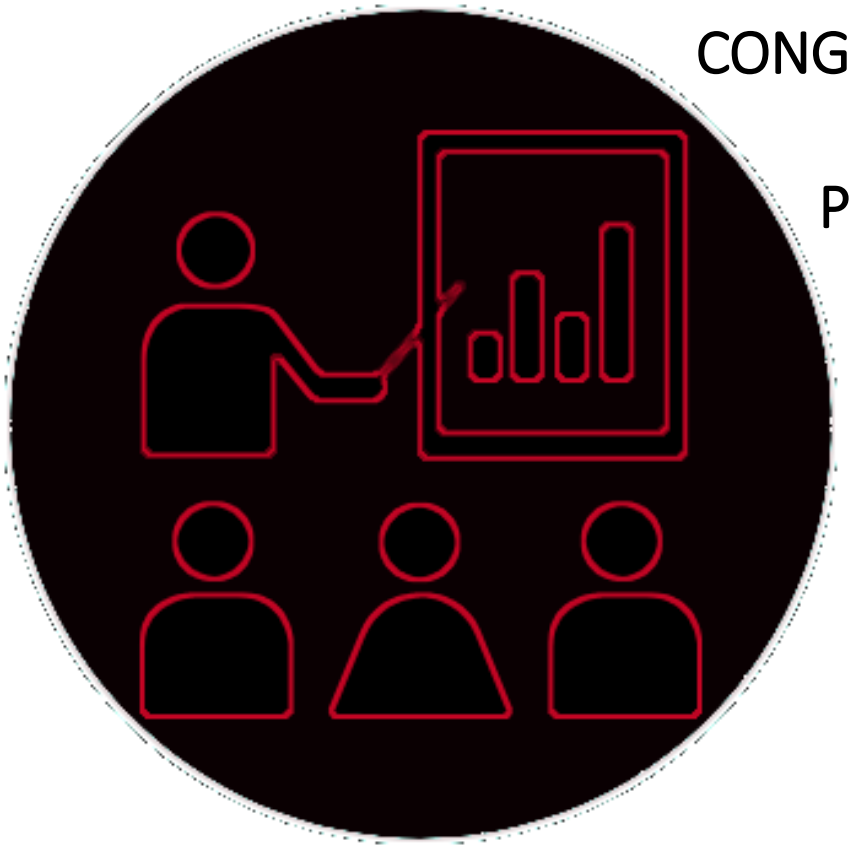
TO UPLOAD MULTIPLE ITEMS
FORMATS: pubmedxml, ris, endnote, bibtex…



Repisalud: Uploading an item



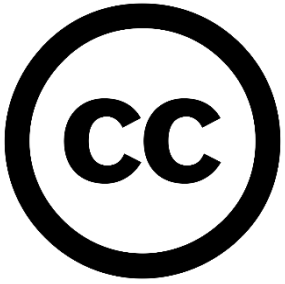
BEYOND JOURNAL ARTICLES

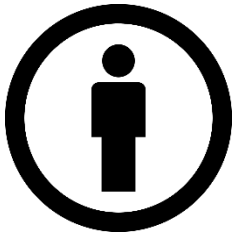


CONGRESS CONTRIBUTIONS

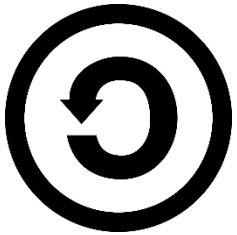
POSTERS

TEACHING MATERIALS



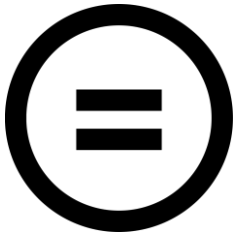


Attribution (BY)



ShareAlike (SA)

NonCommercial (NC)



NoDerivatives (ND)

Do you allow comercial uses of your work?

- Yes ✓
- No

Do you allow modifications of your work?

- Yes ✓
- No
- ShareAlike

Do you allow comercial uses of your work?

Yes

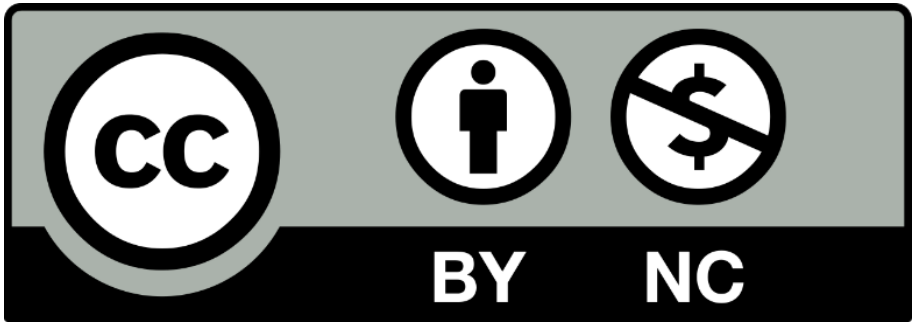
No ✓

Do you allow modifications of your work?

Yes ✓

No

ShareAlike

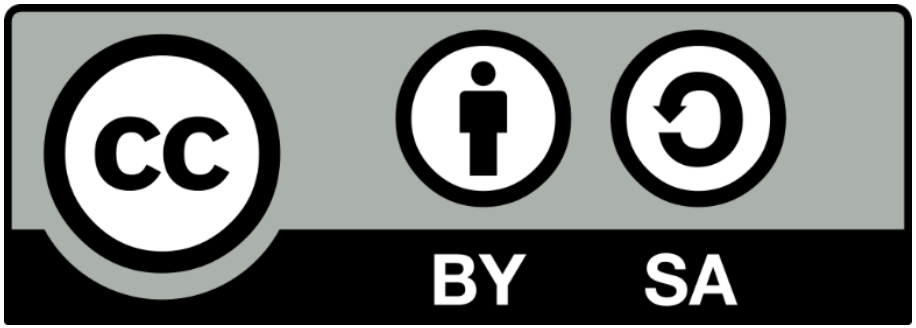


Do you allow comercial uses of your work?

- Yes ✓
- No

Do you allow modifications of your work?

- Yes ✓
- No
- ShareAlike ✓



Do you allow comercial uses of your work?

Yes ✓

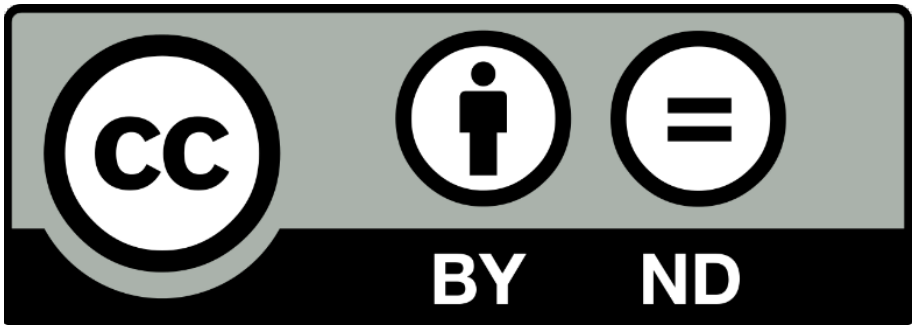
No

Do you allow modifications of your work?

Yes

No ✓

ShareAlike

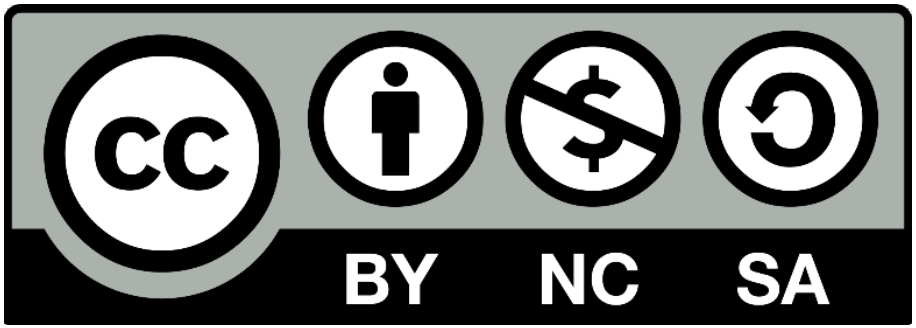


Do you allow comercial uses of your work?

- Yes
- No ✓

Do you allow modifications of your work?

- Yes ✓
- No
- ShareAlike ✓



Do you allow comercial uses of your work?

Yes

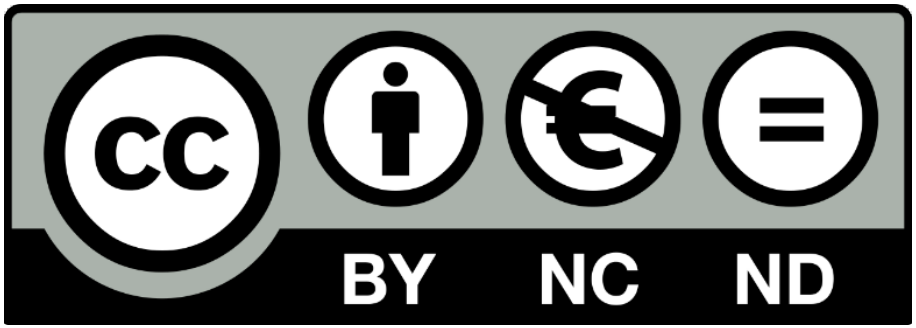
No ✓

Do you allow modifications of your work?

Yes

No ✓

ShareAlike



How far my research will go?

"Open Access is about making research findings freely available to anyone"

Questions about…?

#plagiarism

#pre-prints

#APC

#copyright negotiation