

UTILIDAD Y USABILIDAD de las plataformas de revistas científicas para LECTORES Y AUTORES:

DESCRIPCIÓN Y COMPARACIÓN de cuatro plataformas digitales de editoriales de ciencias de la salud

Blanca San José Montano

Biblioteca de Ciencias de la Salud del Hospital Universitario de Móstoles (Madrid).

María del Carmen Osuna Cortés

Documentación clínica. Hospital Universitario San Cecilio (Granada).

Manuel Espantaleón Agreda.

Biblioteca de Ciencias de la Salud del Hospital Clínico San Carlos (Madrid)

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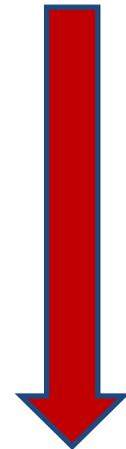
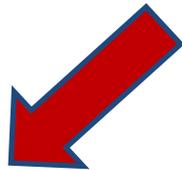


ACCESO: PLATAFORMAS DIGITALES

Arquitectura
Diseños interfaz
Elementos descubrimiento



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IMPORTANT!



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OBJETIVO

✓ **DESCRIBIR Y CONTRASTAR:**

- Funciones, diseño interfaz y soluciones de arquitectura

4

PLATAFORMAS “COMERCIALES” DIGITALES INTERNACIONALES DE BIOMEDICINA

✓ **RELACIONARLO** con su **BENEFICIO** para nuestros **USUARIOS** en sus diferentes roles:

- Autor, Editor, Lector, Revisor, Evaluador, Asesor...

SELECT

4

PLATAFORMAS “COMERCIALES” DIGITALES*:

1. **BMJ journals**  Revista: *Thorax*
2. **Elsevier, ScienceDirect**  Revista: *Lancet Diabetes&Endocrinology*
3. **Springer Medicine Journal**  Revista: *Intensive Care Medicine*
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*No incluimos plataformas “megajournals” con nuevas formas de publicación acceso abierto para homogeneizar resultados.



MÉTODO

✓ Checklist *recogida datos:*

- *Funciones*
- *Diseño interfaz*
- *Soluciones arquitectura*



✓ 2 NIVELES*

- *Revista*
- *Artículo*



✓ Rol usuarios *benefician recursos:*

- **A:** Autor 🗣️
- **B:** Bibliotecario, evaluador, asesor...
- **E:** Editor y revisor
- **L:** Lector



Nivel REVISTA Checklist



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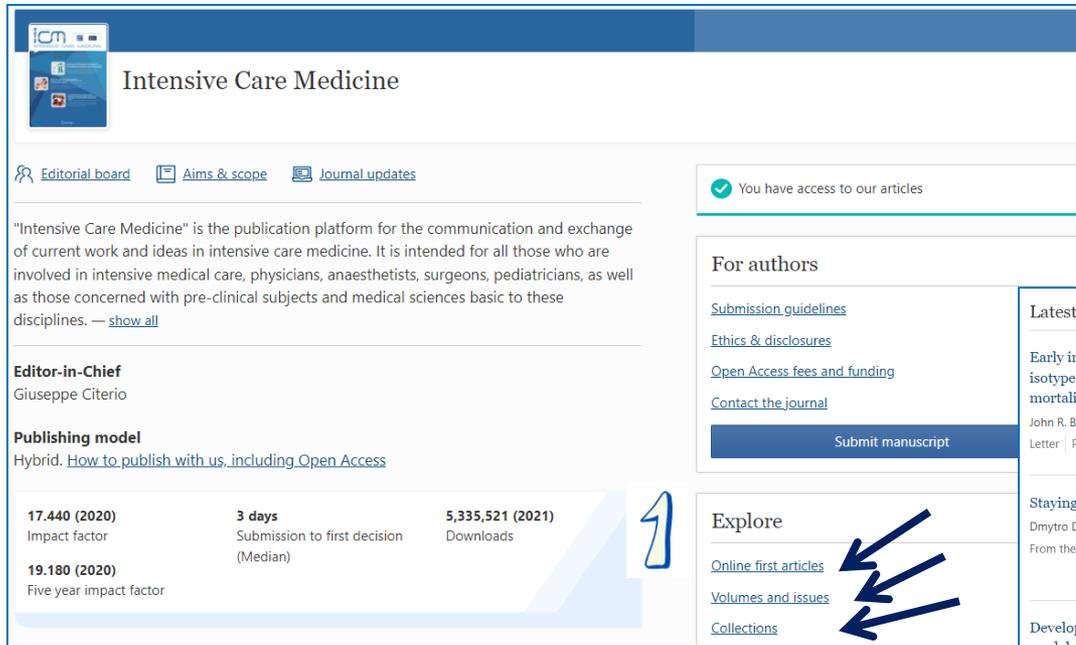
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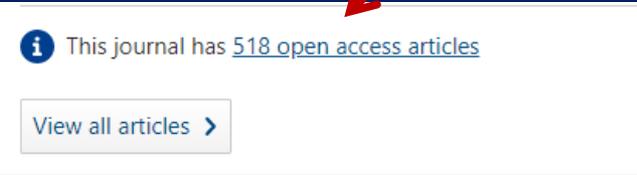
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Impact Factor: 9.250
Citescore: 13.5
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Thorax is an official journal of the British Thoracic Society and is a companion to BMJ Open Respiratory

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Acceptance Rate	Time to first decision	Impact Factor	Impact Factor rank (JCR)	Citescore	2020 total Altmetric mentions
13%	14 days*	9.250	7/64	13.5	9,718

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How we get these metrics

*mean number of days

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Thorax is BMJ's flagship respiratory medicine journal and has an open access companion journal, *BMJ Open Respiratory Research*.

Journal Statistics (2020)

Acceptance rate: 13%

Speed

Time to first decision: 14 days (mean)
Time from acceptance to publication: 26 days (mean)

Impact

Impact Factor category: Respiratory System
Impact Factor (JCR): 9.250
Impact Factor rank: 7/64
5 Year Impact Factor: 11.301
Journal Citation Indicator: 2.30
Eigenfactor Score: 0.02576
Citescore: 13.5
CiteScore rank: 6/133
SciMag Journal Rank (SJR): 3.083

Reach

2020 total content views: 1,705,409
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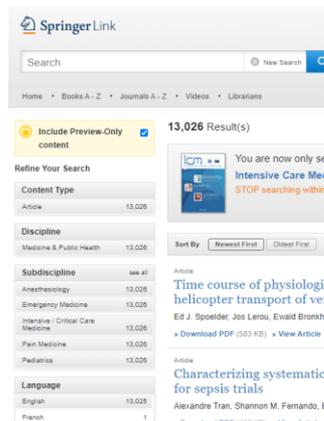


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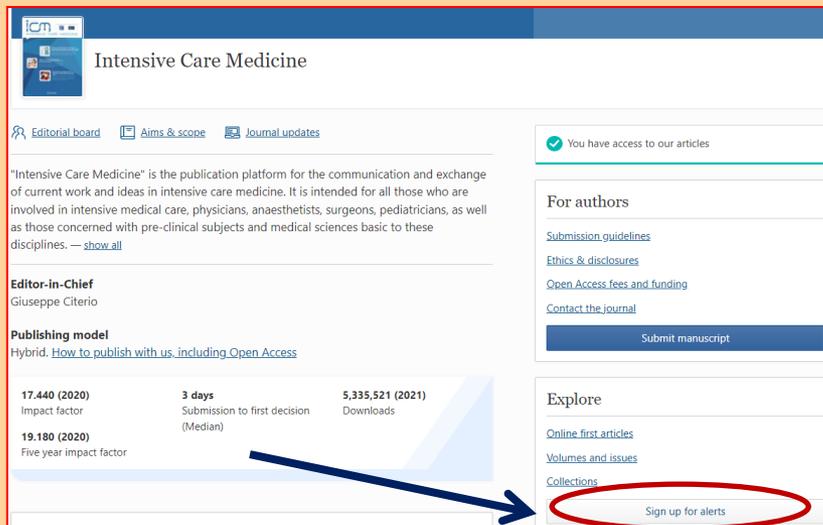
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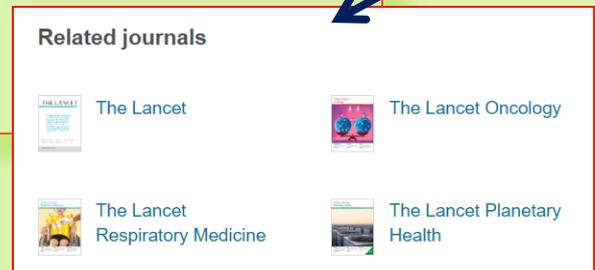


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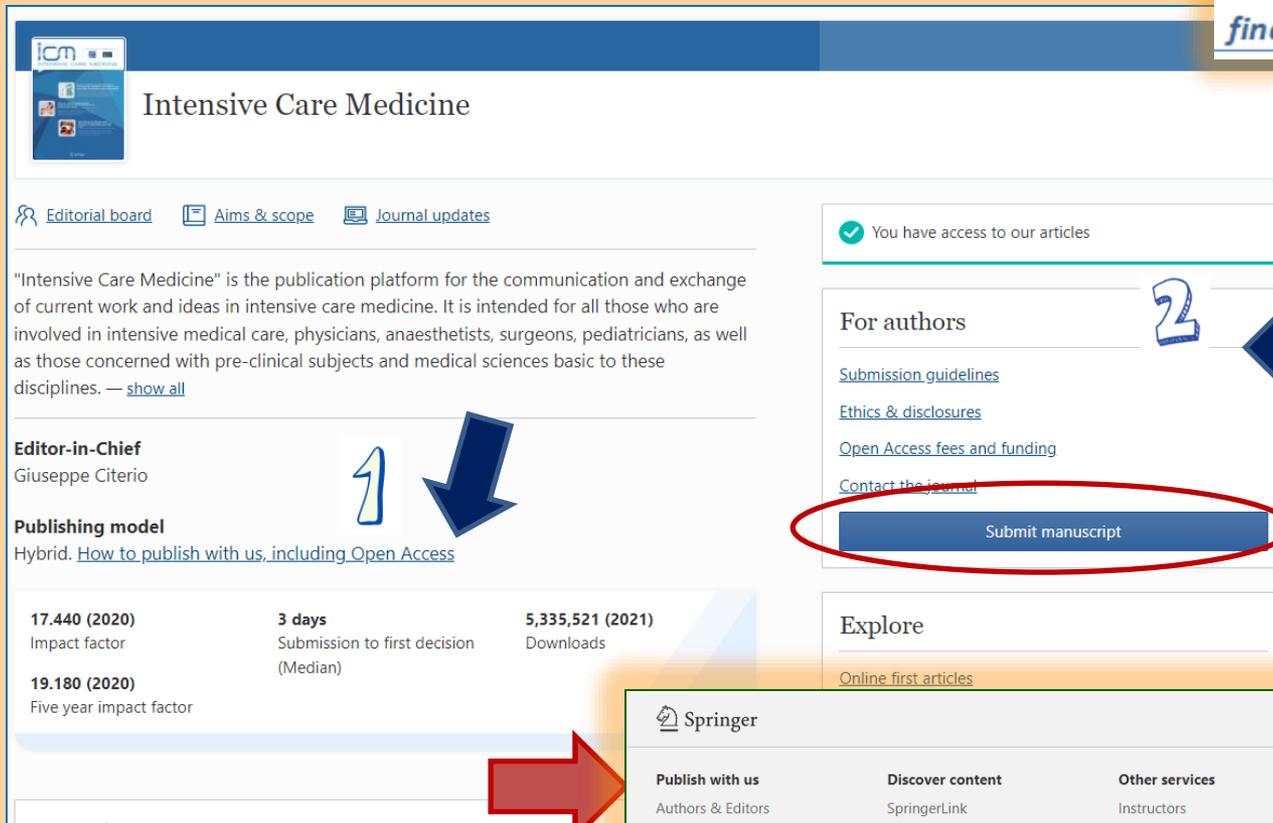
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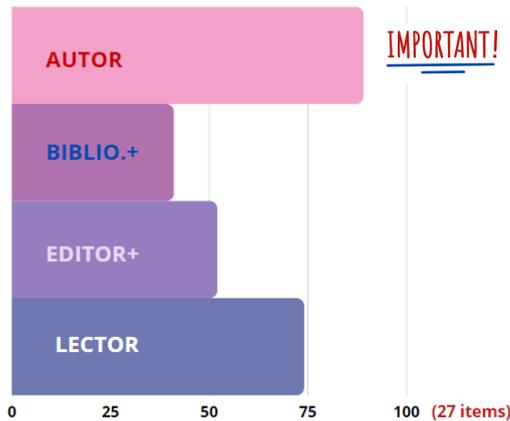
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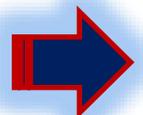


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Ashley K Clift
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Cancer Research UK Oxford Centre, Department of Oncology, University of Oxford, Oxford, UK
Adam von Ende
Clinical Trial Service Unit, Nuffield Department of Population Health, University of Oxford, Oxford, UK
Pui San Tan
Nuffield Department of Primary Care Health Sciences, University of Oxford, Oxford, UK
Hannah M Sallis
MRC Integrative Epidemiology Unit, University of Bristol, Bristol, UK
School of Psychological Science, University of Bristol, Bristol, UK
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Smoking
Original research

Smoking and COVID-19 outcomes: an observational and Mendelian randomisation study using the UK Biobank cohort

Ashley K Clift^{1, 2}, Adam von Ende³, Pui San Tan¹, Hannah M Sallis^{4, 5, 6}, Nicola Lindson¹, Carol A C Coupland⁷, Marcus R Munafò^{4, 5, 6}, Paul Aveyard¹, Julia Hippisley-Cox¹, Jemma C Hopewell³

Correspondence to Dr Ashley K Clift, Nuffield Department of Primary Care Health Sciences, University of Oxford, Oxford, UK; ashley.clift@phc.ox.ac.uk

Abstract

Background Conflicting evidence has emerged regarding the relevance of smoking on risk of COVID-19 and its severity.

Methods We undertook large-scale observational and Mendelian randomisation (MR) analyses using UK Biobank. Most recent smoking status was determined from self-reporting. Outcomes were derived from 18 August 2020. Logistic regression models adjusted for age, sex, education, COVID-19-related infection, COVID-19-related genetic instruments, smoking status, and smoking cessation were used to estimate the association between smoking and COVID-19-related outcomes.

Results There were 421 469 COVID-19-related deaths. Current smoking was associated with a 61% increase in COVID-19-related deaths (OR 1.61, 95% CI 1.35 to 1.92) and mortality (smoking cessation was associated with a 27% reduction in COVID-19-related deaths (OR 0.73, 95% CI 0.57 to 0.94). Genetically predicted smoking was associated with a 2.27-fold increase in COVID-19-related deaths (OR 2.27, 95% CI 1.71 to 3.01). Genetically predicted smoking cessation was associated with a 2.27-fold reduction in COVID-19-related deaths (OR 0.44, 95% CI 0.33 to 0.58).

Interpretation Congruent results from observational and Mendelian randomisation analyses suggest that smoking is associated with an increased risk of COVID-19-related deaths and mortality, and that smoking cessation is associated with a reduced risk of COVID-19-related deaths and mortality.

Keywords COVID-19, Mendelian randomisation, smoking, mortality, observational study, UK Biobank

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Smoking and COVID-19 outcomes: an observational and Mendelian randomisation study using the UK Biobank cohort

Ashley K Clift^{1, 2}, Adam von Ende³, Pui San Tan¹, Hannah M Sallis^{4, 5, 6}, Nicola Lindson¹, Carol A C Coupland^{1, 7}

Marcus R Munafò^{4, 5, 6}, Paul Aveyard¹, Julia Hippisley-Cox¹, Gemma C Hopewell³

Correspondence to Dr Ashley K Clift, Nuffield Department of Primary Care Health Sciences, University of Oxford, Oxford, UK; ashley.clift@phc.ox.ac.uk

Abstract

Background Conflicting evidence has emerged regarding the relevance of smoking on risk of COVID-19 and its severity.

Methods We undertook large-scale observational and Mendelian randomisation (MR) analyses using UK Biobank. Most recent smoking status was determined from primary care records (70.8%) and UK Biobank questionnaire data (29.2%). COVID-19 outcomes were derived from Public Health England SARS-CoV-2 testing data, hospital admissions data, and death certification (18 August 2020). Logistic regression was used to estimate associations between smoking status and confirmed SARS-CoV-2 infection, COVID-19-related hospitalisation, and COVID-19-related death. Inverse variance-weighted MR analyses using established genetic instruments for smoking initiation and smoking heaviness were undertaken (reported per SD increase).

Results There were 421 469 eligible participants, 1649 confirmed infections, 968 COVID-19-related hospitalisations and 44 COVID-19-related deaths. Compared with never-smokers, current smokers had higher risks of hospitalisation (OR 1.80, 95% CI 2.29) and mortality (smoking 1–9/day: OR 2.14, 95% CI 0.87 to 5.24; 10–19/day: OR 5.91, 95% CI 3.66 to 9.54; 20+/day: 6.11, 95% CI 3.59 to 10.42). In MR analyses of 281 105 White British participants, genetically predicted propensity to initiate smoking was associated with higher risks of infection (OR 1.45, 95% CI 1.10 to 1.91) and hospitalisation (OR 1.60, 95% CI 1.10 to 2.29).



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Data availability statement

Data are available upon reasonable request. Data may be obtained from a third party and are not publicly available. The individual participant data from UK Biobank is available on application by bona fide researchers to the UK Biobank (via the Access Management Team). Individual level data is not permitted to be shared by the authorship team. A data dictionary defining ICD-10 codes used to define comorbidities, or Read/SNOMED codes to define smoking exposures from primary care data, can be made available on request to the corresponding author.

Ethics statements

Not required.

Patient consent for publication

Not required.

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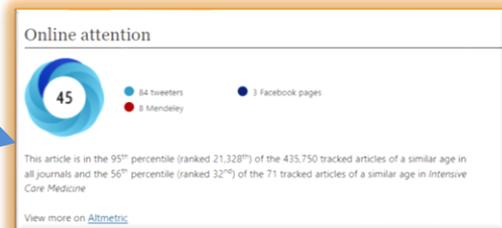
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Thilo C. von Groote

Department of Anaesthesiology, Intensive Care and Pain Medicine, University Hospital Münster, Münster, Germany

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Outline

- Summary
- Introduction
- Metabolic and endocrine pathways linked to SARS-CoV-2
- Predisposition to severe COVID-19 outcomes in patients with metabolic disorders
- Effects of SARS-CoV-2 on the metabolic and endocrine systems
- Implications for medications and therapy
- Importance of risk stratifications
- Breakthrough infections and metabolic disease
- Long-term consequences and monitoring after COVID-19
- Conclusions
- Search strategy and selection criteria
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THE LANCET

Diabetes & Endocrinology

Volume 9, Issue 11, November 2021, Pages 786-798

COVID-19 and metabolic disease: mechanisms and clinical management

Charlotte Steenblock PhD ^a, Prof Peter E H Schwarz MD ^{a, c, d}, Prof Barbara Ludwig MD ^{a, b, c, d, e}, Prof Andreas Linkermann MD ^a, Prof Paul Zimmet MD ^f, Konstantin Kulebyakin PhD ^{a, h}, Prof Vsevolod A Tkachuk DSc ^{a, h}, Prof Alexander G Markov DSc ⁱ, Prof Hendrik Lehnert MD ^j, Prof Martin Hrabě de Angelis MD ^{g, k, l}, Hannes Rietzsch MD ^a, Roman N Rodionov PhD ^a, Prof Kamlesh Khunti MD ^m, David Hopkins FRCP ⁿ, Prof Andreas L Birkenfeld MD ^{a, h, o, p, q}, Prof Bernhard Boehm MD ^{r, s}, Prof Richard I G Holt FRCP ^t, Prof Jay S Skyler MD ^{u, v}, Prof Stefan R Bornstein MD ^{a, c, d, e, g, h, x}

Summary

Up to 50% of the people who have died from COVID-19 had metabolic vascular disorders. Notably, there are many direct links between the metabolic and endocrine systems. Thus, not only are patients with dysfunction (eg, obesity, hypertension, non-alcoholic fatty liver disease, diabetes) at an increased risk of developing severe COVID-19 but with SARS-CoV-2 might lead to new-onset diabetes or aggravated metabolic disorders. In this Review, we provide an update on the

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Up to 50% of the people who have died from COVID-19 had metabolic and vascular disorders. Notably, there are many direct links between COVID-19 and the metabolic and endocrine systems. Thus, not only are patients with metabolic dysfunction (eg, obesity, hypertension, non-alcoholic fatty liver disease, and diabetes) at an increased risk of developing severe COVID-19 but also infection with SARS-CoV-2 might lead to new-onset diabetes or aggravation of pre-existing metabolic disorders. In this Review, we provide an update on the mechanisms of how metabolic and endocrine systems contribute to the development of severe COVID-19.

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Evaluation of the RCOG guideline for the prediction of neonates that are small for gestational age and comparison with the competing risks model

I Papastefanou, U Nowacka, O Buerger, R Akolekar, D Wright, KH Nicolaides

First published: 17 June 2021 | <https://doi.org/10.1111/1471-0528.16815> | Citations: 1

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To examine the predictive performance of the RCOG guideline for neonates small for gestational age (SGA), and to compare the performance of the competing risks model for SGA.

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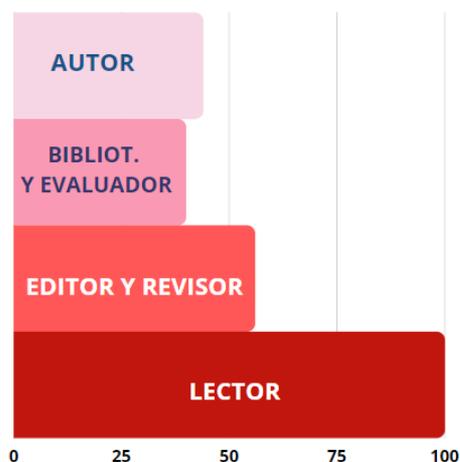
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Gracias



Blanca San José Montano

Bibliotecaria/documentalista y animadora científica en Ciencias de la Salud y Vocal de la Junta Directiva de SEDIC
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