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„NICOLAE TESTEMIȚANU” DIN REPUBLICA MOLDOVA

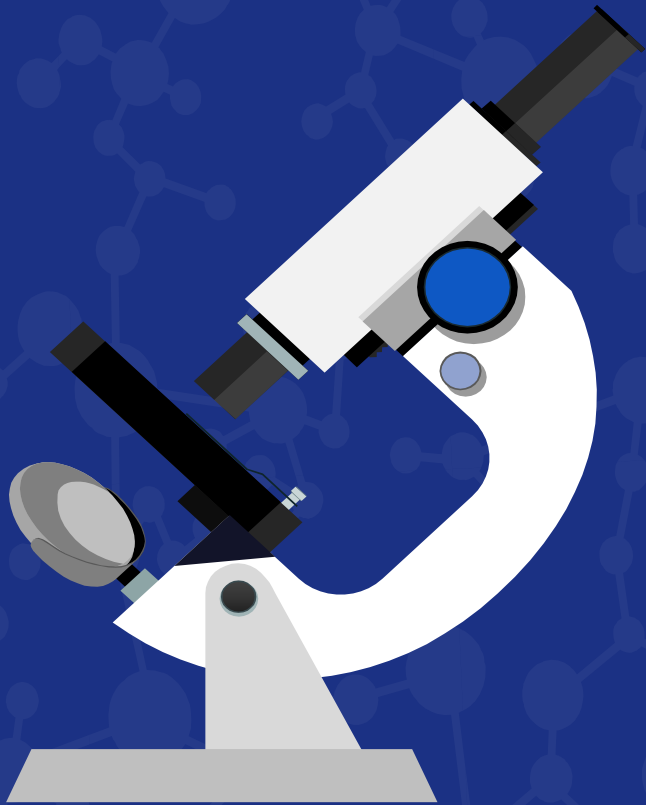
# GALA LAUREAȚILOR



HR EXCELLENCE IN RESEARCH



# *Concursul de performanță*



**IMPACTUL  
ACTIVITĂȚII  
DE CERCETARE**

# Concursul de performanță IMPACTUL ACTIVITĂȚII DE CERCETARE

## *Criterii de evaluare*



- 1 Indicele Hirsch în Scopus
- 2 Indicele Hirsch în Google Academic
- 3 Articol publicat în anul 2020 în reviste cu cel mai mare factor de impact

# Concursul Impactul Activității de Cercetare

## Indicele Hirsch în SCOPUS

*Valoarea Hirsch - 20*

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„NICOLAE TESTEMIȚANU” DIN REPUBLICA MOLDOVA

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# DIPLOMĂ DE ONOARE

Se decernează  
Dnei/Dlui **Mihail Todiraș**  
Laureat al Concursului de performanță  
„Impactul activității de cercetare”

CONFERINȚA ȘTIINȚIFICĂ ANUALĂ  
CERCETAREA ÎN BIOMEDICINĂ ȘI SĂNĂTATE:  
CALITATE, EXCELENȚĂ ȘI PERFORMANȚĂ

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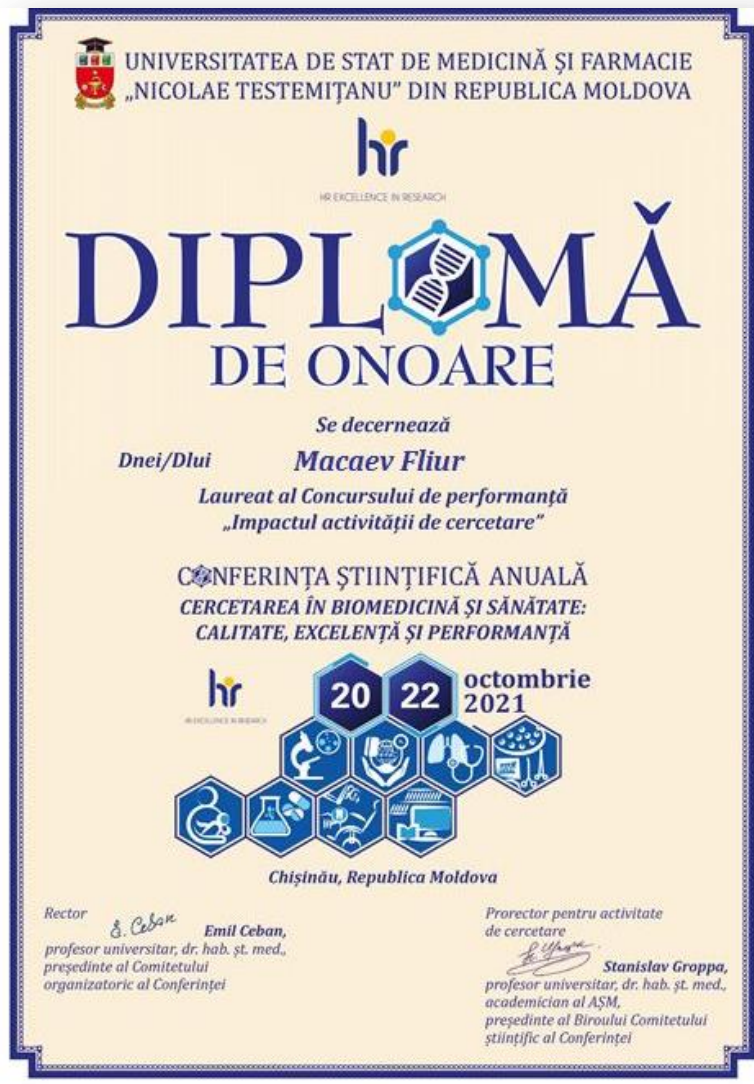
20 22 octombrie  
2021

Chișinău, Republica Moldova

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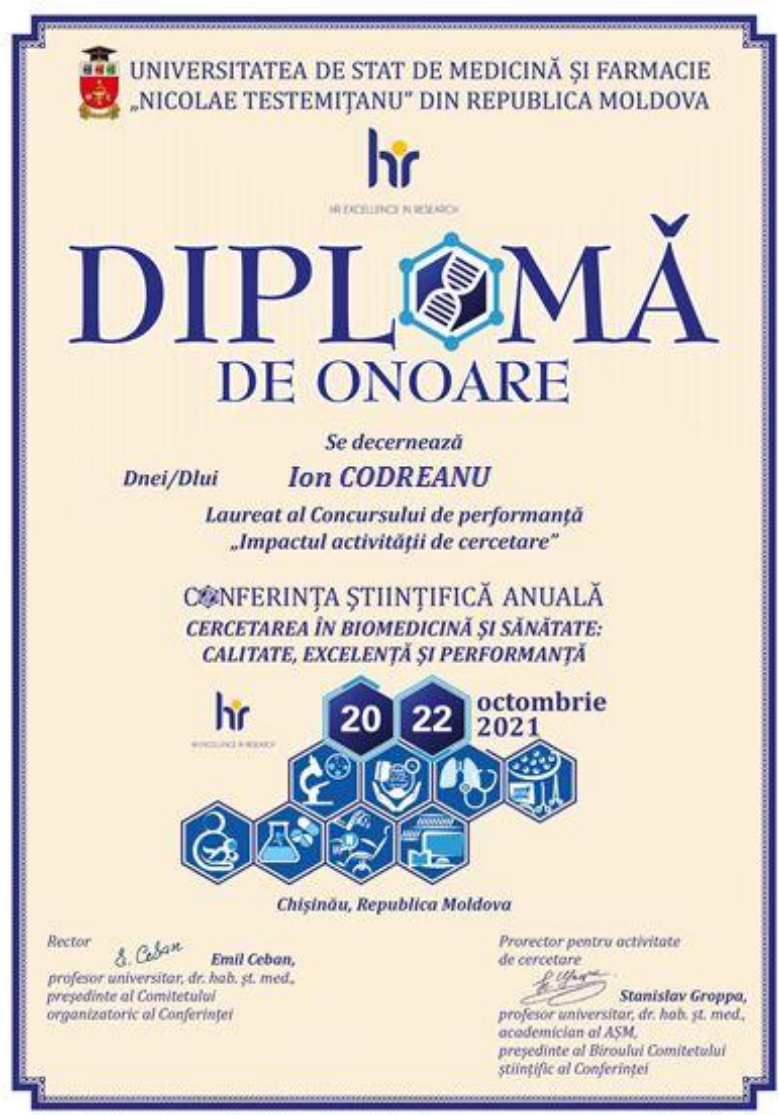
## Indicele Hirsch în SCOPUS

*Valoarea Hirsch - 15*

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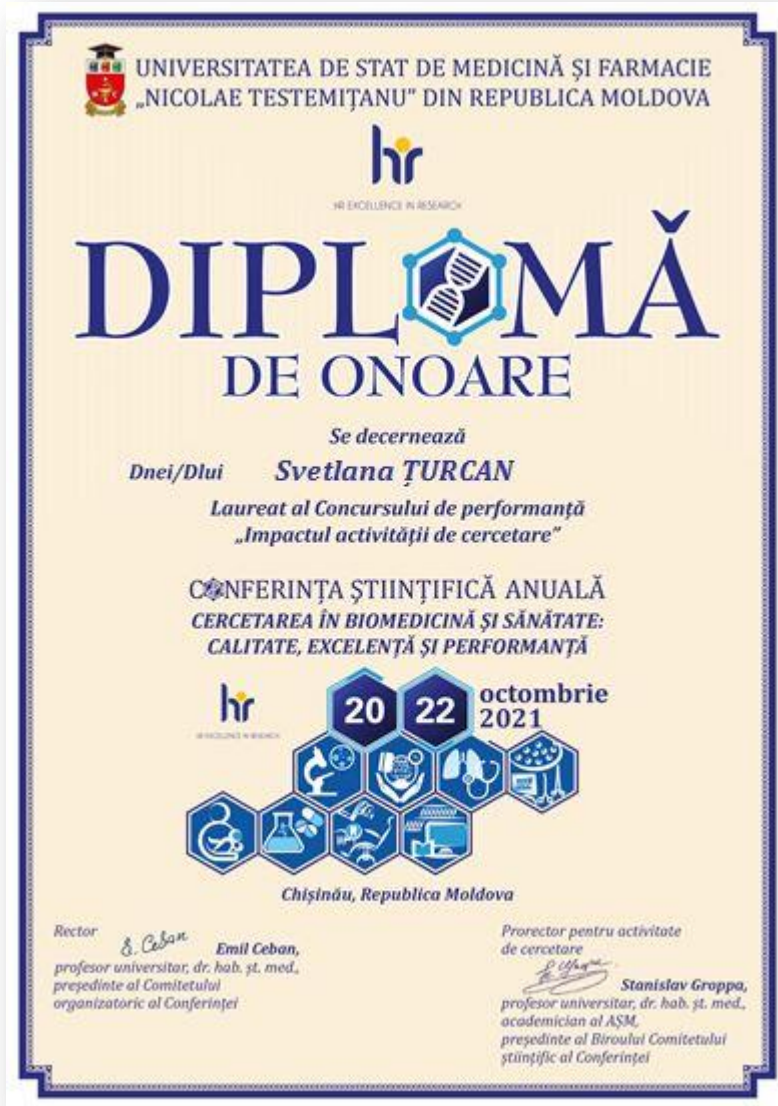
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*Valoarea Hirsch - 14*

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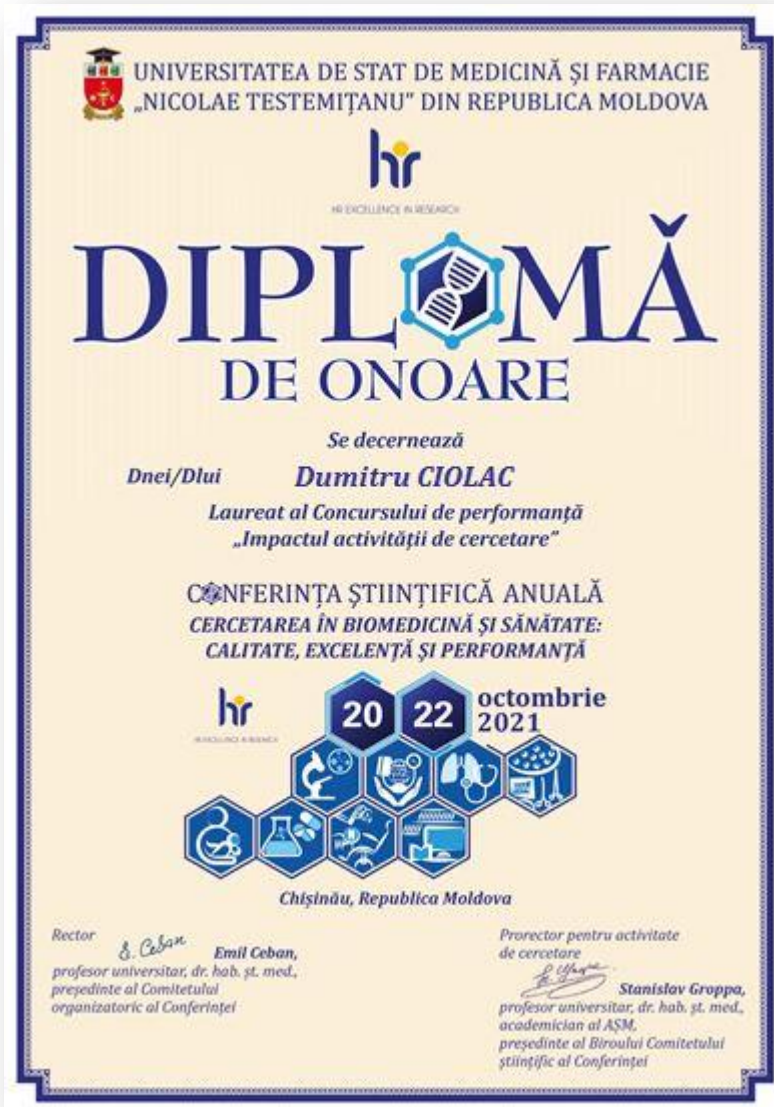
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*Valoarea Hirsch - 6*

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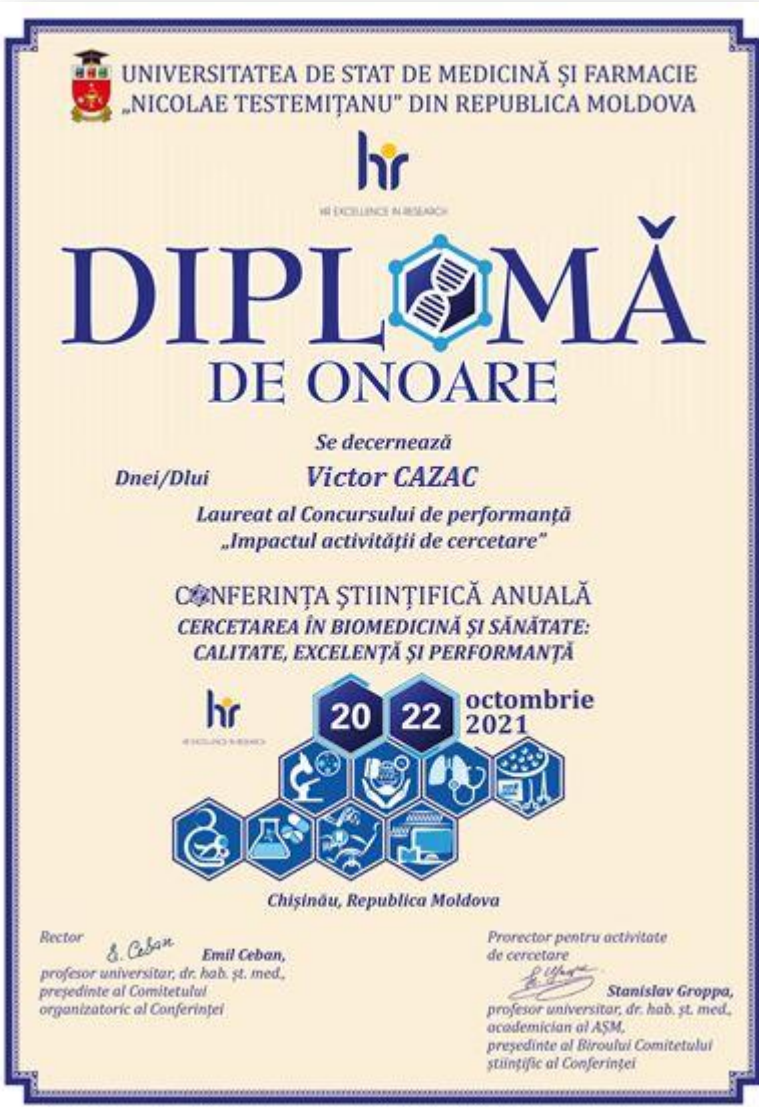
## Indicele Hirsch în SCOPUS

*Valoarea Hirsch - 2*

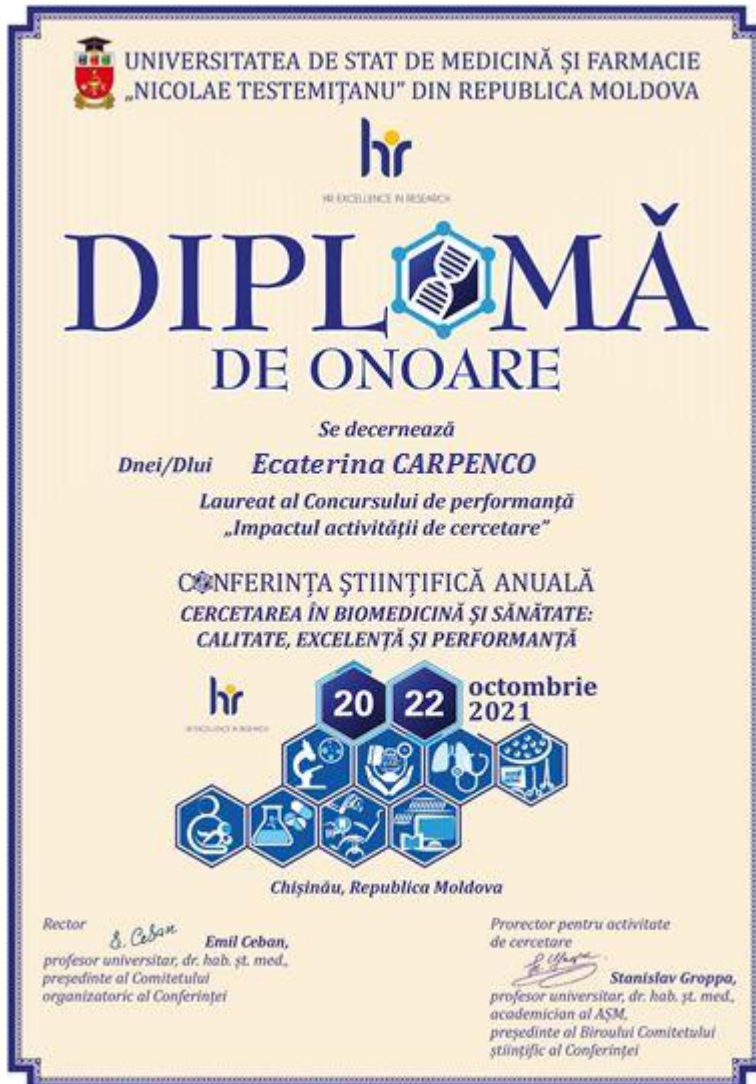
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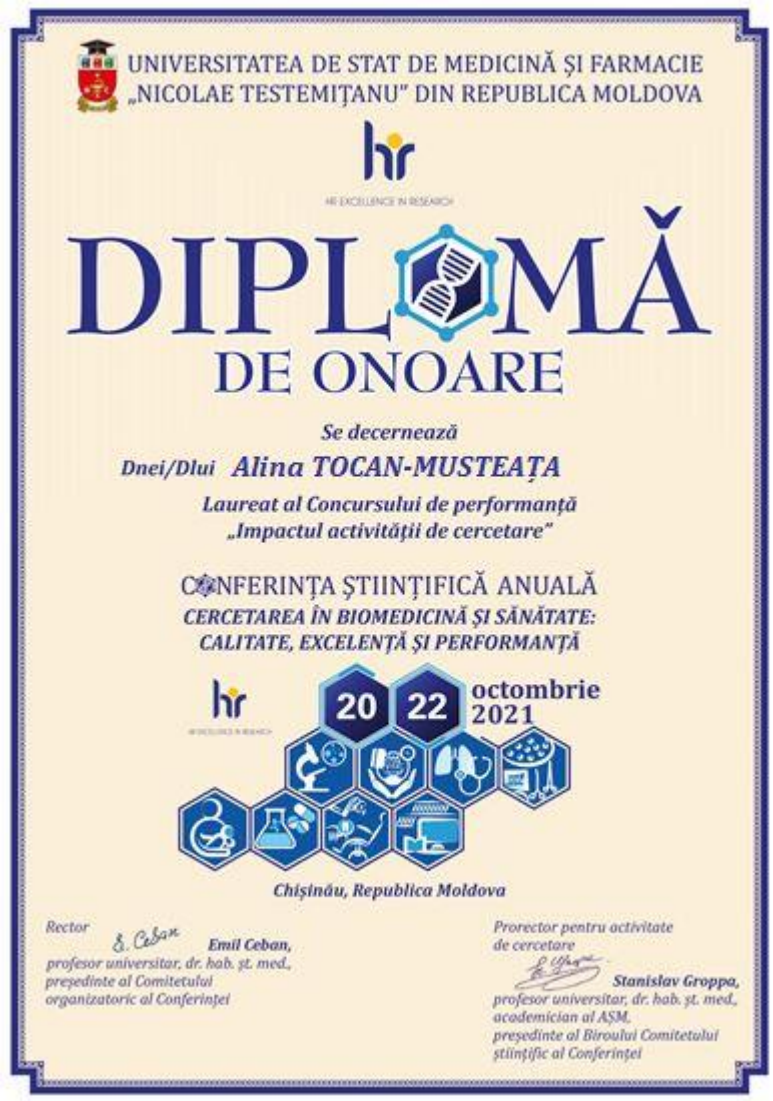
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## Indicele Hirsch în SCOPUS

*Valoarea Hirsch - 2*

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Program de doctorat:  
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Anul III



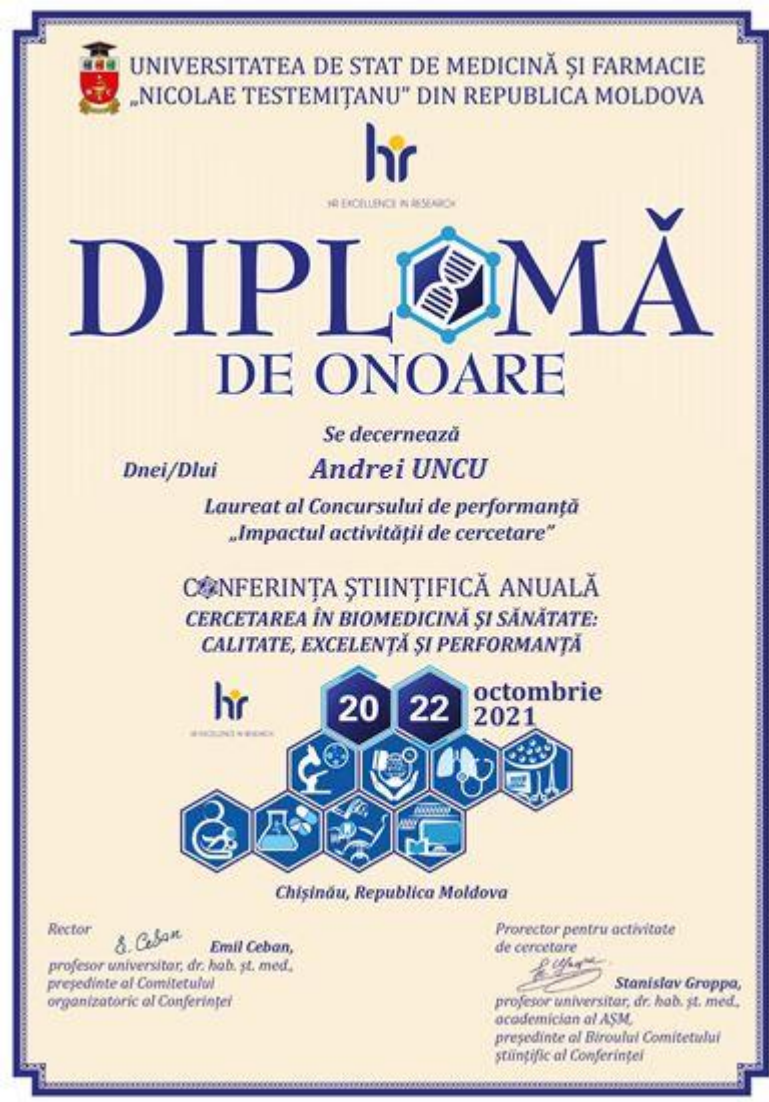
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*Valoarea Hirsch - 2*

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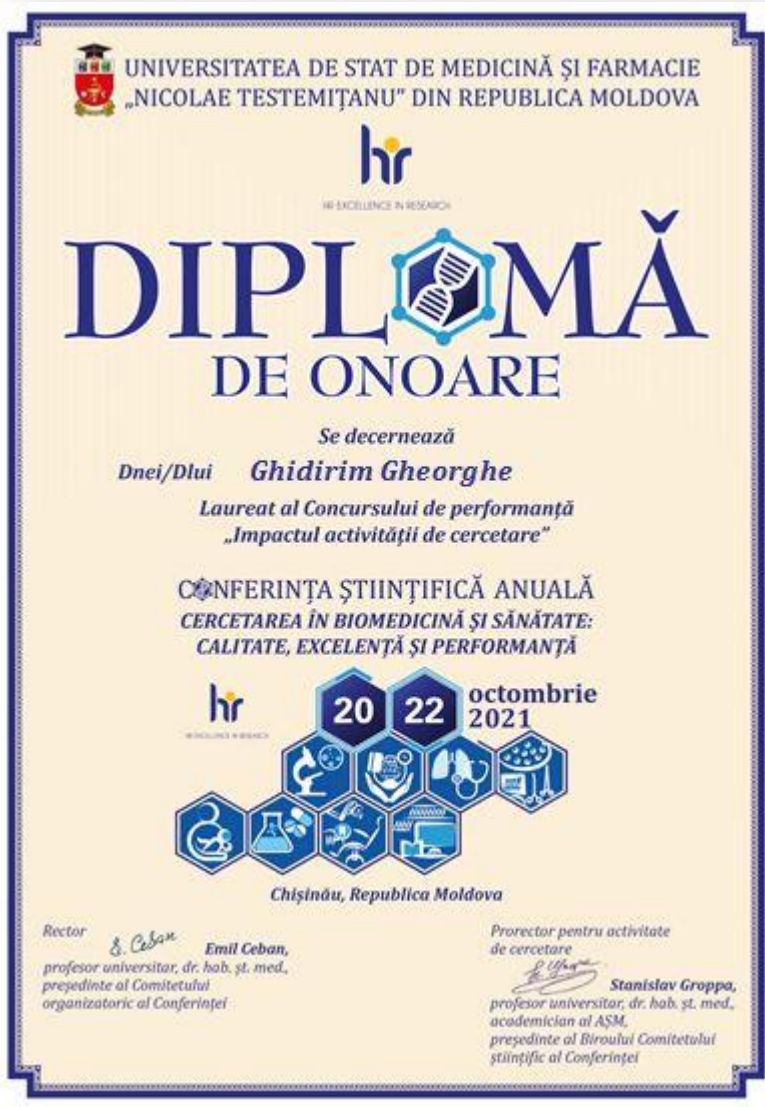
**Indicele Hirsch  
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# Concursul Impactul Activității de Cercetare



**Indicele Hirsch  
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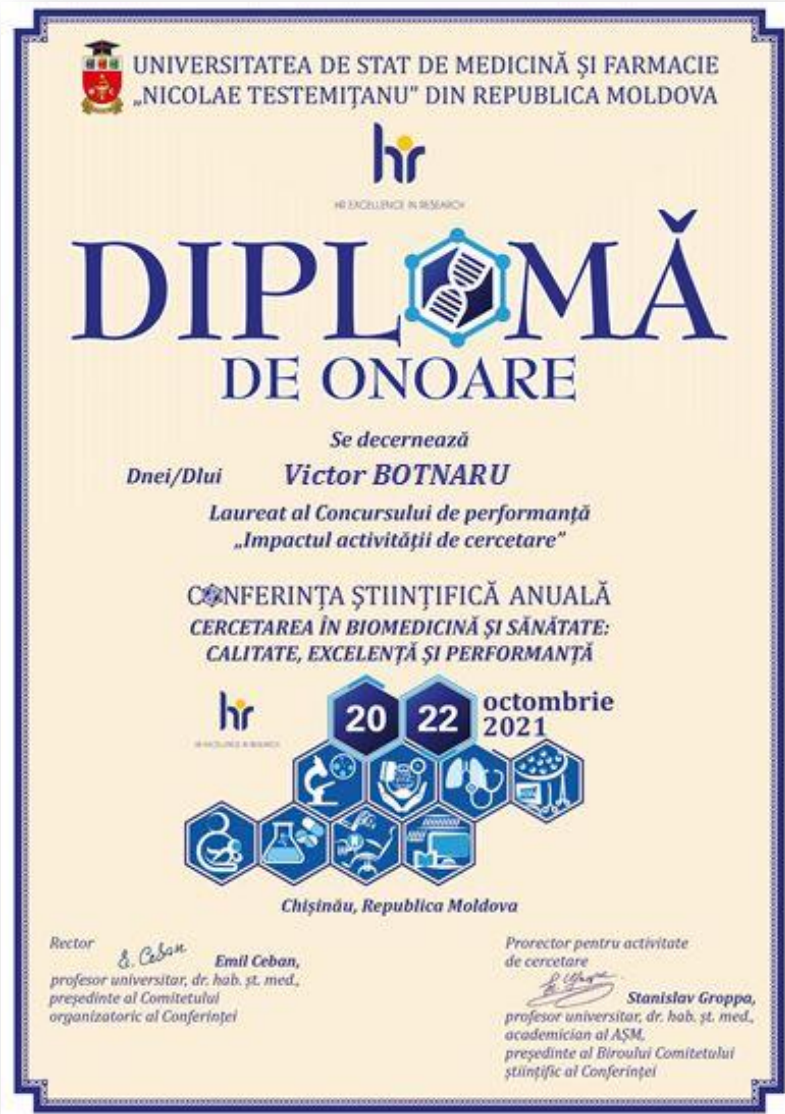
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**Indicele Hirsch în  
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# Concursul Impactul Activității de Cercetare



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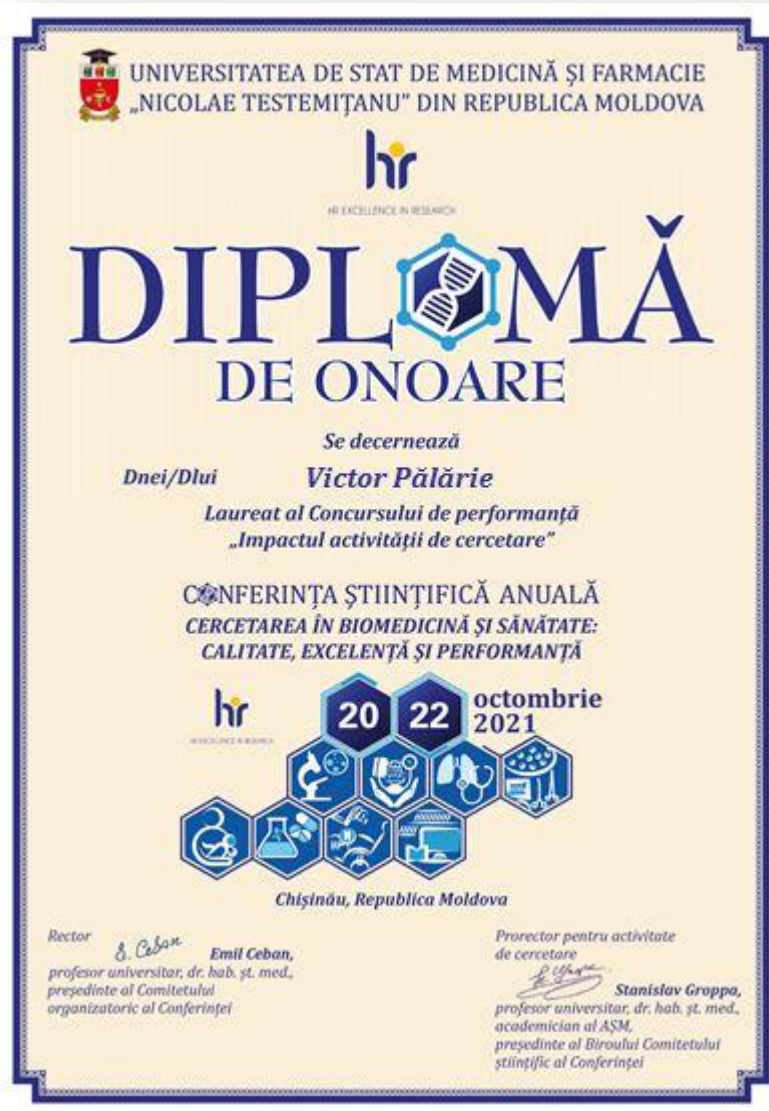
Valoarea Hirsch - 12

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# Concursul Impactul Activității de Cercetare



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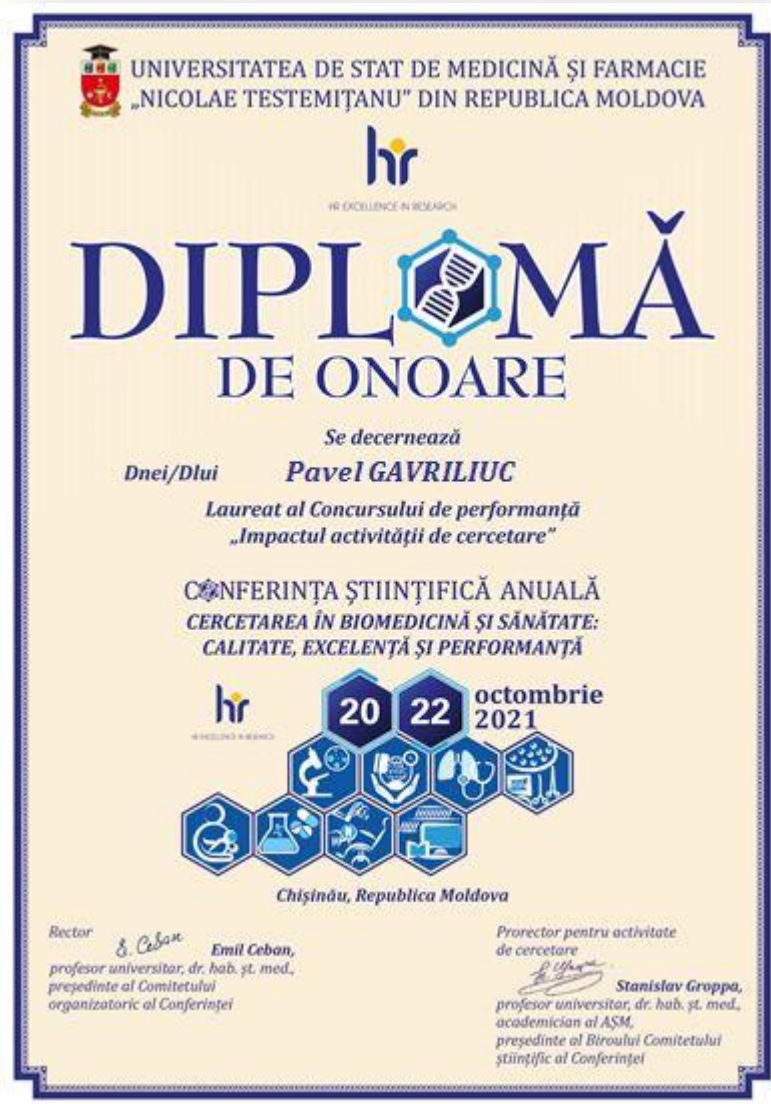
*Valoarea Hirsch - 12*

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# Concursul Impactul Activității de Cercetare



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# Concursul Impactul Activității de Cercetare

## Indicele Hirsch în GOOGLE ACADEMIC

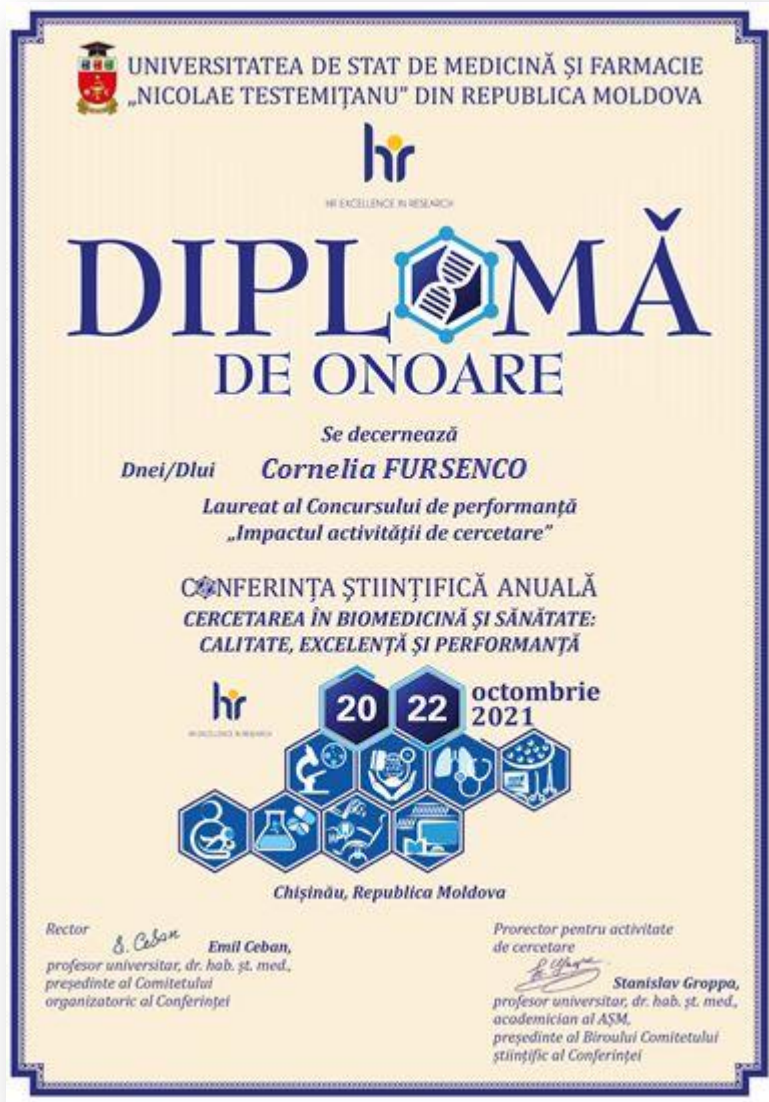
*Valoarea Hirsch - 3*

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# Concursul Impactul Activității de Cercetare



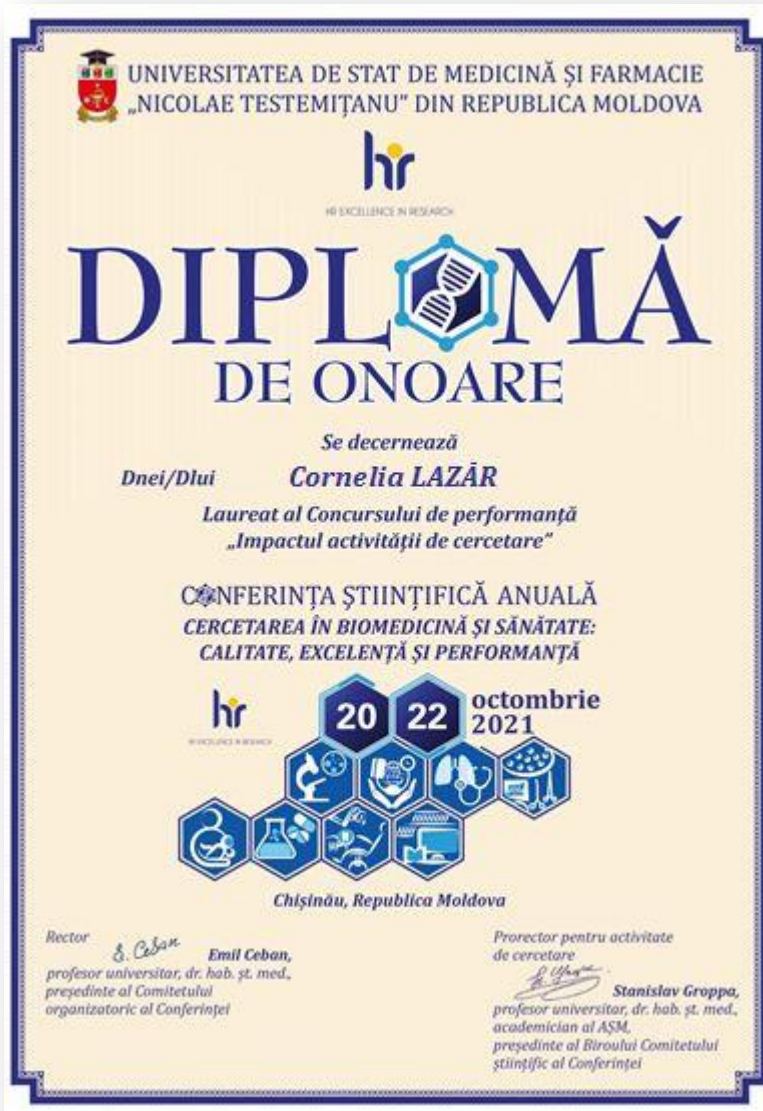
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# Concursul Impactul Activității de Cercetare

Articol publicat în reviste  
cu cel mai înalt Factor de Impact  
*Impact factor – 17.373*

Publicat în *Gastroenterology*,  
2020, 158:2180–2194.

## Grupul de autori:

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Gastroenterology 2020;158:2180–2194

## CLINICAL—LIVER

### Safety and Efficacy of 48 Weeks REP 2139 or REP 2165, Tenofovir Disoproxil, and Pegylated Interferon Alfa-2a in Patients With Chronic HBV Infection Naïve to Nucleos(t)ide Therapy

Michel Bazinet,<sup>1</sup> Victor Pânteu,<sup>2</sup> Gheorghe Plăcintă,<sup>2</sup> Iurie Moscalu,<sup>3</sup> Valentin Cebotarescu,<sup>2</sup> Lilia Cojuhari,<sup>2</sup> Pavlina Jimbei,<sup>4</sup> Liviu Iarovoi,<sup>2</sup> Valentina Smesnoi,<sup>4</sup> Tatiana Musteata,<sup>4</sup> Alina Jucov,<sup>2,3</sup> Ulf Dittmer,<sup>5</sup> Adalbert Krawczyk,<sup>6,8</sup> and Andrew Vaillant<sup>1</sup>

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Therapy	Response rates during therapy	Response rates during treatment-free follow-up
TDF pegIFN	LOW HBsAg loss HBsAg seroconversion	LOW Functional cure
TDF pegIFN NAPs	HIGH HBsAg loss HBsAg seroconversion Asymptomatic transaminase flares	HIGH Functional cure Normal liver function

Gastroenterology

See editorial on page 2051.

**BACKGROUND & AIMS:** Nucleic acid polymers (NAPs) inhibit assembly and secretion of hepatitis B virus (HBV) subviral particles. We performed an open-label, phase 2 study of the safety and efficacy of the NAPs REP 2139 or REP 2165 combined with tenofovir disoproxil fumarate (TDF) and pegylated interferon alfa-2a (pegIFN) in patients with chronic HBV infection who were negative for hepatitis B e antigen. **METHODS:** Following 24 weeks TDF therapy, 40 patients were randomly assigned to groups that received 48 weeks of experimental therapy (TDF + pegIFN + REP 2139-Mg or REP 2165-Mg) or 24 weeks of control therapy (TDF + pegIFN) followed by 48 weeks of experimental therapy. Patients were then followed for a treatment-free period of 48 weeks. Primary outcomes were the safety and tolerability of REP 2139-Mg or REP 2165-Mg in combination with TDF + pegIFN compared with TDF + pegIFN alone through the first 48 weeks of therapy and subsequently throughout 48 weeks of NAP-based

combination therapy (treatment weeks 24–72 in the experimental group and weeks 48–96 in the control group). Secondary outcomes were reductions in hepatitis B surface antigen (HBsAg) in control and experimental groups over the first 48 weeks of the study and throughout 48 weeks of combination therapy and virologic control (HBsAg positive, HBV DNA below 2000 IU/mL, normal level of alanine aminotransferase) or functional cure (HBsAg below 0.05 IU/mL, HBV DNA target not detected, normal level of alanine aminotransferase) after removal of all therapy. **RESULTS:** Levels of HBsAg, anti-HBs, and HBV DNA did not differ significantly between the groups given REP 2139 vs REP 2165. PegIFN-induced thrombocytopenia ( $P = .299$  vs controls) and neutropenia ( $P = .112$  vs controls) were unaffected by NAPs (REP 2139 vs REP 2165). Increases in levels of transaminases were significantly more frequent ( $P < .001$  vs controls) and greater ( $P = .002$  vs controls) in the NAP groups (but did not produce symptoms), correlated with initial decrease in HBsAg, and normalized during therapy and follow-up. During the first 24 weeks of TDF and pegIFN administration, significantly higher proportions of



# Concursul Impactul Activității de Cercetare

Articol publicat în reviste  
cu cel mai înalt Factor de Impact  
*Impact factor – 17.373*

Publicat în *Gastroenterology*, 2020,  
vol.158, issue 3., p. e1-18 2020

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*Gastroenterology* 2020;158:537-549

## Efficacy and Safety of Mirikizumab in a Randomized Phase 2 Study of Patients With Ulcerative Colitis

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See editorial on page 467.

Keywords: EB Dosing; Drug; Cytokine; Inhibitor.

**BACKGROUND & AIMS:** Interleukin 23 contributes to the pathogenesis of ulcerative colitis (UC). We investigated the effects of mirikizumab, a monoclonal antibody against the p19 subunit of interleukin 23, in a phase 2 study of patients with UC. **METHODS:** We performed a trial of the efficacy and safety of mirikizumab in patients with moderate to severely active UC, enrolling patients from 14 countries from January 2016 through September 2017. Patients were randomly assigned to groups given intravenous placebo (N = 63), mirikizumab 50 mg (N = 63) or 200 mg (N = 62) with exposure-based dosing, or mirikizumab 600 mg with fixed dosing (N = 61) at weeks 0, 4, and 8. Of assigned patients, 63% had prior exposure to a biologic agent. Clinical responders (decrease in 9-point Mayo score, including  $\geq 2$  points and  $\geq 35\%$  from baseline with either a decrease of rectal bleeding subscore of  $\geq 1$  or a rectal bleeding subscore of 0 or 1) at week 12 who had received mirikizumab were randomly assigned to groups that received maintenance treatment with mirikizumab 200 mg subcutaneously every 4 weeks (N = 47) or every 12 weeks (N = 46). The primary endpoint was clinical remission (Mayo subscores of 0 for rectal bleeding, with 1-point decrease from baseline for stool frequency, and 0 or 1 for endoscopy) at week 12. A multiple testing procedure was used that began with the 600-mg dose group, and any nonsignificant comparison result ended the formal statistical testing procedure. **RESULTS:** At week 12, 15.9% (P = .066), 22.6% (P = .004), and 11.5% (P = .142) of patients in the 50-mg, 200-mg, and 600-mg groups achieved clinical remission, respectively, compared with 4.8% of patients given placebo. The primary endpoint was not significant (comparison to 600 mg, P > .05). Clinical responses occurred in 41.3% (P = .014), 59.7% (P < .001), and 49.2% (P = .001) of patients in the 50-mg, 200-mg, and 600-mg groups, respectively, compared with 20.6% of patients given placebo. At week 52, 46.0% of patients given subcutaneous mirikizumab 200 mg every 4 weeks and 37.0% given subcutaneous mirikizumab 200 mg every 12 weeks were in clinical remission. **CONCLUSIONS:** In a randomized trial of patients with UC, mirikizumab was effective in inducing a clinical response after 12 weeks. Additional studies are required to determine the optimal dose for induction of remission. Mirikizumab showed durable efficacy throughout the maintenance period. [Clinicaltrials.gov, Number NCT02589665](https://doi.org/10.1053/j.gastro.2018.08.043)

Ulcerative colitis (UC) is a chronic inflammatory disease characterized by mucosal inflammation of the colon and rectum, with typical symptoms of rectal bleeding, diarrhea, and urgency.<sup>1</sup> The goals of medical management are to reduce symptoms by controlling mucosal inflammation and, ultimately, to prevent disability, colectomy, and colorectal cancer.<sup>2</sup> Monotherapy or combination treatment with aminosalicylates, corticosteroids, and thiopurines are often used as initial therapy.<sup>3</sup> Biologic agents targeting tumor necrosis factor, including infliximab, adalimumab, and golimumab, or integrins (eg, vedolizumab), and, more recently, a small molecule inhibitor targeting Janus kinases (eg, tofacitinib) are used in patients refractory or intolerant to conventional therapy or those who have more severe disease activity or worse prognosis.<sup>2-7</sup> Many patients have an inadequate response or lose response over time; thus, new treatment approaches are needed.

Interleukin (IL) 23, a member of the IL12 family of cytokines, has 2 components: the p40 subunit, which is shared by IL12, and the p19 subunit, which is found in IL23 but not IL12. IL23 plays a key role in the maintenance and amplification of T helper 17 (Th17) cells and the stimulation of many innate immune cells, which are important in the pathogenesis of chronic inflammatory diseases, including UC.<sup>8-11</sup> Ustekinumab, a monoclonal antibody directed to the shared p40 subunit of IL12 and IL23, is effective for treatment of Crohn's disease and psoriasis.<sup>12-14</sup> However, multiple studies in patients with psoriasis have suggested that more selective targeting of the IL23 pathway by blocking the p19 subunit of IL23 is more effective than

Abbreviations used in this paper: AE, adverse event; CD, Crohn's disease; CRP, C-reactive protein; EB, exposure-based; IBD, inflammatory bowel disease; QoL, quality of life; IL, interleukin; SAE, serious adverse event; SC, subcutaneous; Th17, T helper 17; UC, ulcerative colitis.

Most current article

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0016-5085  
<https://doi.org/10.1053/j.gastro.2018.08.043>

# Concursul Impactul Activității de Cercetare

Articol publicat în reviste  
cu cel mai înalt Factor de Impact

*Impact factor – 11.627*

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2020, 22: p. 789-799.  
doi:10.1002/ejhf.1747

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European Journal of Heart Failure (2020)  
doi:10.1002/ejhf.1747



**Ischaemic cardiomyopathy. Pathophysiological insights, diagnostic management and the roles of revascularisation and device treatment. Gaps and dilemmas in the era of advanced technology**

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Received 27 August 2019; revised 21 October 2019; accepted 30 December 2019

Ischaemic cardiomyopathy (ICM) represents an important cardiovascular condition associated with substantially increased morbidity and mortality. It is characterised from a broad spectrum of clinical manifestations and pathophysiological substrates and its diagnosis is based on the demonstration of significant left ventricular dysfunction in the context of significant epicardial coronary artery disease. Contemporary management aims at improving prognosis through evidence-based pharmacotherapy and device therapy, where indicated. Whilst the beneficial role of revascularisation remains clear in patients with strong indications such as those with symptoms and/or acute coronary syndromes, for those patients that are asymptomatic and suffer from stable ischaemic heart disease the impact of revascularisation on hard outcomes remains less well defined and currently its adoption is hampered by the lack of robust randomised data. The aim of this review is therefore to provide a constructive appraisal on the pathophysiology of ICM, the role of the various non-invasive imaging techniques in the diagnosis of ICM and the differentiation between viable and non-viable myocardium and finally discourse the potential role of revascularisation and contemporary device therapy in the management of patients with ICM.

**Keywords** Ischaemic cardiomyopathy • Revascularisation • Viable myocardium • Non-invasive imaging

## Introduction

Ischaemic cardiomyopathy (ICM) is associated with considerable morbidity and mortality and currently represents the most common cause of heart failure in the developed world.<sup>1</sup> The predominance of heart failure secondary to ICM has been mainly attributed to the notable advances in the treatment of patients with acute myocardial infarction (AMI) involving the obsolete thrombolytic therapy and the more contemporary percutaneous coronary intervention (PCI) with drug-eluting stents. Successful revascularisation in this context has led to an improved survival of patients that have suffered an AMI but with an inevitable trade-off of an upsurge

in the prevalence of ICM.<sup>2</sup> Although there is no general consensus regarding the definition of ICM, the latter is considered as a left ventricular (LV) dysfunction in the presence of severe coronary artery disease including at least one of the following characteristics: prior revascularisation or AMI; >75% stenosis in the left main stem or the left anterior descending artery; two or more coronary vessels with >75% luminal stenosis.<sup>3</sup>

The pathophysiological milieu behind ICM involves a spectrum of metabolic, neurohumoral and inflammatory changes resulting in an adverse myocardial remodelling and contractile dysfunction in the context of significantly impaired myocardial blood flow and/or reduced coronary flow reserve.<sup>4</sup>

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