ОДБОРУ ЗА НАУЧНОИСТРАЖИВАЧКУ ДЕЛАТНОСТ МЕДИЦИНСКОГ ФАКУЛТЕТА ВОЈНОМЕДИЦИНСКЕ АКАДЕМИЈЕ УНИВЕРЗИТЕТА ОДБРАНЕ У БЕОГРАДУ

На 111. седници Наставно-научног већа одржаној 28.09.2023. године, покренут је поступак за избор др сц. мед. Александра Пантовића, из Клинике за неурологију Војномедицинске академије, у звање **научни сарадник**. На овој седници, Наставно-научно веће је именовало Комисију за оцену испуњености услова за избор у звање.

На основу приложене документације о научно-истраживачком раду, као и увида у целокупни рад кандидата, а у складу са Законом о науци и истраживањима („Службени гласник РС", број 49/2019-3) и Правилником о стицању истраживачких и научних звања (159/2020-82), Одбору за научноистраживачку делатност Медицинског факултета Војномедицинске академије Универзитета одбране у Београду подносимо следећи

ИЗВЕШТАЈ

1. БИОГРАФСКИ ПОДАЦИ

Др Александар Пантовић је рођен 27.09.1982. године у Бару. Дипломирао је на Медицинском факултету у Београду 2008. године са просечном оценом 9.17. Након основних студија медицине био је запослен на Медицинском факултету у Београду у лабораторији за молекуларну медицину Института за микробиологију и имунологију. Радио је на пројектима Министарства просвете науке и технолошког развоја Републике Србије: Имуномодулаторно, цитопротективно и цитотоксично дејство наночестица (број пројекта 145073) и Модулација унутарћелијског енергетског баланса – контрола сигналних путева у терапији тумора и неуро-имуно-ендокриних поремећаја (број пројекта 41025). Специјалистички испит из неуролгије положио је у децембру 2016. године на Клиници за неурологију Војномедицинске академије. Докторску дисертацију под називом „Улога аденозин-монофосфатом активиране протеин-киназе и mTOR комплекса 1 у in vitro цитотоксичном дејству нестероидних анти-инфламаторних лекова на ћелије глиома" одбранио је у септембру 2020. године. Др Александар Пантовић је запослен на Клиници за неурологију Војномедицинске академије. Члан је Друштва неуролога Србије, Друштва за неуронауке Србије, Међунарног удружења за неуроимунологију, Међународног удружења за невољне покрете и Интернационално удружења против главобоља. Ожењен је и отац две ћерке.

2. БИБЛИОГРАФСКИ ПОДАЦИ

2.1 Објављени радови

Поред сваке публикације унешена је позиција на листи часописа и његов импакт фактор из одговарајуће дисциплине са годином у којој је био најповољнији (за период од две године пре публиковања и година публиковања, и то за ону годину у којој је часопис најбоље рангиран, односно ону у којој је имао највећи импакт фактор). Након тога означен је укупан број бодова (ББ) и на крају нормиран број бодова (НББ) и формула која је коришћена за израчунавање према броју коаутора.

Рад у међународном часпоису изузетне вредности (M21a):

1. Markovic Z, Harhaji-Trajkovic L, Todorovic-Markovic B, Kepić D, Arsikin K, Jovanović S, **Pantovic A,** Dramićanin M, Trajkovic V. In vitro comparison of the photothermal anticancer activity of graphene nanoparticles and carbon nanotubes. Biomaterials. 2011;32(4):1121-9. IF 7.404 (10 бодова) НББ: 10/(1+0,2*(9-7)) = 7.142 Biomedical, Multidisciplinary

Рад у врхунском међунарнодном часопису (M21):

2. **Pantovic A,** Bosnjak M, Arsikin K, Kostic M, Mandic M, Ristic B, Tosic J, Suzin-Zivkovic V, Grujicic D, Isakovic A, Trajkovic V, Harhaji-Trajkovic Lj. In vitro antiglioma action of indomethacin is mediated through AMPK/mTOR complex 1 signaling pathway. Int J Biochem Cell Biol. 2017;83:84-96. IF 3.905 (8 бодова) НББ: 8/(1+0,2*(12-7)) = 4 Molecular biology & Neurosciences

3. **Pantovic A,** Krstic A, Janjetovic K, Kocic J, Harhaji-Trajkovic L, Bugarski D, Trajkovic V. Coordinated time-dependent modulation of AMPK/Akt/mTOR signaling and autophagy controls osteogenic differentiation of human mesenchimal stem cells. Bone. 2013;52(1):524-31. IF 4.461 (8 бодова) Metabolism & Endocrinology

4. Wasik AM, Grabarek J, **Pantovic A,** Cieślar-Pobuda A, Asgari HR, Bundgaard-Nielsen C, Rafat M, Dixon IM, Ghavami S, Łos MJ. Reprogramming and carcinogenesis-parallels and distinctions. Int Rev Cell Mol Biol. 2014;308:167-203. Review. IF 4.973 (8 бодова) НББ: 8/(1+0,2*(10-7)) =5 Biochemistry & Molecular medicine

5. Harhaji-Trajkovic L, Arsikin K, Kravic-Stevovic T, Petricevic S, Tovilovic G, **Pantovic A,** Zogovic N, Ristic B, Janjetovic K, Bumbasirevic V, Trajkovic VS. Chloroquine-mediated lysosomal dysfunction enhances the anticancer effect of nutrient deprivation. Pharmaceutical Research. 2012;29(8):2249-63. IF 4.742 (8 bodova) НББ: 8/(1+0,2*(11-7)) =4.44 Pharmacology & Pharmacy, Multidisciplinary

6. Pantic I, Harhaji-Trajkovic L, **Pantovic A,** Milosevic NT, Trajkovic V. Changes in fractal dimension and lacunarity as early markers of UV-induced apoptosis. J Theor Biol. 2012;303:87-92. IF 2.351 (8 бодова) Biology

7. Trpkovic A, Todorovic-Markovic B, Kleut D, Misirkic M, Janjetovic K, Vucicevic L, **Pantovic A,** Jovanovic S, Dramicanin M, Markovic Z, Trajkovic V. Oxidative stress-mediated hemolytic activity of solvent exchange-prepared fullerene (C60) nanoparticles. Nanotechnology. 2010;21(37):375102. IF 3.652 (8 бодова) НББ: 8/(1+0,2*(11-7)) =4.4 Multidisciplinary

Рад у истакнутом међународном часопису (M22):

8. **Pantović A,** Lepic T, Pasovski V, Krsmanovic Z, Raicevic R. Artery of Percheron infarction associated with COVID-19 in the young adult. J Neurovirol. 2021 Dec;27(6):951-953. IF 3.739 (5 bodova) Neurosciences

9. Sumarac-Dumanovic M, Jeremic D, **Pantovic A,** Janjetovic K, Stamenkovic-Pejkovic D, Cvijovic G, Stevanovic D, Micic D, Trajkovic V. Therapeutic improvement of glucoregulation in newly diagnosed type 2 diabetes patients is associated with a reduction of IL-17 levels. Immunobiology. 2013;218(8):1113-8. IF 3.180 (5 бодова) НББ: 5/(1+0,2*(9-7)) =3.57 Immunology

Рад у међународном часопису (М23):

10. **Pantovic A**, Arsikin K, Kosic M, Ristic B, Trajkovic V, Harhaji-Trajkovic L. Data supporting the inability of indomethacin to induce autophagy in U251 glioma cells. Data Brief. 2017;10;11:225-230. IF 0.736 (3 бода) Multidisciplinary

Рад у националном часопису (М53):

11. **Pantović A**, Barjaktarević D, Kostić S, Dinčić E. Spazam konvergencije: izmedju prave i lažne slabosti okulogira – prikaz slučaja. Sinapsa - publikacija Društva mladih neurologa Srbije, jun 2013. (1 бод) Неурологија

12. **Pantović A**, Jovanovski A, Raičević R, Dinčić E. Siringomijelija u longitudinalnom ekstenzivnom transverzalnom mijelitisu kod bolesnika sa oboljenjem iz spektra neuromijelitis optika. Sinapsa - publikacija Društva mladih neurologa Srbije, decembar 2017. (1 бод) Нурологија

Предавајне по позиву на међународном скупу (М32):

13. 5th Symposium of regenerative medicine "Stem cells - a vision beyond borders", Belgrade novembar 2018. Tema: Stem cell application in neurological disorders (1.5 бодова)

Саопштење са међународног скупа штампано у изводу (М34):

14. MDS- ES Winter School for Young Neurologists, Belgrade 2014. Distonic or pyramidal sign in a patient with clinical presentation of Parkinson disease (video prikaz) (0.5 бодова)

15. Adriatic neurology forum, Опатија 2015. Optical coherence tomography in the assessment of structural with functional and cognitive changes in multiple sclerosis (0.5 бодова)

16. Adriatic neurology forum, Бечићи 2016. Siringomyelic presentation of Longitudinal Extensive Transverse Myelitis as first manifestation of Sistemic Lupus Eritematosus and NMO spectrum disorder – a case report (0.5 бодова)

17. International Society of Neuroimmunology congress, Јерусалим 2016. Gene polymorphisms as modifiers of response to interferon beta 1b in Serbian Multiple Sclerosis population (candidate gene study) (0.5 бодова)

18. International Society of Neuroimmunology congress, Јерусалим 2016. Angiotensin-converting enzyme and angiotensin receptors gene polymorphisms as risk factors for multiple sclerosis (0.5 бодова)

Саопштење са скупа националног значаја штампано у изводу (М64):

19. Конгрес неуролога и неуронаука Србије Београд, 2013. Спазам конвергенције – приказ случаја (0.2 бода)

20. Конгрес неуролога и неуронаука Србије Београд, 2013. Повезаност СНП у генима за ренин-ангиотензин систем са ризиком за настанак мултипле скелерозе (0.2 бода)

21. Конгрес неуролога и неуронаука Србије Београд, 2013. Indomethacin exerts in vitro antiglioma effect through modulation of AMPK/mTOR signaling pathway (0.2 бода)

22. Конгрес неурофизиолога Србије 2015. Retinal nerve fiber layer thickness and multifocal visual evoked potentials in optic neuritis associated with multiple sclerosis – A case study (0.2 бода)

23. Конгрес неуролога и неуронаука Србије Нови сад, 2015. Оптичка кохерентна томографија у процени структурних са функционалним и когнитивним променама код пацијента са мултиплом склерозом (0.2 бода)

24. Школа за младе неурологе – Палић 2015. Мултипла склероза на путу до дијагнозе неуролупуса (0.2 бода) (0.2 бода)

25. Школа за младе неурологе – Палић 2015. Неуролупус/Неуромијелитис оптика спектар – диференцијална дијагноза (0.2 бода)

26. Конгрес неуролога и неуронаука Србије Нови сад, 2015. Мултифокални визуелни евоцирани потенцијали и структурне промене на мрежњачи код болесника са лонгитудиналним екстензивним трансферзалним мијелитисом и IgG аквапоринским антителима – НМО, приказ случаја (0.2 бода)

27. Семинар практичног лекара, Игало, 2019. Тумерфактивна мултипла склероза (0.2 бода)

28. Симпозијум Covid-19 и неурологија Врдник, 2021. Биталамички инфаркт повезан са COVID-19 приказ случаја младог одраслог пацијента (0.2 бода)

Одбрањена докторска дисертација (М70):

29. Uloga adenozin-monofosfatom aktivirane protein-kinaze i mTOR kompleksa 1 u in vitro citotoksičnom dejstvu nesteroidnih anti-inflamatornih lekova na ćelije glioma. Медицински факултет, Универзитет у Београду, септембар 2020. (6 бодова)

Табела 1. Преглед публикација др сц. мед. Александра Пантовића од почетка научног и клиничког рада по категоријама, вредности резултата (укупно и нормирано) и импакт фактор.

КАТЕГОРИЈА НАУЧНЕ ПУБЛИКАЦИЈЕ	М	БРОЈ РАДОВА	УКУПАН БРОЈ ПОЕНА	НОРМИР-АН БРОЈ ПОЕНА	ИМПАКТ ФАКТОР
Рад у међународном часопису	M21	7	58	40.98	31.934
	M22	2	10	8.57	6.919
	M23	1	3	3	0.736
Predavanje po pozivu sa međunarodnog skupa štampano u izvodu	M32	1	1.5	1,5	/
Saopštenje sa međunarodnog skupa štampano u izvodu	M34	5	2.5	2.5	/
Рад у научном часопису	M53	2	2	2	/
Саопштење са скупа националног значаја штампано у изводу	M64	10	2	2	/
Одбрањена докторска дисертација	M71	1	6	6	/
УКУПНО		**29**	**85**	**66.55**	**39.583**

3. АНАЛИЗА РАДОВА
(радови штампани у целини, публиковани од почетка каријере)

Објављени радови др сц. мед. Александра Пантовића у истраживачком делу дају објашњења за молекуларне механизме цитотоксичних ефеката нестероидних анти-инфламаторних лекова на ћелије глиома. Посебно се истражује улога сигналних путева који контролишу процесе апоптозе и аутофагије у цитотоксичном деловању ових лекова на ћелије тумора централног нервног система. Истраживања која се односе на активацију процеса аутофагије у контроли диференцијације мезенхимских матичних ћелија су по први пут објављена у раду др Пантовића а ова публикација је цитирана преко 200 пута **(референца 3).** Након отпочињања специјализације из неурологије и након тога др Пантовић је објавио неколико клиничких случајева од којих посебан значај има приказ акутног исемијског можданог удара код младог пацијента у сливу Перчеронове артерије истовремено са оболевањем од Covid-19.

Из радова под називом „In vitro antiglioma action of indomethacin is mediated through AMPK/mTOR complex 1 signaling pathway“ **(референца 2)** и „Data supporting the inability of indomethacin to induce autophagy in U251 glioma cells“ **(референца 10)** коршћен је највећи број резултата за докторску дисретацију кандидата. У поменутим радовима и дисертацији др Александар Пантовић износи податке о примарним туморима централног нервног система глиомима са посебним освртом на најчешћи малигни тумор централног нервног система глиобластом. У уводном делу наведени су подаци о доказима за антитуморско деловање нестероидних анти-инфламаторних лекова (НСАИЛ). Кандидат у уводном делу цитира и основне неурофармаколошке карактеристикаке нестероидног анти-инфламаторног лека индометацина, који је у истраживању показао најизраженији цитотоксични ефекат и једини од испитиваних лекова утицао на активацију AMPK молекула. Описана је улога сигналног пута AMPK/mTOR и модел mTORC1- зависне инхибиције AKT механизмима повратне спреге. Такође описан је молекуларни механизам апоптозе, програмиране ћелијске смрти типа I и аутофагије, катаболичког процеса који омогућује ћелијску деградацију сопствених протеина и органела у аутофаголизозомима.

Имајући у виду улогу AMPK у инхибицији mTOR и индукцији апоптозе и аутофагије, претпостављено је да НСАИЛ своје антиглиомско деловање остварују модулацијом AMPK/mTORC1 сигналног пута. Да би ова претпоставка била испитана постављени су следећи циљеви овог истраживања: (1) испитати утицај НСАИЛ на вијабилитет ћелија хумане ћелијске линије глиома У251 и примарних туморских ћелија изолованих из узорака ткива пацијената оболелих од глиобластома (2) испитати тип и механизме ћелијске смрти индуковане НСАИЛ у ћелијама глиома (3) испитати утицај НСАИЛ на активацију AMPK/mTOR сигналног пута у ћелијама глиома (4) испитати улогу AMPK/mTOR сигналног пута у индукцији ћелијске смрти индуковане НСАИЛ у ћелијама глиома.

У делу о материјалу и методама, др Пантовић описује експерименталне методе и услове под којима су експерименти изведени:

(а) Гајење хумане ћелијске линије глиобластома У251 као и изолацију примарних туморских ћелија изолованих из узорака ткива пацијената оболелих од глиома градуса IV. На Клиници за неурохирургију Клиничког центра Србије у Београду постављена је дијагноза основног обољења и узети су узорци ткива за изолацију примарне културе. Истраживање је спроведено у складу са Хелсиншком Декларацијом и одобрено од стране Етичког комитета Клиничког центра Србије и Етичког комитета Медицинског факултета Универзитета у Београду. Сви оболели су дали пристанак у писаној форми за учешће у истраживању и анализу узорака у дијагностичке и научне сврхе. Поглавље материјал и методе је подељено у следеће целине:

(б) Одређивање вијабилитета ћелија мерењем активности ћелијског ензима лактат дехидрогеназе, мерењем активности митохондријалних дехидрогеназа и кристал виолет тестом.

(е) Испитивање улоге AMPK/mTORC1 сигналног пута у цитотоксичној активности третираних ћелија мерењем вијабилности ћелија код којих је експресија AMPK инхибирана РНК интерференцијом, као и након третмана ћелија активатором mTORC1 леуцином. Такође, у циљу доказивања улоге AMPK/mTORC1 сигналног пута испитивано је да ли фармаколошки активатори AMPK метформин и AICAR попут индометацина остварују антиглиомске ефекте модулацијом овог сигналног пута.

(ф) Утврђивање типа и механизама ћелијске смрти. За ову сврху коришћена је проточна цитофлуориметрија за анализу апоптотске фрагментације ДНК, транслокације фосфатидилсерина, активације каспаза, продукције супероксида, промене потенцијала митохондријалне мембране, ацидификације ћелијске цитоплазме. Real-ime RT-PCR метода је коришћена за анализу експресије инхибитора циклин зависне киназе p21. Имуноблот метода за испитивање експресије или активације сигналних молекула који учествују у регулацији енергетске хомеостазе, ћелијеке пролиферације, апоптозе и аутофагије (AMPKα, ACC, Akt, Raptor, mTOR, S6K, PRAS40, каспазу-3, PARP, LC3, beclin-1).

(г) Утврђивање механизама ативације AMPK. Садржај AMP и ATP у ћелијама линије У251 је анализиран градијентном течном хроматографијом високих перформанси (HPLC) и биолуминисцентним есејем.

(х) Статистичке методе коришћене за обраду добијених података.

У резултатима кандидат јасно и документовано приказује добијене експерименталне податке о антиглиомском ефекту НСАИЛ на ћелије глиома У251 линије и примарне културе ћелија глиома градуса IV (глиобластома).

Први резултати експеримената су показали да индометацин снажније од других COX инхибитора, диклофенака, напроксена и кетопрофена, смањује вијабилитет У251 ћелија хуманог глиобластома. Такође у истраживању кандидата показано је да једино индометацин од коришћених нестероидних анти-инфламаторних лекова статистички значајано активира AMPK. Третман индометацином који је надаље коришћен у истраживању довео је до морфолошких промена на ћелијама У251 линије које су каратеристичне за апоптозу. У ћелијама које су третиране индометацином показане су промене у степену продукције реактивних кисеоничних врста, деполаризацији митохондрија, транслокацији фосфатидилсерина на површину ћелијске мембране, ДНК фрагментацији, активацији каспаза и PARP. Цитотоксичном ефекту индометацина претходило је повећање експресије тумор супресорског протеина p21 и застој ћелијског циклуса у Г2М фази. Приказни разултати директно доводе у везу улогу сигналног пута AMPK/mTORC1 са антиглиомским деловањем индометацина. Наиме, показано је да индометацин повећава фосфорилацију AMPK и нисходних молекула које ензим фосфорилише као што су Raptor и ацетил-CoA карбоксилаза (ACC). Активацију AMPK прати смањење фосфорилације mTOR и молекула чију активност mTORC1 регулише, као што су рибозомална p70S6 киназа (S6K) и PRAS40 (Сер183). Генетска инхибиција експресије AMPK молекула РНК интерференцијом, као и третман ћелија активатором mTOR1 леуцином довде до делимичног поништавања цитотоксичних ефеката изазваних индометацином. Са друге стране, третирање У251 ћелија инхибитором mTOR-а рапамицином смањује број живих ћелија у култури. Осим тога, фармаколошки активатори AMPK метформин и AICAR попут индометацина испољавају антиглиомски ефекат инхибицијом mTORC1. Истражујући механизме којима индометацин активира AMPK измерено је смањење ћелијског ATP и пораст AMP/ATP односа у третираним ћелијама док повезаност фосфорилације AMPK са инхибицијом COX и повећањем интраћелијског нивоа калцијума није нађена.

Резултати показују да је цитотоксични ефекат индометацина на ћелије примарне културе глиобластома такође посредован активацијом AMPK/Rapor/ACC и инхибицијом mTORC1/S6K сигналних молекула. Коначно, истраживана је и способност индометацина да индукује аутофагију у ћелијској линији глиобластома У251. Међутим, није детектовано повећање маркера аутофагије као нити повећање цитоплазматских везикула са киселим садржајем, експресија beclin-1 и конверзија лаког ланца 3-I (LC3-I) у аутофагозомну форму LC3-II у присуству лизозомалних инхибитора. Показано је да генетска и фармаколошка инхибиција аутофагије не утичу на цитотоксични ефекат индометацина, што је потврдило да је антиглиомски ефекат лека независан од аутофагије.

У дискусији резултата кандидат је на систематичан начин упоредио резултате приказане у овој публикацији са резултатима других истраживача. На основу тога, кандидат је изнео своје закључке и хипотезе, који објашњавају резултате ове пуликације у складу са резултатима већине других аутора који су имали исто истраживачко интересовање.

У поглављу закључци су сумирани сви резултати добијени у овом истраживању који у потпуности одговарају на циљеве постављене на почетку.

У најцитиранијој публикацији на којој кандидат партиципира као коаутор **(референца 1)** поређена је фототермална антиканцерска активност графенских наночестица и угљеничних наноцеви побуђених спектром који је близак инфрацрвеној светлости. Упркос нижем капацитету апсорбовања графенских наночестица оне су генерисале више топлоте од угљеничних наноцеви растворених под одоговарајућим условима. Механизми фототермалног убијања канцерских ћелија посредованог графеном су укључивали оксидативни стрес и деполаризацију митохондријалне мембране што је доводило до мешовите апоптотске и некротичне ћелијске смрти.

У наредном истраживању **(референца 3)** аутори су процењивали улогу протеин киназе активиране од AMP (AMPK), молекуле Akt, mTOR и аутофагију као и интеракцију наведних молекула у диференцијацији мезенхимских матичних ћелија. Добијени подаци указују да AMPK контролише диференцијацију хумане мезенхимске матичне ћелије кроз рану аутофагију посредовану инхибицијом mTOR-а и касну активацију Akt/mTOR сигналне осе.

У ревијалном раду у којем је др Пантовић учествовао као део међународне групе истраживача **(референца 4)** дата су разматрања о употреба четири транскрипциона фактора, Oct3/4, Sok2, c-Mic и Klf4 –тзв. "Јаманака фактори" у конверзији диференцираних ћелија, назад у плурипотентни/ембрионални стадијум. Овако "репрограмиране" ћелије отворају велико поље могућности примене укључујући и неурорегенерацију али и етичку дилему медицинске употребе због потенцијала за малигу трансформацију.

Истраживана је способност хлорокина, лизозомског инхибитора аутофагије, како би се побољшао ефекат против малигних ћелија које су изгладњиване у in vitro условима **(референца 5)**. Хлорокин је брзо убио малигне ћелије механизмом независним од аутофагије. Нађено је да су основни механизми цитотоксичности акумулација лизозома изазвана хлорокином, оксидативни стрес, митохондријална деполаризација, активација каспазе и мешана апоптотска/некротична ћелијска смрт ћелије. Хлорокин је инхибирао и раст меланома мишева са ограниченим уносом калорија сличним механизмом. Закључено је да комбиновани третман са хлорокином и калоријском рестрикцијом може бити користан у терапији канцера.

Циљ наредног истраживања **(референца 6)** у којем је др Пантовић учествовао као коаутор био је да се примени фрактална анализа за процену ултраструктурних промена у раним фазама апоптозе. Апоптоза је индукована у У251 ћелијској линији хуманог глиома излагањем утравиолентном (УВ) спектру светлости. Ћелије су визуелизоване оптичком фазно-контрастном микроскопијом и фотографисане пре УВ третмана, непосредно након третмана, као и у интервалима од 30 мин током периода посматрања од 5 сати. За сваку од 32 анализиране ћелије, ћелијска и нуклеарна фрактална димензија, као и нуклеарна лакунарност, одређене су у свакој временској тачки. Наши подаци показују да ћелијска ултраструктурна сложеност одређена фракталном димензијом и лакунарношћу значајно опада након УВ зрачења, при чему је нуклеарна лакунарност посебно осетљив параметар у откривању ране апоптозе. Фрактална анализа била у стању да открије ћелијске апоптотске промене пре конвенционалне проточне цитометријске анализе екстернализације фосфатидилсерина, фрагментације ДНК и пермеабилизације ћелијске мембране. Ови резултати указују да би фрактална анализа могла бити моћна и приступачна метода за неинвазивну рану идентификацију апоптозе у ћелијским културама.

У наредном истраживању испитиване су хемолитичке особине наночестица фулерена (C60) припремљених изменом растварача тетрахидрофурана (nC60THF) или са механохемијски потпомогнутим формирањем комплекса са макроцикличним олигосахаридом гама циклодекстрином (nC60CDKS) или кополимером етилен винил ацетат–етилен винил версататом (nC60EVA–EVV) **(референца 7)**. Спектрофотометријска анализа ослобађања хемоглобина открила је да је само nC60THF, али не и nC60CDKS или nC60EVA–EVV, био у стању да изазове лизу еритроцита на начин који зависи од дозе и времена. Микроскопски је утврђено да хемолизи посредованој nC60THF претходи скупљање еритроцита и повећање храпавости површине ћелије. Проточна цитометријска анализа је потврдила смањење величине еритроцита и показала значајно смањење повећања производње реактивних врста кисеоника у црвеним крвним зрнцима изложеним nC60THF. Хемолитичку активност изазвану nC60THF ефикасно су смањили антиоксиданси Н-ацетилцистеин и бутиловани хидроксианизол, као и серумски албумин, најзаступљенији протеин у крвној плазми човека. Ови подаци указују да nC60THF може изазвати хемолизу која се може спречити у серумском албумину кроз оштећење мембране еритроцита посредовано оксидативним стресом.

Следећа публикација садржи истраживање ефеката лечења нарушене глукорегулације на нивое проинфламаторних Th17 цитокина, интерлеукина IL-17 і IL-23, Th1, интерферона (INF)-γ и IL-12 у серуму код новодијагностикованих пацијената са дијабетесом типа 2 **(референца 9)**. Нивои цитокина у серуму, гликозилираног хемоглобина (HbA1C), индекс процене модела хомеостазе као мера инсулинске резистенције (HOMA-IR), и индекс телесне масе (BMI) су одређени пре и после 12 недеља терапије која се састојала од стандардних модификација начина живота и метформина (1000 mg два пута дневно). Нивои Th17 и Th1 цитокина пре третмана нису били у корелацији са узрастом, BMI или HOMA-IR. Пацијенти са лошом глукорегулацијом (HbA1C >7%), у поређењу са онима са добром глукорегулацијом (HbA1C ≤7%), имали су виши ниво Th17 и Th1 цитокина у серуму, али само разлике у IL-17 и IFN-γ достигле су статистичку значајност (p=0,003 и p=0,012). Смањење вредности HbA1C (са 8,6 на 5,9%, п=0,000) примећено код пацијената са лошом регулацијом глукозе било је повезано са значајним смањењем концентрације IL-17 (са 21,2 на 12,9 pg/ml, p=0,020), али не і INF-γ (50,6 наспрам 52,3, p=0,349). Ови подаци указују на то да терапијско побољшање глукорегулације може допринети смањењу нивоа IL-17 код новодијагностикованих пацијената са дијабетесом типа 2.

Клинички публицистички допинос кандидата је поткрепљен са три публикације у којима су представљени случајеви пацијената са лонгитуиналним екстензивним трансферзлним мијелитисом **(референца 11)**, функционалном слабошћу мишића покретача очних јабучица **(референца 12)** и публикација у којој је претстављен случај пацијента млађе животне доби са можданим ударом у сливу Перчеронове артерија током оболевања од COVD-19 **(референца 10)**.

4. НАУЧНО-ИСТРАЖИВАЧКИ РАД

4.1. Учешће у реализацији научних пројеката и ангажовање у руковођењу научним радом

Др сц. мед. Александдар Пантовић је учествовао у реализацији 2 научноистраживачка пројекта:

1. Имуномодулаторно, цитопротективно и цитотоксично дејство наночестица (број пројекта 145073)
2. Модулација унутарћелијског енергетског баланса – контрола сигналних путева у терапији тумора и неуро-имуно-ендокриних поремећаја (број пројекта 41025)

5. КВАЛИТАТИВНИ ПОКАЗАТЕЉИ НАУЧНОГ УСПЕХА

5.1. Организација научног рада

До сада др Александар Пантовић није руководио пројектима нити пројектним задацима.

5.2. Ангажованост у образовању и формирању научних кадрова

Др сц. мед. Александар Пантовић води практичну наставу као сарадник у настави од марта месеца 2023. г.

5.3. Рецензија радова публикованих у научним часописима и предлога за пројекте

Др сц. мед. Алексанадр Пантовић није имао искусства као рецензент.

5.4. Међународна сарадња

- Др сц. мед. АлександарПантовић је више пута учествовао на домаћим и међународним конгресима као аутор радова
- IHead Acadmemy: Precision medicine approach for migraine management
- COST Action Proposal OC-2023-1-26779, поднета апликација

5.5. Чланства и активност у научним друштвима

Др сц. мед. Александар Пантовић је члан следећих научних друштава:

- Društvo neurologa Srbije (DNS)
- Nacionalno udruženje za glavobolje Srbije (NUGS)
- International Society of Neuroimmunology (ISNI)
- Movement disorder society (MDS)
- European Federation of Neurological Societies (EFNS)
- Federation of European Neuroscience Societies (FENS)
- International Headache Society (IHS)

5.6. Оригиналност научног рада, степен самосталности у научноистраживачком раду и улога у реализацији радова

- Током научноистраживачког рада кандидат је исказао познавање научноистраживачке методологије. Учествовао је у свим фазама научноистраживачког процеса, од дизајнирања истраживања па све до публиковања рада. У неколико радова заузима прво или друго место у списку коаутора, а такође у неколико радова је аутор за кореспонденцију.
- Од почетка научног и клиничког рада, др Пантовић је објавио 29 публицистичких јединица, од тога 10 радова штампаних у целини у међународним часописима (7×M_{21} + 2xM_{22} + 1xM_{23}). Збирни импакт фактор радова које је др Пантовић до сада публиковао је **39,583** (као први аутор збирни импакт фактор је **12,841**). Просечан број коаутора у радовима др Пантовића публикованим у целости износи осам. На неколико радова број аутора је већи од осам, што је условљено мултидисциплинарним истраживчким приступом.
- Др Пантовић је од почетка научног и клиничког рада објавио 12 радова *in extenso*, при чему је на 6 био први аутор. На основу захтева Правилника о избору у звање збир од потребних 10 боова у категоријама $M_{10}+M_{20}+M_{31}+M_{32}+M_{33}+M_{41}+M_{42}$ износи 72.5 (нормирано 54.04), односно, од потребних 6 бодова у категоријама $M_{11}+M_{12}+M_{21}+M_{22}+M_{23}$, збир износи 71 (нормирано 52,55).
- Пуубликације у којима је Др Пантовић први аутор или коаутор до сада су цитиране преко 900 пута (хетероцитати): Scopus 901; Web of Science 850

1. ПЕТ НАЈЗНАЧАЈНИХ НАУЧНИХ ОСТВАРЕЊА

Према мишљењу Комисије међу најважнијим научним остварењима др сц. мед. Александра Пантовића истичу се следећи радови:

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6.1.Утицајност

- Др Пантовић је до сада цитиран 901 пута (хетероцитати): Scopus 901; Web of Science 850

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Markovic Z, Harhaji-Trajkovic L, Todorovic-Markovic B, Kepić D, Arsikin K, Jovanović S, **Pantovic A,** Dramićanin M, Trajkovic V. In vitro comparison of the photothermal anticancer activity of graphene nanoparticles and carbon nanotubes. Biomaterials. 2011;32(4):1121-9. IF 7.404 (10 бодова)

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7. КВАЛИТЕТ НАУЧНИХ РЕЗУЛТАТА

Др сц. мед. Александар Пантовић публиковао је значајне радове на пољу неуронауке, молекуларне медицине и клиничке неурологије. Већина радова је публикован у међународним часописима. Током научноистраживачког рада кандидат је исказао познавање научноистраживачке методологије. Учествовао је у свим фазама научноистраживачког процеса, од дизајнирања истраживања па све до публиковања рада. У неколико радова заузима прво или друго место у списку коаутора, а такође је у неколико радова и аутор за кореспонденцију.

Од почетка научног и клиничког рада, др Пантовић је објавио 29 публицистичких јединица, од тога 10 радова штампаних у целини у међународним часописима (7×M21 + 2xM22 + 1xM23). Збирни импакт фактор радова које је др Пантовић до сада публиковао је 39,583 (као први аутор збирни импакт фактор је 12,841). Просечан број коаутора у радовима др Пантовића публикованим у целости износи осам.

Др Пантовић је од почетка научног и клиничког рада објавио 12 радова in extenso, при чему је на 6 био први аутор. На основу захтева Правилника о избору у звање збир од потребних 10 у категоријама M10+M20+M31+M32+M33+M41+M42 износи 72.5 (нормирано 54.04), односно, од потребних 6 у категоријама M11+M12+M21+M22+M23, збир износи 71 (нормирано 52,55).

Др Пантовић је до сада цитиран 901 пута (хетероцитати): Scopus 901; Web of Science 850

Табела 3. Укупне вредности М коефицијента кандидата од почетка каријере према категоријама прописаним у Правилнику за област природно-математичих и медицинских наука.

КРИТЕРИЈУМИ МИНИСТАРСТВА		РЕЗУЛТАТИ КАНДИДАТА		
			Укупно	НББ
УКУПНО	16	УКУПНО	85	66,55
M10+M20+M31+M32+M33+M41+M42	10	M10+M20+M31+M32+M33+M41+M42	72.50	54.04
M11+M12+M21+M22+M23	6	M11+M12+M21+M22+M23	71	52,55

8. ЗАВРШНО МИШЉЕЊЕ И ПРЕДЛОГ КОМИСИЈЕ

Истраживачки рад др сц. мед. Александра Пантовића је везан за област молекуларне медицине и неурологије. Од почетка каријере, кандидат је објавио укупно 29 публицистичких јединица, од тога је 10 радова штампано у целини, са укупним фактором утицаја од 39,53 и са преко 900 хетероцитата.

Приказани резултати научног рада указују да је др сц. мед. Александар Пантовић својим истраживањима допринео развоју научне области којом се бави. Посебно треба истаћи да је део резултата његових истраживања по први пут објашњава цитотоксичне механизме појединих нестероидних антиинфламатарних лекова на ћелије глиома и молекуларне механизме активације и улогу процеса аутофагије у диференцијацији матичних ћелија. Радови др сц. мед. Александра Пантовића су објављени и у значајним међународним часописима. Током свог истраживачког рада, др сц. мед. Александар Пантовић је био самосталан истраживач, способан да осмисли и реализује истраживање. Др сц. мед. Пантовић је објавио и неколико клиничких приказа случајева из области неурологије у домаћим и страним часописима.

Узимајући у обзир квантитет и квалитет публикованих резултата као и остале квалитативне показатеље успеха у научном раду, Комисија сматра да кандидат испуњава све законом прописане критеријуме за стицање научног звања научни сарадник, донетих од стране Министарства просвете, науке и технолошког развоја Републике Србије, те са великим задовољством предлаже Научном већу Војномедицинске академије да усвоји извештај и предлог Комисије да се др сц. мед. Александар Пантовић, специјалиста неурологије, изабере у звање научни сарадник.

ЧЛАНОВИ КОМИСИЈЕ:

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У Београду, 27.10.2023.

РЕПУБЛИКА СРБИЈА
МИНИСТАРСТВО ОДБРАНЕ
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