

Międzywydziałowe Koło Naukowe Biochemii Medycznej

Wydział Nauk Medycznych & Wydział Biologii i Biotechnologii



Opiekun Koła Naukowego:
prof. dr hab. Elżbieta Kostyra, prof. zw.

Przewodniczący Koła Naukowego:
mgr Michał Matysiewicz

Kim jesteśmy?

- Międzywydziałowe Koło Naukowe Biochemii Medycznej istnieje **od 2011 roku**.
- Wcześniej Koło funkcjonowało jako *Koło Naukowe Biochemików* (założone w 2002 roku)
- Jesteśmy Kołem Naukowym zrzeszającym:
 - **studentów** Wydziału Biologii i Biotechnologii oraz Wydziału Nauk Medycznych
 - **doktorantów** Wydziału Biologii i Biotechnologii z Katedry Biochemii
- Obecnie Koło **zrzesza 9 członków**



Profil badawczy koła

Prowadzimy badania z zakresu biochemii medycznej, wykorzystując do tego posiadaną wiedzę, kreatywność, metody biologii molekularnej oraz technik hodowli komórkowych *in vitro*.

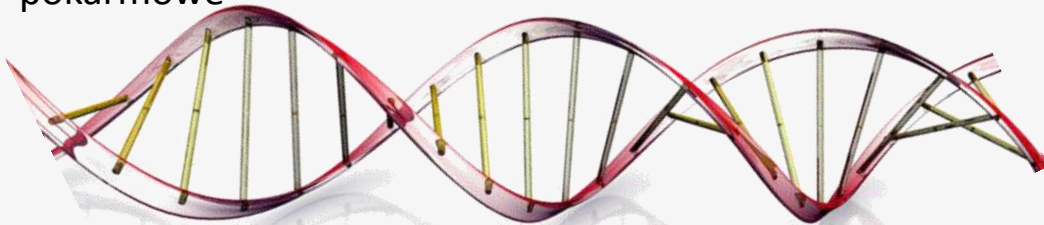
Prowadzimy badania z zastosowaniem nowotworowych linii komórkowych:

- **Caco-2** – linia raka okrężnicy,
- **HCT116** – linia raka okrężnicy,
- **MCF7** – linia raka piersi,
- **HepG2** – linia raka wątroby,

oraz **PBMC** (ang. peripheral blood mononuclear cel),

na których sprawdzamy cytotoksyczność związków biologicznie czynnych, transport związków przez monowarstwę komórkową, wpływ na proliferację komórek, wpływ na sekrecję cytokin oraz zmianę ekspresji wybranych genów (gł. *MOR* - *mu-opioid receptor*).

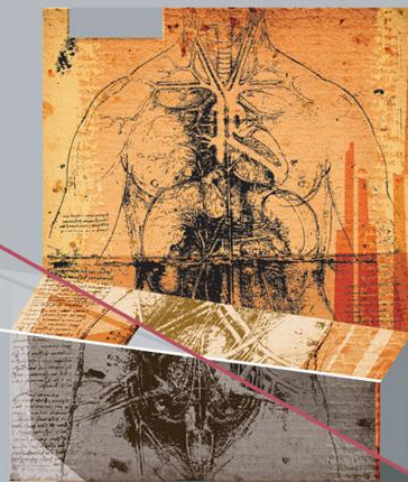
Dodatkowo prowadzimy badania nad wykrywaniem **mutacji punktowych SNP** u ludzi z uzależnieniami oraz różnymi zespołami chorobowymi takimi jak: ostre zapalenie trzustki, autyzm, nowotwory, alergie pokarmowe



Przegląd działalności koła naukowego w roku akademickim 2013/2014



	HEMII MEDYCZNEJ Współpraca z: AMC.edu.pl/pierwszokola	
		Członkowie - Anna Cieślinska - Joanna Koscuszk - Mateusz Jarczuk - Ania Janicka - Lukasz Jasiewicz - Katarzyna Regin - Ewa Fiedorowicz - Berta Białko - Michał Mayskiwicz - Konrad Patyra - Natalia Śmukała



Poster (Warszawa, 18-21 wrzesień) I miejsce w XII sekcja posterowej

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Selected cytokines and opioid peptides in the colorectal cancer

Jadwiga Śniarska, Michał Malszewicz, Michał Tenderenda, Bartłomiej Kocbach, Konrad Włoriski, Eżbieta Kostyra

Zespół Biochemii Medycznej – Sekcja Peptydów i Białek, Wydział Biologii i Biochemologii; Uniwersytet Warmińsko-Mazurski w Olsztynie
Katedra Chirurgii, Wydział Nauk Medycznych; Uniwersytet Warmińsko-Mazurski w Olsztynie
Katedra Onkologii, Wydział Nauk Medycznych; Uniwersytet Warmińsko-Mazurski w Olsztynie

Wstęp

Zgodnie z informacjami z międzynarodowej statystycznej bazy danych GLOBOCAN, każdego roku na świecie odnotowuje się 1,234 mln nowych przypadków raka jelita grubego, a więcej niż 800 tys. pacjentów umiera corocznie z powodu tego nowotworu złośliwego. Także według danych GLOBOCAN, w 2008 roku w Polsce odnotowano 16 374 nowych przypadków raka jelita grubego oraz 10 407 zgonów z powodu tego nowotworu. Wyniki leczenia są nadal niezadowalające co wynika ze zbyt późnego rozpoznania tej choroby.

Cel

- Celem podjętych badań było określenie stężenia 31-amino-kwasowego peptydu opioidowego - ludzkiej β -endorfiny, w moczu, surowicy krwi oraz homogenacie nowotworowo zmienionej tkanki jelita grubego.
- Dodatkowo w materiale oznaczono wybrane cytokiny: IL-6, TNF- α (cytokiny prozapalne) oraz IL-10 (cytokina inhibitorowa).

Pacjenci i metody

- Badaniem objęto 21 chorych z potwierdzonym histopatologicznie nowotworem jelita grubego (14 kobiet, 7 mężczyzn; wiek 46-82, średnia wieku: 65 lat), leczonych w Klinice Chirurgii Ogólnej lub Klinice Chirurgii Onkologicznej działającymi przy Wydziale Nauk Medycznych UWM w Olsztynie.
- Do badań wykorzystano materiał przedoperacyjny (mocz i surowica krwi) oraz materiał śródoperacyjny (nowotworowo zmieniony fragment tkanki jelita grubego).
- Stężenie ludzkiej β -endorfiny w moczu, surowicy krwi oraz homogenacie nowotworowo zmienionej tkanki jelita grubego oznaczono za pomocą metodą enzymatyczną ELISA (EIAab, China); podobnie jak stężenie wybranych cytokin (Mabtech, Szweden).
- Na przeprowadzenie badań uzyskano zgodę Okręgowej Komisji Bioetycznej.

Wyniki

- Oznaczone stężenie β -endorfiny w moczu i surowicy wyniosło odpowiednio: 85,8pg/ml i 96,2pg/ml, natomiast w homogenacie tkanki nowotworowo zmienionej - 24,8pg/g. Obecność ludzkiej β -endorfiny w tkance nowotworowej potwierdzono także wykonując immunocytochemię (ICC) ^{tab.1}.
- Ocena stężenia wybranych cytokin w moczu wyniosła: 24,0pg/ml (IL-6), 70,5pg/ml (TNF- α) oraz 3,9pg/ml (IL-10); w surowicy poziom cytokin zapalnych IL-6 i TNF- α odpowiednio 159,7pg/ml i 79,6pg/ml a cytokiny inhibitorowej IL-10 - 60,9pg/ml. W homogenacie tkanki nowotworowej IL-6 i TNF- α odpowiednio 589,1pg/g, 102,4pg/g oraz IL-10 - 63,5pg/g ^{tab.1}.
- Przeprowadzona analiza uzyskanych wyników między sobą wykazała silną korelację pomiędzy stężeniem β -endorfiny w homogenacie guza i jej stężeniem w surowicy krwi ($r = 0,7548$; $p < 0,001$) ^{rys.4}.

	Mocz	Surowica	Homogenat tkanki nowotworowej
β -endorfina (norma 20-30pg)	85,8pg/ml (n=21)	96,2pg/ml (n=21)	24,8pg/g
IL-6 (norma 1,4-14,1pg)	24,0pg/ml (n=21)	159,7pg/ml (n=21)	589,1pg/g
TNF- α (norma 0,2-10pg)	70,5pg/ml (n=21)	79,6pg/ml (n=21)	102,4pg/g
IL-10 (norma 0,1-10pg)	3,9pg/ml (n=21)	60,9pg/ml (n=21)	63,5pg/g

Tablica 1. Stężenie średnich stężeń oznaczanych markerów. W nawiasach podano zakres i liczbę wzrostu; ¹ - dane literaturowe.

Wykres 1. Średnie stężenie oznaczanych markerów w homogenacie tkanki nowotworowej.
Legenda: β -endorfina (ciemnoniebieski); IL-6 - niebieski; TNF- α - zielony; IL-10 - fioletowy.

Wykres 2. Średnie stężenie oznaczanych markerów w moczu.
Legenda: β -endorfina (ciemnoniebieski); IL-6 - niebieski; TNF- α - zielony; IL-10 - fioletowy.

Wykres 3. Średnie stężenie oznaczanych markerów w surowicy krwi.
Legenda: β -endorfina (ciemnoniebieski); IL-6 - niebieski; TNF- α - zielony; IL-10 - fioletowy.

Wykres 4. Korelacja zachodząca pomiędzy stężeniem β -endorfiny w homogenacie tkanki nowotworowej a jej stężeniem w surowicy krwi. Wskazano na korelację silną dodatnią. R² = 0,568; p < 0,001; współczynnik korelacji = 0,7548.

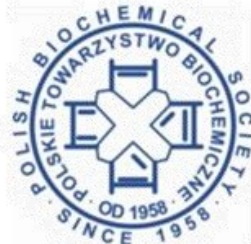
Wniosek:

Wstępne wyniki badań wskazują na przydatność oznaczeń wybranych cytokin oraz ludzkiej β -endorfiny we wczesnej diagnostyce raka jelita grubego.

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The link between A118G polymorphism in the μ -opioid receptor (MOR) gene and predisposition to the occurrence of addictions

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- Introduction -

The opioid receptors belong to G-protein coupled family. There are four types of opioid receptors: MOR, NMR, KOR, and ORR. These receptors are mainly located in the nervous system. Furthermore, they can also be found in other body tissues such as smooth muscles. They send signals associated with pain perception, mood, and the regulation of mood control. The opioid receptors also play a role in the addictiveness of opiates, cocaine, or alcohol, especially the μ -opioid receptor (MOR). A high number of single nucleotide polymorphisms (SNPs) has been detected in the μ -opioid receptor (MOR) gene. One of the most extensively studied SNPs is a substitution, serine to guanine at position 118 of nucleotide chain which results in the substitution of asparagine to aspartate at position 40 of amino acid chain (diagram 1). This mutation occurs with the allele frequency of 10-32% in different ethnic groups. Some of A118G polymorphisms studies suggest that there is a contribution of the G allele at the increased risk of addiction to heroin and alcohol. Moreover, the occurrence of G allele may decrease receptor binding ability in smokers.

- Aim of study -

The aim of the study was to determine the relationship between A118G polymorphism occurrence in the μ -opioid receptor (MOR) gene and the predisposition to addiction to different substances (alcohol, energy drink, cigarette, coffee, cigarettes) among students. Furthermore, the percentage of genotypes AA, AG, and GG in the study population was estimated.

- Materials and methods -

The questionnaire was distributed among second students. Based on the survey, the frequency of drug use was estimated. In addition, the DNA was isolated from students' blood. The A118G polymorphism of MOR gene was determined using the PCR-RFLP method (Figure 1). The percentage of examined genotypes was estimated. The obtained results were correlated with the questionnaire answers. The outline of procedure is shown in the diagram 2.

Diagram 1: Expression of μ -opioid receptor gene with A118G polymorphism

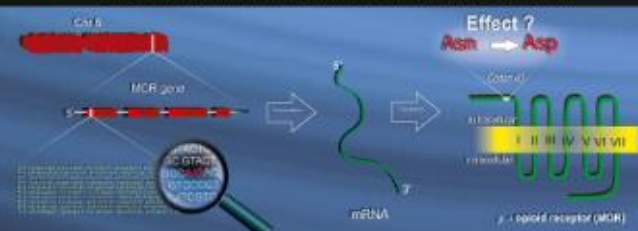


Diagram 2: Outline of the experiment



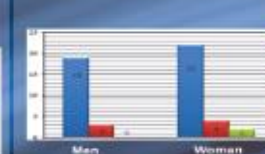
- Results -

On the basis of the results, the percentage of genotype AA was 82%, AG - 14% and GG 4% in tested population. Due to the low share of one of the genotypes, there was no association between the occurrence of the genotype and predisposition to addiction, but one can notice some relations. The obtained results and data on the necessity to continue working on the μ -opioid receptor (MOR) gene and its susceptibility to addictions.

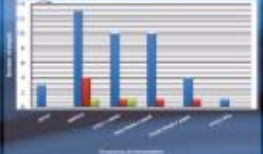
Legend



Comparison of gender and the obtained genotype



Frequency of alcohol consumption



Frequency of sweet consumption



Frequency of coffee consumption



Frequency of energy drink consumption



Frequency of smoking



- Study conclusion -

- There was no association between A118G polymorphism and predisposition to addiction among consumers of coffee, alcohol and sweets.
- Among of energy drink consumers and smokers only AA genotype was present.
- The rarity of GG genotype makes it difficult to accurately investigate the tendency to addiction among heterozygotes and homozygotes.

Poster

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IDENTIFYING THERMOSTABILITY OF MIXES USED IN PCR

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- INTRODUCTION -

PCR (Polymerase Chain Reaction) is one of the most frequently used methods of genetic material duplication. It allows specific amplification of DNA fragment in the laboratory. The reaction mixture includes: DNA - which is a matrix to reproduce, two primers - short nucleotide sequences designating the amplified nucleic acid fragment, polymerase - an enzyme involved in the synthesis of newly synthesized DNA, deoxyribonucleotides triphosphates - the building blocks for the synthesis of the PCR product and a buffer with magnesium ions, which provides a suitable chemical environment. These components (except DNA and primers) may be included in the final set of so-called reagent - mix. Manufacturers of the mixes suggest that they should be stored at -20°C, otherwise they might lose their activity. Component of the mix, the most sensitive to temperature is the polymerase. Frequently used is Taq polymerase, isolated from bacteria *Thermus aquaticus*.

- MATERIALS AND METHODS -

The first stage of the study was to make standard PCR reactions with the six commercially available mixes stored at temperatures recommended by the manufacturer (-20°C) to check if a product PCR is present (control probe). After PCR reaction, electrophoresis in 1.5% agarose gel was carried out. Obtained band showed the correct PCR reaction. No band in the path with probe marked degradation of the reagent. In order to check the thermal stability of the reagent six mixes were incubated for 12, 24, 48, 72 hours and 7, 13, 18, 19, 23 and 28 days at two temperatures: at +4°C and at room temperature (+22°C). Reagent resistance to freezing thawing was examined by multiple (approximately 50 and 100 times) freezing (-20°C) and thawing. The scheme below shows terms of the procedure and steps of the experiment.

- THE AIM OF THE RESEARCH -

The aim of the experiment was to check whether the storage of mixes at different temperatures than those recommended by the manufacturers may affect the quality of the PCR reaction. Furthermore, it was examined whether repeated freezing and thawing the mix will affect his performance in the reaction.



- RESULTS AND DISCUSSION -

During the PCR reaction mixture components must be sufficiently resistant to high temperatures (about +50 to +95°C for about 30 seconds - few minutes), so as not to degrade during subsequent stages of the reaction. In addition, polymerase occurs in species of bacteria, the living environment temperature exceeds +40°C. The conducted experiments show that the tested reagents for PCR (despite the manufacturer's instructions to store at -20°C) are able to stand at room temperature for some time. At 4°C some of them did not lose its activity even after 28 days. At room temperature these reagents can be stored for 72 h without affecting their activity. After 7 days of incubation, some of the tested mixes have lost their functionality. In the case of multiple freezing, four of the six tested mixes were useful in the PCR reaction. PCR results obtained from the incubation mixes at two temp. at different time and by freeze thawing are shown in the table 1. Sample images of electrophoresis are given below for mix 1 (Fig. 1) and mix 6 (Fig. 2).



Figure 1: The electrophoresis of PCR reactions using mix 1. Paths 1, 2, 3, 4, 5 - incubation at 4°C, for 12, 24, 48, 72 hours and 7 days, respectively. Path 6, 7, 8 - incubation at 22°C, for 12, 24, 48, 72 hours and 7 days, respectively. Path 9 - blank. Path 10 - DNA size marker.



Figure 2: The electrophoresis of PCR reactions using mix 6. Paths 3, 4, 5, 6, 7, 8 - incubation at 4°C, respectively for 7, 13, 18, 19, 23, 28 days. Path 9, 10, 11, 12, 13, 14 - incubation at 22°C for 7, 13, 18, 19, 23, 28 days, respectively. Path 1 and 15 - DNA size marker. Path 2 - blank.

Table 1: Results of the PCR reaction using mixes incubated at 4°C and +22°C at a specified time and by freeze thawing

	Mix 1	Mix 2	Mix 3	Mix 4	Mix 5	Mix 6		Mix 1	Mix 2	Mix 3	Mix 4	Mix 5	Mix 6
Control (BPC)	+	+	+	+	+	+	Control (BPC)	+	+	+	+	+	+
4°C/12 h	+	+	+	+	+	+	22°C/12 h	+	+	+	+	+	+
4°C/24 h	+	+	+	+	+	+	22°C/24 h	+	+	+	+	+	+
4°C/48 h	+	+	+	+	+	+	22°C/48 h	+	+	+	+	+	+
4°C/72 h	+	+	+	+	+	+	22°C/72 h	+	+	+	+	+	+
4°C/7 days	+	+	+	+	+	+	22°C/7 days	+	+	+	+	+	+
4°C/13 days	+	+	+	+	+	+	22°C/13 days	+	+	+	+	+	+
4°C/19 days	+	+	+	+	+	+	22°C/19 days	+	+	+	+	+	+
4°C/23 days	+	+	+	+	+	+	22°C/23 days	+	+	+	+	+	+
4°C/28 days	+	+	+	+	+	+	22°C/28 days	+	+	+	+	+	+
4°C/50 days	+	+	+	+	+	+	22°C/50 days	+	+	+	+	+	+
4°C/100 days	+	+	+	+	+	+	22°C/100 days	+	+	+	+	+	+
4°C/150 days	+	+	+	+	+	+	22°C/150 days	+	+	+	+	+	+
4°C/200 days	+	+	+	+	+	+	22°C/200 days	+	+	+	+	+	+
4°C/250 days	+	+	+	+	+	+	22°C/250 days	+	+	+	+	+	+
4°C/300 days	+	+	+	+	+	+	22°C/300 days	+	+	+	+	+	+
4°C/350 days	+	+	+	+	+	+	22°C/350 days	+	+	+	+	+	+
4°C/400 days	+	+	+	+	+	+	22°C/400 days	+	+	+	+	+	+
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4°C/600 days	+	+	+	+	+	+	22°C/600 days	+	+	+	+	+	+
4°C/650 days	+	+	+	+	+	+	22°C/650 days	+	+	+	+	+	+
4°C/700 days	+	+	+	+	+	+	22°C/700 days	+	+	+	+	+	+
4°C/750 days	+	+	+	+	+	+	22°C/750 days	+	+	+	+	+	+
4°C/800 days	+	+	+	+	+	+	22°C/800 days	+	+	+	+	+	+
4°C/850 days	+	+	+	+	+	+	22°C/850 days	+	+	+	+	+	+
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4°C/2150 days	+	+	+	+	+	+	22°C/2150 days	+	+	+	+	+	+
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4°C/2250 days	+	+	+	+	+	+	22°C/2250 days	+	+	+	+	+	+
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4°C/2900 days	+	+	+	+	+	+	22°C/2900 days	+	+	+	+	+	+
4°C/2950 days	+	+	+	+	+	+	22°C/2950 days	+	+	+	+	+	+
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4°C/3050 days	+	+	+	+	+	+	22°C/3050 days	+	+	+	+	+	+
4°C/3100 days	+	+	+	+	+	+	22°C/3100 days	+	+	+	+	+	+
4°C/3150 days	+	+	+	+	+	+	22°C/3150 days	+	+	+	+	+	+
4°C/3200 days	+	+	+	+	+	+	22°C/3200 days	+	+	+	+	+	+
4°C/3250 days	+	+	+	+	+	+	22°C/3250 days	+	+	+	+	+	+
4°C/3300 days	+	+	+	+	+	+	22°C/3300 days	+	+	+	+	+	+
4°C/3350 days	+	+	+	+	+	+	22°C/3350 days	+	+	+	+	+	+
4°C/3400 days	+	+	+	+	+	+	22°C/3400 days	+	+	+	+	+	+
4°C/3450 days	+	+	+	+	+	+	22°C/3450 days	+	+	+	+	+	+
4°C/3500 days	+	+	+	+	+	+	22°C/3500 days	+	+	+	+	+	+
4°C/3550 days	+	+	+	+	+	+	22°C/3550 days	+	+	+	+	+	+
4°C/3600 days	+	+	+	+	+	+	22°C/3600 days	+	+	+	+	+	+
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4°C/4000 days	+	+	+	+	+	+	22°C/4000 days	+	+	+	+	+	+
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4°C/4150 days	+	+	+	+	+	+	22°C/4150 days	+	+	+	+	+	+
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4°C/4250 days	+	+	+	+	+	+	22°C/4250 days	+	+	+	+	+	+
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Transport of the opioid peptides across Caco-2 monolayer

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1. Introduction

Milk is the source of β -casomorphins (BCMs) – biologically active peptides with opioid activity – which are suspected to play various roles in the human body. The local influence of exogenous opioid peptides on gastrointestinal functions has been widely reported. After passing the gut barrier, β -casomorphins may affect the functions of immunological system, as well as dopaminergic, serotonergic and GABA-ergic systems in brain, regulate the opioid receptor development and elicit behavioral effects. However, possibilities and mechanisms of the intestinal transport of β -casomorphins in human body *in vivo* have not been accurately described so far. However, it is known that in the gastrointestinal tract as well as in the blood, β -casomorphins can be degraded by the dipeptidyl peptidase IV (DPP-IV; EC 3.4.14.5), a highly specified aminopeptidase that cleaves dipeptides (X-Pro) from N-terminus fragments of peptide.

2. Aim

The aim of this research was to determine the permeability coefficients of the μ -opioid agonists human β -casomorphin-5 and -7 in time intervals, and to compare them with the permeability coefficient of μ -opioid antagonist peptide, human lactoferrin A.

4. Results

The permeability coefficients (Papp) were calculated according to:

$$P_{app} = \frac{dQ}{dt} \frac{1}{A C_0}$$

where: Q is the final amount of the peptide in the basolateral container (ng ml⁻¹), t is the time of the experiment (s), C₀ is the initial amount of the peptide in the apical container (ng ml⁻¹), A is the area of the insert (cm²).

4.1. Transepithelial flux of peptides across the Caco-2 monolayer

Table 1. Comparison of permeability coefficients for three investigated peptides in two transport directions

Peptide	Papp [$\times 10^{-4}$ cm s ⁻¹] $\pm$ S.D.	
	Apical-basolateral	Basolateral-apical
β -Casomorphin-5	0.63 $\pm$ 0.03	0.19 $\pm$ 0.03
β -Casomorphin-5 with diprotin A	5.12 $\pm$ 0.12	2.08 $\pm$ 0.12
β -Casomorphin-7	6.70 $\pm$ 0.83	4.95 $\pm$ 0.84
β -Casomorphin-7 with diprotin A	14.04 $\pm$ 0.80	19.07 $\pm$ 0.85
Lactoferrin A	1.32 $\pm$ 0.15	0.81 $\pm$ 0.18

5. Conclusions

Our studies have shown that the transport of opioid peptides across epithelial cells may be more efficient in the case of lower DPP-IV activity. These data have also confirmed that DPP-IV is the main factor limiting the half-life of opioid peptides in the intestine. Intestinal transport of μ -opioid receptor agonist and antagonist peptide has the potential to regulate the access of those peptides to their target sites in the body. It prompts the need to investigate the transport abilities of opioid peptides, especially in the cases of diseases such as allergy, mucosal immunity, celiac disease, autism and schizophrenia.

3. Materials and methods

The human colon adenocarcinoma cell line, Caco-2, was obtained from American Type Culture Collection.

Human β -casomorphin-5 (Tyr-Pro-Phe-Val-Glu), β -casomorphin-7 (Tyr-Pro-Phe-Val-Glu-Pro-Ile), and lactoferrin A (Tyr-Leu-Gly-Ser-Gly-Tyr) have been custom synthesized at Gdansk University (Poland).

3.1. Cell culture

Caco-2 cells were cultured in DMEM with 10% FBS, 1 % NEAA and 50 mg/dm³ of gentamicin. They were incubated at 37°C in an atmosphere of 5% CO₂ in air. For transport studies Caco-2 cells were seeded in cell culture inserts with polycarbonate membranes (pore sizes 0.4 μ m; area 4.2 cm²) at a cell density of 4.5 $\times 10^5$ /cm² and incubated in six-well culture plates with medium change every second day. The confluent monolayers were used between days 21 and 25 after seeding.

3.2. Transport studies

The peptide transport has been determined in both directions: apical to basolateral (A-B) and basolateral to apical (B-A).

After incubation for 30 min, the Hank's solution was replaced with peptide solutions: β -casomorphin-5, β -casomorphin-7 and lactoferrin A (300 μ l in apical side and 400 μ l in basolateral side) to final concentrations of 400 μ g/mL for each peptide. To determine the rate of A-B transport, the peptide solutions (respectively in HBSS with diprotin A) were added to the apical chamber and to determine the (B-A) flux the peptide solutions were added to basolateral chamber. The samples of apical or basolateral solutions were taken after 15, 30, 45 and 60 min of incubation at 37°C and replaced by a fresh buffer. Peptide concentration in apical and basolateral solutions was determined using high-performance liquid chromatography (HPLC).

4.2. Effect of diprotin A on β -casomorphins transport

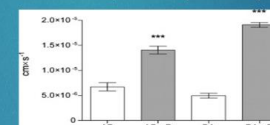


Fig. 1. Permeability coefficients for β -casomorphin-5 with the presence or absence of diprotin A. AB apical to basolateral transport of BCM5; AB + D apical to basolateral transport of BCM5 with diprotin A; BA basolateral to apical transport of BCM5; BA + D apical to basolateral transport of BCM5 with diprotin A.

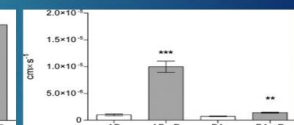


Fig. 2. Permeability coefficients for β -casomorphin-7 with the presence or absence of diprotin A. AB apical to basolateral transport of BCM7; AB + D apical to basolateral transport of BCM7 with diprotin A; BA basolateral to apical transport of BCM7; BA + D apical to basolateral transport of BCM7 with diprotin A.

4.3. Concentration of peptides in donor chambers after 60-min incubation

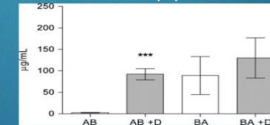


Fig. 3. Concentration of β -casomorphin-5 in initial chamber after 60 min of incubation with Caco-2 monolayer in the presence or absence of diprotin A. AB apical to basolateral transport of BCM5; AB + D apical to basolateral transport of BCM5 with diprotin A; BA basolateral to apical transport of BCM5; BA + D apical to basolateral transport of BCM5 with diprotin A.

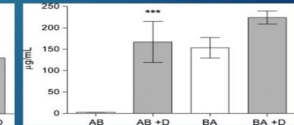


Fig. 4. Concentration of β -casomorphin-7 in initial chamber after 60 min of incubation with Caco-2 monolayer in the presence or absence of diprotin A. AB apical to basolateral transport of BCM7; AB + D apical to basolateral transport of BCM7 with diprotin A; BA basolateral to apical transport of BCM7; BA + D apical to basolateral transport of BCM7 with diprotin A.





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- Wpływ ekstraktów roślinnych na proliferację komórek nowotworowych linii komórkowych Caco-2
- Badań nad częstością występowania mutacji punktowych SNP w genie MOR w różnych stadiach chorobowych.

PREZENTACJE WYNIKÓW NA STUDENCKICH
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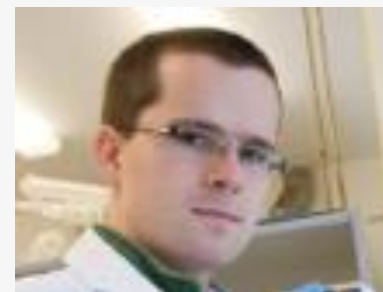
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Opiekun Koła Naukowego
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Przewodniczący Koła Naukowego
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