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It is our great pleasure to present this Supplement Issue on "*Macedonian Pharmaceutical Bulletin*" to the scientific and professional community. This supplement includes the short communications from the *Sixth Congress of Pharmacy in Macedonia with International participation*, as the largest gathering for the pharmacy profession held in the Republic of Macedonia. The main theme of the Congress was "Modern pharmacist - bridging science with practice".

A broad spectrum of topics within the pharmaceutical sciences and practice carefully selected for this special occasion in order to build up a highly interesting and comprehensive program were covered. The contributions submitted to the Congress included 6 plenary lectures, 84 section lectures, and more that 240 posters. This Congress, followed the excellent international tradition, was attended by close to 1000 domestic and foreign participants. We received 326 short paper submissions from more than 25 countries. These numbers show that our Congress is aiming for the highest scientific standards, and that it can be considered a well-established venue for researchers in the broad fields of Pharmaceutical sciences and practice.

We would like to thank all internationally prominent researchers for their contribution to reinforcing the overall quality of the Congress. They give the state of the art of the recent advances in the field of pharmacy research.

Sincere thanks to the hosts of the Sixth Congress of Pharmacy in Macedonia with International participation, Macedonian Pharmaceutical Association and Faculty of Pharmacy, Ss Cyril and Methodius University in Skopje for their vision and commitments.

We acknowledge the sponsoring companies: the platinium sponsor AD ALKALOID, Skopje, the golden sponsor PLIVA, the silver sponsor EUROFARM and the bronze sponsor SEPTIMA, for the permanent support to our efforts during the organization.

We would also like to thank our members of the Scientific Committee for their volunteer time and dedication to the critical peer review process and in the organization of the program. We also wish to thank all the members of the Organizing Committee, whose work and commitment was invaluable.

On behalf of the Advisory and Scientific Committees, we would like to especially thank the authors, whose work was the essential part of the congress and contributed to a very successful event. Besides the many academic staff and professionals who contributed to the success of the Congress, we are grateful to the students who participated with oral presentations and posters.

The pharmaceutical sciences continue to grow as dynamic scientific interdisciplinary fields. We believe that published short communications will be an excellent source of scientific material in the fast evolving fields in Pharmaceutical sciences and practice.

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The present issue *of Macedonian Pharmaceutical Bulletin* is a special issue of the 6th Congress of Pharmacy in Macedonia with international participation.

This issue of *Macedonian Pharmaceutical Bulletin* contains short papers accepted by the scientific committee for the presentation at the Congress.

The authors are fully responsible for the contents of their short papers.

All reviewers that were involved in the short papers revision process are sincerely acknowledged.

Opening lecture

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Short communication

Molecular mechanisms of capsaicin mediated cytotoxic activity

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Introduction

In the last few decades, capsaicin has exerted potent anti-cancer properties by enouncing anti-proliferative, apoptotic and anti-metastatic activity. To the best of our knowledge, the specific cell targets and successive mechanism of cytotoxic activity of capsaicin in different type of malignant cells, still remains unclear and contradictory. Therefore, this mini review, explains some of the most significant chemopreventive mechanisms of the action of capsaicin at a cellular level, reported in the recent literature.

The specific cytotoxic activity of capsaicin is usually a result of targeting of capsaicin toward two aims: TRPV1 (transient receptor potential cation channel subfamily V member 1) and tumor associated NADPH-oxidase. Some researchers reported that capsaicin provokes its anticancer activity through the interaction with TRPV1 receptor (Kim et al., 2006). Amantini et al., (2007) reported that capsaicin elicits apoptosis in U373 glioma cells, because the TRPV1 receptor was highly expressed and oppositely the cytotoxic effect in U87 cell line in which the TRPV1 receptor was very low expressed, capsaicin did not show cytotoxic effects (Amantini et al., 2007). Hu et al., (2008) have shown that TRPV1 receptor is included in capsaicin induced Ca^{2+} influx, generation of reactive oxygen species (ROS), depolarization of the mitochondrial membrane, and ultimately cell death on the synovial fibroblasts in rats. On the other side, capsaicin is one of the molecules which could inhibit the activity of tumor associated NADPH-oxidase, which is related to the inhibition of proliferation of cancer cells (Hedges et al., 2003).

The ability of capsaicin to inhibit the growth of different cancer cells is primary mediated by its ability to induce apoptosis. Apoptosis represents a type of programmed cell death, which is one of the physiological mechanisms for maintaining the homeostasis in the organism. It has been reported that two different pathways are mainly mediating the process of activation of apoptosis. First one is extrinsic pathway which is accomplished by activating of the "death receptor", and the other is intrinsic pathway which is followed by activation of cascade of caspase enzymes (Chou et al., 2007).

Extrinsic pathway

The extrinsic mechanism of apoptosis is characterized by activation of the external cell surface receptors, namely TRAIL (Tumor necrosis factor (TNF)-related apoptosis-inducing ligand) and DR (death receptor, Fas/CD95), leading to downstream caspase-mediated apoptosis (Codesido et al., 2014). These receptors can be activated by a signal that activates the enzymes procaspase 8 and 3, and therefore consequently triggers the apoptosis of cells. The number of studies, which included the extrinsic pathway into the mechanism of capsaicin mediated apoptosis, is much lower than numbers of reported studies which indicated the intrinsic pathway as the main mechanism of apoptosis.

Moon et al., 2012 notified that capsaicin induced the surface expression of TRAIL-receptor D5 through the activation of SP1 due to a calcium influx-dependent SP1 (specific protein 1) in kidney cancer cells. In multiple malignant glioma cells, subtoxic concentrations of capsaicin sensitized TRAIL-induced apoptosis mediated through ER Stress proteins CHOP/GADD153. DR5 and surviving contribute to amplification of the caspase cascade, thus restoring TRAIL sensitivity (Kim et al., 2010).

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Intrinsic pathway

The mechanism of intrinsic pathway of apoptosis is distinguished by intracellular activation of apoptosis through intracellular stressors, in which the most common are reactive oxygen species (ROS) and excess intracellular calcium. Disruption of the mitochondrial membrane and alterations in bcl-2, Bax, bcl-xL, and bad proteins, can cause a release of cytochrome C in cell cytosol and subsequent activation of the caspase cascade. These processes are the initial triggers that are eventually leading to apoptosis (Zhang et al., 2008). Cytochrom C together with some other factors can induce activation of caspase-9. Activated capsasa-9 can lead to activation of caspasa 3 and 7, which cleaves the inhibitor of caspasa activated DNAase and results in DNA fragmentation. Many studies examining the cytotoxic effects of capsaicin on prostate and other malignant cell lines, have reported that capsaicin induced apoptosis is linked to intrinsic mechanisms.

Pramanik et al., (2011) have evaluated the mechanism of capsaicin-mediated ROS generation in pancreatic cancer cells and they suggested that mitochondrial complex-I and III are involved in capsaicin mediated ROS generation. They demonstrated that capsaicin inhibited the enzymatic activity of antioxidant enzymes superoxide dismutase (SOD), catalase and glutathione reductase, which resulted in oxidative stress.

According to Kryston et al., (2011), the agonistic effect of capsaicin on TRPV1, can evoke intracellular influx of calcium, which leads to further intracellular stress, activating apoptosis in various cell lines, namely prostate cancer. They found that the generation of ROS induced by capsaicin correlated with the dissipation of the inner mitochondrial transmembrane potential and the release of cytochrome-c into the cytosol. Activation of the caspase-3 cascade resulted in the cleavage of poly(ADP-ribose)polymerase (PARP) and resultant apoptosis.

Conclusion

A large number of investigators clearly demonstrated that capsaicin inhibits the growth of cancer cells by inducing apoptosis and cell cycle arrest, but its molecular mechanisms in some types of cancers are not well understood. Therefore, additional studies are required to elucidate and to supplement the missing part of this data.

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