Faculty of pharmacy, Mansoura University, would like to appreciate a great thanks to

Prof. Dr. Ashraf Mohamed Abdel-Basset

For his great support to the faculty of conference

Faculty Administrative and Conference Organization Staff.

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Hagar Attia

Ibrahim Ashraf

I am pleased to extend a warm greeting to all presenters and participants in the 8thScientific

Conference of Pharmacy students. The annual Scientific Student Conference is one of the most important and successful activities of Faculty of Pharmacy- Mansoura University. The Conference aims to shed light on the new pharmaceutical sciences, and research exchanging among the students and the experts of this field. In fact, the Scientific Student Conference provides an opportunity to display results of research projects, gain new knowledge, and exchange ideas on the latest developments. The conference agenda of the conference combines discussion panels related to oral and poster presentations of 59 scientific research from 7 universities in addition to keynote speeches. This year, we will celebrate the 8thScientific Conference. I am very glad to thank all keynote speakers, moderators and students.

Thank you for joining us at8thScientific Conference (Pharmacy students).

Dean of the Faculty

Prof. Dr. Manal M. Eid

It gives me great pleasure to extenda warm welcome to all presenters and participants of the 8thScientific Conference (Pharmacy students). I consider the Scientific Student Conference one of the most important and successful activities of Faculty of Pharmacy- Mansoura University.Important, because it is up to the future generation of scientists to make a difference in solving the major challenges facing the global community today.The conference agenda conference combines discussion groups related to oral and poster presentations and keynote speeches in order to keep you engaged and give you an opportunity to express yourself on every topic that comes up.This year, we will celebrate the 8thScientific Conference. Students play a critical role in a development and successof the conference. The Scientific Student Conference provides a forum to share results of research projects, acquire new knowledge,and exchange ideas on the latest developments in addition to identifying new directions.I warmly thankall keynote speakers and students for their contributions and the session chairs for their dedication.

Thank you for joining us at8thScientific Conference (Pharmacy students).

Vice Dean of Education and Students Affairs

Prof. Dr. Rasha Barwa

On behalf of the Organizing Committee and the board members, we are honored and delighted to welcome you to the 8th Scientific Conference (Pharmacy students) which will be held in Faculty of Pharmacy, Mansoura University, Egypt. The Conference provides an opportunity for future scientists, and researchers to exchange experience, gain new knowledge and establish contact.

For the last seven years, the Conference attracted young researchers and undergraduate students from all the national universities (governmental and private). They were attracted by the high research level, competition rush and valuable knowledge in all pharmaceutical areas.

Year after year, number of contributed universities increased confirming the success of the conference. Increasing number of submitted posters, oral presentations confirmed the importance of this annual event.All the submitted projected will be examined and evaluated by a number of highly qualified scientists

We are looking forward to meet all of you in Mansoura University to make 8th Scientific Conference (Pharmacy students)a grand success.

Prof. Ghada Sameh Hafez Hassan

Rapporteur of the 8th Scientific Conference

It's a great pleasure for me to organize 8th version of scientific conference encouraging my student to play a role in scientific research which represents a major career path for majority of pharmacist all over the world . As a leading people, entrepreneurs.. You should start from now.. Read, think, create, then you can be a unique Always try to be the only as when you fails you will be the first

DR. Ahmed Ramadan

SECRETARY OF THE CONFERENCE

➢ Schedule of the day

Time	
09.00 : 10.00 AM	**Registration**
10.00 : 10.30 AM	**Opening Ceremony**
10.30 : 11.15 AM	**Lecture (Research Methodology in Medicine) Prof, DR/ Ahmed Shokeir**
11.15:12.00 PM	**Lecture (Scientific Writing) Prof , Dr / Khalid Beshir**
12.00 : 12.30 PM	**Break**
12.30 : 04.00 PM	**Posters and oral presentations judgment**
04.00 : 05.00 PM	**Closing Ceremony (Awards Announcement)**

Blue Hall

Biochemistry (B)

NO	Code	Time	Presentation
1	B1	12:30-12:50	Cancer vaccine.
2	B3	12:50-1:10	Neuropathy is the starting point of diabetes mellitus
3	B2	1:10-1:30	Micro RNA

Medicinal Chemistry (MC)

NO	Code	Time	Presentation
1	MC2	1:30-1:50	Chalcones derivatives as anticancer.
2	MC4	1:50-2:10	Fighting MRSA&VRSA: The war has just begun
3	MC1	2:10-2:30	Design, synthesis and molecular docking of novel benzoxazole derivatives as antiviral agent
4	MC3	2:30-2:50	FerriIridium: A Lysosome-Targeting Iron(III)-Activated Iridium(III) Prodrug for Chemotherapy in Gastric Cancer Cells

Pharco Hall

MICROBIOLOGY (M)

NO	Code	Time	Presentation
1	M3	12:30-12:50	Antibody-mediated delivery of viral epitopes to tumors harnesses CMV-specific T cells for cancer therapy.
2	M8	12:50-1:10	Scientists find ally in fight against brain tumors: Ebola
3	M6	1:10-1:30	Sequential LASER ART and CRISPR Treatments Eliminate HIV-1 in a Subset of Infected Humanized Mice
4	M7	1:30-1:50	Phage therapy as a novel therapeutic strategy for multi-drug resistant pathogens.

Pharmaceuticals and pharmacy practice (T)

NO	Code	Time	Presentation
7	T1	3:00-3:20	Pharmacogenetic and Clinical Predictors of Response to Clopidogrel Plus Aspirin After Acute Coronary Syndrome in Egyptians.

AL-Nahdi Hall

MICROBIOLOGY (M)

NO	Code	Time	Presentation
1	M1	12:30-12:50	New Strategies To Reverse MRSA
2	M2	12:50-1:10	Discovery of new T cell raises a prospect of universal cancer therapy.
3	M9	1:10-1:30	T cell receptor gene therapy targeting WT1 prevents acute myeloid leukemia relapse post-transplant
4	M4	1:30-1:50	Quorum sensing inhibition as a novel therapeutic strategy for multi-drug resistant pathogens.
5	M5	1:50-2:10	.A Requirement for Argonaute 4 in Mammalian Antiviral Defense.

LIGHT FLOOR HALL (Exams Hall)(6th floor)

Pharmacology and Toxicology (p)

NO	Code	Time	Presentation
1	P1	12:30-12:50	multiple sclerosis MS
2	P3	12:50-1:10	Hypoxia and the extracellular matrix:drivers of tumour metastasis
3	P4	1:10-1:30	Autophagy- dependent generation of Axin2+ cancer stem- like cells promotes hepatocarcinogenesis in liver cirrhosis.
4	P5	1:30-1:50	Scorpion venom component can reduce severity of rheumatoid arthritis
5	P6	1:50-2:10	Efficacy and safety of ipilimumab as sequential therapy to nivolumab for treatment advanced melanoma patients
6	P8	2:10-2:30	Hepcidin Deficiency Protects Against Atherosclerosis.
7	P2	2:30-2:50	Telmisartan modulates NFkB and ERK1/2 cross-talk in n-nitrosodiethyla mine-induced hepatocellular carcinoma in mice

LIGHT FLOOR HALL (conferences Hall)(6th floor)

Pharmacology and Toxicology (p)

NO	Code	Time	Presentation
1	P14	12:30-12:50	Molecular 'switch' reverses chronic inflammation and aging
2	P15	12:50-1:10	Targeted pharmacological therapy restores Î²-cell function for diabetes remission.
3	P16	1:10-1:30	Self-powered implant boosts weight loss Via vagus nerve stimulation.
4	P17	1:30-1:50	Hope for Diabetes: Boosting Human Beta Cells proliferation With Drug Combination.
5	P18	1:50-2:10	Stem cell therapies for liver fibrosis and regeneration.
6	P19	2:10-2:30	Clozapine reliably increases the motivation for food: parsing the roleof the 5-HT2c and H1 receptors.
7	P10	2:30-2:50	Diosmin enhances the anti-angiogenic activity of sildenafil and pentoxifylline against hepatopulmonary syndrome via regulation of TNF-Î±/ VEGF, IGF-1/PI3K/AKT, and FGF-1/ANG-2 signaling pathways.

Hall 4 (Clinical Building – D)

Pharmacology and Toxicology (p)

NO	Code	Time	Presentation
1	P9	1:00-1:20	Model-Based Nanoengineered Pharmacokinetics of Iron-Doped Copper Oxide for Nanomedical Applications.
2	P11	1:20-1:40	Cannabinoids in treatment of epilepsy.
3	P12	1:40-2:00	Human induced pluripotent stem cell-derived GABAergic interneuron transplants attenuate neuropathic pain.
4	P13	2:00-2:20	Orphan G-protein coupled receptor 183 (GPR183) potentiates insulin secretion and prevents glucotoxicity-induced Î²-cell dysfunction
5	P7	2:20-2:40	Generation of induced neural stem cells from peripheral mononuclear cells and differentiation toward dopaminergic neuron precursors for transplantation studies..

POSTER (LIGHT FLOOR , 6th floor)

NO	Department	Time	Code
1	Medicinal Chemistry	12:30	PMC1 PMC2*
2	Microbiology & immunology	12:30	PM1 PM2* PM3*
3	Biochemistry	1:30	PB1
4	Pharmacology & Toxicology	12:30	PP1* PP4* PP7 PP2* PP5 PP8 PP3 PP6
5	Pharmaceuticals and pharmacy practice	1:00	PT1

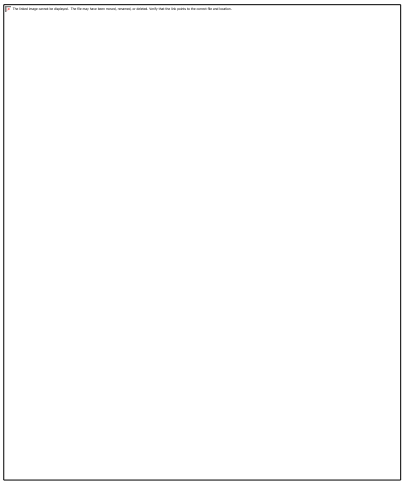
Scientific Sections

Oral presentations :

Opening Lecture

Prof. Dr: Ahmed A. Shokeir

- Professor of Urology, Urology and Nephrology center.
- X-director of Urology & Nephrology center.
- X-chairman of Urology department editor in chief, Arab Journal of Urology.
- Director of center of excellence for genome and cancer research.
- Head of institutional review board (IRB) of faculty of medicine
- Mansoura University, mansoura – Egypt.
- E-mail: ahmed.shokeir@hotmail.com



Prof.DR: Khalid B. salim

- Pharmaceutical organic chemistry.
- Director of pharm D, faculty of pharmacy, Mansoura University.
- PhD Kyoto University.
- Post. Doc, KISI.S.Korea.
- Post. Doc, le mane, France.
- Associate Professor, Faculty of pharmacy, Mansoura University.

Oral Presentations

Section MC

Medicinal Chemistry (MC1-MC4)

MC1	Medicinal Chemistry	Faculty of pharmacy Horus University

Amany Ahmed Sarhan.
Nada Aniss Ashour.
Israa Shawqii Ibrahim.

Design, synthesis and molecular docking of novel benzoxazole derivatives as antiviral agents

Abstract

A novel coronavirus (2019-nCoV) originating in Wuhan, China presents a potential respiratory viral pandemic to the world population. Current efforts are focused on containment and quarantine of infected individuals. Ultimately, the outbreak could be controlled with a protective vaccine to prevent 2019-nCoV infection. While vaccine research should be pursued intensely, there exists today no therapy to treat 2019-nCoV upon infection, despite an urgent need to find options to help these patients and preclude potential death.

In our research to develop a new drug for the treatment of coronavirus (2019-nCoV), we designed a new compound, explained its binding mode using MOE drug design program and started to synthesis it as a part of our graduation project.Hoping to aid in finding a new active drug against this deadly virus.

MC2	Medicinal Chemistry	Faculty of pharmacy Mansoura University

Ghada Samir Hashim.
Hager Attia Mohammed.

Chalcones derivatives as anticancer.

Abstract

Benzthiazole -as a sulfur-containing heterocycle- is widely represented in many biologically active materials, such as antibacterial, anticonvulsant, antiviral and anticancer active agents. The groove binding agents dactinomycin, netropsin and thia-netropsin also represent a group of compounds that contains thiazole moiety and were used as antitumor drugs. On the other hand, chalcones constitute an important class of natural products displayed interesting biological activities including anti-inflammatory, antioxidant, cytotoxic, antimicrobial, anti-leishmanial, and anti-tuberculosis. In addition, they have a recognized synthetic utility in the preparation of numerous pharmacologically-interesting heterocyclie systems such as pyrimidines that have drawn the attention of medicinal chemists as chemotherapeutic agents. Several members of this class have earned valued places in chemotherapy as effective agents. Various literature reports displayed numerous fused ring systems and their chemotherapeutic activities as anticancer, antibacterial, antifungal and antiviral agents. In the present work, new derivatives were synthesized through molecular hybridization between benzthiazole ring and chalcones that were expected to produce active agents as antitumor activity. The anticancer activity was performed against many selected cell lines and the percentage inhibition against these cell lines was determined.

MC3	Medicinal Chemistry	Faculty of pharmacy Mansoura University

Ali Mohsen Ali Steta

FerriIridium: A Lysosome-Targeting Iron(III)-Activated Iridium(III) Prodrug for Chemotherapy in Gastric Cancer Cells

Abstract

In recent years, the potential of organometallic complexes in medicinal chemistry has emerged and expanded across the periodic table. In particular, iridium-based complexes. Many studies show a positive correlation between iron storage and the risk of cancer including stomach cancers. Interestingly, some studies indicate that the labile iron pool (LIP) levels in lysosomes are increased in gastric cancers and highlights the LIP as a promising anticancer target. In this research they present a novel synthetic ferric-ion-activated, anticancer, iridium-based smart prodrug and theranostic agent (FerriIridium). It contains a meta-imino catechol group that can selectively bond to, and be oxidized by, free FeIII inside the cell. Subsequent rearrangement releases FeII and hydrolyses the amine bond under acidic conditions, forming an aminobipyridyl Ir complex and 2-hydroxybenzoquinone. Thus, FeII catalyzes the Fenton reaction, transforming hydrogen peroxide into hydroxyl radicals, the benzoquinone compounds interfere with the respiratory chain, and conversion of the prodrug into the Ir complex leads to an increase in phosphorescence and toxicity. These properties, combined with the high FeIII content and acidity of cancer cells, make FerriIridium a selective and efficient theranostic agent (IC50 = 9.22 mm for AGS cells vs. > 200 mm for LO2 cells). FerriIridium is the first metal-based compound that has been developed for chemotherapy using FeIII to enhance both selectivity and potency.

MC4	Medicinal Chemistry	Faculty of pharmacy HorusUniversity

Eman Nasser Akl.
Hamada S. Abulkhair.

Fighting MRSA&VRSA: The war has just begun.

Abstract

Methicillin-resistant Staphylococcus aureus (MRSA) and Vancomycin-resistant Staphylococcus aureus (VRSA) are a critical health problem and cause a high rate of morbidity and mortality worldwide. The management of infectious diseases caused by such microorganisms is extremely difficult due to the inherent resistance of MRSA & VRSA causative agents to presently used antibacterial agents. The development of novel potent antibacterial is essential to combat the emergence of antimicrobial resistance. In the last decade, many scaffolds were designed and constructed with the objective of fighting, overcoming the resistance such pathogens, as phenylthiazoles which were reported as a new scaffold with wide antimicrobial activity against multidrug-resistant Gram-positive strains, including MRSA, VRSA. In this review, we are shedding the light on the chemistry and the potency of these scaffolds.

Section M

Microbiology (M1-M9)

M1	Microbiology	Faculty of pharmacy Mansoura University

Lamees Abd El-Razik Goda.
Fatima Medhat Saad.

New Strategies To Reverse MRSA.

Abstract

Staphylococcus aureus is the leading cause of infections worldwide, and methicillin-resistant strains (MRSA) are emerging. New strategies are urgently needed to overcome this threat. Using a cell-based screen of ~45,000 diverse synthetic compounds, we discovered a potent bioactive, MAC-545496, that reverses Î²-lactam resistance in the community-acquired MRSA USA300 strain. MAC-545496 could also serve as an antivirulence agent alone; it attenuates MRSA virulence in Galleria mellonella larvae. MAC-545496 inhibits biofilm formation and abrogates intracellular survival in macrophages. Mechanistic characterization revealed MAC-545496 to be a nanomolar inhibitor of GraR, a regulator that responds to cell-envelope stress and is an important virulence factor and determinant of antibiotic resistance. The small molecule discovered herein is an inhibitor of GraR function. MAC-545496 has value as a research tool to probe the GraXRS regulatory system and as an antibacterial lead series of a mechanism to combat drug-resistant Staphylococcal infections.

M2	Microbiology	Faculty of pharmacy Mansoura University

Shaimaa Yahia Foaud Ghaly.
Ahmed Zaher Ebrahim.

Discovery of new T cell raises a prospect of universal cancer therapy.

Abstract

Human leukocyte antigen (HLA)-independent, T cell-mediated targeting of cancer cells would allow immune destruction of malignancies in all individuals. Here, we use genome-wide CRISPR-Cas9 screening to establish that a T cell receptor (TCR) recognized and killed most human cancer types via the monomorphic MHC class I-related protein, MR1, while remaining inert to noncancerous cells. Unlike mucosal-associated invariant T cells, recognition of target cells by the TCR was independent of bacterial loading. Furthermore, concentration-dependent addition of vitamin B-related metabolite ligands of MR1 reduced TCR recognition of cancer cells, suggesting that recognition occurred via sensing of the cancer metabolome. An MR1-restricted T cell clone mediated in vivo regression of leukemia and conferred enhanced survival of NSG mice. TCR transfer to T cells of patients enabled killing of autologous and non-autologous melanoma. These findings offer opportunities for HLA-independent, pan-cancer, pan-population immunotherapies.

M3	Microbiology	Faculty of pharmacy Mansoura University

Noha Ashraf Mohamed.

Antibody-mediated delivery of viral epitopes to tumors harnesses CMV-specific T cells for cancer therapy.

Abstract

Several cancer immunotherapy approaches, such as immune checkpoint blockade and adoptive T-cell therapy, boost T-cell activity against the tumor, but these strategies are not effective in the absence of T cells specific for displayed tumor antigens. Here we outline an immunotherapy in which endogenous T cells specific for a noncancer antigen are retargeted to attack tumors. The approach relies on the use of antibodyâ€“peptide epitope conjugates (APECs) to deliver suitable antigens to the tumor surface for presention by HLA-I. To retarget cytomegalovirus (CMV)-specific CD8+ T cells against tumors, we used APECs containing CMV-derived epitopes conjugated to tumor-targeting antibodies via metalloprotease-sensitive linkers. These APECs redirect pre-existing CMV immunity against tumor cells in vitro and in mouse cancer models. In vitro, APECs activated specifically CMV-reactive effector T cells whereas a bispecific T-cell engager activated both effector and regulatory T cells. Our approach may provide an effective alternative in cancers that are not amenable to checkpoint inhibitors or other immunotherapies.

M4	Microbiology	Faculty of pharmacy Horus University

Heba Sherif Radi Elmetwally.
Hend Mostafa Abdelwahab.
Hend Mohsen Mohamed.

Quorum sensing inhibition as a novel therapeutic strategy for multi-drug resistant pathogens.

Abstract

High incidence of antibiotic resistance among bacterial clinical isolates necessitates the discovery of new targets for inhibition of microbial pathogenicity, without stimulation of microbial resistance. This could be achieved by targeting virulence determinants, which cause host damage and disease. Many pathogenic bacteria elaborate signaling molecules for cellular communication. This signaling system is named quorum sensing system (QS), and it is contingent on the bacterial population density and mediated by signal molecules called pheromones or autoinducers (AIs). Bacteria utilize QS to regulate activities and behaviors including competence, conjugation, symbiosis, virulence, motility, sporulation, antibiotic production, and biofilm formation. Hence, targeting bacterial communicating signals and suppression of QS exhibit a fundamental approach for competing microbial communication. In this project, we illustrate the common up to date approaches to utilize QS circuits in pathogenic bacteria as novel therapeutic targets.

M5	Microbiology	Faculty of pharmacy Tanta University

Ghada Mohamed Barakat.

A Requirement for Argonaute 4 in Mammalian Antiviral Defense.

Abstract

While interferon (IFN) responses are critical formammalian antiviral defense, induction of antiviralRNA interference (RNAi) is evident. To date, individual functions of the mammalian RNAi and microRNA (miRNA) effector proteins Argonautes 1â€“4(AGO1â€“AGO4) during virus infection remain undetermined. AGO2 was recently implicated in mammalian
antiviral defense, so we examined antiviral activity ofAGO1, AGO3, or AGO4 in IFN-competent immunecells. Only AGO4-deficient cells are hyper-susceptible to virus infection. AGO4 antiviral function is
both IFN dependent and IFN independent, sinceAGO4 promotes IFN but also maintains antiviral capacity following prevention of IFN signaling or production. We identified AGO-loaded virus-derivedshort interfering RNAs (vsiRNAs), a molecular marker of antiviral RNAi, in macrophages infected with influenza or influenza lacking the IFN and RNAi suppressor NS1, which are uniquely diminished withoutAGO4. Importantly, AGO4-deficient influenza-infected mice have significantly higher burden and viraltiters in vivo. Together, our data assign an essentialrole for AGO4 in mammalian antiviral defense.

M6	Microbiology	Faculty of pharmacy Mansoura University

Mohamed Ramadan Awad.
Fatma El-zahraa Gamal Fathy Helmy Abd El-wahab.
Salma Hassan Mohamed Solaiman Dawood.

Sequential LASER ART and CRISPR Treatments Eliminate HIV-1 in a Subset of Infected Humanized Mice.

Abstract

Elimination of HIV-1 requires clearance and removal of integrated proviral DNA from infectedcells and tissues. Here, sequential long-acting slow-effective release antiviral therapy (LASERART) and CRISPR-Cas9 demonstrate viral clearance in latent infectious reservoirs in HIV-1
infected humanized mice. HIV-1 subgenomic DNA fragments, spanning the long terminalrepeats and the Gag gene, are excised in vivo, resulting in elimination of integrated proviralDNA; virus is not detected in blood, lymphoid tissue, bone marrow and brain by nested anddigital-droplet PCR as well as RNAscope tests. No CRISPR-Cas9 mediated off-target effects
are detected. Adoptive transfer of human immunocytes from dual treated, virus-free animalsto uninfected humanized mice fails to produce infectious progeny virus. In contrast, HIV-1 isreadily detected following sole LASER ART or CRISPR-Cas9 treatment. These data provideproof-of-concept that permanent viral elimination is possible.

M7	Microbiology	Faculty of pharmacy Horus University

Nabila Ahmed Ghazy.
Dina Abdelazim Galal.
Aya Safy Salem.

Phage therapy as a novel therapeutic strategy for multi-drug resistant pathogens.

Abstract

Nowadays the most important problem in the treatment of bacterial infections is the appearance of MDR (multidrug-resistant) bacteria and the scarce prospects of producing new antibiotics. There is renewed interest in revisiting the use of bacteriophage to treat bacterial infections. The practice of phage therapy, the application of phages to treat bacterial infections, has been around for approximately a century. Phage therapy relies on using lytic bacteriophages and purified phage lytic proteins for treatment and lysis of bacteria at the site of infection. Current research indicates that phage therapy has the potential to be used as an alternative to antibiotic treatments. It is noteworthy that, whether phages are used on their own or combined with antibiotics, phages are still a promising agent to replace antibiotics. So, this project focuses on an understanding of challenges of MDR bacteria and phages mechanism for treating bacterial infections and the most recent studies on potential phages, cocktails of phages, and enzymes of lytic phages in fighting these resistant bacteria.

M8	Microbiology	Faculty of pharmacy Tanta University

Fatma Alzahraa Mohamed Ibrahim

Scientists find ally in fight against brain tumors: Ebola

Abstract

Glioblastomas are relentless, hard to treat, and often lethal brain tumors. So, the scientists found that Ebola virus have MLD, protein, this protein can damage the tumor cell without any effect to normal cell. but Ebola virus is very killer, so cannot inject it in the cell. So, the scientists make virus has the same protein, which in Ebola virus, so MLD damage the target cell without effect to normal cell.

M9	Microbiology	Faculty of pharmacy Mansoura University

Eman Mahmoud Khalifa.
Basma Taher Shohip.

Tcell receptor gene therapy targeting WT1 prevents acute myeloid leukemia relapse post-transplant

Abstract

Relapse after allogeneic hematopoietic cell transplantation (HCT) is the leading cause of death in patients with acute myeloid leukemia (AML) entering HCT with poor-risk features1â€“3. When HCT does produce prolonged relapse-free survival, it commonly reflects graft-versus-leukemia effects mediated by donor TÂ cells reactive with antigens on leukemic cells4. As graft TÂ cells have not been selected for leukemia specificity and frequently recognize proteins expressed by many normal host tissues, graft-versus-leukemia effects are often accompanied by morbidity and mortality from graft-versushost disease5. Thus, AML relapse risk might be more effectively reduced with TÂ cells expressing receptors (TCRs) that target selected AML antigens6. We therefore isolated a high-affinity 1,2,3,6* CD34+ stem cells11. Based on similar over-expression in other malignancies, WT1 was identified as a high-priority antigen target by the US National Cancer Institute12. The binding affinity of a TCR for its target antigen largely determines the avidity of the TÂ cell carrying that TCR; that is, its ability to mediate anti-tumor effector functions13. In a previous clinical study, we demonstrated direct anti-leukemia activity of WT1-specific CD8+ TÂ cell clones transferred post-HCT14. Although the highest avidity TÂ cell clones were selected from each patientâ€™s HLA-matched donor, most endogenous WT1-specific TÂ cells were of low avidity. To increase potency in the anti-WT1 TCR study described here, we screened many donors to identify a high-affinity, HLA-A*0201+ Wilmsâ€™ Tumor Antigen 1-specific TCR (TCRC4) from HLA-A2+ normal donor repertoires, inserted TCRC4 into Epsteinâ€“Bar virus-specific donor CD8+ TÂ cells (TTCR-C4) to minimize graftversus-host disease risk and enhance transferred TÂ cell survival7,8, and infused these cells prophylactically post-HCT into 12 patients (NCT01640301). Relapse-free survival was 100% at a median of 44â€‰months following infusion, while a concurrent comparative group of 88 patients with similar risk AML had 54% relapse-free survival (Pâ€‰=â€‰0.002). TTCR-C4 maintained TCRC4 expression, persisted long-term and were polyfunctional. This strategy appears promising for preventing AML recurrence in individuals at increased risk of post-HCT relapse.

Section P

Pharmacology and Toxicology (P1-P20)

P1	pharmacology and Toxicology	Faculty of pharmacy Mansoura University

Aseel Mitwally Awad.

multiple sclerosis MS.

Abstract

Multiple sclerosis is an autoimmune disease including demylination and inflammation in CNS . it's triggered by environmental factors and genetic factors that form a susceptible host .Autoimmunity of the disease is associated with pathology of TH1 and TH17 response .one of the changes in the demyelinated axons in MS is mitochondrial changes required for the myelination specifically in progressive MS , but myelin is thought to regenerate and this required the study of dynamics of oligoendrocyte generation in MS .the treatment aims at restablishing the normal complex interaction in the immune system in relapsive multiple sclerosis while progressive multiple sclerosis treatment aims to minimize the symptoms .

P2	pharmacology and Toxicology	Faculty of pharmacy Delta University

Authors
Saber S1, Khodir AE2, Soliman WE3,4, Salama MM5, Abdo WS6, **Elsaeed** B5, Nader K5, Abdelnasser A5, Megahed N5, Basuony M5, Shawky A5, Mahmoud M5, Medhat R5, Eldin AS5.

Telmisartan modulates NFkB and ERK1/2 cross-talk in n-nitrosodiethyla mine-induced hepatocellular carcinoma in mice

Abstract

Hepatocellular carcinoma are major worldwide health problems and due to the complicated molecular pathogenesis. The currently available therapeutic interventions are yet limited. Therefore, the discovery of new therapeutic strategies is essentially required for improving the prognosis and survival of patients with HCC. Treatment with sorafenib (SRF) alone increases patient survival by only a few months. Angiotensin-converting enzyme inhibitors and angiotensin II receptor blockers, having well established safety profiles, may provide synergistic effects to existing chemotherapies as anti-tumor effects. However, the mechanisms of Ang-II remain to be elucidated. N-Nitrosodiethylamine was utilized to examine the effects of Telmisartan (TEL) (15 mg/kg), SRF(30 mg/kg), and a combination of these two agents on HCC mice. Downregulation of NF-ÐºBP65 mRNA expression and inhibition of the phosphorylation-induced activation of both ERK1/2 and NF-ÐºB P65 were implicated in the anti-tumor effects of TEL and SRF. Consequent improvement in liver function with reduced levels of AFP, TNF-Î±, and TGF-Î²1 were confirmed. Anti-proliferative, anti-metastatic, and anti-angiogenic effects of treatment were indicated by reduced hepatic cyclin D1 mRNA expression, reduced MMP-2 levels, and reduced VEGF levels, respectively.TEL, but not SRF, demonstrated agonistic activity for PPARÎ³ receptors, as evidenced by increased PPARÎ³ DNA binding activity, upregulating of CD36, and HO-1 mRNA expression followed by increased liver antioxidant capacity. Both TEL and SRF inhibited TAK1 phosphorylation-induced activation, indicating that TAK1 might act as a central mediator in the interaction between ERK1/2 and NF-ÐºB. TEL,. Therefore, TEL is an encouraging agent for further clinical trials regarding the management of HCC.

P3	pharmacology and Toxicology	Faculty of Pharmacy Mansoura University

Abdelrahman alaa labib

Hypoxia and the extracellular matrix:drivers of tumour metastasis

Abstract

Of the deaths attributed to cancer, 90% are due to metastasis, and treatments that prevent or cure metastasis remain elusive. Emerging data indicate that hypoxia and the extracellular matrix (ECM) might have crucial roles in metastasis. During tumour evolution, changes in the composition and the overall content of the ECM reflect both its biophysical and biological properties and these strongly influence tumour and stromal cell properties, such as proliferation and motility. Originally thought of as independent contributors to metastatic spread, recent studies have established a direct link between hypoxia and the composition and the organization of the ECM, which suggests a new model in which multiple microenvironmental signals might converge to synergistically influence metastatic outcome.

P4	pharmacology and Toxicology	Faculty of pharmacy Mansoura University

Shrouk Abdelhai Mosbah

Autophagy- dependent generation of Axin2+ cancer stem- like cells promotes hepatocarcinogenesis in liver cirrhosis.

Abstract

Autophagy is a pathophysiological phenomenon in liver cirrhosis that can further progress into hepatocarcinoma. Liver cancer stem cells (CSCs) are believed to initiate hepatocarcinogenesis. To investigate the precise mechanism related to the origin of CSCs in liver cirrhosis and hepatocarcinogenesis, we labeled Axin2+ hepatic cells with EGFP in Axin2Cre;Rosa26EGFP transegenic rats, and then stratified clinical and rat liver cirrhosis samples by autophagy flux. Clinical follow-Up and lineage tracing in transegenic rat liver cirrhosis revealed that while Axin2/EGFP+ hepatic cells were present in normal livers and cirrhotic livers without aberrant autophagy, hepatic Axin2/EGFP+CD90+cells were generated exclusively in cirrhotic livers with aberrant autophagy and promoted hepatocarcinogenesis. Aberrant autophagy in liver cirrhosis resulted in hepatocyte growth factor HGF expression leading to activation of Met/JNK and Met/STAT3 signaling in sorted hepatic Axin2/EGFP+ cells and their transition into Axin2/EGFP+CD90+ cells that posses CSC properties. In a transegenic rat liver cirrhosis model, induction or inhibition of autophagy in cirrhotic livers by systemic administration of rapamycin or chloroquine or transfection with Atg3-and Atg7-shRNAs significantly induced or suppressed HGF expression, which in turn increased or reduced generation of EGFP+CD90+ hepatic cells by activation inactivation of Met/JNK and Met/STAT3 signaling, therapy promoting or preventing hepatocarcinogenesis. Systemic treatment with HGF-shRNA,SP600125 or stattic also reduced generation of EGFP (Axin2)+hepatic cell-originated CD90+CSCs in aberrant autophagic cirrhotic livers by inactivation of HGF/Met/JNK or HGF/Met/STAT3 signaling, further preventing hepatocarcinogenesis. The data suggest that activation of Met/JNK and Met/STAT3 signaling in Axin2+ hepatic cells Via autophagy-dependent HGF expression and the resultant generation of Axin2+CD90+ CSCs is a major mechanism of hepatocarcinogenesis in cirrhotic livers.

P5	pharmacology and Toxicology	Faculty of pharmacy Mansoura University

Mohamed Abdulla Elshewy.

Scorpion venom component can reduce severity of rheumatoid arthritis

Abstract

A group of researchers has found that one of the hundreds of components in scorpion venom can reduce the severity of rheumatoid arthritis in animal models, without inducing side effects associated with similar treatments.

A treatment that improves the lives of nearly 1.3 million people with rheumatoid arthritis might one day originate from scorpion venom. A group of researchers led by Dr. Christine Beeton at Baylor College of Medicine has found that one of the hundreds of components in scorpion venom can reduce the severity of the disease in animal models, without inducing side effects associated with similar treatments. The study appears in the Journal of Pharmacology and Experimental Therapeutics.

"Rheumatoid arthritis is an autoimmune disease -- one in which the immune system attacks its own body. In this case, it affects the joints," said Beeton, associate professor of molecular physiology and biophysics and member Dan L Duncan Comprehensive Cancer Center at Baylor College of Medicine. "Cells called fibroblast-like synoviocytes (FLS) play a major role in the disease. As they grow and move from joint to joint, they secrete products that damage the joints and attract immune cells that cause inflammation and pain. As damage progresses, the joints become enlarged and are unable to move."

Current treatments target the immune cells involved in the disease and none are specific for FLS. Beeton and her colleagues studied FLS looking for an 'Achilles' heel' that would allow them to prevent or stop them from damaging the joints.

P6	pharmacology and Toxicology	Faculty of pharmacy Mansoura University

Amira Ragab Matareek.
Islam Khaled Moukhtar.

Efficacy and safety of ipilimumab as sequential therapy to nivolumab for treatment advanced melanoma patients

Abstract

Introduction: Immunotherapy has made a revolution in the world of medicine as it treats several cancers and targets the immune system rather than the tumor itself, thereby immunotherapy has proven its effectiveness in treating advanced melanoma. There are various immunotherapies. But, the most common immunotherapy used to treat melanoma is immune checkpoint inhibitors. Immune Checkpoint inhibitors work by regulating T-cell activity and giving an effective antitumor response rate. Three checkpoint inhibitors are used currently to treat advanced melanoma. Those are ipilimumab (anti-CTLA-4), nivolumab(anti-PD-1) and pembrolizumab(anti-PD-1).

Areas covered: Anti-CTLA-4 and anti-PD-1 treatments promote antitumor immunity through blocking the binding of the ligands(PDL-1, PDL-2, CD80, CD86) and inhibitory receptors(CTLA-4, PD-1), so boosting T-cell activation and proliferation with consequent up-regulation of antitumor immunity(Lipson & Drake, 2011).

The combination of the anti-CTLA-4 agent ipilimumab and the anti-PD-1 agent nivolumab has been shown to enhance objective response rate and progression-free survival compared with either agent alone as monotherapy in patients with advanced melanoma. However, the combination was associated with significant toxicity (Grimaldi et al., 2016).

In this literature review, we investigated ipilimumab efficacy and safety as sequential therapy to nivolumab in advanced melanoma patients that associated with reasonable overall survival and objective response rate with a ratio[3,6 : 16%] and similar toxicity with concurrent therapy.

P7	pharmacology and Toxicology	Faculty of pharmacy Zagazig University

Mohab Ali Abdelqawy Ali

Generation of induced neural stem cells from peripheral mononuclear cells and differentiation toward dopaminergic neuron precursors for transplantation studies.

Abstract

Parkinson's disease is a common neurodegenerative disorder, caused by degeneration of dopaminergic neurons at the substantia nigra pars compacta in the ventral mesencephalon.
Over the past decade, cell therapy has shown potential in treatment of Parkinson's disease.
Reprogramming technology has made significant progress, which provides a promising cellular source for replacement therapy. Human induced pluripotent stem cells and embryonic stem cells have been proven to be to differentiate into dopaminergic neural cells, which could survive, maturate and improve the motor functions.
There's a cellular source for cell transplantation which is "lineage-committed adult stem cells" obtained through direct reprogramming, such as induced neural stem cells, which can be derived from the unstable intermediates, bypassing by pluripotent stage.
Both types of stem cells can be reprogrammed from autologous cellular sources, such as fibroblasts, peripheral blood mononuclear cells and various other types of cells, these cells are inherent with reduced risk of tumor formation and lineage-committed plasticity, only able to differentiate into neurons and glia.
Initial reprogramming studies employed integrative delivery systems such as lentiviral or retroviral vectors, which are efficient and easy to implement in many types of cells, however these delivery systems may cause mutations and reactivation of residual transgenes, which present safety issues for clinical therapeutic purposes. Sendai virus is a non integrative RNA virus with a negative sense, single stranded genome that doesn't integrate into the host genome but only replicates in the cytoplasm of infected cells, offering an efficient and safe vehicle for reprogramming.

P8	pharmacology and	Faculty of pharmacy

	Toxicology	Mansoura University

Nada mohamed albaioumy.
Ghada mohamed abd-elsatar.
fatima mohamed mostafa.

Hepcidin Deficiency Protects Against Atherosclerosis.

Abstract

Objective: Inflammatory stimuli enhance the progression of atherosclerotic disease. Inflammation also increases the expression of hepcidin, a hormonal regulator of iron homeostasis, which decreases intestinal iron absorption, reduces serum iron levels and traps iron within macrophages. The role of macrophage iron in the development of atherosclerosis remains incompletely understood. The objective of this study was to investigate the effects of hepcidin deficiency and decreased macrophage iron on the development of atherosclerosis.

Approach and Resultsâ€"Hepcidin- and LDL (low-density lipoprotein) receptor-deficient (Hampâˆ’/âˆ’/Ldlrâˆ’/âˆ’) mice and Hamp+/+/Ldlrâˆ’/âˆ’ control mice were fed a high-fat diet for 21 weeks. Compared with control mice, Hampâˆ’/âˆ’/Ldlrâˆ’/âˆ’ mice had decreased aortic macrophage activity and atherosclerosis. Because hepcidin deficiency is associated with both increased serum iron and decreased macrophage iron, the possibility that increased serum iron was responsible for decreased atherosclerosis in Hampâˆ’/âˆ’/Ldlrâˆ’/âˆ’ mice was considered. Hamp+/+/Ldlrâˆ’/âˆ’ mice were treated with iron dextran so as to produce a 2-fold increase in serum iron. Increased serum iron did not decrease atherosclerosis in Hamp+/+/Ldlrâˆ’/âˆ’ mice. Aortic macrophages from Hampâˆ’/âˆ’/Ldlrâˆ’/âˆ’ mice had less labile free iron and exhibited a reduced pro-inflammatory (M1) phenotype compared with macrophages from Hamp+/+/Ldlrâˆ’/âˆ’ mice. THP1 human macrophages treated with an iron chelator were used to model hepcidin deficiency in vitro. Treatment with an iron chelator reduced LPS (lipopolysaccharide)-induced M1 phenotypic expression and decreased uptake of oxidized LDL.

In summary, in a hyperlipidemic mouse model, hepcidin deficiency was associated with decreased macrophage iron, a reduced aortic macrophage inflammatory phenotype, and protection from atherosclerosis. The results indicate that decreasing hepcidin activity, with the resulting decrease in macrophage iron, may prove to be a novel strategy for the treatment of atherosclerosis.

P9	pharmacology and Toxicology	Faculty of pharmacy Mansoura University

Nermeen Ahmed Lotfy Mo'men

Model-Based Nanoengineered Pharmacokinetics of Iron-Doped Copper Oxide for Nanomedical Applications.

Abstract

The progress in nanomedicine (NM) using nanoparticles (NPs) is mainly based on drug carriers for the delivery of classical chemotherapeutics. As low NM delivery rates limit therapeutic efficacy, an entirely different approach was investigated. CuO NPs are known for their cell toxic potential so a homologous series of engineered CuO NPs was designed for dual purposes (carrier and drug) with a direct chemical compositionâ€“biological functionality relationship. Model-based dissolution kinetics of CuO NPs in the cellular interior at post-exposure conditions were controlled through Fe-doping for intra/extra cellular Cu2+ and biological outcome. Through controlled ion release, as cytotoxicity of an exposure of cultured cells to dispersions of CuO NPs are mediated by the release of intracellular copper ions from CuO-NPs. Iron-doping has been reported to reduce the susceptibility of CuO-NPs to dissolution,which in turn lowers the cell toxic potential of iron-doped CuO-NPs. And the reactions taking place in the cellular interior, tumors could be treated selectively, in vitro and in vivo. Locally administered NPs enabled tumor cells apoptosis and stimulated systemic anti-cancer immune responses. We clearly show therapeutic effects without tumor cells relapse post-treatment with 6% Fe-doped CuO NPs combined with myeloid-derived suppressor cell silencing.

P10	pharmacology and Toxicology	Al-Azher University

Osama Magdy El-Sayed Farahat

Diosmin enhances the anti-angiogenic activity of sildenafil and pentoxifylline against hepatopulmonary syndrome via regulation of TNF-Î±/ VEGF, IGF-1/PI3K/AKT, and FGF-1/ANG-2 signaling pathways.

Abstract

Hepatopulmonary syndrome (HPS) is a severe complication of hepatic cirrhosis, which is characterized by hypoxia, intrapulmonary vasodilation, inflammation, and angiogenesis. In this study, we aimed to investigate the regulatory effects of diosmin (DS) on selected phosphodiesterase inhibitors against chronic bile duct ligation (CBDL)-induced HPS. Experimentally, Wistar Albino rats were used and HPS was induced by CBDL for 28 days.

DS (100 mg/kg, daily, P.O.), sildenafil (Sild; 10 mg/kg, twice daily, P.O.), and pentoxifylline (PTX; 50 mg/kg, daily, P.O.) were evaluated either alone or in combinations for their anti-angiogenic activity. CBDL significantly altered oxidative stress biomarkers and up-regulated pulmonary mRNA expressions of VEGF, IGF-1, ET-1, iNOS, eNOS, and ANG-2 as well as the protein expressions of vWF, FGF-1, PI3K, AKT, p-AKT, TGF-Î², HYP, MPO activityand circulating TNF-Î±. Treatment with DS, Sild, PTX, and their combinations significantly attenuated molecular and cellular changes due to CBDL. Improvement of histopathological changes was also observed after drug treatment which further supported our results. Furthermore, DS combination with Sild or PTX exhibited an improvement in HPS in comparison to each drug alone. Collectively, DS can augment the anti-angiogenic activity of Sild and PTX during HPS through regulation of TNF-Î±/VEGF, IGF-1/PI3K/AKT, and FGF-1/ANG-2signaling pathways.

P11	pharmacology and Toxicology	Faculty of pharmacy Mansoura University

Ghada Fathy Mohamed Alabd.
Merna Magdy Basala.

Cannabinoids in treatment of epilepsy.

Abstract

It is estimated that 30% of people with epilepsy continue to have seizures despite treatment. The approval of many new antiseizure drugs during the past two decades has not substantially reduced the proportion of patients with medically refractory disease. Patients need new treatments. Many families choose to try alternative therapy options. An abundance of preclinical evidence and anecdotal human data support the use of cannabinoids in the treatment of epilepsy. The present review paper aims to present the current state of knowledge regarding the effectiveness and safety of cannabinoids in the treatment of epilepsy. And it was found that Cannabidiol has shown anticonvulsant activity in many acute animal models of seizures. Recently three well controlled randomized trails focused on the potential usefulness of cannabinoids in the treatment of epilepsy have been published. Based on these publications, the US Food and Drug Administration approved in 2018 a purified, plant-derived cannabinoid for the treatment of seizures in patients with Dravet syndrome and Lennox-Gastaut syndrome.

P12	pharmacology and Toxicology	Faculty of pharmacy Mansoura University

Samar Abd Al-Rahman El-Gendy.
Alaa Ibrahim Gaafar.
Sara Moustafa Labib.

Human induced pluripotent stem cell-derived GABAergic interneuron transplants attenuate neuropathic pain.

Abstract

Neuropathic pain causes severe suffering, and most patients are resistant to current therapies. A core element of neuropathic pain is the loss of inhibitory tone in the spinal cord. Previous studies have shown that foetal GABAergic neuron precursors can provide relief from pain. However, the source of these precursor cells and their multipotent status make them unsuitable for therapeutic use. Here, we extend these findings by showing, for the first time, that spinally transplanted, terminally differentiated human

P13	pharmacology and Toxicology	Faculty of pharmacy Mansoura University

Amira Mohamed Abd ElMaboud.

Orphan G-protein coupled receptor 183 (GPR183) potentiates insulin secretion and prevents glucotoxicity-induced Î²-cell dysfunction

Abstract

The expression and functional impact of most orphan G-protein coupled receptors (GPCRs) in Î²-cell is not fully understood. Microarray expression indicated that 36 orphan GPCRs are restricted in human islets, while 55 receptors overlapped between human islets and INS-1â€¯cells. GPR183 showed higher expression in diabetic compared to non-diabetic human islets. GPR183 expression co-localized with Î²-cells while it was lacking in Î±-cells in human islets. The GPR183 agonist (7Î±-25-DHC) potentiated insulin secretion and protected against glucotoxicity-induced Î²-cell damage in human islets. Silencing of GPR183 in INS-1cells decreased the expression of proinsulin genes, Pdx1, Mafa and impaired insulin secretion with a concomitant decrease in cAMP generation. Cultured INS-1â€¯cells with 7Î±-25-DHC were associated with increased proliferation and expression of GPR183, INS2, PDX1, NeuroD, and INSR. In conclusion, the beneficial impact of GPR183 activation on Î²-cell function makes it a potential therapeutic target to prevent or reverse Î²-cell dysfunction.

P14	pharmacology and Toxicology	Faculty of pharmacy Mansoura University

Aly Hussein El Gabria.
Manal Mohamed Ali.

Molecular 'switch' reverses chronic inflammation and aging.

Abstract

Scientists have identified a molecular 'switch' that controls the immune machinery responsible for chronic inflammation in the body. The finding could lead to new ways to halt or even reverse many age-related conditions, from from Alzheimer's and Parkinson's to diabetes and cancer.

P15	pharmacology and Toxicology	Faculty of pharmacy Mansoura University

Aya El-Sayed Hassan Ragab.
Aya Ayman El-Sayed Saber.
Alaa El-Sayed Mohammed El-Kelany

Non alcoholic steatohepatitis (NASH): epidemiology and therapies.

Abstract

Non-alcoholic steatohepatitis (NASH) is associated with liver fibrosis and cirrhosis, which eventually leads tohepatocellular carcinoma. Although several animal models were developed to understand the mechanisms of NASH pathogenesis and progression, it remains obscure.Nonalcoholic steatohepatitis has become one of the most common liver-related health problems. This condition has been linked to an unhealthy diet and weight gain, but it can also be observed in nonobese people. The standard of care is represented by the lifestyle intervention.Global urbanisation and modernisation in the 20th and 21st centuries have been linked to unhealthy lifestyle changes. Consequently, the last 3 decades have seen significant increases inthe mean global body mass index (BMI) and the prevalence of obesity, which are the pathophysiological drivers of NAFLD.1 This is exemplified by the rapid increase in the prevalence of NAFLD in Asia over the past 15 years â€“ related to urbanisation and the adoption of Westerntype foods.2 The driving forces behind unhealthy lifestyle habits and choices are complex and multi-faceted, how-ever they can be successfully combated to induce significant health benefits. When successful, lifestyle changes that lead to weight loss are highly effective at reducing fibrosis and the necroinflammatory changes of non-alcoholic steatohepatitis (NASH), surpassing the efficacy of drugs currently being evaluated in phase III trials. However, sustained lifestyle changes and weight loss are difficult to achieve and, unfortunately, lifestyle changes alone are not successful in every individual.

P16	pharmacology and Toxicology	Faculty of pharmacy Mansoura University

Khaled Muhammad Zakaria Al-Badrawy.

Targeted pharmacological therapy restores Î²-cell function for diabetes remission.

Abstract

Dedifferentiation of insulin-secreting Î² cells in the islets of Langerhans has been proposed to be a major mechanism of Î²-cell dysfunction. Whether dedifferentiated Î² cells can be targeted by pharmacological intervention for diabetes remission, and ways in which this could be accomplished, are unknown as yet. Here we report the use of streptozotocin-induced diabetes to study Î²-cell dedifferentiation in mice. Single-cell RNA sequencing (scRNA-seq) of islets identified markers and pathways associated with Î²-cell dedifferentiation and dysfunction. Single and combinatorial pharmacology further show that insulin treatment triggers insulin receptor pathway activation in Î² cells and restores maturation and function for diabetes remission. Additional Î²-cell selective delivery of oestrogen by Glucagon-like peptide-1 (GLP-1â€“oestrogen conjugate) decreases daily insulin requirements by 60%, triggers oestrogen-specific activation of the endoplasmic-reticulum-associated protein degradation system, and further increases Î²-cell survival and regeneration. GLP-1â€“oestrogen also protects human Î² cells against cytokine-induced dysfunction. This study not only describes mechanisms of Î²-cell dedifferentiation and regeneration, but also reveals pharmacological entry points to target dedifferentiated Î² cells for diabetes remission.

P17	pharmacology and Toxicology	Faculty of pharmacy Zagazig University

Asmaa Adel Ebrahim Mohamed Saeed.

Self-powered implant boosts weight loss Via vagus nerve stimulation.

Abstract

Self-powered implant boosts weight loss Via vagus nerve stimulationObesity is an epidemic in the United States. This condition puts people at a higher risk for serious diseases, such as type 2 diabetes, heart disease, and cancer.

Globally, an estimated 4 million people died of conditions related to a high body mass index (BMI) in 2015 alone.These worrying trends mean scientists are focused on understanding the causes, risk factors, and implications of obesity.

Recently, researchers from the University of Wisconsin-Madison tested a groundbreaking, high-tech solution.The scientists designed a small, implantable device that they hope will reduce hunger pangs and help people lose weight.

The device, which is less than 1 centimeter across, can be implanted using a minimally invasive technique.Consisting of a flexible nanogenerator, it sends small pulses of electricity through the vagus nerve, which passes messages between the stomach and the brain.

This mild stimulation convinces the brain that the stomach is full and reduces feelings of hungerIn vivo vagus nerve stimulation holds great promise in regulating food intake for obesity treatment. Here we present an implanted vagus nerve stimulation system that is battery-free and spontaneously responsive to stomach movement.

The vagus nerve stimulation system comprises a flexible and biocompatible nanogenerator that is attached on the surface of stomach. It generates biphasic electric pulses in responsive to the peristalsis of stomach.

The electric signals generated by this device can stimulate the vagal afferent fibers to reduce food intake and achieve weight control.

Key words ;
Obesity , (BMI) , vagus nerve , weight control

P18	pharmacology and Toxicology	Faculty of pharmacy Mansoura University

Raghda Lotfy.

Hope for Diabetes: Boosting Human Beta Cells proliferation With Drug Combination.

Abstract

Scientists have gone from saying "it's impossible to make beta cells proliferate," to, in 2015, "it's possible, but not at a fast enough rate," to now, "it's possible at rates that are fast enough," senior author of the new study, Andrew F. Stewart, MD, Icahn School of Medicine at Mount Sinai, New York City.

Mount Sinai research team published preclinical study about drug cocktail enhance beta-cell proliferation.

The GLP-1 agonistâ€“DYRK1A inhibitor combination enhanced human beta cell proliferation, human insulin secretion, and blood glucose control not only in the cell cultures, but also in further studies of human islets transplanted into mice that were and were not diabetic.

No adverse events were observed in the mouse studies during a 1-week period.

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P19	pharmacology and Toxicology	Faculty of pharmacy Mansoura University

Hagar Attia Mohamed.
Amr Mahmoud Gepril.

Stem cell therapies for liver fibrosis and regeneration.

Abstract

Progression of fibrosis and the development of cirrhosis are responsible for the liver related morbidity and mortality associated with chronic liver diseases, and lead to limiting liver regenerative capacity. The clinical standard of care is transplantation, although stem cell therapy is an alternative option.

Either by inducing endogenous hematopoietic stem cell (HSC) with granulocyte colony stimulating factor (G-CSF) or induced pluripotent stem cells (iPSC) or mesenchymal stem cells (MSC) that can be cultured relatively easily and can be obtained not only from bone marrow, but also from medical wastes such as adipose tissue and umbilical cord tissue and because of its antigenicity allogenic MSC is safe.

Recently, the function of exosome (extracellular nanovesicles) have gained attention, and cell free therapy may become possible as alternative therapy, ENV isolated from iPSC or MSC.

P20	pharmacology and Toxicology	Faculty of pharmacy Mansoura University

Eiad Hatem Ali Elkaluoby.
Ahmed Ashraf Elsaid Ghazy.

Clozapine reliably increases the motivation for food: parsing the roleof the 5-HT2c and H1 receptors.

Abstract

Rationale and objectives: Although clozapine is effective in treating schizophrenia, it is associated with adverse side effects including weight gain and metabolic syndrome. Despite this, the role of clozapine on feeding behaviour and food intake has not been thoroughly characterized. Clozapine has a broad pharmacological profile, with affinities for several neurotransmitter receptors, including serotonin (5-hydroxytriptamine, 5-HT) and histamine. Given that the serotonin 5-HT2C receptor and histaminergic H1 receptor are involved in aspects of feeding behaviour, the effect of clozapine on feeding may be linked to its action at these receptors.

Methods: We assessed, in rats, the effect of acute and sub-chronic administration of clozapine on responding for food under a progressive ratio (PR) schedule under conditions of food restriction and satiety. We also examined the effect of antagonists of the serotonin 5-HT2C and histaminergic H1 receptors on the same schedule. Clozapine reliably increased responding for food, even when rats had ad libitum access to food. The effect of clozapine on responding for food was reproduced by combined (but not individual) antagonism of the serotonin 5-HT2C and histaminergic H1 receptors.

Conclusion: These findings show that clozapine enhances the motivation to work for food, that this effect is stable over repeated testing, and is independent of hunger state of the animal. This effect may relate to a combined action of clozapine at the serotonin 5-HT2C and histaminergic H1 receptors.

Section B

Biochemistry
(B1-B3)

B1	Biochemistry	Faculty of pharmacy Mansoura University

Sara Adel Ahmed Mohamed.

Cancer vaccine.

Abstract

Most of us know about vaccines given to healthy people to help prevent infections, such as measles and chicken pox. These vaccines use weakened or killed germs like viruses or bacteria to start an immune response in the body. Getting the immune system ready to defend against these germs helps keep people from getting infections.

Most vaccines used to treat cancer work the same way, but they make the personimmune system attack cancer cells. The goal is to help treat cancer or to help keep it from coming back after other treatments. But there are also some vaccines that may actually help prevent certain cancers.

Vaccines to help prevent cancerSome cancers are caused by viruses. Vaccines that help protect against infections with these viruses might also help prevent some of these cancers.

Some strains of the human papillomavirus (HPV) have been linked to cervical, anal, throat, vaginal, vulvar, and penile cancers. In fact, most cervical cancers are caused by infection with HPV. Vaccinating certain people against HPV helps protect against cervical cancer and the other 5 cancers. Read more in Protect Against HPV.

People who have chronic (long-term) infections with the hepatitis B virus (HBV) are at higher risk for liver cancer. Getting the vaccine to help prevent HBV infection may lower some peopleâ€™s risk of getting liver cancer.

These are traditional preventive vaccines that target the viruses that can cause certain cancers. They may help protect against some cancers, but they donâ€™t target cancer cells directly because cancer cells have not yet been formed or found.

These types of vaccines are only useful for cancers known to be caused by infections. But most cancers, including colorectal, lung, prostate, and breast cancers, are not thought to be caused by infections.

Vaccines to treat cancerCancer treatment vaccines are different from the vaccines that work against viruses. These vaccines try to get the immune system to mount an attack against cancer cells in the body. Instead of preventing disease, they are meant to get the immune system to attack a disease that already exists.

B2	Biochemistry	Faculty of pharmacy Delta University

Hazem Mohamed Salah.
Mahmood Rashad.
Nada Ali Mansour.

Neuropathy is the starting point of diabetes mellitus.

Abstract

Brain is important organ that control all body functions. Stress and depression induce neuronal death and directlyaffect hippocampus which induces neurogenesis and affect hypothalamo-pituitary-adrenal axis (HPA) which induces hyperglycemia, triglycerides and decrease in HDL that it is most characteristics of Type 2Diabetes Mellitus. Moreover, the insulin resistance is most cause of Type 2 Diabetes Mellitus.The brain is directly affected bythe resistance of liver, muscles and adipose tissues. Moreover, the brain is directly connected to beta cells in pancreas. When neurons degenerate and neurogenesis is decreased, centrally and peripherally neuropathy occur. In this article, we show imagine about neuropathy of Type 2 Diabetes Mellitus and insulin resistance. when neurons continue to degenerate, beta cells of pancreas are affected leading toType 1 Diabetes Mellitus.Inducing neurogenesis,assists the patient getting better.

B3	Biochemistry	Faculty of pharmacy Kafrelsheikh University

Huda Anas.

Micro RNA.

Abstract

MicroRNAs (miRNAs) are small, non-coding RNAs found throughout the eukaryotes that control the expression of a number of genes involved in commitment and differentiation of hematopoietic stem cells and tumorigenesis. Widespread dysregulation of miRNAs have been found in hematological malignancies, including human acute myeloid leukemia (AML). A comprehensive understanding of the role of miRNAs within the complex regulatory networks that are disrupted in malignant AML cells is a prerequisite for the development of therapeutic strategies employing miRNA modulators. Herein, we review the roles of emerging miRNAs and the miRNAs regulatory networks in AML pathogenesis, prognosis, and miRNA-directed therapies.

Section T

Pharmacy Practice (T1)

T1	Pharmacy Practice	Faculty of pharmacy Mansoura University

Alaa Ayman Mohammed.
Shorouq Ehab Ahmed Samra.

Pharmacogenetic and Clinical Predictors of Response to Clopidogrel Plus Aspirin After Acute Coronary Syndrome in Egyptians.

Abstract

Dual antiplatelet therapy (DAPT) with aspirin and clopidogrel reduces the risk for recurrent cardiovascular events after acute coronary syndrome (ACS). However, there is significant variation in response to DAPT that may be influenced by both genetic and nongenetic factors. This study aimed to assess the effect of genetic polymorphisms in PON-1, PEAR-1, P2Y12, CES1, and CYP2C19, along with clinical, demographic, and social factors, on variation in response to DAPT in Egyptians.

This study included 230 Egyptians treated with clopidogrel 75 mg/day and aspirin 81 mg/day for at least 12 months following their first ACS.Using multivariable logistic regression analysis, the CYP2C19*2 polymorphism was the only genetic predictor of MACE [odds ratio (OR): 2.23, 95% confidence interval (CI): 1.15-4.33, P=0.01]. In addition, proton pump inhibitor use (OR: 4.77, 95% CI: 1.47-15.54, P=0.009) and diabetes (OR: 1.83, 95% CI: 1.03-3.26, P=0.03) were associated with higher cardiovascular risk, whereas statin use was associated with lower risk (OR: 0.43, 95% CI: 0.25-0.76, P=0.003). The contribution of these four genetic and nongenetic factors explained 19% of the variability in risk for MACE in Egyptians treated with DAPT.

These results highlight that CYP2C19*2, along with diabetes, and use of proton pump inhibitor and statin are important factors jointly associated with variability in clinical response to DAPT following ACS in Egyptians.

Another study of genetic and nongenetic factors associated with clopidogrel response in Egyptians. Inall, 190 Egyptians with acute coronary syndrome (ACS) and/or percutaneous coronary intervention (PCI), treated with clopidogrel (75 mg/day) for at least a month.

CYP2C19 loss-of-function (LOF) alleles carriers had increased risk of MACE vs. noncarriers (odds ratio 2.52; 95% confidence interval 1.23â€“5.15, P = 0.011). In a logistic regression, CYP2C19 LOF variants (P = 0.011), age (P = 0.032), and bodymass index (BMI, P = 0.039) were significantly associated with the incidence of MACE in patients taking clopidogrel. CYP2C19genetic variants, age, and BMI are potential predictors associated with variability to clopidogrel response in Egyptians.

Poster

Section PP

Pharmacology and Toxicology

PP1	pharmacology and Toxicology	Faculty of pharmacy Delta University

Shereen Alaa.
Rania Khalil.

Serum MIF level as a clinically biomarker at the early pre-symptomatic stages of Alzheimer's disease in diabetic patients.

Abstract

Neurodegenerative processes begin several years before clinical symptoms. Pre-symptomatic stages of dementia could be more frequent at early onset of other diseases as diabetes mellitus (DM). DM represents a risk factor for the ongoing neurodegenerative damage through a non-enzymatic glycation reaction associated with hyperglycemia. This reaction could be represented through Alzheimerâ€™s disease (AD) pathology. Macrophage migration inhibitory factor (MIF) is a cytokine that is expressed both by immune and by non-immune cells. Early glycation and oxidation of MIF in AD brain was previously identified. This modification inhibits MIF enzyme activity and ability to stimulate glial cells. MIF is involved in immune response and insulin regulation, hyper-glycaemia, oxidative stress and glycation are all implicated in AD. Therefore, the current study aimed to investigate that serum MIF could predict brain neurodegeneration at the early pre-symptomatic stages of Alzheimer's disease in diabetic patients. Patients & Methods: Subjects were divided into: Group 1 (n=10): Normal control Non-diabetic subjects. Group 2 (n=10): Diabetes mellitus patients with non-symptomatic AD (DMNAD). Group (3) (n=20): Diabetes mellitus patients with Pre-symptomatic AD (DMPAD). Blood samples were collected from all groups. In this study, adults with T2DM attending Al Batinah Specialized Hospital in Mansoura were examined and compared with volunteers adults without T2DM. The participants were assessed using a short form of the IQCODE, containing 16 items that translated to Arabic version to evaluate for dementia in an Arabic-speaking study population.

Additionally, all the participants had a physical examination, including assessment of glycated haemoglobin, fasting blood glucose and lipid profile. MIF level was measured by ELISA. The data were analyzed using SPSS version 20 for Windows. Results: This study included 30 diabetic patients and 10 controls non-diabetic. A total of 24 diabetic patients (80%) had pre-symptomatic dementia, 19 of whom were women. Pre-symptomatic dementia in the diabetic patients was significantly associated with advancing age, female gender, duration of diabetes and hypertension. MIF level in serum are negatively correlated with symptoms of dementia that it diminished at the pre-symptomatic stages of Alzheimer's disease. Conclusion: Diagnosis processes could not be used as routine examinations for still pre-symptomatic AD.

This may be due to time-consuming and largely depend on physicianâ€™s experience. So, serum MIF level could predict brain neurodegeneration at the early pre-symptomatic stages of Alzheimer's disease in diabetic patients which may support its potential utility as a clinicall

PP2	pharmacology and Toxicology	Faculty of pharmacy Mansoura University

Abdalla Mohamed Anwar Elsayed Ali.
Abdelmonem Elsayed Abdelmonem.

Symptom improvement in children with autism spectrum disorder following bumetanide administration is associated with decreased GABA/glutamate ratios

Abstract

Bumetanide has been reported to alter synaptic excitationinhibition (E-I) balance by potentiating the action of Î³-aminobutyric acid (GABA), thereby attenuating the severity of autism spectrum disorder (ASD) in animal models.However, clinical evidence of its efficacy in young patients with ASD is limited. This was investigated in the present clinical trial of 83 patients, randomised to the bumetanide group (bumetanide treatment, 0.5 mg twice daily) or the control group (no bumetanide treatment). Primary [Children Autism Rating Scale (CARS)], secondary [Clinical Global Impressions (CGI)], and exploratory [inhibitory (Î³-aminobutyric acid, GABA) and excitatory (glutamate, Glx) neurotransmitter concentrations measured in the insular cortex (IC) and visual cortex (VC) by magnetic resonance spectroscopy (MRS)] outcome measures were evaluated at baseline and at the 3-month follow-up. Side effects were monitored throughout the treatment course. Compared with the control group, the bumetanide group showed significant reduction in symptom severity, as indicated by both total CARS score and number of items assigned a score. The improvement in clinical symptoms was confirmed by CGI. GABA/Glx ratio in both the IC and VC decreased more rapidly over the 3-month period in the bumetanide group than that in the control group. This decrease in the IC was associated with the symptom improvement in the bumetanide group. Our study confirmed the clinical efficacy of bumetanide on alleviating the core symptoms of ASD in young children and it is the first demonstration that the improvement is associated with reduction in GABA/Glx ratios. This study suggests that the GABA/Glx ratio measured by MRS may provide a neuroimaging biomarker for assessing treatment efficacy for bumetanide.

PP3	pharmacology and Toxicology	Faculty of pharmacy Mansoura University

Ola Atef Mostafa Belal.
Asmaa Mohamed Abd El_rahman.

CRISPR-engineered T cells in patients with refractory cancer.

Abstract

CRISPR-Cas9 gene editing provides a powerful tool to enhance the natural ability of human T cells to fight cancer. We report a first-in-human phase I clinical trial to test the safety and feasibility of multiplex CRISPR-Cas9 editing to engineer T cells in three patients with refractory cancer. Two genes encoding the endogenous T cell receptor (TCR) chains, TCRÎ± (TRAC) and TCRÎ² (TRBC) were deleted in T cells to reduce TCR mispairing and to enhance the expression of a synthetic, cancer-specific TCR transgene (NY-ESO-1).
Removal of a third gene encoding PD-1 (PDCD1), was performed to improve anti-tumor immunity. Adoptive transfer of engineered T cells into patients resulted in durable engraftment with edits at all three genomic
loci. Though chromosomal translocations were detected, the frequency decreased over time. Modified T cells persisted for up to 9 months suggesting that immunogenicity is minimal under these conditions and demonstrating the feasibility of CRISPR gene-editing for cancer immunotherapy.

PP4	pharmacology and Toxicology	Faculty of pharmacy Mansoura University

Esraa hesham fouda.
Aliaa Ali Abdelhady

Neutrophil activation and NETosis are the major drivers of thrombosis in heparin-induced thrombocytopenia.

Abstract

Heparin-induced thrombocytopenia/thrombosis (HIT) is a serious immune reaction toheparins, characterized by thrombocytopenia and often severe thrombosis with high morbidity and mortality. HIT is mediated by IgG antibodies against heparin/platelet factor 4antigenic complexes. These complexes are thought to activate platelets leading to thrombocytopenia and thrombosis. Here we show that HIT immune complexes induce NETosis via
interaction with FcÎ³RIIa on neutrophils and through neutrophil-platelet association. HITimmune complexes induce formation of thrombi containing neutrophils, extracellular DNA,citrullinated histone H3 and platelets in a microfluidics system and in vivo, while neutrophildepletion abolishes thrombus formation. Absence of PAD4 or PAD4 inhibition with GSK484
abrogates thrombus formation but not thrombocytopenia, suggesting they are induced byseparate mechanisms. NETs markers and neutrophils undergoing NETosis are present in HITpatients. Our findings demonstrating the involvement of NETosis in thrombosis will modifythe current concept of HIT pathogenesis and may lead to new therapeutic strategies.

PP5	pharmacology and Toxicology	Faculty of pharmacy Mansoura University

Fatma fathy El-sayed Abd-elfatah El-habr.

Effect of mTOR inhibitors on neuropathic pain revealed by optical imaging of the insular cortex in rats.

Abstract

In the pain matrix, the insular cortex (IC) is mainly involved in discriminative sensory and motivative emotion.Abnormal signal transmission from injury site causes neuropathic pain, which generates enhanced synapticplasticity. The mammalian target of rapamycin (mTOR) complex is the key regulator of protein synthesis; it isinvolved in the modulation of synaptic plasticity. To date, there has been no report on the changes in opticalsignals in the IC under neuropathic condition after treatment with mTOR inhibitors, such as Torin1 and XL388.

Therefore, we aimed to determine the pain-relieving effect of mTOR inhibitors (Torin1 and XL388) and observethe changes in optical signals in the IC after treatment. Consequently, the inhibitors showed the most effective alleviation 4 h after microinjection intothe IC. In optical imaging, peak amplitudes of optical signals and areas of activated regions were reduced aftertreatment with Torin1 and XL388. However, there were no significant optical signal changes in the IC before andafter vehicle application. These findings suggested that Torin1 and XL388 are associated with the alleviation ofneuronal activity that is excessively manifested in the IC, and is assumed to diminish synaptic plasticity.

PP6	pharmacology and Toxicology	Faculty of pharmacy Mansoura University

Amira Bahaa Ismail.
Merna Tarek Elsaeed.
Samar Mohamed Mahmoud.

Stem cell therapy in autism.

Abstract

Autism spectrum disorders (ASDs) are characterized by core domains: persistent deficits in social communication and interaction; restricted, repetitive patterns of behavior, interests, or activities. ASDs comprise heterogeneous and complex neurodevelopmental pathologies with well-defined inflammatory conditions and immune system dysfunction. Due to neurobiologic changes underlying ASD development, cell-based therapies have been proposed and applied to ASDs. Indeed, stem cells show specific immunologic properties, which make them promising candidates in ASD treatment. This comprehensive up-to-date review focuses on ASD cellular/ molecular abnormalities, potentially useful stem cell types, animal models, and current clinical trials on the use of stem cells in treating autism. Limitations are also discussed.

PP7	pharmacology and Toxicology	Faculty of pharmacy Mansoura University

Mohamed Ibrahim Mourad.

Selenium nanoparticles act as an intestinal p53 inhibitor mitigating chemotherapy-induced diarrhea in mice.

Abstract

Selenium, at high-dose levels approaching its toxicity, protects tissues from dose-limiting toxicities of many cancer chemotherapeutics without compromising their therapeutic effects on tumors, there by allowing the delivery of higher chemotherapeutic doses to achieve increased cure rate. In this regard, selenium nanoparticles (SeNPs), which show the lowest toxicity among extensively investigated selenium compounds including methylselenocysteineand selenomethionine, are more promising for application. The key issue remains to be resolved is whether low-toxicity SeNPs possess a selective protective mechanism. p53 or p53-regulated thrombospondin-1 has each been confirmed to be an appropriate target for therapeutic suppression to reduce side effects of anticancer therapy. The present study demonstrated that SeNPs transiently suppressed the expression of many intestinal p53-associated genes in healthy mice. SeNPs did not interfere with tumor-suppressive effect of nedaplatin, a cisplatin analogue; however, effectively reduced nedaplatin-evoked diarrhea. Nedaplatin-induceddiarrhea was associated with activation of intestinal p53 and high expression of intestinal thrombospondin-1. The preventive effect of SeNPs on nedaplatin-induced diarrhea was correlated with a powerful concomitant suppression of p53 and thrombospondin-1. Moreover, the high-dose SeNPs used in the present study did not suppress growth nor caused liver and kidney injuries as well as alterations of hematological parameters in healthy mice. Overall, the present study reveals that chemotherapeutic selectivity conferred by SeNPs involves adual suppression of two well-documented targets, the p53 and thrombospondin-1, providing mechanistic and pharmacologic insights on low-toxicity SeNPs as a potential chemoprotectant for mitigating chemotherapy-induced diarrhea.

Section (PM)

Microbiology

(PM1-PM3)

PM1	Microbiology	Faculty of pharmacy Mansoura University

Mariam Yahya Hussein Abu-Elenin
Nafesa Yousef Abdelhady Abdelkader.

Semaphorin 3A Is Effective in Reducing Both Inflammation and Angiogenesis in a Mouse Model of Bronchial Asthma.

Abstract

Semaphorin 3A (sema3A) belongs to the sub-family of the immune semaphorins that function as regulators of immune-mediated inflammation. Sema3A is a membrane associated molecule on T regulatory cells and on B regulatory cells. Being transiently ligated to the cell surface of these cells it is suggested to be a useful marker for evaluating their functional status. In earlier studies, we found that reduced sema3A concentration in the serum of asthma patients as well as reduced expression by Treg cells correlates with asthma disease severity. Stimulation of Treg cells with recombinant sema3A induced a significant increase in FoxP3 and IL-10 expression. To find out if sema3A can be of benefit to asthma patients, we evaluated the effect of sema3A injection in a mouse model of asthma. BALB\c-mice were sensitized using ovalbumin (OVA) + adjuvant for 15 days followed by OVA aerosol inhalation over five consecutive days. Four hours following air ways sensitization on each of the above days-15 of these mice were injected intraperitoneally with 50 Âµg per mouse of recombinant human sema3A-FR and the remaining 15 mice were injected with a similarly purified vehicle. Five days later the mice were sacrificed, broncheo-alveolar lavage (BAL) was collected and formalin-fixed lung biopsies taken and analyzed. In sema3A treated mice, only 20% of the bronchioles and arterioles were infiltrated by inflammatory cells as compared to 90% in the control group ($p = 0.0079$). In addition, eosinophil infiltration was also significantly increased in the control group as compared with the sema3A treated mice. In sema3A treated mice we noticed only a small number of mononuclear and neutrophil cells in the BAL while in the control mice, the BAL was enriched with mononuclear and neutrophil cells. Finally, in the control mice, angiogenesis was significantly increased in comparison with sema3A treated mice as evidenced by the reduced concentration of microvessels in the lungs of sema3Atreated mice. To conclude, we find that in this asthma model, sema3A functions as apotent suppressor of asthma related inflammation that has the potential to be further developed as a new therapeutic for the treatment of asthma.

PM2	Microbiology	Faculty of pharmacy Horus University

Dina mohamed Abdrabo Awad.

Plastic antibodies for cancer Therapy

Abstract

One of the most promising strategies to treat cancer is the use of therapeutic antibodies that disrupt cell-cell adhesion mediated by dysregulated cadherins. The principal site where cell-cell adhesion occurs encompasses Trp2 found at the N-terminal region of the protein. Herein, we employed the naturally exposed highly conserved peptide Asp1-Trp2-Val3-Ile4-Pro5-Pro6-Ile7, as epitope to prepare molecularly imprinted polymer nanoparticles (MIP-NPs) to recognize cadherins. Since MIP-NPs target the site responsible for adhesion, they were more potent than commercially available therapeutic antibodies for inhibiting cell-cell adhesion in cell aggregation assays, and for completely disrupting three-dimensional tumor spheroids as well as inhibiting invasion of HeLa cells. These biocompatible supramolecular anti-adhesives may potentially be used as immunotherapeutic or sensitizing agents to enhance antitumor effects of chemotherapy.

PM3	Microbiology	Faculty of pharmacy Mansoura University

Hamsa Hassan Helaley.
Amira Ali Ali Mustafa Elgendy.
Baraaha Hossam.

Therapeutic applications of lytic phages in human medicine.

Abstract

The emergence and spread of antibiotic-resistant bacteria constitute a critical issue for modern medicine. Patients with antibiotic-resistant bacterial infections consume more healthcare resources and have worse clinical outcomes than patients with antibiotic-sensitive bacterial infections. Phages are natural predators of bacteria and may therefore be a source of useful antibacterial drugs. Phage therapy possess availability for oral administration, penetration through the bacteria cell wall, and eradication bacterial biofilms. All of these advantages give phage therapy the possibility to turn into applications for infectious diseases. In this mini-review, we focus on the brief history of lytic phage therapy, the life cycles of lytic phages and the therapeutic effects of lytic phages.

Section (PB)

Biochemistry

PB1

PB1	Biochemistery.	Faculty of pharmacy Mansoura University

Aya Ahmed Abbas Al-Anany.

Anticancer effects of silver nanoparticles encapsulated by Taxus baccata extracts.

Abstract

Cancer is one of the main causes of human fatality and finding effective anticancer drugs is a high priority. Modified anticancer medicines have advantages compared to traditional drugs including; better delivery and lower doses. In the present study, the anticancer activity of silver nanoparticles (Ag NPs) synthesized using Taxus baccata extracts was studied on Caov-4 and HeLa cancer cell lines as well as normal human fibroblast cells. Microscopic studies showed significant morphological changes of cancer cells following the exposure to the Ag NPs while MTT assay revealed dose, time and cell line dependent cytotoxicity. The toxic effect of Ag NPs on Caov-4 cells was considerably higher than HeLa cells as N98% mortality obtained after 72 h incubation of Caov-4 cells with 5 and 20 Î¼g/mL Ag NPs encapsulated by aqueous and ethanolic extract, respectively. Interestingly, aqueous extract encapsulated Ag NPs had no significant toxicity on normal fibroblast cells suggesting potent and selective anticancer activity on cancer cell lines. Flow cytometric analysis revealed a combination of apoptosis and necrosis following the exposure of Ag NPs to cancer cells. Apoptosis was determined as main mechanism of cell death in low concentration of Ag NPs while considerable necrosis (up to 41%) was observed by increasing the Ag NPs dose (up to 10 Î¼g/mL) and incubation time (up to 72 h). The significant DNA damage and change in expression level of caspase 8, caspase 9, bcl-2 and c-Abl genes indicated the induction of mitochondrial death pathway following cell exposure to Ag NPs. It can be concluded that the preparation method and stabilizing agents may have major effects on the biological activity of nanoparticles as well as their physicochemical properties.

Section (PMC)

Medicinal Chemistry.

(PMC1-PMC2)

PMC1	Medicinal Chemistry.	Faculty of pharmacy Mansoura University

Mariam Sabry Abd-allah El-baz.

Design,synthesis and anticancer evaluation of 1H-pyrazolo[3,4-d]pyrimidine derivatives as potent EGFR WT and EGFR T790M inhibitors and apoptosis inducers.

Abstract

In our attempt to develop effective EGFR-TKIs, two series of 1H-pyrazolo[3,4-d]pyrimidine derivatives were designed and synthesized. All the newly synthesized compounds were evaluated in vitro for their inhibitory activities against EGFRWT. Compounds 15b, 15j, and 18d potently inhibited EGFRWT at sub-micro molar IC50 values comparable to that of erlotinib. Moreover, thirteen compounds that showed promising IC50 values against EGFRWT were tested in vitro for their inhibitory activities against mutant EGFRT790M. Compounds 17d and 17f exhibited potent inhibitory activities towards EGFRT790M comparable to osimertinib. Compounds that showed promising IC50 values against EGFRWT were further tested for their anti-proliferative activities against three cancer cell lines bearing EGFRWT (MCF-7, HepG2, A549), and two cancer cell lines bearing EGFRT790M (H1975 and HCC827). Compounds 15g, 15j, 15n, 18d and 18e were the most potent anticancer agents against the EGFRWT containing cells, while compounds15e, 17d and 17f showed promising anti-proliferative activities against EGFRT790M containing cells. Furthermore, the most active compound 18d was selected for further studies regarding to its effects on cell cycle progression and induction of apoptosis in the HepG2 cell line. The results indicated that this compound is good apoptotic agent and arrests G0/ G1and G2/M phases of cell cycle. Finally, molecular docking studies were performed to investigate binding pattern of the synthesized compounds with the prospective targets, EGFRWT (PDB: 4HJO) and EGFRT790M (PDB: 3W2O).

-Key words: Anticancer; EGFR-TKIs; Docking; NSCLC; 1H-Pyrazolo[3,4-d]pyrimidine; EGFRWT; EGFRT790M

PMC2	Medicinal Chemistry.	Faculty of pharmacy Horus University

Amr Mahmoud Mostafa Abdo Sakr

Design, synthesis and molecular docking of novel benzothiazole derivatives as antiviral agents.

Abstract

A novel coronavirus (2019-nCoV) originating in Wuhan, China presents a potential respiratory viral pandemic to the world population. Current efforts are focused on containment and quarantine of infected individuals. Ultimately, the outbreak could be controlled with a protective vaccine to prevent 2019-nCoV infection. While vaccine research should be pursued intensely, there exists today no therapy to treat 2019-nCoV upon infection, despite an urgent need to find options to help these patients and preclude potential death.

In our research to develop a new drug for the treatment of coronavirus (2019-nCoV), we designed a new compound, explained its binding mode using MOE drug design program and started to synthesis it as a part of our graduation project.

Hoping to aid in finding a new active drug against this deadly virus.

Section PT

Pharmacy Practice (PT1)

PT1	Pharmacy Practice	Faculty of pharmacy Mansoura University

Alaa Atef Abdulkhalik Shaheen.

25% of Egyptians have mental health issues and the rest could be lying

Abstract

A national survey conducted by the Ministry of Health found one fourth of the Egyptians to be suffering from mental health-related illnesses.
The study showed the predominance of depression and anxiety among that quarter of the participants, with approximately 43.7% showing anxiety disorders, while 30.1% suffered depression that is linked to substance abuse.
Among the samples of 22,000 random families across Egypt, Minya presented the highest percentage of people suffering from anxiety.
The results of the survey prove the serious need to be more attentive and to expand health services dedicated to mental problems, especially in the disadvantaged areas that have insufficient access to facilities and treatment for such issues.
A family history of mental issues is shown to be among the main factors to the high incidence of an individual developing mental illness, accentuating the prominence of incorporating various activities and sports into the daily routine of citizens, which can considerably help the cause.
The community view of seeking help and addressing mental illness as diseases with specific pathogenesis is also questionable and needs multiple actions and efforts of every person in the health care field in aim to control the case.

Research is to see what everybody else has seen, and to think what nobody else has thought. Research means to investigate something you do not know or understand. While advances in scientific research have led to some new and exciting treatments that have enlarged and enhanced the quality and length of human life, we must not lose sight as to what we are trying to accomplish