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AN AWARD-WINNING, STUDENT-OPERATED NEWSLETTER BY THE
ST. JOHN'S UNIVERSITY COLLEGE OF PHARMACY AND HEALTH SCIENCES' RHO CHI BETA DELTA CHAPTER

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Meet Corlanor®: A New Drug for Chronic Heart Failure

By: Svetlana Akbasheva, Staff Editor

For years, the medications that have been the mainstay of therapy for chronic heart failure have been angiotensin converting enzyme (ACE) inhibitors (or angiotensin receptor blockers), beta-blockers, loop diuretics, and aldosterone antagonists, with the occasional addition of digoxin or the hydralazine/isosorbide dinitrate combination.¹ Now, a novel medicine is trying to make its way into this exclusive circle. On April 15th 2015, the FDA approved Amgen's ivabradine (Corlanor®) for the indication of reducing hospitalizations in chronic heart failure patients.²

The utility of ivabradine in heart failure is attributed to its mechanism of slowing down the heart rate by blocking the activation pathway of the cardiac pacemaker *I_f* current. Unlike other medications that exhibit negative chronotropic effects, ivabradine is unique in that it exclusively affects heart rate and has no effect on the force of contraction of the heart. It is important to note that ivabradine is not indicated for all chronic heart failure patients. Ideal candidates are those with a left ventricular ejection fraction $\leq 35\%$ and a baseline heart rate of at least 70 beats per minute who are at maximum therapeutic doses of or have contraindications to beta-blocker therapy.³

Ivabradine has several important contraindications that must be ruled out prior to initiating therapy. Patients with acute decompensated heart failure should not receive ivabradine. In addition, patients with a baseline resting heart rate of less than 60 beats per minute or whose blood pressure is below 90/50 mmHg are excluded from therapy. Patients with sick sinus syndrome, sinoatrial block, or 3rd degree AV block may receive this medication only if they have a functioning demand pacemaker. However, any patient that is fully dependent on a pacemaker to maintain their heart rate should not receive ivabradine.

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Since ivabradine is primarily metabolized by CYP3A4, concurrent use of strong inhibitors of this enzyme is contraindicated due to the risk of drug buildup and toxicity. Although moderate CYP3A4 inhibitors such as diltiazem, verapamil, and grapefruit juice are not contraindicated, they should be avoided. Finally, severe hepatic impairment, indicated by a Child-Pugh score of C, precludes the use of ivabradine.³

For patients starting ivabradine therapy, the target is to achieve a heart rate of 50 to 60 beats per minute. Initiation with a dose of 5 mg twice daily with food is recommended. However, an exception is patients with a history of conduction defects or in whom bradycardia could be dangerous; this population should begin with the lower dose of 2.5 mg twice daily. After initiating therapy, dose adjustments should be made until the target heart rate is achieved. For patients with a heart rate above 60 beats per minute on their current regimen, the dose may be increased by 2.5 mg twice daily up to the maximum recommended dosage of 7.5 mg twice daily.

Conversely, patients whose heart rate drops below 50 beats per minute should have their dose decreased by 2.5 mg twice daily. If a patient's heart rate remains under 50 even at the lowest dose of 2.5 mg twice daily, then ivabradine should be discontinued. Although ivabradine does not require any dosage adjustments in mild to moderate hepatic or renal impairment, it has not been studied in patients with a creatinine clearance less than 15 ml/min.³

The most severe adverse effects of ivabradine seen in clinical trials are atrial fibrillation and bradycardia. Patients' cardiac rhythm should be assessed periodically while on ivabradine and the medication should be discontinued if atrial fibrillation is confirmed. Bradycardia is particularly risky in patients

who are also on beta-blockers or other negative chronotropes. Ivabradine can also cause visual disturbances, often characterized by increased visual brightness, due to its effects on receptors in the retina. In addition, animal studies have shown that ivabradine can cause fetal toxicity, so women of childbearing age should avoid getting pregnant while on this medication.³

It is important to distinguish that the Systolic Heart Failure Treatment with I_f inhibitor Ivabradine Trial (SHIFT), which showed a decreased rate of hospitalizations with ivabradine compared to placebo, showed no effect of ivabradine on mortality from heart failure. In addition, two other trials showed no benefit of ivabradine in delaying cardiovascular death or myocardial infarction in patients with stable coronary artery disease, both with and without heart failure.³ Thus, each patient must be carefully assessed to see whether the anticipated benefit with this new medication is justified for his or her particular condition.

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Miltefosine (Impavido®) Approved to Treat Tropical Disease

By: Sang Hyo Kim, Staff Editor

On March 19, 2014, the U.S Food and Drug Administration (FDA) approved miltefosine (Impavido®) for the treatment of leishmaniasis. Leishmaniasis is a disease caused by *leishmania*, a parasite that is transmitted to humans through sand fly bites. Although the majority of people affected with leishmaniasis are from tropical and subtropical regions, many U.S patients contract the disease when overseas.¹ Leishmaniasis is found in over 80 countries, and about 350 million people are considered to be at risk. There are currently 12 million people infected and an estimated 70,000 deaths annually.²

Miltefosine, manufactured by Paladin Therapeutics, is the first oral drug approved to treat the three main types of leishmaniasis: visceral leishmaniasis (affecting internal organs), cutaneous leishmaniasis (affecting the skin), and mucosal leishmaniasis (affecting the nose and throat).¹ Miltefosine has a direct toxic effect on the parasite by stimulating immune cells such as macrophages to cause parasite elimination via oxidative radicals.³

The efficacy of miltefosine was tested in four clinical trials with a total of 547 patients receiving the treatment drug and 183 patients receiving an alternative drug or placebo.¹ The overall results showed that miltefosine can successfully treat the three main types of leishmaniasis. The FDA also granted miltefosine a fast track designation, priority review, and orphan designation. In other words, the drug demonstrated the potential to fill an unmet medical need in a serious disease state, the potential to be a significant improvement in a treatment of a disease, and the potential to treat a rare disease, respectively.⁴

Miltefosine is intended for patients 12 years of age and older.¹ The most common side effects seen in clinical trials were nausea, vomiting, diarrhea, headache, decreased appetite, dizziness, abdominal pain, itching, drowsiness, and elevated levels of liver enzymes (transaminases) and creatinine.¹ Miltefosine is not recommended for pregnant women; this drug

bears a black box warning highlighting the harm it could cause to a fetus. The FDA therefore strongly urges healthcare professionals to advise women to use effective contraception during and for five months after miltefosine therapy.¹

Although leishmaniasis is prevalent in tropical areas, it is reassuring to know that there is a treatment available here if necessary. The release of drugs to treat diseases that are rare in the U.S. elucidates the relentless efforts of researchers and the FDA.

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Stem Cells May Halt Progression of Multiple Sclerosis

by: Nehali Parikh, PharmD Candidate c/o 2016

Multiple sclerosis (MS) is an autoimmune disease that results in interrupted neurotransmission throughout the body. It occurs when the immune system mistakenly attacks myelin, the insulating layer surrounding nerve cells, causing the formation of scar tissue, called sclerosis. Fatigue, numbness, tingling, difficulty in walking, and dizziness are all common symptoms of MS.¹

While there are various stages and forms of MS, relapsing-remitting MS (RRMS) is the most common form, in which mild or no symptoms occur within certain periods of more severe symptoms.³ MS is not a curable disease, but there are certain medications that have been approved to help treat relapsing forms of MS. These immunosuppressant agents aim to modify the disease course by treating exacerbations and alleviating symptoms.¹

A non-randomized, multicenter clinical trial was conducted by the National Institute of Allergy and Infectious Diseases (NIAID)-funded Immune Tolerance Network, in which participants with RRMS received autologous stem cell transplants after treatment with a high dose of immunosuppressive therapy. The clinical trial, known as HALT-MS, is a prospective, open-label, single-arm, phase 2 clinical trial.⁵ The primary endpoint of this study is the time to treatment failure within 5 years after HCT, which is defined as death or disability by: change in EDSS score of more than 0.5 points compared to baseline, relapse or recurring symptoms lasting more than 48 hours, and 2 or more MS disease-related lesions on brain MRI that occurred 1 year or more after HCT.⁵

These participants were patients who experienced relapses with loss of neurologic function while

they received disease-modifying therapies during 18 months prior to enrollment, including interferon beta-1a, glatiramer acetate, mitoxantrone, natalizumab, interferon beta-1b, methylprednisolone or dexamethasone, cyclophosphamide, methotrexate, minocycline, and plasma exchange.⁵ Thirty-six patients with RRMS were screened and 25 were enrolled in this study.⁵

Eligible patients were aged 18 to 60 years and had a diagnosis of MS according to the McDonald criteria with: RRMS, Krutzke Expanded Disability Status Scale (EDSS) scores between 3.0 to 5.5, lesions that were shown on a brain MRI, duration of the disease for less than 15 years, and failure of disease modifying therapies. The failure of previous treatment was defined as 2 or more clinical relapses during 18 months of therapy that were shown with an increase in the EDSS score.⁵

In this trial, patients were initially given 1 mg/kg of prednisone for 10 days, which was completed 1 day before filgrastim therapy was initiated, in order to prevent MS relapse. Peripheral blood stem cells were collected from patients and treated with filgrastim for four days; CD34 cells were selected and cryopreserved.⁵ Patients then received high dose chemotherapy, which was composed of 300 mg/m² of carmustine on day -6, 200 mg/m² of etoposide, 200 mg/m² of cytarabine daily from days -5 to -2, and 140 mg/m² of melphalan was on day-1.⁵ Rabbit antithymocyte globulin, 2.5 mg/kg/d, was given on days -2 and -1. On day 0, CD34+ cells were thawed and infused in the patients. Filgrastim was administered from day 5 until the neutrophil count was greater than 500/ μ L.⁵ Prednisone 0.5 mg/kg/d

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was given to patients on days 7 to 21 to prevent engraftment syndrome, then tapered over the following two weeks.

A three-year interim analysis of this five year trial showed how high-dose immunosuppressive therapy with autologous hematopoietic cell transplant (HDIT/HCT) might lead to a sustained remission of RRMS.² The disease activity was controlled, according to the primary endpoint criteria, through three years after HCT in most patients except in two patients, who had a failure of treatment.⁵

Previous clinical trials revealed that HDIT/HCT treatment was not effective in participants who had progressive MS. However, this data shows that it may be effective in participants who are in the early stages of RRMS. In fact, three years after the HDIT/HCT clinical trial, about 80% of the patients survived without experiencing any symptoms of MS, including any neurological disabilities.^{2,3} during these three years of the trial, the investigators did not find many unexpected side effects. Most of the side effects that participants experienced were gastrointestinal as well as infections, both of which are common adverse effects of high dose immunosuppression.⁴

Daniel Rotrosen, MD, director of the NIAID Division of Allergy, Immunology and Transplantation, states, "Participants did not receive any MS drugs after transplant, yet most remained in remission after three years. In contrast, other studies have shown that the best alternative MS treatments induce much shorter remissions and require long-term use of immunosuppressive drugs that can cause serious side effects."⁴

The researchers plan to follow the participants for a total of 5 years. The results from this will help determine what further studies need to be done to use HDIT/HCT as an effective alternative treatment in patients with MS.

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FDA Approves Vorapaxar

By: Azia Tariq, Section Editor

Myocardial infarctions, more commonly known as heart attacks, are prevalent in the United States. Each year, approximately 720,000 Americans suffer a heart attack.¹ The approval of vorapaxar by the Food and Drug Administration (FDA) presents an additional treatment option for patients at high risk for myocardial infarction and stroke.^{1,2}

Myocardial infarctions largely occur due to a blood flow blockage in the coronary arteries. The blood flow blockage can be caused by a blood clot or from the buildup of plaque in the coronary arteries. The lack of oxygen to the heart causes the cells to die, resulting in the heart attack. Hallmark symptoms include chest pain or numbness that spreads to the shoulder blades, neck, and jaw. Additional symptoms may include anxiety, coughing, fainting, light-headedness, dizziness, nausea or vomiting, palpitations, shortness of breath, and sweating.^{3,4}

Immediate treatment includes nitroglycerin and morphine to help reduce chest pain, in addition to aspirin to prevent blood clots. Subsequent steps may include angioplasty, a procedure to open narrowed or blocked blood vessels that supply blood to the heart. An angioplasty should be done within 90 minutes of the patient's arrival to the hospital and usually no later than 12 hours after a heart attack. Pharmacologic treatment is also given in order to break up the clot during thrombolytic therapy. Some patients may undergo bypass surgery to restore blood flow to the heart by diverting the flow around a section of a blocked artery.³

Vorapaxar (ZontivityTM) is an oral protease-activated receptor-1 (PAR-1) antagonist that inhibits thrombin-induced platelet activation and is FDA approved for to reduce the risk of heart attack, stroke, and cardiovascular death.⁵ The 2.08 mg tablet is taken orally once daily in addition to aspirin.⁵ It will also be used for procedures that restore blood flow to the heart in patients with a previous heart attack or blockages in the arteries to the legs.⁶ As an anti-platelet agent designed to decrease the tendency of platelets to clump together to form a blood clot, vorapaxar decreases the risk of heart attack and stroke.²

In the clinical trial referred to as TRA2P TIMI 50, 26,449 patients were randomly selected to receive either vorapaxar 2.5mg daily in addition to standard of care, or placebo in addition to standard of care. The primary efficacy endpoint was the composite rate of cardiovascular death, MI, stroke, or urgent coronary revascularization. Eligibility criteria required a diagnosis of atherosclerosis—specifically MI, PAD, or ischemic stroke during the prior period of 2 weeks to 12 months. Baseline characteristics of the patients indicated that 67% had a previous MI, 19% had a previous stroke, and 14% had PAD.

In January 2011, after an increase in intracranial hemorrhage (ICH) was seen in patients with a history of stroke, The Data and Safety Monitoring Board (DSMB) recommended that these individuals stop taking vorapaxar. Other patient groups continued the trial as expected, with a median follow-up of 2.5 years. At 3 years, the results showed a reduction in the primary endpoint in the vorapaxar group (9.3% vs. 10.5%; HR 0.87; 95% CI 0.8-0.94, $p<0.001$). A greater reduction was observed in patients without a history of stroke (8.3% vs. 9.6%; HR 0.84; 95% CI 0.76-0.93). Moderate to severe bleeding was increased in the vorapaxar group (4.2% vs. 2.5%; HR 1.66; 95% CI 1.43-1.93; $p<0.001$), with a significant increase in ICH (1% vs. 0.5%, $p<0.001$).^{7,8}

An independent companion trial, the TRACER study, analyzed whether the addition of vorapaxar to standard of care would lead to a reduction in atherothrombotic ischemic events compared to standard of care alone. A total of 12,944 non-ST-segment elevation acute coronary syndrome (NSTEMI) patients were randomized to receive either vorapaxar in addition to standard of care or standard of care alone. NSTEMI refers to a number of conditions ranging from unstable angina to NSTEMI myocardial infarction (MI) which have an underlying cause of atherosclerotic plaque disruption and differing degrees of associated thrombosis and distal embolization.⁹ Of those patients, 8750 (67.6%) had undergone percutaneous coronary intervention or coronary artery bypass grafting and 4194 patients (32.4%) had not undergone revascularization during the tri-

al.⁷ Patients who were managed medically were heterogeneous with different risk profiles, including 1137 (27.1%) who did not undergo coronary angiography.⁹

In the medically managed group, two year primary outcome (cardiovascular death, myocardial infarction, stroke, recurrent ischemia with re-hospitalization, and urgent coronary revascularization) event rates were 16.3% with vorapaxar and 17.0% with only standard of care (HR 0.99, 95% CI 0.83-1.17) with no interaction between drug and management strategy ($p=0.75$). Secondary endpoint (cardiovascular death, myocardial infarction, stroke) rates were 13.4% with vorapaxar and 14.9% with only standard of care (HR 0.89, 95% CI 0.74-1.07) with no interaction ($p=0.58$). Vorapaxar also increased moderate to severe bleeding in medically managed patients (adjusted HR 1.46, 95% CI 0.99-2.15).⁹

In patients undergoing coronary artery bypass grafting (CABG) during the trial, vorapaxar-treated patients had a 45% lower rate of the primary endpoint (composite of death, myocardial infarction, stroke, recurrent ischemia with rehospitalization, or urgent coronary revascularization during index hospitalization) (HR: 0.55; 95% CI: 0.36 to 0.83; $p = 0.005$), with a significant interaction ($p = 0.012$) compared to CABG patients with only standard of care.¹⁰ The trial follow-up was terminated early after a safety review by the DSMB. After a median follow-up of 502 days, the primary end point occurred in 1031 of 6473 patients receiving vorapaxar versus 1102 of 6471 patients receiving standard of care (18.5% vs. 19.9%; HR 0.92; 95% CI 0.85 to 1.01; $P=0.07$). A composite of death from cardiovascular causes, myocardial infarction, or stroke occurred in 822 patients in the vorapaxar group versus 910 in the placebo group (14.7% and 16.4%, respectively; HR 0.89; 95% CI 0.81 to 0.98; $P=0.02$).

Rates of moderate/severe bleeding were 7.2% in the vorapaxar group and 5.2% in the placebo group (HR 1.35; 95% CI 1.16 to 1.58; $P<0.001$). Rates of nonhemorrhagic adverse events were similar in the two groups. Intracranial hemorrhage rates were 1.1% and 0.2%, respectively (HR 3.39; 95% CI, 1.78 to 6.45; $P<0.001$).⁶ Trial follow-up was terminated in January 2011 after a safety review by

the DSMB.

Rosser et al examined the beneficial effect of vorapaxar on global thrombotic and thrombolytic status in patients with coronary disease by testing the blood of 57 patients who were given vorapaxar with the point-of-care global thrombosis test. This is an automated test that employs non-anti-coagulated blood to assess thrombotic and thrombolytic status by measuring the time required to form a shear-induced thrombus under physiological conditions, called the occlusion time (OT), and the time to achieve endogenous lysis of the thrombus, called the lysis time (LT).

A longer OT on and off treatment was observed for patients on vorapaxar [median 561 seconds (interquartile range 422-654) vs. 372 s(338-454), $P = 0.003$] and shorter LT on treatment than off treatment [1,158 s(746-1,492) vs. 1,733 s(1,388-2,230), $P = 0.016$]. Patients on placebo showed no difference in OT [419 s(343-514) vs. 411 s(346-535), $P = 0.658$] or LT [1,236 s(985-1,594) vs. 1,400 s(1,092-1,686), $P = 0.524$] on and off treatment. During treatment, OT was longer in patients taking vorapaxar [561 s(422-654) vs. 419 s(343-514), $P = 0.009$], but LT was similar in vorapaxar and placebo arms [1,158 s(746-1,492) vs. 1,236 s(985-1,594), $P = 0.277$]. It was observed that vorapaxar prolongs OT and shortens LT and has favorable antiplatelet effects on thrombotic and thrombolytic status. In addition, vorapaxar enhances thrombolysis, which is often impaired in coronary disease.¹¹

Director of the Office of Drug Evaluation I in the FDA's Center for Drug Evaluation and Research, Ellis Unger, M.D., stated, "In patients who have had a heart attack or who have peripheral arterial disease, this drug will lower the risk of heart attack, stroke, and cardiovascular death. In the study that supported the drug's approval, Zontivity™ lowered this risk from 9.5 percent to 7.9 percent over a 3-year period – about 0.5 percent per year."²

As with any medication, vorapaxar has a range of adverse effects which include life-threatening and fatal bleeding, intracranial hemorrhage, anemia, depression, rash, and exanthema. Usage is contraindicated in patients with history of stroke, TIA, or ICH and with patients with active bleeding such as ICH or peptic ulcer. Withholding vorapaxar for a brief pe-

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riod will not be useful in managing an acute bleeding event due to its long half-life. There is no known treatment to reverse the antiplatelet effect of vorapaxar. Platelet aggregation inhibition can be expected for weeks after discontinuation of normal dosing. Significant inhibition of platelet aggregation remains 4 weeks after discontinuation.

No clinically significant pharmacokinetic or pharmacodynamic interactions were observed in an interaction study with vorapaxar and warfarin in healthy subjects. No pharmacokinetic effects were noted between vorapaxar and prasugrel following multi-dose administration as well.⁵

Healthcare professionals must consider underlying risks of bleeding before initiating vorapaxar. Risk factors for bleeding include older age, low body weight, reduced renal or hepatic function, history of bleeding disorders, and use of certain concomitant medications that increase the risk of bleeding (anticoagulants, fibrinolytic therapy, chronic non-steroidal anti-inflammatory drugs [NSAIDs], selective serotonin reuptake inhibitors, serotonin norepinephrine reuptake inhibitors). Avoid concomitant use of vorapaxar and warfarin or other anticoagulants.

No renal or hepatic dose adjustment is required, however, vorapaxar is not recommended for patients with severe hepatic impairment due to increased risk of bleeding. No adequate studies were performed for use of vorapaxar in pediatric populations or pregnant women. The potential for serious adverse reactions and the possibility of being excreted in human milk suggests nursing mothers should not take vorapaxar.⁵

With recent FDA-approval of vorapaxar to help reduce the risk of myocardial infarction and stroke through the mechanism of inhibited thrombin-induced platelet activation, patients who are at risk now have an additional efficacious treatment option.

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Puzzle: Word Scramble



odWr
IembaScr

By: Sang Hyo Kim
Section Editor

Can **YOU**
Unscramble
these common
prescription
medications?

Answers
on next page

1. Doluentiex
2. nFsloae
3. rPatixeoem
4. uneeloDit
5. niexoteFlu
6. oenFvtl
7. aeamrF
8. pazemOolre
9. liAoiFcdc
10. apCxe
11. iuvanaJ
12. feexKl
13. eparKp
14. evLaquin
15. uaLsnt
16. paivrSi
17. ratlmU
18. noMirt

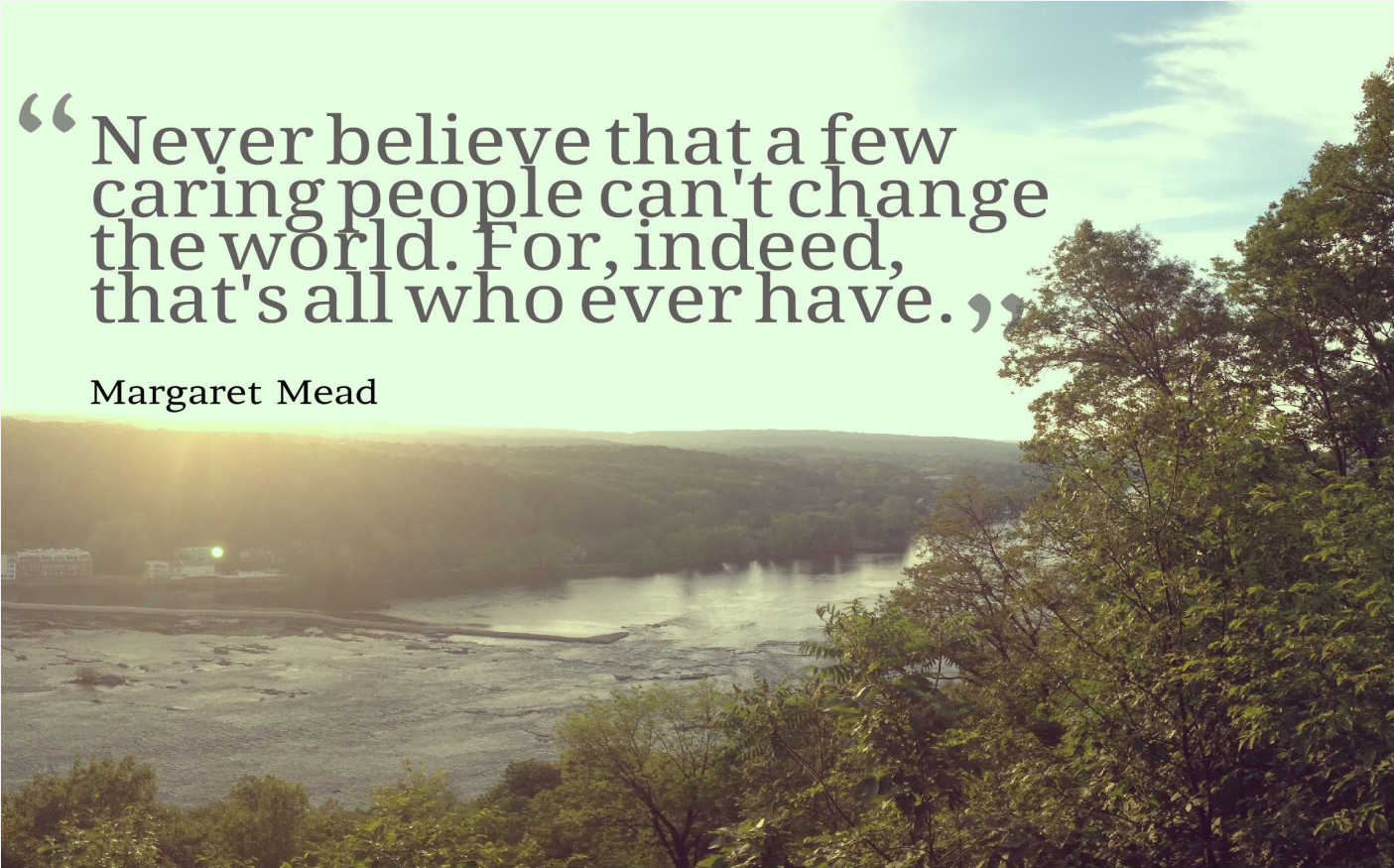
How Did You Do???

Answers to Word Scramble

1) Duloxetine 2) Flonase 3) Paroxetine 4) Duloxetine 5) Fluoxetine 6) Flovent 7) Femara 8) Omeprazole 9) Folic Acid 10) Capex 11) Januvia 12) Keflex 13) Keppra 14) Levaquin 15) Lantus 16) Spiriva 18) Ultram 19) Motrin

Quote of Month

By: Nicollette Pacheco, Staff Editor [Graphics-focused]



“Never believe that a few caring people can't change the world. For, indeed, that's all who ever have.”

Margaret Mead

PUZZLES

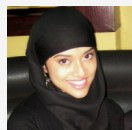
Do you enjoy our puzzle?

Send us a suggestion for a brainteaser at

RhoChiPost@gmail.com

We will feature your work in our next issue!

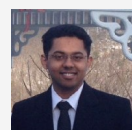
RHO CHI POST: TEAM MEMBERS

**@ Tasnima Nabi (6th Year, STJ; Editor-in-Chief)**

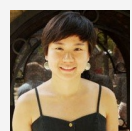
Writing has always been my greatest outlet for experience and knowledge, through which I hope to keep you engaged and informed. It is imperative to keep up with our changing profession and community, and I look forward to bringing pertinent information to the newsletter.

**@ Katharine Cimmino (PharmD; Graduate Copy Editor [Content-Focused])**

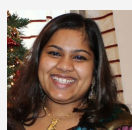
I have always been an avid reader and writer. As a member of the Rho Chi Post I am able to merge my passions with the professionalism that comes with aspiring to be a healthcare provider. I am eager to be a part of a publication that promotes my interests and vocation.

**@ Bharat Kirthivasan (PhD; Graduate Copy Editor [Content-Focused])**

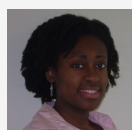
I received my doctorate in Industrial Pharmacy researching nanoparticles for delivery to the brain. The only thing I enjoy more than reading a well-written piece of work is writing it. I am glad to work for the Rho Chi Post, and I encourage others to do the same.

**@ Hayeon Na (PharmD; Graduate Copy Editor [Content-Focused])**

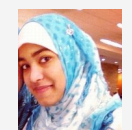
Hello! My name is Hayeon Na. I am one of the Graduate Copy Editors for the Rho Chi Post. I hope the information I present will be helpful, or at least interesting. If you have any comments regarding my contribution, feel free to contact me at any time!

**@ Melissa Roy (PharmD; Graduate Copy Editor [Graphics-Focused])**

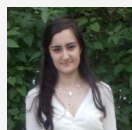
We as future healthcare professionals owe it to our patients and ourselves to be aware and current on the events affecting our profession. The Rho Chi Post is our way to learn new things and stay in touch with the pharmacy world, on- and off-campus. Feel free to reach out to me with suggestions and comments.

**@ Davidta Brown (5th Year, STJ; Copy Editor [Content-Focused])**

My two great loves are innovative science and quality writing, and the Rho Chi Post is an insightful combination of both. As an editor, I look forward to bringing relevant information and fresh perspectives to the student and faculty of St. John's University, as well as to making the Rho Chi Post a newsletter that offers something new to every reader.

**@ Fatema Elias (6th Year, STJ; Copy Editor [Content-Focused])**

I am honored to be a part of the Rho Chi Post team. In this age of technology and the continuously changing healthcare profession, I hope to engage like-minded students and professionals. Writing is something that I hold dear to my heart and I hope with this newsletter we can all stay well informed, interested, and educated.

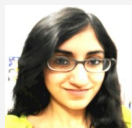
**@ Tamara Yunusova (5th Year, STJ; Section Editor: Clinical)**

My name is Tamara Yunusova, and I am a 5th year Pharm D candidate at St. John's University. I enjoy articulating information in a captivating and insightful way. I hope to make this publication more informative, student-friendly, and innovative.

**@ Sang Hyo Kim (4th Year, STJ; Section Editor: Puzzles)**

Advancements of technology and developments of new medicines, prolonging the lifespan and improving the quality of life, have increased the geriatric population. In years to come, pharmaceutical industries and healthcare systems will persistently work to find solutions to changing demands and new problems of the society. Through the Rho Chi Post, I wish to learn, educate, and prepare myself and others for the future.

RHO CHI POST: TEAM MEMBERS



@ Azia Tariq (5th Year, STJ; Section Editor: News)

The Rho Chi Post is a prominent and highly esteemed resource for pharmacy students and professionals. I am privileged to be a part of the team and hope to contribute informative and engaging pieces to the newsletter.



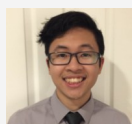
@ Nicollette Pacheco (5th Year, STJ; Staff Editor [Graphics-Focused])

As a new member of the Rho Chi Post team, I have a vast appreciation of what it means to be a future pharmacist in the rapidly evolving world of healthcare. I am looking forward to being on the team as a graphics-focused staff editor, and I hope to bring my passion for science and creativity to the Rho Chi Post.



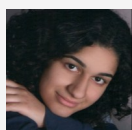
@ Svetlana Akbasheva (6th Year, STJ; Staff Editor [Content-Focused])

I am very excited and honored to be part of the Rho Chi Post! In a profession that is constantly evolving with new developments, it is so important to remain informed and current. The Rho Chi Post helps do just that, and I look forward to contributing to this unique publication.



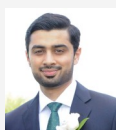
@ Andrew Leong (6th Year, STJ; Staff Writer)

Students have to do more than what is required of us in classes to truly learn about our profession. That's why I joined the Rho Chi Post. This publication represents a channel by which our team members, faculty, and readership can share information - something I believe is important in this ever-changing pharmacy world.



@ Sylva Ohanian (5th Year, STJ; Staff Writer)

The Rho Chi Post is a refreshing outlook on our profession. I am thrilled and grateful to be able to work with the other members in continuing its success, and hopefully to bring greater attention to it, which it deserves.



@ Fawad Piracha (6th Year, STJ; Finance and Outreach Manager)

I am delighted to join the editorial team. I have the firm intention of broadening readership and facilitating growth of the Rho Chi Post.



@ Joshua Bliss (6th Year, STJ; Social Media Manager)

By providing student-organized, reliable healthcare information, the Rho Chi Post helps us all in fulfilling our education both in and out of the classroom. Education is the tool we use to set paths for our futures, and every chance to expand our education is a chance at building a better future. I am honored to be a part of the Rho Chi Post & look forward to the future!



@ You!

We are always looking for creative and motivated students to join our team!
If you are interested in becoming a Rho Chi Post editorial team member, please visit:
<http://rhochistj.org/RhoChiPost/Application>

RHO CHI

The Rho Chi Society encourages and recognizes excellence in intellectual achievement and advocates critical inquiry in all aspects of Pharmacy.

The Society further encourages high standards of conduct and character and fosters fellowship among its members.

The Society seeks universal recognition of its members as lifelong intellectual leaders in Pharmacy, and as a community of scholars, to instill the desire to pursue intellectual excellence and critical inquiry to advance the profession.

THE RHO CHI POST

MISSION

The Rho Chi Post is an award-winning, monthly, electronic, student-operated, faculty-approved publication that aims to promote the pharmacy profession through creativity and effective communication. Our publication is a profound platform for integrating ideas, opinions, and innovations from students, faculty, and administrators.

VISION

The Rho Chi Post aims to become the most exciting and creative student-operated newsletter within St. John's University College of Pharmacy and Health Sciences. Our newsletter continues to be known for its relatable and useful content. Our editorial team continues to be known for its excellence and professionalism. The Rho Chi Post essentially sets the stage for the future of student-operated publications in pharmacy.

VALUES

Opportunity, Teamwork, Respect, Excellence

GOALS

1. To provide the highest quality student-operated newsletter with accurate information
2. To maintain a healthy, respectful, challenging, and rewarding environment for student editors
3. To cultivate sound relationships with other organizations and individuals who are like-minded and involved in like pursuits
4. To have a strong, positive impact on fellow students, faculty, and administrators
5. To contribute ideas and innovations to the Pharmacy profession

CURRENT EXECUTIVE BOARD



Michael, Lina, Julia, Jessica, Davidta, Zachary at the 2015 Induction Ceremony

President: Michael Bosco

Vice President: Lina Lin

Secretary: Jessica Langton

Treasurer: Julia Kamuda

Historian: Davidta Brown

Media Relations Coordinator: Zachary Piracha

Chapter Advisor: S. William Zito, PhD

UPCOMING EVENTS

International Conference on Influenza

August 24-26; West Drayton, UK

European Pharma Congress

August 25-27; Valencia, Spain

Best of ASCO Chicago

August 28-29; Chicago, IL

17th Asia Pacific League of Associations for Rheumatology Congress (APLAR)

September 6-9; Chennai, India

9th Neurodegenerative Conditions Research & Drug Discovery Conference

September 9-10; Philadelphia, PA

World CDx Boston

September 8-11; Boston, MA

CNS Diseases World Summit

September 9-11; Philadelphia, PA