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President's Message

Greetings! It has been a great honor and privilege to serve you for the past 6 months! As I am half way through my presidential term, I would like to take this opportunity once again to express my gratitude for everyone's continuous support! Without your commitment and effort, our chapter will not be the same! I always marvel at many of the great ideas and initiatives that have emerged from our board meetings each month! It truly has been an enriching experience for me!

Fall was an extremely busy season for our chapter with a whirlwind of scheduled fun events. This year, one of my goals is to expand our networking/social events and collaborate with our neighboring chapters (Royal Counties, Westchester, and Long Island chapters). We have had 3-4 networking/continuing education dinner programs every month to ensure all members had the opportunity to participate.

In September and October, we had two combined networking dinner programs with the Royal Counties chapter in Little Italy, Manhattan and Williamsburg, Brooklyn. Both programs were well attended and highly received by members from both chapters. In November, we also had our first combined CE dinner program with the Westchester chapter at Montefiore Medical Center, Bronx, NY. Close to 100 members from both chapters and 8 exhibitors joined us for this 2-hour CE program. This is one of the ways in which we created opportunities to bring members from different chapters together to network and build friendships with each other.

In addition to the CE and networking dinner program, Director-at-Large Dr. Zane Last organized our very first hiking trip with spectacular views over the Hudson River at Breakneck Ridge in early fall. We extended the invitation of the hiking trip to other chapters as well. Twenty-five chapter members and friends from New York City, Royal Counties, and Long Island chapters joined the hike. Did I mention that the New York City chapter flag football team led by our captain, Dr. Karen Berger, also played well for the fall season? Can't wait for the spring season to come for more outdoor activities! What a great way to network and interact with each other outside of the traditional professional meetings!

In order to give back to the community, we also had two volunteering opportunities in the fall. What a rewarding experience from the beautification of Adopt-A-Highway led by Director-at-Large Dr. Charrai Byrd. And it was so awesome to paint staircases and hallways at public school (PS 47X) in the Bronx for New York Cares Day with our members! We are a fun group of artistic and creative health care professionals!

Moreover, we also received close to $20,000 in grants for continuing education programs this fall. Kudos to our Grant Committee under the leadership of Dr. Evangelina Berrios-Colon. This year we also expanded our Grant Committee so we can engage in more grant writing opportunities in order to prepare for 2016 and 2017 continuing education programs.

What great team work! So stay tuned for more fun events and new ideas to come for 2016. I am so excited and looking forward to an awesome 2016 as we continue to further our pharmacy profession! So come join us; let's do this!!!

Best,

Yi Guo, PharmD, President, NYCSHP, 2015-2016

President-elect's Message

I hope you all enjoyed the holidays with family and friends! Although we are just a few weeks into 2016, I have already started planning the monthly CE programs for the society and Nikki Bhogal has been hard at work organizing networking dinner programs. In the past few years, pharmaceutical companies have been facing financial and regulatory burdens with supporting our society's programs; however, we have still been able to work with them in new ways to continue to provide continuing education. I would also like to acknowledge Eva Berrios-Colon and Kanika Ballani-Lala, co-chairs of the Grant Committee, who have been working diligently to submit grant applications for three CE programs this year. We all hope to continue to provide educational programs that you find pertinent to your practice.

- January 21st (CE Program)
  - Topic: Clinical Adoption of Pharmacogenomics and its Implications for Clinical Pharmacists
- January 26th (Networking Program)
  - Topic: Highlights in Management of Heart Failure with Reduced EF and Sacubitril/Valsartan (Entresto®) Prescribing Information
- February 18th (Networking Program)
- February 25th (CE Program)
- March 15th (Networking Program)
- March 22nd (CE Program)
- April 6th (Networking Program)
- May 12th ((Networking Program)

These meetings are an excellent venue for networking and sharing best practices with others. I look forward to seeing you all at our meetings! If you have any CE topic ideas or would like to present a CE at a meeting, please feel free to email me at Jason.babby@mountsinai.org.

Thank you,

Jason Babby, PharmD, BCPS, President-Elect, NYCSHP

All Star Rotation Team: Students, Residents, Practitioners—all on one service!

Nicholas Jandovitz, PharmD, PGY-2 Solid Organ Transplant Pharmacy Resident, New York-Presbyterian Hospital
Jonathan Sin, PharmD, PGY-1 Pharmacy Resident, New York-Presbyterian Hospital
Demetra Tsapepas, PharmD, Clinical Pharmacy Manager, Solid Organ Transplantation, New York-Presbyterian Hospital

Academic medical centers are in a unique position to offer essential resources necessary to train and develop pharmacy learners: pharmacy students, residents, and even practicing clinical pharmacy specialists. The collaboration of pharmacy learners during various experiences allows each individual to build upon their pharmacotherapy knowledge and critical thinking skills. The pharmacy team is led by the clinical pharmacy specialist who oversees the guidance and progression of the experience for all learners. A post graduate year-2 (PGY-2) specialty resident also plays a major role in teaching and helping to train the post graduate year-1 (PGY-1) resident and pharmacy student. In addition, the PGY-1 gains experience being a co-preceptor for the student and explores various aspects of clinical practice. Finally, the student is able to integrate didactic education into daily clinical practice.

The various facets of learning experiences and the interplay of each pharmacy learner during a solid organ transplant rotation at a large, urban, academic medical center in New York City are described below.

Patient Care

- Pharmacy learners are trained to deliver a variety of clinical pharmacy services as part of the multi-disciplinary care team, including medication reconciliation, pharmacotherapeutic and pharmacokinetic drug monitoring, medication teaching, and documentation in the medical record.

Patient Education

- As medication experts, pharmacy learners are able to properly educate patients on drug therapy, adverse effects, drug-drug and drug-food interactions, proper administration, and the importance of adhering to their prescribed regimens.

Presentations

- Pharmacy learners are able to share their medication expertise through formal and informal presentations to other members of multi-disciplinary care teams.

Career Development

- The field of pharmacy is versatile and there are an increasing number of sub-specialties in which pharmacists can practice. An important aspect of career development involves membership in professional organizations for networking and continuing education, as well as education opportunities to explore new interests.

Feedback

- Feedback is important to all pharmacy learners for personal and professional growth. It should be incorporated into every pharmacy practice experience and involve open and honest discussion.

Activity-Interplay of Pharmacy Learners				
	Student	**PGY-1**	**PGY-2**	**Clinical Pharmacy Specialist**
Patient Care	• Follow one patient initially and increase as rotation progresses • Review recommendations with PGY-1 and PGY-2 residents • Make recommendations to the medical team	• Follow half of the patients on service • Build clinical knowledge of immunosuppression and transplantation • Develop teaching style by precepting students	• Follow all patients on service • Precept a PGY-1 resident and a student to build clinical knowledge • Discuss patients with pharmacy learner group before acute care rounds and review interventions	• Facilitate integration of other pharmacy learners into the multidisciplinary team • Serve as a role model and provide guidance throughout rotation
Patient Education	• Shadow PGY-1 resident to gain experience and exposure • Participate in a group patient education class with PGY-1 and PGY-2 residents	• Shadow PGY-2 to gain experience • Lead a group patient education class with PGY-2 • Educate individual patients	• Introduce patient handouts to a PGY-1 and a student • Organize mock education sessions to assess competency of PGY-1 and student	• Facilitate training process for effective patient education
Presentations	• Present patients to pharmacy learner group • Provide an in-service to the multi-disciplinary care team with supplemental handout	• Present patients to pharmacy learner group • Serve as the primary preceptor for student presentations • Provide an in-service to the multi-disciplinary care team with supplemental handout	• Serve as the primary preceptor for PGY-1 resident presentations • Provide an in-service to the team with supplemental handout	• Lectures to practitioners and trainees to share clinical knowledge
Career Development	• Lead discussions on how to increase involvement in local, state, and national pharmacy organizations	• Lead discussion on professional networking	• Lead discussion on process and benefit of board certification	• Lead discussion on how to build collaborative relationships with other institutional departments
Feedback	• Provide feedback on teaching style to the PGY-1 resident • Provide secondary feedback on teaching style to the PGY-2 resident and the specialist • Receive input on performance from senior members of the pharmacy team	• Provide feedback on performance to the student • Provide feedback on teaching style and the overall experience to the PGY-2 and the specialist • Receive input on performance from senior members of the pharmacy team	• Provide feedback on performance to the student and PGY-1 • Provide feedback on teaching style and overall experience to the specialist • Receive input on performance from senior members of the pharmacy team	• Oversee all evaluations • Receive feedback from all pharmacy learners and integrate into practice for the next learning cycle

2015 Lobby Day: Student Reflection

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Preceptor: Evangelina Berrios-Colon, PharmD, MPH, BCPS, CACP

With Pharmacy Lobby Day approaching, it is important to advocate for our profession as pharmacists. On numerous occasions, I have heard of pharmacy evolving and shaping into a multifaceted profession. Laws are an essential part of our career and as pharmacists; we are responsible for knowing and following all federal and state laws in our place of practice. However, pharmacists and pharmacy students are not only responsible for abiding by the laws, but are also co-creators of these laws.

Recognizing the importance of pharmacy laws and the role they play in pharmacists' daily practice, I wanted to make a difference by participating in last year's Pharmacy Lobby Day, which took place in Albany, New York on April 21, 2015. I did not know what to expect, but I believed it would be an educational and productive experience. This event included students and faculty from seven New York State Pharmacy Schools: Albany College of Pharmacy, University of Buffalo, Long Island University, Touro College of Pharmacy, St. John's University, Saint John Fisher College, and D'Youville College.

Pharmacy Lobby Day is held annually by the Pharmacists Society of the State of New York (PSSNY). Students from different schools and regions are divided into groups and are led by pharmacists who are fully versed on the issues discussed. Students were provided a detailed agenda and key talking points on legislative matters in advance.

The two key bills lobbied for that day were Immunization Expansion and Reform A-123 Paulin/S-4739 Hannon and Collaborative Drug Therapy Management (CDTM) A-5805 McDonald. The Immunization Expansion and Reform bill focused on granting pharmacists the authority to become permanent administrators of vaccines to adults 18 years and older and adding Tdap vaccine into the already existing list of vaccines which includes influenza, pneumococcal, meningococcal, and shingles. This bill also advocated for making the vaccination process for pharmacists easier by removing county restrictions for non-patient specific orders and permitting either patient-specific or non-patient specific authorization for all five vaccines. It also included removing the mandatory annual Department of Health (DOH) survey and providing the DOH Commissioner with the authority to issue statewide standing orders in case of disease outbreaks.

The CDTM bill advocated for lifting the restrictions that limit current CDTM practice to teaching hospitals. In addition, the bill would grant the Board of Pharmacy the authority to provide CDTM certification and would make it a requirement for practicing pharmacists. During our visit to the assemblymen's offices, we were able to reinforce the importance of passing these bills and request their support. It was a wonderful opportunity to educate the governor and assemblymen on pharmacists' roles in healthcare and lobby for the expansion of the profession.

As a result of our efforts, Governor Cuomo signed the Immunization Expansion S-4739-A Hannon bill into law, which is now Chapter 46 of the Laws of 2015. Senator LaValle submitted a SAME AS S-04857-A bill to the McDonald CDTM bill. One of the assemblymen that my group had the pleasure of meeting that day was

Andrew R. Garbarino from District 7. He seemed very interested in learning about the bills and obtaining more information, and in fact became a co-sponsor for that bill. Then on September 14, 2015, Governor Cuomo signed the CDTM S-4857-A LaValle bill into law, which became Chapter .238 of the Laws of 2015. There are further expansions and improvements for pharmacy practice, which pharmacists could lobby for during the next Pharmacy Lobby Day on April 12, 2016 in Albany, New York. I truly encourage everyone to experience this event first hand because remarkable achievements can be produced though the collaborative work of our professional group. By working together, we can accomplish unlimited possibilities that can positively impact the health of our patients.

Picture: Lobby Day with Assemblyman Garbarino

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Simplifying Chronic Kidney Disease

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Preceptor: Keith T. Veltri, BPharm, PharmD

Background

Chronic kidney disease (CKD) is a current public health problem due to the silent nature of the disease. Patients in early stages of the disease tend to be asymptomatic and diagnosed only through routine blood and urine tests. It is not until later stages that patients develop symptoms. In fact, it is estimated that more than 10% of adults in the US have CKD[1]. Due to the increased costs of CKD and end-stage renal disease (ESRD), timely recognition of this easily identifiable disease is essential in delaying the progression of the disease, preventing complications, and reducing healthcare costs. The purpose of this summary is to provide a basic clinical review of CKD.

Definition

CKD is defined as either:

- Structural or functional kidney damage for ≥ 3 months regardless of glomerular filtration rate (GFR)[2,4]

OR

- All individuals with a GFR < 60 mL/min/ 1.73m^2 for ≥ 3 months regardless of kidney damage* [2,4]

[*] GFR < 60 mL/min/ 1.73^2 is less than half the normal value in young adults, which is approximately 125 mL/min/ 1.73^2

Table 1 outlines structural and functional kidney damage

Table 1: Structural vs. Functional Kidney Damage [3,4]

Structural Damage	Functional Damage
• Albuminuria > 30 mg/day • Abnormalities in urine sediment • Electrolyte and other abnormalities due to tubular disorders • Damage detected by imaging History of kidney transplantation	• Decreased GFR

Classification of CKD

CKD is classified based on the cause of kidney disease, GFR category, and albuminuria category (CGA) according to the **2012 Kidney Disease: Improving Global Outcomes (KDIGO)** clinical guidelines[2]. It can also be classified according to the **Kidney Disease Outcomes Quality Initiative (KDOQI)** clinical guidelines[4]. **Table 2** outlines the classifications according to both guidelines (KDIGO and KDOQI) based on GFR.

Table 2: Classification of CKD [2,4]

GFR (mL/min/1.73m^2)	KDOQI Stage	KDIGO Category	Terms
> 90	Stage 1	G1	Normal or high
60 – 89	Stage 2	G2	Mildly decreased
45 – 59	Stage 3	G3a	Mildly to moderately decreased
30 – 44		G3b	Moderately to severely decreased
15 – 29	Stage 4	G4	Severely decreased
< 15	Stage 5 **OR** ESRD if requiring dialysis	G5	Kidney failure

Etiology

Diabetes mellitus (DM) is the leading cause of CKD and ESRD in the United States. Hypertension and glomerulonephritis are the second and third leading causes of ESRD.

Complications of CKD

Damage to the kidneys prevents proper filtration of blood and leads to accumulation of waste products. Complications of CKD contribute to significant morbidity and mortality and affect all organ systems.
The most common complications of CKD are the following:

1. **Microalbuminuria**
2. **Anemia of chronic kidney disease**
3. **Bone mineral disorders**
4. **Metabolic acidosis**

Microalbuminuria

Microalbuminuria is defined as urine albumin levels of 30-300 mg in a 24-hour urine collection[4]. Structural damage to the glomerular membrane causes protein to abnormally leak into the urine. The first protein to leak into the urine is albumin due to its size and negative charge and indicates early signs of kidney damage[5]. The presence of microalbuminuria is therefore a sensitive test in detecting early kidney damage[5]. Patients with microalbuminuria should be started on an angiotensin-converting enzyme inhibitor (ACE-I) or angiotensin receptor blocker (ARB) as first-line therapy, given that these agents lower glomerular capillary blood pressure and protein filtration[4]. Hyperkalemia and slight worsening of renal function within the first weeks may occur with these agents and need to be monitored during therapy. Patients may be normotensive during initiation of ACE-I or ARB and therefore BP must also be monitored as hypotension may occur. According to the 2014 Eighth Joint National Committee (JNC-8) evidence-based guideline, goal blood pressure is < 140/90 mmHg in all patients with CKD regardless of age and with or without diabetes[8].

Anemia of Chronic Kidney Disease

Erythropoietin (EPO) is an essential hormone produced by the kidneys in response to low hemoglobin levels. Damage to the kidneys and reduction in kidney function leads to reduced production of erythropoietin, contributing to low hemoglobin levels and shortened erythrocyte life span. Anemia of chronic kidney disease is considered normocytic, normochromic anemia with a normal ferritin and TSAT [5]. It is diagnosed in adults and children > 15 years old with CKD when the hemoglobin (Hgb) concentration is < 13 g/dL in males or < 12 g/dL in females[2,4].
Anemia is associated with higher rates of hospitalization, cardiovascular disease, and mortality[4]. Therefore, it is essential that CKD patients be screened annually (Stage 3), at least twice a year in severe CKD cases (Stage 4 and 5), and at least every three months for patients on dialysis[2,6]. Treatment of anemia in CKD patients involves erythropoietin stimulating agents (ESAs) such as *darbepoeitin alfa (Aranesp)* and *erythropoietin alfa (Epogen),* along with iron therapy to ensure adequate response to ESA. It is also important to rule out other causes of anemia. The following labs must be performed prior to initiating ESA: CBC, stool for occult blood, iron studies, and folate and vitamin B12 levels[2,6]. ESA therapy is initiated when hemoglobin is < 10 g/dL. The goal of therapy is to maintain hemoglobin levels between 10-12 g/dL. Hemoglobin levels > 13g/dL have shown to increase cardiovascular mortality[4-6]. As a result, ESA therapy should be individualized and risk versus benefit must be assessed.

Bone Mineral Disorders

Changes in phosphate and calcium homeostasis occur in the early stages of CKD and progress further in the late stages. Patients with CKD often present with **hyperphosphatemia, hyperparathyroidism**, and **hypocalcemia** due to decline in kidney function[4].

Hyperphosphatemia

Phosphate absorption is regulated by the parathyroid hormone (PTH) and is excreted by the kidneys. In CKD patients, kidney function decline causes excess phosphate to be accumulated in the blood (hyperphosphatemia). In order to compensate for the excess phosphate, the parathyroid secretes PTH which acts to block phosphate reabsorption in the kidneys, leading to increased excretion of phosphorus. Normal phosphate level is between 2.5–4.5 mg/dL. However, in hyperphosphatemia, total serum calcium-phosphorus (Ca-P) products should be maintained at < 55 mg^2/dL^2 as this prevents metastatic calcification of joints and vesicles seen in uncontrolled hyperphosphatemia[7]. Treatment for hyperparathyroidism includes both non-pharmacological therapy (diet restriction, dialysis) and pharmacological therapy (phosphate binders). Two categories of phosphate binders are available: calcium-based phosphate binders such as *calcium carbonate* and *calcium acetate (PhosLo)* and non-calcium, non-aluminum, and non-magnesium phosphate binders such as *sevelamer HCl (Renagel), sevelamer carbonate (Renvela), and lanthanum carbonate (Fosrenol)*. Goal target levels for phosphate are listed in **Table 3** according to the National Kidney Foundation[7].

Hyperparathyroidism

PTH is an essential hormone which maintains the following functions in the kidney: 1) increases calcium reabsorption, 2) increases phosphorus excretion, and 3) activates 1-hydroxylase which converts vitamin D to its active form, 1-25 hydroxyl vitamin D. Along with its effects on the kidneys, PTH also causes bones to release calcium and increase activation and proliferation of osteoclasts. Secondary hyperparathyroidism is a compensatory mechanism which increases PTH production in response to hypocalcemia and hyperphosphatemia, as seen in CKD patients[5]. Adequate levels of vitamin D and calcium can prevent increased PTH production. Calcimimetics such as *cinacalcet (Sensipar)* decrease PTH production by increasing calcium sensitivity and are reserved for patients in ESRD with serum calcium levels > 8.4 mg/dL. Hyperparathyroidism can be due to either secondary hyperparathyroidism or vitamin D deficiency. Serum vitamin D levels should be measured prior to initiation of phosphate binders. Patients who present with normal vitamin D levels (> 30 ng/mL) are recommended to have annual screenings. Vitamin D supplementation is necessary when serum vitamin D levels are < 30 ng/mL[7]. Vitamin D supplementation causes an increase in calcium and phosphate levels and it is therefore essential that calcium and phosphate levels are at goal prior to therapy. If the calcium levels exceed 10.2 mg/dL during therapy, vitamin D therapy and use of calcium-based phosphate binders should be stopped[7].

Hypocalcemia

Declining renal function in CKD patients leads to hypocalcemia due to the kidneys' inability to reabsorb calcium. Total elemental calcium intake (both dietary and calcium-based phosphate binders) should not exceed 2,000 mg/day [7]. If a patient experiences clinical symptoms of hypocalcemia such as paresthesia, bronchospasm, tetany, or seizures and has a plasma intact PTH level above the target range **(see Table 3)**, therapy should be initiated[7]. Hypocalcemia treatment includes calcium salts, such as *calcium carbonate* and/or vitamin D sterols[7]. Target goals of calcium depend on the stage of CKD and are listed in **Table 3**. Calcium in serum is bound to albumin. Low levels of albumin (< 4 g/dL) may not accurately reflect free calcium and the **corrected calcium equation** must be used.

Corrected Calcium = Serum calcium + 0.8 (4 – Serum albumin*)
* if serum albumin is < 4 g/dL

Metabolic Acidosis

Patients with CKD should have their serum levels of CO_2 measured. According to the National Kidney Foundation, CO_2 levels should be monitored every 12 months (Stage 3), every 3 months (Stage 4 and 5), or every month during dialysis. Target CO_2 total for these patients is **22 mEq/L**. If levels drop below 22 mEq/L, supplemental alkali salts such as sodium bicarbonate should be considered[7].

Prevention of CKD

Understanding the risk factors of CKD is important in preventing the progression of the disease. Regular screening of high-risk groups (patients with diabetes, hypertension, family history of kidney disease, age > 60 years, ethnic minorities, presence of hematuria, and proteinuria) can help in early detection and initiation of interventions to prevent and delay complications[2,4].

Table 3: Goal Target Levels for Bone Mineral Disorders [7]

CKD Stage	Phosphorus (mg/dL)	Intact PTH (pg/mL)	Corrected Calcium (mg/dL)	Ca- P Product (mg^2/dL^2)
3	2.7 – 4.6	35 – 70	"Normal"	< 55
4	2.7 – 4.6	70 – 110	"Normal"	< 55
5 or dialysis	3.5 – 5.5	150 – 300	8.4 – 9.5	< 55

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The Risks and Benefits of Pioglitazone Therapy: From Type 2 Diabetes to Alzheimer's Disease

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Preceptor: Bardia Askari, Ph.D. Touro College of Pharmacy

Pioglitazone (PIO) is a peroxisome proliferator-activated receptor-gamma (PPARγ) agonist currently used in the treatment of Type 2 Diabetes Mellitus (T2DM). It is classified as a thiazolidinedione (TZD) and works as an insulin sensitizer by increasing glucose utilization and decreasing its production.[1] In a recent article published by the *Journal of American Medical Association*, a study was conducted to determine whether PIO increases the risk for bladder cancer. This posed concerns in patients who were currently taking PIO for treatment of T2DM. The incidence of bladder cancer per 100,000 person-years was 89.8 for those on PIO and 75.9 for those not on PIO.[2] Interestingly, studies have also demonstrated the potential benefit of using PIO in Alzheimer's disease (AD), a neurodegenerative disorder affecting millions of Americans worldwide. A major risk factor of AD is increasing age, although it may occur in younger adults as well.[3] The causes of AD are not clear, but symptoms include loss of memory, cognition, and changes in behavior (Fig 1).

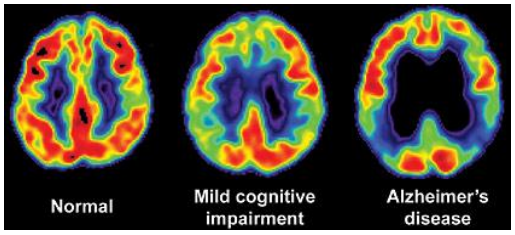
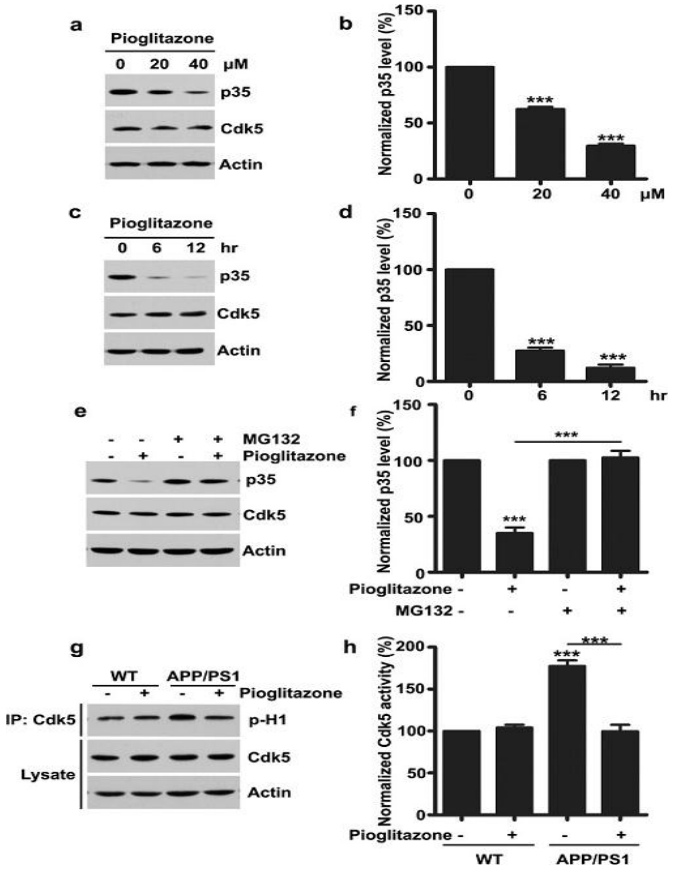


Figure 1: The progression to Alzheimer's disease
http://www.berkeley.edu/news/media/releases/2009/07/14_alzheimers.shtml. Accessed October 25, 2015.

There is a growing body of evidence relating the development of AD as a result of insulin resistance[4], a relationship that may have great implications in drug therapy. As an insulin-sensitizing agent, PIO may alleviate synaptic dysfunctions in patients suffering from AD. In a recent study, mice were used as models to indicate whether PIO is able to inhibit cyclin-dependent kinase 5 (CDK5) activity, an important pathological marker in AD development.[5] Results demonstrated beta amyloid-induced dendritic spine loss mediated by CDK5 hyperactivation, contributing to the severity and progression of the disease[4]. In people with AD, this triggered a neuroinflammatory response causing damage to cell signals and increased production of proinflammatory agents. PIO suppression of CDK5 hyperactivation in the APP/PS1 mutant mouse hippocampus led to decreased p35 (neuronal-specific activator of CDK5) protein levels.[5] Therefore, PIO may reverse the long-term potentiation deficits and improve impaired spatial memory in mice models of AD, implicating an exciting possibility that the anti-diabetes drug PIO can be a promising regimen for AD mitigation (Fig 2).

Figure 2: Pioglitazone inhibits CDK5 activity by reducing p35 level



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Public Relations	Kara Kubli, Jessica Snead	
Special Projects	Demetra Tsapepas, Daryl Glick, Jason Chan	
Student Relations	Christopher Mendoza, Mehwish Mahmood, Sefa Kploanyi, Karissa Johnson	
Legislative Affairs	Leanne Svoboda, Mitch Dorn, Peter Hoang, Sefa Kploanyi	
Supportive Personnel	Mitch Dorn	
Installation Program	Alla Khaytin, George Bugayenko, Sasha Libman	
Industry Relations	Michelle Lee, Benjamin Georgiades, Sefa Kploanyi, Vita Rozenfeld	
Grant Writing	Evangelina Berrios-Colon, Kanika Ballani	
Social Media Coordinator	Elsie Wong	
Historian	Jamie Chin	
Community Outreach	Marie DiMicco	
Residents Collaborative	Loriel Solodokin, Jamie Chin	
Membership	Priyasha Uppal, Jamee Maase, Jordan Keen, Karin Koseck, Iryna Poktylyuk	
New York State Council of Health-System Pharmacist State Representatives		
State Liaisons	Monica Mehta, Joe Pinto, Philip Manning, Vincent Giambanco	
Faculty Liaisons		
Touro College of Pharmacy: Mary Choy Mary.Choy@touro.edu	St. Johns University: Khusbu Patel Khusbu.Patel@va.gov	Long Island University: Antony Pham Antony.Pham@liu.edu

Beautification Adopt-the-Highway: September 13, 2015

Breakneck Ridge Hiking-with 25 Members and Friends from NYC, Royals, Long Island Chapters: September 27, 2015

1st Combined Networking Dinner Program by NYC and Royal Chapters-Il Cortile, Little Italy, NY: September 22, 2015

NYSCHP Downstate Critical Care Program: October 2, 2015

2nd Combined Networking Dinner Program by NYC and Royal Chapters, at Wythe Hotel, Brooklyn, NY: October 13, 2015

NYCSHP Networking Dinner Program at Water Club: October 8, 2015

New York Cares Day at PS 47X, Bronx, NY: October 17, 2015

NYCSHP on NBC Today's Show: October 21, 2015

NYCSHP October CE Dinner Program, Heartland Brewery and Chophouse, Times Square: October 29, 2015

Student and Resident Workshop: November 3, 2015

First November CE Dinner Program: November 22, 2015

Brasserie 8 ½ NYCSHP Networking Dinner Program: November 17, 2015

Combined CE Dinner Program by NYC and Westchester Chapters: November 12, 2015

BOD Appreciation Holiday Dinner