JANUARY/FEBRUARY 2008

GULF COAST SOCIETY OF HEALTH-SYSTEM PHARMACISTS
SPECIAL EDITION

ANNUAL SEMINAR
MOODY GARDENS
GALVESTON, TX FEBRUARY 23, 2008

Inside this issue:

PRESIDENT'S REPORT

Ogechi Eshleman, PharmD
President-GCSHP

Happy New Year!

Since we last spoke, The GCSHP Mentor Program has been featured in the Winter issue of the TSHP Journal in the Best Practices Section.

I caught up with some program mentors to get their feedback on the status of the program. Rodney Hunter, MD Anderson Cancer Center Fellow states that "everything is going really well" with his student. "She has been and is still eager to know exactly what I do."

Says Sylvester Agbahiwe, Pharmacy Practice Resident at the VA, "The program has allowed me to give back, not just to the school where I got my degree, but to everyone who has helped me along the way. I could not be happier."

Thank you to all GCSHP mentors for contributing towards the success of the program.

I'll see you February 23rd at the Annual Seminar !

Until then, be well!

Ogechi

ANNOUNCEMENTS

Technician Section

"Under Construction"

GCSHP would like to add a

-Technician Section-

to the newsletter

If you are a technician and interested in contributing towards the Technician Section of the newsletter, please email topics and/or submissions to the editor at

Monica_Robinson@hchd.tmc.edu

Calendar of Events

- **January 1—Happy New Year**
- **January 17—Residency Mentoring Social**
- **January 21—MLK Day**
- **February 3—National Wear Red Day**
- **February 14—Happy Valentine's Day**
- **February 23—GCSHP Annual Seminar—Moody Gardens, Galveston, Texas**

JANUARY 2008

Sun	Mon	Tue	Wed	Thu	Fri	Sat
		1	2	3	4	5
6	7	8	9	10	11	12
13	14	15	16	17	18	19
20	21	22	23	24	25	26
27	28	29	30	31		

FEBRUARY 2008

Sun	Mon	Tue	Wed	Thu	Fri	Sat
					1	2
3	4	5	6	7	8	9
10	11	12	13	14	15	16
17	18	19	20	21	22	23
24	25	26	27	28	29	

Annual Seminar Registration Information

Advanced Registration Deadline—February 11, 2008

Registration Fees:

GCSHP Pharmacist Members $35.00

Non-GCSHP Pharmacist Members or onsite registration $50.00

GCSHP Student & Technician Members $15.00

Non-GCSHP Student & Technician or onsite registration $20.00

Hotel rooms have been blocked off for Friday, February 22nd.

Reservation deadline is February 1st.. For reservations call 1-888-388-8484. Room Rate: $135.00 + tax

Gulf Coast Society of Health-System Pharmacists

Annual Seminar

Moody Gardens
Galveston, Texas
February 23, 2008

Breakfast
7:15-8:00 am

Opening remarks
8:00-8:10 am

Top Five Significant Infectious Disease Issues for 2008
Richard J. Hamill, MD
Baylor College of Medicine
8:10-9:00 am

Questions
9:00-9:10 am

Break / Time to review resident posters and pharmaceutical exhibits
9:10-9:30 am

Pain Management Control in the Cancer Patient
Allen W. Burton, MD
UT MD Anderson Cancer Center
9:30-10:20 am

Questions
10:20-10:30 am

Break / Time to review resident posters and pharmaceutical exhibits
10:30-10:50 am

Optimizing the Safe Use of Anticoagulants:
The New National Patient Safety Goals on Anticoagulation
William Dager, PharmD
University of California Davis Medical Center

10:50-11:40 am

Questions
11:40-11:50 am

Lunch
12:00-1:00 pm

For additional information please contact:
Dr. Richard Cadle or Dr. Eddie Lee
Email: cadle.richardmark@va.gov OR
lelee@utmb.edu

STUDENT SECTION

TEXAS SOUTHERN UNIVERSITY SSHP
PAMELA DYER, PRESIDENT

TSU-SSHP reflects positively on the events and activities that have occurred during the fall semester. The month of August contained informative and educational events: the first year professional pharmacy student orientation and a brown bag community service event at Acres Homes Clinic Annual Health Fair. In September, to increase participation and awareness in professional organizations, SSHP and SNPhA hosted a social for the class of 2011.

During National Pharmacy Month, TSU-SSHP joined the University of Houston chapter in hosting the 2nd Annual Fall Seminar. The event featured an Italian themed buffet dinner as well as presentations regarding residencies, interview skills, and curriculum vitae preparation. The guest speakers were TSU-COPHS alumni,

Drs. Monica Robinson Green, Desiree Dunlop, & Michael Piñón. The event also included recognition of the GCSHP Mentor Program.

The chapter also conducted its local Clinical Skills Competition, which provided students the opportunity to compete at the national competition and sharpen their clinical knowledge. Fourth year professionals students, Patrick Starks and Ralph Bunch, were selected as the winners of the local competition and competed at the ASHP Midyear Clinical Meeting. We are extremely proud of the effort they put forth at the national competition. Also, members attended a CE dinner sponsored by ASHP Advantage that featured Dr. John Fanikos. To conclude the month, Dr. Patti Peyman, from Memorial Herman Hospital, spoke to the students about residency program preparation and opportunities.

In November, Vijaya Henry, M.S, R.Ph, from Pfizer Inc. Global Research & Development gave a presentation to the students regarding pharmacist opportunities in the pharmaceutical industry.

Forty-three students attended 2007 ASHP Midyear Clinical Meeting. Allyson Davis, Pamela Dyer, Dwayne Moore, Shalondria Simpson, and Jacque Washington presented posters. In addition, Pamela Dyer was presented with a Special Recognition Award from the Association of Black Health System Pharmacists (ABHP) for her achievement and involvement in the pharmacy profession.

TSU Students: Brian Mitchell, Candice Wiggins, Janae Williams, Adora Obiechina, Carlos Conway

In conclusion, the Texas Southern University chapter of the Student Society of Health System Pharmacists is pleased with its accomplishments thus far and anticipates the spring semester to emulate and exceed the fall semester.

ASHP- Texas Reception—TSU Alumni & Students

ASHP Midyear Clinical Meeting Las Vegas, Nevada

TSU Students: Carissa Mathis, Allyson Davis, Brian Mitchell, Shalondria Simpson, Janelle Harris, Ekaette Ebong

Jasper W. Watkins III ABHP President & Pamela Dyer (TSU)

Pamela Dyer (TSU), Jack Iskander (Xavier University), Ann Marie Prazak (UH)

UH Students: Constance Reyes, Ann Marie Prazak, James Tyler

STUDENT SECTION

UNIVERSITY OF HOUSTON SSHP
ANN MARIE PRAZAK, PRESIDENT

During the Fall 2007 semester, the UH SSHP chapter held meetings on both the UH main campus (P1 and P2 students) and at the Texas Medical Center (TMC) campus (P3 and P4 students). We would like to extend a special "Thank you!" to the following speakers for taking time from their busy schedules to share information about their areas of expertise:

- Hospital Pharmacy- Thuy Huynh, Pharm.D., The Methodist Hospital
- Pediatrics- Karen Severson, Pharm.D., Pediatric Clinical Specialist, Children's Memorial Hermann Hospital
- Medical Cardiology and Heart Failure- Stephen Michaud, RPh, St. Luke's Episcopal Hospital
- ASHP Involvement- Andy Laegeler- ASHP Pharmacy Student Forum Executive Committee
- Michael E. DeBakey Veterans Affairs Medical Center Residency Program- Martin Ziska, Pharm.D., Lara S. Picard, Pharm.D., and Sylvester Agbahiwe, Pharm.D., PGY1 residents
- Kelsey-Seybold Residency Program- Denise Martinez, Pharm. D., R.Ph. Administrator of Clinical Services; Amera Soliman, Pharm.D. and Natasha M. S. Jackson, Pharm.D., PGY1 residents
- UTMB-Galveston Residency Program- Eddie Lee, Pharm.D., BCPS, Residency Program Director; Denisse I. Martinez, Pharm.D. and Jigna A. Patel, Pharm.D., PGY1 residents
- The Methodist Hospital Residency Program- Sharla Tajchman, Pharm.D. and Sarah Rodriguez, Pharm.D., PGY1 residents
- The University of Texas MD Anderson Cancer Center Residency Program- Brian Dee, Pharm.D., PGY2 resident

During the fall semester, we participated in numerous service activities. On October 6, 2007 we joined the University of Houston College of Pharmacy Team in the Susan G. Komen Race for the Cure. Next, on October 23, 2007, SSHP members provided osteoporosis screenings, healthy bone counseling, blood pressure measurements, and drug information at The Methodist Hospital in the TMC. The event was arranged to celebrate and promote our profession during National Hospital & Health-System Pharmacy Week. Finally, from November 1 – December 1, 2007, we held our annual shoe and sock drive, where we collected new and gently used items from the College of Pharmacy students for the Houston Outreach Medicine, Education, and Social Services (H.O.M.E.S.) clinic.

Approximately 30 teams participated in our local Clinical Skills Competition on October 19, 2007. The finalists were Jason Lovero & Jon Arends, Aimee Hammerstrom & Mike George, Dhara Shah & Rina Patel, Ann Marie Prazak & James Tyler, and Saba Syed & Jaffar Syed. Congratulations to Ann Marie Prazak and James Tyler, who represented our chapter at the National Clinical Skills Competition during the ASHP Midyear Clinical Meeting in Las Vegas, NV, on December 1, 2007. In addition, we sincerely appreciate the diligent efforts of Simi Bassett (P2) for organizing a workshop to teach P1 and P2s about the competition and for promoting the event.

On November 9, 2007, we had a remarkable turn-out of students and residency programs at the UH College of Pharmacy Residency Showcase that we hosted at the UH Pharmacy Building in the TMC. Thank you to all of the local and out-of-town programs who participated and to our advisors, Drs. Kimberly Birtcher and Steven Pass, and Aimee Hammerstrom (P3), for organizing such a successful event!

In December, 55 students attended the ASHP Midyear Clinical Meeting. SSHP members Van-Anh Le (P4) and Cheryl Holt (P3) presented posters on research they conducted during the past year. In addition, our UH SSHP chapter presented a poster at the ASHP Student Society Showcase. The following students are members of the ASHP Student Forum Advisory Groups for 2007-2008: Meetings and Programming Advisory Group- Daniel Ortiz (P1), Erin Ressler (P2), and Elaine DePrang (P4); Policy and Legislative Affairs Advisory Group- Simi Bassett (P2); Student Society and Leadership Development Advisory Group- Ann Marie Prazak (P3) and Tri Nguyen (P3); and Communications Advisory Group- Aimee Hammerstrom (P3).

We will kick off the spring semester with the 3rd Annual Residency Mentoring Social on January 17, 2008 from 5 – 8 PM at the UH Alumni Center. This is a joint event with Texas Southern University that is sponsored by Cardinal Health and GCSHP. We will also host meetings on infectious diseases, nuclear pharmacy, hospital administration, and investigational new drug programs, and continue holding our monthly journal club on the TMC campus and promote Poison Prevention Week and World Asthma Day as our service projects.

Good luck to all of the P4s who are applying for residencies and to everyone for a successful semester!

2nd Annual SSHP Fall Seminar October 2007

STUDENT SECTION

Clindamycin Phosphate 2% Single-Dose Vaginal Cream for the Treatment of Bacterial Vaginosis in Non-Pregnant Women

Van-Anh Le, Pharm.D. Candidate 2008

University of Houston College of Pharmacy

Bacterial Vaginosis (BV) is a vaginal discharge disease characterized by multiple bacterial overgrowths, which displace the normal bacterial flora in the vagina.[1] The usual predominant bacterium, *Lactobacillus sp.* is replaced by anaerobic bacteria such as *Prevotella sp.* and *Mobiluncus sp.*, *Gardnerella vaginalis*, and *Mycoplasma hominis*.[1] About 29.2% of women in the US develop BV, and its prevalence is associated with multiple sex partners, new sex partners, and douching.[1] However, non-sexually active women can experience BV due to a hormonal imbalance in the body or a lack of adequate vaginal lactobacilli. About 50% of patients with BV are asymptomatic and treatment is not recommended.[1] On the other hand, symptomatic patients should be treated in order to minimize the risk of HIV transmission, other STDs, and complications associated with abortion and hysterectomy. Diagnosis for BV is a positive three out of four in the Amsel criteria, which includes: thin, white, homogeneous discharge around the vaginal walls; vaginal pH > 4.5; a fishy odor from the vaginal discharge with or without addition of 10% potassium hydroxide; and the presence of >20% of clue cells.[2]

According to the 2006 CDC guidelines for the management of BV, metronidazole taken orally or vaginally or clindamycin vaginal cream is the treatment of choice.[2] Clindamycin phosphate 2% once a day vaginal cream was FDA approved in 2004 for the treatment of BV. Vaginal application avoided complications of systemic adverse effects, such as nausea and taste alteration as compare to oral medications.

Stein et al showed that clindamycin phosphate 2% vaginal cream had a cure rate of 77% as apposed to 25% for placebo in patients with BV ($P < 0.001$).[3] When Ferris et al compared oral metronidazole, metronidazole vaginal gel, and clindamycin vaginal cream, there was not a statistically significant difference in cure rates among the three groups ($P = 0.548$).[4] Vulvovaginal candidiasis was a major side effect in these treatments with a higher occurrence in the vaginal applicator group; nevertheless, these patients expressed higher satisfaction than to oral metronidazole.[4] Another placebo controlled study conducted by Ahmed-Jushuf and Shahmanesh demonstrated the effectiveness of a 3 day course of 2% clindamycin phosphate cream in the treatment of BV; however, there was a 25% recurrence rate after a 28 day follow-up.[5]

Clindamycin phosphate 2% single-dose vaginal cream (CSDVC) has a unique formulation compared to the conventional clindamycin phosphate 2% vaginal cream (CVC).[6] The new bioadhesive formulation allows a slower release of the active drug in the vagina over an extended period of time.[6] According to a clinical study conducted by Faro and Skokos, treatment outcomes from clindamycin phosphate 2% vaginal cream once a day are comparable in safety and efficacy to a seven day dose of clindamycin phosphate 2% vaginal cream.[7] The single day dose has a therapeutic cure of 42.1% vs 45.6% in the seven days dose ($P = 0.572$).[7]

Clindamycin phosphate is one of the drugs of choice for the treatment of BV. The single dose vaginal cream applicator is the only sustained release formulation in its class in managing BV. With non-compliance being a major issue in patients, this new therapy is another option for managing care. However, higher cost is a deterrent with this latest therapy.

REFERENCES:

Sobel JD. Bacterial vaginosis [database on the Internet]. Waltham (MA): UpToDate. C2007 [updated 2007; cited 2007Aug 7]. Available from: http://www.uptodate.com

Centers for Disease Control and Prevention. Diseases Characterized by Vaginal Discharge. MMWR. 2006 Aug;55(No. RR11):1-10.

Stein GE, Christensen SL, Mummaw NL, Soper DE. Placebo-controlled trial of intravaginal clindamycin 2% cream for the treatment of bacterial vaginosis. Ann Pharmacother. 1993 Nov;27(11):1343-5.

Ferris DG, Litaker MS, Woodward L, Mathis D, Hendrich J. Treatment of bacterial vaginosis: a comparison of Oral Metronidazole, Metronidazole Vaginal Gel, and Clindamycin Vaginal Cream. J Fam Pract. 1995 Nov;41 (5):443-9.

Ahmed-Jushuf IH, Shahmanesh M, Arya OP. The treatment of bacterial vaginosis with a 3 day course of 2% clindamycin cream: results of a multicentre, double blind, placebo controlled trial. Genitourin Med. 1995 May;71:254-6.

.Levinson RS, Mitan SJ, Steinmetz JI, Gattermeir DJ, Schumacher RJ, Joffrion JL. An Open-Label, Two-period, Crossover Study of the Systemic Bioavailability in Healthy Women of Clindamycin Phosphate from Two Vaginal Cream Formulations. Clin Ther. 2005 Dec;27(12):1894-900.

Faro S, Skokos CK. The efficacy and safety of a single dose of Clindesse™ vaginal cream versus a seven-dose regimen of Cleocin® vaginal cream in patients with bacterial vaginosis. Infect Dis Obstet Gynecol. 2005 Sep;13(3):155-60.

STUDENT SECTION

Lapatinib: a novel agent in the treatment of breast cancer

Elaine Bernardo, Pharm. D. Candidate 2008

University of Houston College of Pharmacy

Metastatic breast cancer is one of the leading causes of cancer deaths in women worldwide[1]. It is often associated with increased expression and activation of the epidermal growth factor receptor tyrosine kinases, EGFR (ErbB-1) and HER2 (ErbB-2). About 20% to 30% of women with invasive breast cancer over-express the HER2 gene, placing them at greater risk for metastatic progression and death than those women who do not over-express the HER2 gene[3]. Trastuzumab is a recombinant, humanized, monoclonal antibody that exerts its action by binding to the extracellular domain of the human epidermal growth factor receptor 2 (HER2) protein, and has proven to be a key component, and is the current standard, against HER2-over-expressing metastatic breast cancer[4]. However, most patients who initially respond to trastuzumab acquire resistance within one year of treatment[5,6]. For this reason, there is a need for identifying alternatives to inhibit HER2 signaling and the growth of trastuzumab-resistant cells.

Lapatinib is an intracellular small molecule dual tyrosine kinase inhibitor at the EGFR and HER2 domains. Because Lapatinib acts on two tyrosine kinases as opposed to just one as with Trastuzumab, it possesses several theoretical advantages. Lapatinib can inhibit heterodimers containing both EGFR and HER2[12]. It can also recognize the tumors that possess truncated forms of both EGFR and HER2 that lack an extracellular domain[14]. The promising mechanism of action of lapatinib has led researchers to conduct studies on its efficacy in breast cancer patients.

Several studies have shown lapatinib to be active as monotherapy in HER2-positive breast cancer. Lapatinib has also shown to be active in trastuzumab-resistant breast cancer patients. In addition, lapatinib has demonstrated efficacy in combination with capecitabine for HER2-positive advanced breast cancer. Geyer et al. concluded that the combination is superior to capecitabine alone in those patients whose disease progressed after treatment with an anthracycline, a taxane, or trastuzumab[25]. One group of patients received 1250 mg/day continuously of lapatinib plus 2000 mg/m^2 of capecitabine on days 1 through 14 of a 21-day cycle. The other group of patients received 2500 mg/m^2 capecitabine alone on days 1 through 14. One hundred sixty-three subjects received combination therapy, and 161 received monotherapy. In the interim analysis, it was reported that 49 disease progression events occurred in the combination-therapy group, and 72 occurred in the monotherapy group. The median time to progression due to breast cancer, which was the primary endpoint in this study, was 8.4 months with combination therapy and 4.4 months with monotherapy. The average hazard ratio for the combination therapy versus capecitabine alone was 0.49 and statistically significant. With no major concerns about safety, the data and safety monitoring committee recommended terminating enrollment and reporting the study results. Furthermore, numerous safety and toxicology data have been collected supporting the oral administration of lapatinib in humans. Overall, the most common drug-related events are grade 1 and 2 diarrhea and rash. Several phase I and II studies suggest that lapatinib is safe and well tolerated at doses up to 1800 mg daily[17,26].

Other possible benefits of lapatinib are emerging as more studies arise. A study conducted by Lin et al. suggests a potential benefit in patients with brain metastases, with the reason being that lapatinib is a small molecule that is able to cross the blood-brain barrier, as opposed to trastuzumab, which is a larger molecule unable to reach sufficient concentrations in the CNS[27]. Furthermore, lapatinib appears to have very little to no cardiotoxic effects, as seen with trastuzumab[28]. With findings of effectiveness in advanced breast cancer and its numerous possible benefits, lapatinib is being eagerly studied by researchers and drug developers. Two large ongoing phase III trials, TEACH and ALTTO, are testing the drug's efficacy as adjuvant therapy in patients with HER2-positive early breast cancer. With its recent FDA approval, lapatinib has shown and continues to represent a promising breakthrough in the treatment of breast cancer.

REFERENCES:

Parkin DM, Bray F, Felay J, Pisani P. Global Cancer Statistics, 2002. CA Cancer J Clin. 2005;55:74-108.

Burris HA 3rd. Dual kinase inhibition in the treatment of breast cancer: initial experience with the EGFR/ErbB-2 inhibitor lapatinib. Oncologist. 2004;9(suppl 3):10-15.

Slamon DJ, Clark GM, Wong SG, Levin WJ, Ullrich A, McGuire WL. Human breast cancer: correlation of relapse and survival with amplification of the HER-2/neu oncogene. Science. 1987;235:177-82.

Vogel CL, cobleigh MA, Tripathy D, Gutheil JC, Harris LN, Fehrenbacher L, et al. Efficacy and safety of trastuzumab as a single agent in first-line treatment of HER2-overexpressing metastatic breast cancer. J Clin Oncol. 2002;20:719-26.

.

STUDENT SECTION

Albanell J, Baselga J. Unraveling resistance to trastuzumab (Herceptin): insulin-like growth factor-I receptor, a new suspect. J Natl Cancer Inst. 2001;93(24):1830-2.

Esteva FJ, Valero V, Booser D, Guerra LT, Murray JL, Pusztai L, et al. Phase II study of weekly docetaxel and trastuzumab for patients with HER-2-overexpressing metastatic breast cancer. J Clin Oncol. 2002;20(7):1800-8.

Nahta R, Yuan LX, Zhang B, Kobayashi R, Esteva FJ. Insulin-like growth factor-I receptor/human epidermal growth factor receptor 2 heterodimerization contributes to trastuzumab resistance of breast cancer cells. Cancer Res. 2005;65(23):11118-28.

Nahta R, Takahashi T, Ueno NT, Hung MC, Esteva FJ. P27kip1 down-regulation is associated with trastuzumab resistance in breast cancer cells. Cancer Res. 2004;64 (11):3981-6.

Nagata Y, Lan KH, Zhou X, Tan M, Esteva FJ, Sahin AA, et al. PTEN activation contributes to tumor inhibition by trastuzumab, and loss of PTEN predicts trastuzumab resistance in patients. Cancer Cell. 2004;6(2):117-27.

Moy B, Goss PE. Lapatinib: current status and future directions in breast cancer. Oncologist. 2006;11(10):1047-57.

Suo Z, Risberg B, Kalsson MG, Willman K, Tierens A, Skovlund E, Nesland JM. EGFR family expression in breast carcinomas: c-erbB-2 and c-erbB-4 receptors have different effects on survival. J Pathol. 2002;196(1):17-25.

Rusnak DW, Affleck K, Cockerill SG, Stubberfield C, Harris R, Page M, et al. The characterization of novel, dual ErbB-2/EGFR, tyrosine kinase inhibitors: potential therapy for cancer. Cancer Res. 2001;61(19):7196-203.

Lin NU, Winner EP. New targets for therapy in breast cancer: small molecule tyrosine kinase inhibitors. Breast Cancer Res. 2004;6(5):204-10.

Xia W, Liu LH, Ho P, Spector NL. Truncated ErbB2 receptor (p95ErbB2) is regulated by heregulin through heterodimer formation with ErbB3 yet remains sensitive to the dual EGFR/ErbB2 kinase inhibitor GW572016. Oncogene. 2004;23(3):646-53.

Rusnak DW, Lackey K, Affleck K, Wood ER, Alligood KJ, Rhodes N, et al. The effects of the novel, reversible epidermal growth factor receptor/ErbB-2 tyrosine kinase inhibitor, GW2016, on the growth of human normal and tumor-derived cell lines in vitro and in vivo. Mol Cancer Ther. 2001;1(2):85-94.

Konecny GE, Pegram MD, Venkatesan N, Finn R, Yang G, Rahmeh M, et al. Activity of the dual kinase inhibitor lapatinib (GW572016) against HER-2-overexpressing and trastuzumab-treated breast cancer cells. Cancer Res. 2006;66(3):1630-9.

Burris HA III, Hurwitz HI, Dees EC, Dowlati A, Blackwell KL, O'neil B, et al. Phase I safety, pharmacokinetics, and clinical activity study of lapatinib, a reversible dual inhibitor of epidermal growth factor receptor tyrosine kinases, in heavily pretreated patients with metastatic carcinomas. J Clin Oncol. 2005;23(23):5305-13.

Blackwell KL, Kaplan EH, Franco SX, Marcom PK, Maleski JE, Sorensen MJ, et al. A phase II, open-label, multicenter study of GW572016 in patients with trastuzumab-refractory metastatic breast cancer. J Clin Oncol. 2004;22:14S.

Spector NL, Blackwell K, Hurley J, Harris JL, Lombardi D, Bacus S, et al. EGF103009, a phase II trial of lapatinib monotherapy in patients with relapsed/refractory inflammatory breast cancer (IBC): Clinical activity and biologic predictors of response. J Clin Oncol. 2006;24:18S.

Iwata H, Toi M, Fujiwara Y, Ito Y, Fuji H, Nakamura S, Aogi K, et al. Phase II clinical study of lapatinib (GW572016) in patients with advanced or metastatic breast cancer. Breast Cancer Res and Treat. 2006;100:S68.

Zhou H, Kim YS, Peletier A, McCall W, Earp HS, Sartor CI. Effects of the EGFR/HER2 kinase inhibitor GW572016 on EGFR- and HER2-overexpressing breast cancer cell line proliferation, radiosensitization, and resistance. Int J Radiat Oncol Biol Phys. 2004;58(2):344-52.

Xia W, Mullin RJ, Keith BR, Liu LH, Ma H, Rusnak DW, et al. Anti-tumor activity of GW572016: a dual tyrosine kinase inhibitor blocks EGF activation of EGFR/erbB2 and downstream Erk1/2 and AKT pathways. Oncogene. 2002;21(41):6255-63.

Nahta R, Yuan LX, Du Y, Esteva FJ. Lapatinib induces apoptosis in trastuzumab-resistant breast cancer cells: effects on insulin-like growth factor I signaling. Mol Cancer Ther. 2007;6(2):667-74.

Tripathy D. Capecitabine in combination with novel targeted agents in the management of metastatic breast cancer: underlying rationale and results of clinical trials. Oncologist. 2007;12(4):375-89.

Geyer CE, Forster J, Lindquist D, Chan S, Romieu CG, Pienkowski T, et al. Lapatinib plus capecitabine for HER2-positive advanced breast cancer. N Eng J Med. 2006;355 (26):2733-43.

Versola M, Burris HA, Jones S, Wilding G, Taylor C, Pandite L, et al. Clinical activity of GW572016 in EGF 10003 in patients with solid tumors. J Clin Oncol. 2004;22:3047.

Lin NU, Carey LA, Liu MC, Younger J, Come SE, Bullitt E, et al. Phase II trial of lapatinib for brain metastases in patients with HER2+ breast cancer. J Clin Oncol. 2006;24:503.

Perez EA, Byrne JA, Hammond IW, Rafi R, Martin AM, Berger MS, et al. Results of an analysis of cardiac function in 2,812 patients treated with lapatinib. J Clin Oncol. 2006;24:583.

Has your contact information changed?

Please send all changes to Allison Wilson at **awilson@tmhs.org**

You can access a membership application at **www.gcshp.org**

STUDENT SECTION

New Guidelines for the Treatment of Hypertension

Kim Nguyen, Pharm.D. Candidate 2008

University of Houston College of Pharmacy

Hypertension is a major risk factor for coronary artery disease (CAD) and affects about 25% of adults living in the United States. The National High Blood Pressure Education Program has published guidelines on the classification and treatment of hypertension. According to its latest guideline, the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7), a normal blood pressure is a systolic blood pressure (SBP) of <120 mmHg and a diastolic blood pressure (DBP) of <80 mmHg. A SBP of >140 mmHg or DBP of >90 mmHg is considered hypertension. In patients being treated for hypertension, JNC 7 recommends that hypertension patients be treated to a goal of <140/90 mmHg. However, in patients with diabetes mellitus or chronic renal disease, the blood pressure goal is <130/80 mmHg.

A thiazide-type diuretic is usually the treatment of choice for patients without compelling indications. However, ACE inhibitors, angiotensin receptor blockers, beta-blockers, calcium channel blockers, or combination therapy may be considered. JNC 7 also has guidelines for treating hypertension in patients with compelling indications. In patients with high risk for CAD, treatment with a diuretic, beta-blocker, ACE inhibitor, or calcium channel blocker is recommended.

The American Heart Association (AHA) recently published guidelines on treating hypertension in patients with ischemic heart disease. The AHA recommends that patients with a high risk of developing CAD (patients with diabetes mellitus, chronic kidney disease, known CAD or CAD equivalent, or a ten-year Framingham risk score ≥10%) be treated to a goal of <130/80 mmHg. These patients should be treated with a thiazide-type diuretic, ACE inhibitor, angiotensin receptor blocker, calcium channel blocker, or combination therapy. In hypertension patients with stable or unstable angina, non-ST-segment elevation myocardial infarction, or ST-segment elevation myocardial infarction, the blood pressure should also be treated to <130/80 mmHg. Treatments for these patients include a beta-blocker along with an ACE inhibitor or angiotensin receptor blocker. Lastly, in patients with left ventricular dysfunction, the blood pressure goal is <120/80 mmHg. These patients should be treated with an ACE inhibitor or angiotensin receptor blocker, beta-blocker, aldosterone antagonist, thiazide or loop diuretic, and hydralazine/isosorbide dinitrate.

The AHA hypertension guideline and JNC 7 differ in their blood pressure goals for patients with hypertension and other comorbid conditions. In patients at high risk for CAD or with known CAD, blood pressure control is of great importance. Adequate antihypertensive therapy and blood pressure control are vital in reducing cardiovascular morbidity and mortality. With the new hypertension guideline from the AHA, aggressive treatment in patients at high risk for CAD or known CAD is appropriate.

REFERENCES:

1. Chobanian AV, Bakris GI, Black HR, Cushman WC, Green LA, Izzo JL, et al; Joint National

Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure;

National Heart, Lung, and Blood Institute; National High Blood Pressure Education Program

Coordinating Committee. Seventh Report of the Joint National Committee on Prevention,

Detection, Evaluation, and Treatment of High Blood Pressure. Hypertension. 2003;42:1206-

52.

2. Rosendorff C, Black HR, Cannon CP, Gersh BJ, Gore J, Izzo JL, et al. Treatment of

hypertension in the prevention and management of ischemic heart disease: a scientific

statement from the American Heart Association Council for High Blood Pressure Research

and the Counsils on Clinical Cardiology and Epidemiology and Prevention. Circulation. 007;115:2761-88 l

Are Statins Safe? Recommendations from The National Lipid Association Statin Safety Assessment Task Force

Diana Thai, Pharm.D. Candidate 2008

University of Houston College of Pharmacy

The National Lipid Association (NLA) Statin Safety Task Force released new recommendations regarding the safety of therapy with 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase inhibitors or statins. After an extensive analysis of results of many different trials and information from the New Drug Application (NDA), the US Food and Drug Administration (FDA) Adverse Event Reporting System (AERS), and administrative claims database, the Task Force has provided recommendations to help guide health professionals in managing coronary artery disease risk in patients undergoing statin therapy.

STUDENT SECTION

Liver: Elevations in the liver enzymes, alanine aminotransferase (ALT) or aspartate aminotransferase (AST), are not very common, usually transient, and resolve spontaneously in most cases even when the statin regimen has not changed. Continuation of measurement of liver enzyme levels before starting therapy, 12 weeks after initiation, after dose increases, and regularly thereafter is still appropriate until a change in the prescribing information of statins occurs. The NLA Statin Safety Task Force did not find any evidence to support routine monitoring of liver function tests and recommends the FDA to review available data and reconsider the monitoring recommendations of statins. Clinicians should watch out for signs and symptoms of hepatotoxicity, such as jaundice, fatigue, and increased levels of indirect bilirubin and prothrombin time. If significant liver injury is evident, discontinue the statin and determine the etiology. Two consecutive occasions of elevations in ALT and AST >3 times the upper limit of normal (ULN) is needed to more accurately recognize patients with persistent liver test abnormalities and other etiologies should also be ruled out before considering changing statin therapy. Discontinuation of statins is not needed if liver enzymes are elevated 1-3 times the ULN and the patient is asymptomatic. The Expert Liver Panel concluded statins can be safely administered in patients with chronic liver disease, nonalcoholic fatty liver disease or nonalcoholic steatohepatitis.

Muscle: The NLA Task Force has provided new definitions regarding muscle symptoms in patients taking statins. Myopathy can be described as complaints of myalgia (muscle pain or soreness), weakness, and/or cramps with elevations in serum creatine kinase (CK) >10 times the ULN. The patient can also be experiencing myopathy without CK >10 times the ULN if muscle symptoms are intolerable. Rhabdomyolysis can be identified as CK >10,000 IU/L or CK >10 times the ULN with an increase in serum creatinine or necessary administration of IV hydration therapy. Other etiologies should be ruled out first anytime muscle symptoms or elevations in CK levels are reported. Because significant elevations of CK levels are rare, routine monitoring of CK levels in asymptomatic patients is not warranted. In symptomatic patients, CK levels can help to assess the severity of muscle damage to determine if statin therapy should be continued or altered. If patients experience intolerable muscle symptoms regardless of CK levels and other causes can not be found, statin therapy should be withdrawn. If symptoms resolve, the same or another statin can be restarted at the same or reduced dose. If symptoms return after use with other statins and doses, another lipid-lowering drug may be needed. Statins can be continued at the same or lower dose if patients are asymptomatic or have tolerable symptoms with CK levels <10 times the ULN. However, if rhabdomyolysis develops, statin therapy should be discontinued and IV hydration administered if necessary. Clinicians should assess the risk vs benefits of statins once symptoms resolve.

Kidney: Unless the patient is experiencing rhabdomyolysis, an association between statins and acute renal failure or insufficiency has not been found. Thus, routine monitoring of serum creatinine and proteinuria during statin therapy is not advised. Discontinuation of statins is not necessary in cases of increased serum creatinine levels without rhabdomyolysis or development of unexpected proteinuria. However, the prescribing information does suggest consideration of dose adjustments in those situations. Chronic kidney disease is not a contraindication of statin therapy; however, doses may need to be modified if there is moderate or severe renal insufficiency.

Neurologic Disorders: Because the risk of peripheral neuropathy with statins is very small and possibly non-existent and no evidence supports a relation of statins to dementia and cognitive impairment, routine neurologic monitoring is not recommended. Secondary causes should be ruled out if symptoms of peripheral neuropathy or impaired cognition are reported. If none are found, withdraw statin therapy for a certain period (3-6 months for peripheral neuropathy and 1-3 months for impaired cognition) to evaluate if statins are the cause. If neurologic symptoms improve after discontinuation, the cause could be related to statins, but consideration can still be given to rechallenge with a different statin and dose because of the benefits of statin therapy. If symptoms do not improve after withdrawing the statin, clinicians should assess risk vs benefit to determine if the statin should be restarted.

REFERENCE:

McKenney JM, Davidson MH, Jacobson TA, Guyton JR. Final conclusions and recommendations of the National Lipid Association Statin Safety Assessment Task Force. Am J Cardiol 2006;97[suppl]:89C-94C.

The Role of Rivastigmine Transdermal Patch in Treatment of Alzheimer's Disease Dementia

Sarah Elizabeth Hansen, Pharm.D. Candidate 2008

University of Houston College of Pharmacy

Alzheimer's disease (AD) dementia is a potentially devastating neurological disease that has few available options for treatment. Almost three decades ago, investigators started researching the idea that increasing acetylcholine levels in the brain, a major neurotransmitter which is depleted in AD, might improve memory and cognition in patients with this disease.[1]

STUDENT SECTION

Acetylcholinesterase inhibitors are one of the only approved drug classes used to treat mild to moderate dementia. In the past, the only available dosage forms of these agents were oral and led to significant cholinergic side effects. The latest development in this drug class is the new formulation of rivastigmine as a transdermal patch.

The benefits of transdermal therapy vs. oral therapy are many. Transdermal patches provide continuous drug delivery, avoid first pass metabolism, offer improved tolerability, reduce side effects, and the convenience of once daily dosing.[2] The oral form of rivastigmine is associated with cholinergic side effects, particularly in the gastrointestinal tract (GIT), including nausea, vomiting and diarrhea.[2]

Lefevre et al compared the pharmacokinetic and pharmacokinetic profile of rivastigmine transdermal patch and oral capsule.[5] They found that the patch offers lower C_{max} and longer T_{max}, which yields more continuous drug levels and reduced side effects.[5] Based on the nonlinear kinetics exhibited by rivastigmine, the study showed that oral administration has two distinct peak and trough levels with each dose while the patch provides continuous and steady drug levels. [5] This study showed that the 10 cm^2 patch exhibited similar drug exposure compared with 6mg twice daily oral therapy and that the 20 cm^2 patch would yield similar exposure to drug as a dose of 9-10mg twice daily orally.[5] The study determined the individual release rates for each patch size as follows: 4.6mg/day (5 cm^2), 9.5mg/day (10 cm^2), 13.3mg/day (15 cm^2), and 17.4mg/day (20 cm^2). [5] Lefevre et al concluded that the recommended maintenance dose is 10 cm^2 daily.[5]

The Investigation of transdermal Exelon in Alzheimer's disease (IDEAL) trial, completed by Winblad et al, was a multi-center, randomized controlled-trial, double-blind, double-dummy, placebo and active controlled 24-week study which looked at the safety and efficacy of rivastigmine transdermal patch vs. oral capsules and placebo. The safety analysis showed that the 10 cm^2 patch produced less nausea and vomiting than the other treatment groups, similar to that of placebo.[3] Commonly reported adverse events in all treatment groups included nausea, vomiting, diarrhea, weight reduction, decreased appetite, dizziness, headache and asthenia.[3] Side effects may be managed by halting titration or decreasing the dose.[3]

The IDEAL trial also looked at primary efficacy outcomes of change in ADAS-cog and ADCS-CGIC, which assess multiple areas including cognition, memory, language, and behavior changes.[3,8,10] The 20 cm^2 patch showed the most improvement in cognition compared to placebo, while the 10 cm^2 patch and oral capsule showed the same improvement. On the CGIC all treatment groups showed little improvement, but were considered statistically significant. The 10 cm^2 and 20 cm^2 patches also showed statistically significant improvements in ADL, MMSE scores, and Trail making Part A. While the results were not statistically significant on the Neuropsychiatric Inventory (NPI), all groups including placebo showed improvements over the 6-month period.[3]

Although the effects of treatment on patients with AD dementia are small, they should not be discounted as insignificant. Each added day of normal functioning is precious from the perspective of both the patient and the caregiver. Transdermal rivastigmine may delay nursing home placement and improve cognition as well as increase compliance by causing less side effects than traditional oral therapy. Large head to head clinical trials are needed to see which cholinesterase inhibitors are most effective. The rivastigmine transdermal patch is a promising therapeutic option for patients with AD dementia.

REFERENCES:

Faulkner JD, Bartlett J, Hicks P. Alzheimer's Disease. In: DiPiro JT, Talbert RL, Yee GC, Matzke GR, Wells BG, Posey LM, editors. Pharmacotherapy: a pathophysiologic approach, 6th Ed. New York: McGraw-Hill, 2005. p.1157-73.

Oertel W, Ross JS, Eggert K, Adler G. Rationale for transdermal drug administration in Alzheimer disease. Neurology 2007; 69(Suppl 1):S4-9.

Winblad B, Grossberg L, Frolich M, Farlow M, Zechner S, Nagel J, et al. IDEAL: A 6-month, double-blind, placebo-controlled study of the first skin patch for Alzheimer disease. Neurology 2007; 69:14-22.

Winblad B, Cummings J, Andreasen N, Grossberg G, Onofrj M, Sadowsky C, et al. A six-month double-blind, randomized, placebo-controlled study of a transdermal patch in Alzheimer's disease-rivastigmine patch versus capsule. Int J Geriatr Psychiatry 2007; 22:456-67.

Lefevre G, Sedek G, Jhee SS, Leibowitz MT, Huang HL, Enz A, et al. Pharmacokinetics and pharmacodynamics of the novel daily rivastigmine transdermal patch compared with twice-daily capsules in Alzheimer's disease patients. Clin Pharmacol Ther 2007 May 23:1-9.

Mace NL and Rabins PV. The 36-hour day: a family guide to caring for persons with Alzheimer disease, related dementing illnesses, and memory loss in later life. New York: Warner Books, 2006.

McEvoy GK, Snow EK, Kester L, Litvak K, Miller J, Welsh OH, et al, editors. AHFS Drug information essentials. Bethesda, Maryland: ASHP;2006.

McKeith IG, Cummings JL, Lovestone S, Harvey RJ, Wilkinson DG. Outcome measures in Alzheimer's disease. 1999. London: Martin Dunitz Ltd.

Folstein MF, Folstein SE, McHugh PR. Mini-mental state: a practical method for grading the cognitive state of patients for the clinician. J Psychiatr Res. 1975; 12:189-201.

STUDENT SECTION

Schneider LS, Olin JT, Doody RS, Clark CM, Morris, JC, Reisberg B, et al. Validity and reliability of the alzheimer's disease cooperative study-clinical global impression of change. Alzeimer Dis Assoc Disorders. 1997;11 Suppl 2: S22-32.

Galasko D, Bennett D, Sano M, Ernesto C, Thomas R, Grundman M, et al. An inventory to assess activities of daily living for clinical trials in Alzheimer's disease. Alzeimer Dis Assoc Disorders. 1997;11 Suppl 2: S33-39.

Cummings JL, Mega M, Gray K, Rosenberg-Thompson S, Carusi, DA, Gornbein, J. The neuropsychiatric inventory: comprehensive assessment of psychopathology in dementia. Neurology. 1994; 44:2308-14.

The Use of Topiramate in Migraine Prophylaxis

Sana Memon, Pharm.D. Candidate 2008

University of Houston College of Pharmacy

The International Headache Society (IHS) defines migraine as a disorder characterized by intermittent attacks of headache combined with nausea, photophobia, and/or phonophobia.[1] Approximately 11% of the US population experiences migraines; however, many patients are undiagnosed and under treated.[2] Preventive therapy can reduce the frequency of migraines by 50% or more.[4] The US Headache Consortium recommends preventive treatment for migraines in certain situations.[5] The goal of preventive therapy is to improve patients' quality of life by reducing migraine frequency, severity and duration.[5] Research indicates that topiramate, an antiepileptic agent, has been used for other neurologic disorders and may also be effective in migraine prevention.

In 1999, an open label study of adults with migraine headaches conducted by Shuaib and colleagues suggested that topiramate may be efficacious in migraine prevention.[9] Topiramate may modulate the neuronal hyperexcitability which underlies the pathogenesis of migraine. Recently, three large, double-blind, placebo-controlled trials on migraine prevention have shown that topiramate resulted in significant benefits when compared to placebo and is effective in migraine prophylaxis in adults.[10-12]

Silberstein and colleagues conducted a 26 week, randomized, double-blind, placebo-controlled, parallel-group, multicenter US study to evaluate the safety and efficacy of topiramate versus placebo in migraine prevention. The primary efficacy measures showed that the mean monthly migraine frequency decreased for the 100 mg/day group ($p < 0.001$) and the 200 mg/day group ($p<0.001$) versus placebo within the first treatment month. Topiramate treated patients experienced a high responder rate ($\geq$ 50% reduction in monthly migraine frequency), a reduction in mean monthly migraine days, and a reduction in mean monthly acute rescue medication days. Side effects included paresthesia, fatigue, and nausea. The mean body weight in all topiramate groups showed a statistically significant reduction.[10]

Brandes and colleagues conducted another 26 week, randomized, placebo-controlled, parallel-group, multicenter US study to evaluate the safety and efficacy of topiramate versus placebo in migraine prevention. The mean monthly migraine frequency decreased significantly for patients receiving topiramate 100 mg/day ($p=0.008$) and topiramate 200 mg/day ($p<0.001$) versus placebo. Within the first month, there were statistically significant reductions in migraines. The responder rate was significantly greater with topiramate. There was a significant decrease in mean reduction in the monthly number of migraine days and in rescue medication use. Adverse events included paresthesia, fatigue, and nausea. Patients experienced a mean reduction in weight percentage that was also statistically significant with topiramate compared to placebo.[11]

Lastly, Diener and colleagues conducted a 26 week, randomized, double-blind, parallel-group, multicenter trial to evaluate the efficacy and safety of topiramate versus placebo for migraine prophylaxis, with propranolol as an active control. The topiramate 100 mg/day group had a significant reduction in average monthly migraine frequency compared with placebo ($p=0.011$) within the first month. The topiramate 100 mg/day and propranolol groups had similar results with respect to reductions in migraine frequency, migraine days, and daily rescue medication usage. Both groups had high responder rates. The most frequent adverse events included paresthesia, difficulty with concentration, and nausea. Patients in the topiramate 100 mg/day group experienced a 2.7% weight decrease compared to a 2.3% weight increase in the propranolol 160 mg/day group.[12]

Topiramate has been shown to be effective in migraine prevention based on these three large, placebo-controlled, dose-ranging trials. These studies indicate that topiramate was superior to placebo as measured by the reduction in mean monthly migraine frequency, monthly migraine days, and monthly migraine duration.[10-12] In 2004, topiramate became the most recent drug approved by the FDA for migraine prevention among adults.[13]

STUDENT SECTION

Based on the evidence presented, these three trials support the use of topiramate 100 mg/day as an appropriate choice for migraine prevention.[10-12]

REFERENCES:

Headache Classification Committee of the International Headache Society. Classification and diagnostic criteria for headache disorders, cranial neuralgias and facial pain. Cephalalgia. 1988;8:1-93.

Lipton RB, Stewart WF, Diamond S, Diamond ML, Reed M. Prevalence and burden of migraine in the United States: data from the American migraine study II. Headache. 2001;41:646-57.

Hu XH, Markson LE, Lipton RB, Stewart WF, Berger ML. Burden of migraine in the United States: disability and economic costs. Arch Intern Med. 1999;159:813-18.

Modi S, Lowder DM. Medications for migraine prophylaxis. Am Fam Physician. 2006;73:72-8.

Silberstein SD. Practice parameter: evidence-based guidelines for migraine headache (an evidence-based review):

Appropriate Use and Duration of Antiplatelet Therapy After Percutaneous Coronary Intervention with Stent Placement

Amy M. Savarino, Pharm.D. Candidate 2008

University of Houston College of Pharmacy

Dual antiplatelet therapy, with aspirin and clopidogrel, is essential after coronary artery stent placement. All or part of the antiplatelet therapy is sometimes discontinued prematurely by the patient or by a healthcare provider who does not realize the risks of discontinuing the antiplatelet therapy prematurely. The most common problem associated with prematurely discontinuing the antiplatelet therapy is stent thrombosis leading to acute myocardial infarction or death. The pharmacist can play a vital role in helping patients understand the need and duration of the dual antiplatelet therapy.

Stents are placed in a variety of coronary and non-coronary arteries to form a scaffold that allows an occluded artery to remain forced open. Types of stents include a bare metal stent and drug eluting stents that release small amount of drug for a short duration of time after placement. Taxus ® (paclitaxel), an antineoplastic agent, and Cypher ® (sirolimus), an immunosuppressant, are the two approved drug eluting stents. These drug eluting stents were created to prevent restenosis of the artery, a common problem of bare metal stents. Restenosis occurs when the body forms a thrombosis around the foreign object (stent). Further harm occurs when a thrombosis breaks off and moves to other critical areas of the body. Drug eluting stents appear to delay this restenosis problem rather than prevent it, demonstrating the importance of antiplatelet therapy duration.

Clopidogrel 75mg and aspirin 325mg can and should be used together for all patients after coronary stent placement, unless the patient is at high risk of bleeding. Increasing age, presence of diabetes, kidney disease, congestive heart failure, acute coronary syndrome and previous intravascular brachytherapy predispose patients to thrombosis. Other high risk predictors for thrombosis include: multiple, long, overlapping stents especially in small vessels.

The current recommendation for the duration of dual antiplatelet therapy is 12 months for all coronary artery stents. Dual antiplatelet therapy may be used for a minimum of one month with bare metal stents only. Patients with drug-eluting stents require at least twelve months of dual antiplatelet therapy. Premature discontinuation of the dual antiplatelet therapy has been associated with life-threatening thrombosis. Once clopidogrel is discontinued, aspirin should be continued indefinitely. However, the dose of aspirin can be reduced from 325mg to 81mg after one month for bare metal stents, and three months for sirolimus stents and 6 months after paclitaxel stents.

Dual antiplatelet therapy requires other considerations. Clopidogrel and aspirin are both associated with an increased risk of bleeding. The treating cardiologist should be consulted before stopping all or part of the dual antiplatelet therapy, even if instructed to do so by another healthcare provider. All elective procedures should be postponed until 12 months after drug-eluting stent placement, rather than discontinuing the dual antiplatelet therapy. Before drug-eluting stent placement, patients should be instructed about the need for dual antiplatelet therapy and the expected level of compliance.

As a pharmacist, it is important to educate the patient on dual antiplatelet therapy duration, appropriateness of aspirin dose, and compliance. Communication between the patient, the pharmacist and other healthcare providers is essential to maximize the benefits and minimize the risks associated with coronary artery stents and dual antiplatelet therapy.

REFERENCES:

1. Grines CL, Bonow RO, Casey DE, et al. Prevention of premature discontinuation of dual antiplatelet therapy in patients with coronary artery stenosis. Circulation. 2007;115.

2. Smith SC, Feldman TE, Hirshfield JW, et al. ACC/AHA/SCAI 2005 Guideline update for percutaneous coronary intervention; a report of the American College of Cardiology/ American Heart Association task force on practice guidelines (ACC/AHA/SCAI writing committee to update the 2001 guidelines for P C I). www.acc.org.

If you are interested in serving on any of the councils, please contact the appropriate chairperson below. Get Involved Today!

We're on the web!
www.gcshp.org

GCSHP Newsletter is published quarterly

Monica Robinson Green, Pharm.D., Editor

phone: (281) 260-3375

e-mail: Monica_Robinson@hchd.tmc.edu

Submit contributions

(preferably via e-mail or CD in Word format)

Monica Robinson Green, Pharm.D.

Acres Home Health Center

818 Ringold

Houston, TX 77088

Office/Position	Name	Contact Information	Reason to Contact
President	Ogechi Eshleman	ogechi_eshleman@hchd.tmc.edu	
President-Elect	Todd Canada	tcanada@mdanderson.org	
Immediate Past President	David Hayes	jdhayes@uh.edu	
Director	Richard Cadle	cadle.richardmark@med.va.gov	Annual Seminar
Recording Secretary	Judith Feltman	judithlin@aol.com	
Treasurer	Laura Gerard	gerardln@yahoo.com	Payment inquiries
Membership Secretary	Allison Wilson	awilson@tmhs.org	Membership status; Address change
Communication Council	Monica Robinson Green	monica_robinson@hchd.tmc.edu	Newsletter article submission Mentorship program
Education Council	Jennifer Christensen	jennifer_christensen@hchd.tmc.edu	Continuing education events or credit
Professional and Legal Affairs	Ryan Roux	ryan_roux@hchd.tmc.edu	
Technician Section	Theresa Batiste Kathleen Thomas	theresa_batiste@hchd.tmc.edu kathleen_thomas@hchd.tmc.edu	Technician membership
Student Section—TSU	Pamela Dyer	pdyer315@hotmail.com	Student membership
Student Section—UH	Ann Marie Prazak	abprazak@uh.edu	Student membership
Members-at-Large	Chad Hardy Eddie Lee Dan Metzen Michael Pinon Douglas Rasmussen	Chad_hardy@hchd.tmc.edu lelee@utmb.edu dmetzen@tmhs.org drmpinon@sbcglobal.net douglas.rasmussen@cardinal.com	Website issues Annual Seminar Annual Seminar Annual Seminar Annaul Seminar
Industry Representative	Rowland Harris		Annual Seminar