AMERICAN PSYCHOSOMATIC SOCIETY

Dedicated to the Integration of Biological Psychological and Social Factors in Medicine

VOL. 21, NO. 2 NEWSLETTER FOR APS MEMBERS Summer 2011

President's Column

Michael R. Irwin, MD

A Record of Success and Building a Framework for the Future

The American Psychosomatic Society is an extraordinary organization. Its broad and diverse membership truly embraces the interdisciplinary strengths needed to promote and advance understanding of the biological, behavioral, and psychosocial factors in health and disease. Increasingly, we have seen the Society's membership take the lead in translating cutting-edge research in the mechanisms of biobehavioral medicine toward the development and dissemination of interventions for the prevention and treatment of disease in humans. Indeed, the success of the Society's members is evident in numerous ground-breaking publications in leading biomedical journals, including the Society's premiere venue *Psychosomatic Medicine*. More than ever, the success of our Society is intricately linked to the success of our members. In turn, the success of the Society is measured by how effective we, as an organization, can work to identify the means to help our members be more successful, and to deliver value by aligning resources to achieve these goals. Moreover, this challenge has evolved in the midst of a dynamic and rapidly changing biomedical and healthcare environment, making it is all the more critical that we strategically define our goals and harness the talent of our membership to move our Society forward.

Towards that end, we have seen an increasing momentum in achieving the priorities of the Society, with numerous changes in the organizational and volunteer leadership of committees as detailed by Past-President Paul Mills in the Spring 2011 newsletter. Furthermore, arising from the Strategic Planning Retreat, the Council and its leadership has refined and focused this agenda, with articulation of seven working goals. From these strategic goals, each of the Society's committees and task forces have identified action items to serve as tangible steps in realizing these broad goals. Furthermore, an engaged effort is now in place to work across committees to integrate shared purposes in relation to goals of membership, liaison, and dissemination, for example.

> *"More than ever, the success of our Society is intricately linked to the success of our members."*

The inter-disciplinary and demographic diversity of the membership is a distinct strength of the Society. Increasingly, it is recognized that the cross-talk between disciplines is critical in advancing our understanding of the biobehavioral mechanisms that promote health and prevent disease; in contrast, artificial silos that separate investigators are an impediment to such progress. The collaborative interactions that are forged by formal and informal mechanisms at our annual meeting, for example, bridge disciplines and accelerate such advances. To build upon this disciplinary breadth of the Society, I have constituted with Council's permission a Membership Task Force. This Task Force is co-chaired by Tene Lewis, PhD and Scott Matthews, MD and includes Paige MacDonald, PhD and Karen Weihs, MD, as well as myself as liaison to Council. This Task Force is charged with developing short- and long-term initiatives that will target non-member constituencies who share a high level of common interests with the Society, but who are not yet aware of the value of membership in our organization; define how we can best focus our energy and resources on membership core needs to ensure a high level of retention across disciplines; and identify programmatic mechanisms to serve and facilitate the transition of Associate Members to full Membership status. The work of this Task Force will be brought to the Fall Council meeting, so that membership priorities can proceed with targeted initiatives this coming year.

Consonant with these membership goals, the Liaison Committee, co-chaired by Benjamin Natelson, MD, and Urs Nater, PhD, is striving to establish bridges to other professional societies and organizations, which share many of our interests. Indeed, this Committee is interacting closely with the Program Committee, chaired by Suzanne Segerstrom, PhD, so that non-members who might have a leadership role in other societies can be fully engaged in the American Psychosomatic Society annual meeting. The goals of this initiative are several fold: to introduce our membership to innovative science that can complement our activities; to bridge informal interactions between societies; and to forge interest of non-members in the American Psychosomatic Society, with the goal of continuing to strengthen the depth of inter-disciplinary interactions at our annual meeting.

To foster the dissemination of psychosomatic research to our membership, as well as the public at large, several organizational entities within the Society are now working together. First, under the leadership of the new Editor-in-Chief, Wijo Kop, PhD, *Psychosomatic Medicine*, is continuing to publish

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From the Editor

John M. Ruiz, PhD

The Personal Importance of Professional Representation

As we go to press with this edition of the newsletter, the collective US and much of the world is focused on the budget negotiations taking place in Washington DC. Although these discussions often seem rather esoteric, the outcomes will have important implications for us as professionals and individuals. For example, if you are sitting in your office at a state-funded institution or at a medical center where a portion of your time is funded through soft-money, your chances of receiving a pay raise, receiving summer funding, or holding onto your position may be a function of these negotiations. Moreover, the availability of grant funding may influence the timing and scope of your research, ability to incorporate novel biomarkers, test clinical interventions, conduct longitudinal follow-ups, or employ trainees. Hence, most of us have a vested interest in the scenes playing out on CNN.

Regardless of how much of the national income is ultimately dedicated to deficit reduction, the outcome will be less money available for the rest of the nation's business including funding of research. The allocation of the smaller research funding pie to psychosocial research, basic science, space exploration etc., is moderated by subjective priorities influenced through representation. So who represents you and your interests in psychosocial and biobehavioral health research? And, what are you doing to ensure vigorous advocacy?

Today is a great day to begin personally addressing the challenge of adequate representation. There are a number of options that you can do that will take you less time than reading this newsletter. Send an email to the NIH director or better; contact the larger societies such as the AMA and APA who have representatives at the research budget negotiating table. Within the larger professional societies, nominate and vote into office candidates who have a demonstrated appreciation for psychosocial health research. Rally others through a listserve posting or through various social media. Contact the APS leadership and find out what can be done to improve the Society's political representation. And of course, invite your colleagues and students to join APS so that we can speak with a louder voice.

Doing great science is a fantastic way to contribute to your field. However, advocacy that enables the science is an equally important contribution.

This Edition of the Newsletter

On a much brighter note, here is your Summer 2011 APS Newsletter. On the preceding page, our new president, Dr. Michael Irwin discusses a comprehensive effort to increase the Society's membership and diversity and to use technology to improve dissemination with the goal of keeping APS at the forefront of biobehavioral medicine. As he points out, any success in these efforts is due to the strength of the membership and the volunteer work of so many. Speaking of being at the forefront of biobehavioral research, Drs. Nicolas Rohleder and Jutta Wolf of Brandeis University discuss the conceptual and operational challenges in measuring inflammation in this edition's *Practical Science*. We also have the privilege of *getting to know* Drs. Steve Manuck of the University of Pittsburgh and John Burns from Rush University in Chicago who discuss their current work, local food recommendations, and in one case, how he is perceived by primates. The *Awards section* of the newsletter features 2011 Herbert Weiner Award winner, Dr. Naomi Eisenberger of UCLA who shares with us her career developmental pathway leading to her research program into the physiological concomitants of social connectedness. Finally, we have updates from the Liaison Committee, the Professional Education Committee, as well as the 2012 Conference Program Committee which detail their extraordinary efforts on behalf of the membership and exemplify the vibrancy of APS.

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Practical Science: Quantifying and Understanding Inflammation

Nicolas Rohleder, PhD, & Jutta M. Wolf, PhD, Department of Psychology, Brandeis University

Inflammation is an immune response that comes in various forms. What we often think of when talking about inflammation is a contained local response to an invading pathogen that might be resolved within hours or days, but can spread to the rest of the body, initiating a systemic inflammatory response with deleterious outcomes. Another, more recently described form of systemic inflammation is chronic low-grade inflammation (Danesh, 1999). This phenomenon is characterized by increases in concentrations of the same plasma mediators that signal and contribute to local and other forms of systemic inflammation, i.e. mainly interleukin-6 (IL-6) and C-Reactive Protein (CRP). However, concentrations are markedly lower than those measured for example in sepsis, but still increased in comparison to the non-inflamed state of a young and healthy organism. Importantly, chronic low-grade inflammation seems to play a role in a huge array of (often age-related) diseases, such as cardiovascular disease, insulin resistance, osteoporosis, and cancer (see (Ershler, 1993). For example, several prospective studies have found that increased concentration of IL-6 and CRP in older adults is a strong predictor of morbidity and all-cause mortality (Ferrucci et al., 1999; Harris et al., 1999; Bruunsgaard et al., 2003).

While the pathways between inflammatory processes and disease outcomes have been and are studied extensively, the behavioral and psychosocial antecedents of chronic low-grade inflammation are less well understood. For example, do psychosocial processes increase inflammation? Do stress, depression, trauma, loneliness, social inequalities, stimulate inflammation? Answering these questions would allow us to draw a pathway between psychosocial processes and disease. To solve this task, we need to quantify inflammation during or following psychosocial processes, and we need to understand how the central nervous system (CNS) acts on inflammation.

Quantifying inflammatory activity and reactivity

Central to this task is first and foremost the accurate quantification of inflammatory activity. The most straightforward and most established approach is to measure IL-6 or CRP concentrations in blood. Until a few years ago, these assays only allowed detection of high concentrations indicative of acute inflammation. More recently, however, high-sensitivity assays became available, allowing detection of more subtle differences such as of interest in the context of chronic low-grade inflammation.

The highest stability in concentrations of inflammatory mediators can be expect with fasting morning blood draws, although studies have been successful in finding relationships with psychosocial processes using afternoon non-fastened blood draws as well. However, given that some markers (e.g. IL-6) show circadian variations, it is important to keep the timing of the blood draws constant. With regard to the question whether to use serum or EDTA plasma, serum is the classical clinical approach, but in our experience, both work well. EDTA plasma has the advantage of faster handling post blood draw, i.e., can immediately be transferred to storage because no clotting time is required. For storage, -80C is highly recommended, since storage at higher temperatures (e.g., -30C) will result in accelerated degradation. Lastly, distributions of both IL-6 and CRP are usually skewed and benefit from log-transformation.

Measures obtain this way address only peripheral inflammatory activity and give no information about where the respective inflammatory mediator comes from. Sources can include circulating leukocytes, adipose tissue, endothelial cells, or muscle, implying different causes for elevated concentrations. However, this does in no way invalidate assessment of inflammatory activity in peripheral blood, because as noted above, these simple and easy to obtain outcomes have strong predictive power for long-term health.

Understanding inflammatory activity from a biobehavioral perspective

As mentioned before, while health relevance of inflammation is well studied, psychosomatic research is more concerned with establishing the links between CNS processes and inflammation. Efforts along those lines have revealed, for example, that higher IL-6 levels are cross-sectionally associated with and predicted by depressive symptoms (Rohleder and Miller, 2008), and associated with chronic stress of caregiving (Kiecolt-Glaser et al., 2003; Rohleder et al., 2009)). Research in the context of inflammatory reactivity, a phenomenon only recently assessed, shows that IL-6 but not CRP responds to acute psychosocial stress and that most individuals' IL-6 stress responses do not habituate (von Kanel et al., 2006). On the contrary, we recently found sensitization upon repeated exposure, and a significant relationship of sensitization with undesirable psychosocial conditions such as lower subjective social status, and lower meaning and purpose in life (Rohleder et al, in prep.).

All of these findings clearly suggest CNS influences on inflammatory (re-)activity. However, in order to develop strategies to control inflammatory processes and subsequent diseases, we need to go a step further and not only understand under which conditions but also exactly how the CNS affects relevant tissues.

> *"Central to this task is first and foremost the accurate quantification of inflammatory activity."*

Pathways between the CNS and peripheral inflammation

Stress systems, including sympathetic and parasympathetic nervous system (SNS and PNS) as well as hypothalamus-pituitary-adrenal (HPA) axis, are currently the best-described pathways between the CNS and peripheral tissues capable of influencing the production and secretion of inflammatory mediators. This includes effects on inflammatory processes activated through true inflammatory stimuli (e.g. (Sternberg, 2006)). While it is important to differentiate between basal activity versus acute reactivity of stress systems as these states differentially affect inflammatory processes, the opposite is true as well, i.e., the state of the inflammatory system

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can determine the effect stress system mediators will have. Some promising targets for studying CNS effects on inflammation are discussed below along with examples of current findings from acute stress studies.

DNA binding activity of NF-kB

A key factor in inflammatory activation is the transcription factor nuclear factor-kappaB (NF-kB). NF-kB can be activated via three different intracellular pathways and once translocated to the nucleus and bound to DNA, regulates a wide variety of genes, including pro-inflammatory cytokines such as Il-6. Interestingly, glucocorticoids (GCs), the end hormone of the HPA axis, interfere with NF-kB activity in various ways, thereby acting as anti-inflammatory agents. Among the more direct ways, GCs interfere with NF-kB DNA binding activity, examples for more indirect GC effects include GCs stimulation of the expression of the NF-kB inhibiting protein I-kappaB. Importantly, direct effects of GCs on NF-kB-DNA binding activity have been found for the increases seen in GC levels in response to acute psychosocial stress as well (Wolf et al., 2009). Contrary, norepinephrine secreted upon SNS activation has been shown to stimulate NF-kB-DNA binding activity in non-activated leukocytes via adrenergic receptor-initiated pathways (Bierhaus et al., 2003). However, catecholamines can also suppress inflammatory activity in the context of mitogen-stimulated leukocytes (Elenkov et al., 2000), providing an example of inflammatory status-dependency of stress effects.

NF-kB-DNA binding activity is usually assessed by electromobility shift assays (EMSA). While being the method of choice, not every laboratory is set-up and equipped to implement this method. Fortunately, non-radioactive ELISA-based methods have become available, and some of our colleagues have successfully used these to show stress-effects on NF-kB activity (see for example Thaddeus Pace's work).

Expression of pro- and anti-inflammatory intracellular proteins

While assessing NF-kB-DNA binding activity provides a functional and in a way composite measure, another approach may be to actually assess the various signals and factors that initially played into this measure, allowing to identify potential key switches in the initiation of this process. For example, Rohleder et al (2009) used targeted assessment of specific proteins (NF-kB, IkB, GRa, GRb, etc) to describe changes in those proteins associated with severe chronic stress (caring for a brain cancer patient) and to develop a potential cascade of events leading up to the negative health consequences seen under those conditions.

However, the complexity, multitude, and interdependency of pathways involved in intracellular stress signal transduction as well as inflammatory activation make it important to not only choose such targets wisely, but also to be aware of the limitations of the various methods available to assess those targets. One solution might be to use microarrays to detect any possible change in gene expression in combination with statistical approaches that actually allow interpretation of the complex set of findings, for example, by grouping changes in gene expressions within known pathways or DNA binding motifs (see Steve Cole's work).

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In vitro assessment of stress system control of inflammation

Alternatively, a lot can also be learned from functional assays that test how well the stress mediators of choice can down-regulate a mitogen-activated inflammatory response. Functional assays like these can be done with simple, untreated venous blood and are reasonably straight-forward to perform in a well-equipped laboratory. The major advantage of these approaches is to use a model system of mitogen-induced inflammatory activation to test the ability of stress systems to control this activated inflammatory response, without exposing human research participants to actual infectious stimuli.

One example for what we can learn using this approach comes from the context of chronic stress and depression. We struggle to explain why unaltered or increased plasma GC availability can co-exist with inflammatory disinhibition. Studies including a measure of GC sensitivity have repeatedly shown that chronic stress induces decreases in GC sensitivity, which together with unchanged cortisol, might be sufficient to permit disinhibition of inflammatory cytokine production (Miller et al., 2002; Rohleder et al., 2009).

We have piloted methods to extend this successful approach to the regulatory efficiency of ANS mediators, and preliminary data shows that catecholamine sensitivity is responsive to acute and chronic stress, showing a resistance to SNS signaling in chronic stress, similar to GC sensitivity.

Summary and conclusions

Taken together, we think that psychosomatic research can benefit from assessing peripheral inflammation in relation to or in consequence of psychosocial states, even more so if directed at understanding why for example inflammation is upregulated in chronic stress. Given the role of inflammation in so many pathophysiological processes, and the emerging role of psychosocial processes to control – or fail to control – inflammation, focusing our efforts on better understanding these pathways bears great potential for our field. There are a number of open questions, many of them related to the fact that we are often limited to peripheral blood and the immune cells within, but many of these might be addressed by creative solutions, for example by targeting specific markers secreted by specific cells.

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American Psychosomatic Society

2012 Annual Meeting
March 14 - 17
Athens, Greece

Symptoms and Patient Reported Outcomes

About the Society
The American Psychosomatic Society is primarily comprised of PhD and MD investigators and clinicians with backgrounds in differing specialties who collaborate on mechanistic research and clinical trials.

About the Meeting
The premier international psychosomatic and behavioral medicine meeting is attended by over 500 researchers and clinicians from around the world and held in different locations across the globe to reflect the geographic diversity and international composition of the membership.

About the Program Format
The majority of the program features symposia, oral paper and poster presentations which are selected from submitted abstracts. Also included are award lectures, invited plenary sessions and symposia given by internationally-recognized scientists and clinicians, pre-conference didactic workshops, informal roundtable discussions and ample time for informal networking.

About the Program Content
This year's theme "Symptoms and Patient Reported Outcomes" emphasizes outcomes such as distress, pain, fatigue, and cognitive dysfunction, drawing on the basic and clinical focus of the Society while capitalizing on expertise in evidence-based primary prevention and public policy.

For abstract submissions, please visit:

www.psychosomatic.org

Topics being highlighted include:

Cardiovascular disease
Diabetes
Obesity
Psycho-oncology
Sleep and fatigue
Pain
Cognitive difficulties
Distress/depression/emotion
Psychoneuroimmunology
Pregnancy
Relationships
Mindfulness

Abstract Deadline: October 11, 2011

The APS Meeting
.... advancing the scientific understanding and multidisciplinary integration of biological, psychological, social and behavioral factors in human health and disease.
.... integrating the fields of biological, psychological, social and behavioral science in education and improved health care.
.... providing a forum for a multidisciplinary understanding of how mind and body interact in the maintenance of health and the causation of disease.

Getting to Know You Interviews with . . .

Stephen Manuck, PhD

Dr. Steve Manuck is the Distinguished University Professor of Health Psychology and Behavioral Medicine in the Department of Psychology at the University of Pittsburgh. He completed his doctorate in Psychology at Vanderbilt University in 1974 before going on to conduct landmark studies in cardiovascular reactivity and atherosclerotic risk and more recent work in gene-environment interactions.

1. Tell us a little about your current research interests and where you see it going over the next 5 years.
For a number of years we were focused on central nervous system serotonergic function as a neurobiologic mechanism of potential influence on multiple, often correlated, behavioral and biological risk factors for heart disease. This work stemmed from studies in which we observed diminished (or dysregulated) serotonergic responsivity – as assessed with neuropharmacologic challenges – to be associated with intercurrent depression, heightened aggressiveness, physical inactivity, and elevated blood pressure. Subsequently, we found reduced brain serotonergic responsivity similarly related to central adiposity, insulin resistance and presence of the metabolic syndrome, and extent of preclinical vascular disease. We always assumed that sertonergic neurotransmission and its variability among individuals (as well as other transmitter systems) affect behavior and CVD risk by biasing activities of functional brain circuitries subserving critical psychological and biological processes, such as emotional experience, behavioral motivation, metabolic function, and cardiovascular regulation. For this reason, we recently redirected our research program from neuropharmacologic methodologies to functional neuroimaging.

Here, we are studying two neural systems of behavioral relevance. The first is a corticolimbic defense system that encompasses interactions among several prefrontal and limbic structures. This network underlies detection of environmental threats and the generation, expression, and regulation of emotion and its autonomic and neuroendocrine correlates. The second is the mesocorticostrial reward system, which centers on dopaminergic projections from the midbrain ventral tegmental area to forebrain structures implicated in the processing of reward-related stimuli and activation of goal-directed behavior. Promising preliminary work suggested two specific hypotheses, for instance, that we are now pursuing: 1) that as a component of emotional processing, reactivity of the amygdala underlies, in part, individual differences in the propensity to experience states of negative affect and promotes risk for heart disease via correlated alterations in autonomic and neuroendocrine activity (a "stress" pathway); and 2) that as a component of appetitive motivation, reactivity of the ventral striatum engenders impulsive choice and abets disease risk via health-impairing behaviors associated with heightened reward sensitivity and their biological sequelae. As part of our on-going program of genetic research, we also seek sources of genetic variation that may interact with contemporaneous or early life environmental exposures to modulate patterns of activation and connectivity within these behaviorally informative circuitries.

Finally, I cannot mention the work we are doing without acknowledging who the "we" are. One of the great benefits of Pittsburgh is the opportunity to work with so many wonderful collaborators, in my case including notably Matt Muldoon, Dick Jennings, Karen Matthews, Sheldon Cohen, Tom Kamarck, Pete Gianaros, and Ahmad Hariri (three of whom, by the way, are past APS presidents).

2. You and Jay Kaplan conducted a landmark study where you examined threat of capture on cardiovascular reactivity and atherosclerotic progression in non-human primates. Which of you was better in the role of the antagonizer and why?
Jay acted as the provocative stimulus in the several studies we published on behaviorally induced cardiac reactivity and atherosclerosis, parading before the animals with arm raised and wearing a large "capture glove." I always thought this was a clever maneuver to create a reproducible, acute stressor for an animal apparently incapable of serial subtraction. Of course, we needed to standardize the stimulus, which accounts for relying on Jay to perform the threatening display. As you may know, Jay has an imposing presence, and I was not surprised to find that our high-reactive animals responded markedly even to a comparison condition in which Jay again paraded before the monkeys, but absent the glove. When I pass the monkey enclosures myself, on the other hand, I tend just to elicit mocking stare-threats and screeches (which I imagine as monkey laughter).

> *"I think it has been shown satisfactorily that an elevated CV reactivity is associated with the presence and progression of preclinical atherosclerosis."*

3. Do you think the hypothesis that cardiovascular reactivity is a plausible mechanism leading to disease is a settled issue?
I think it has been shown satisfactorily that an elevated CV reactivity is associated with the presence and progression of preclinical atherosclerosis. Whether it predicts incident CVD is not yet clear, although some population-based epidemiologic studies with reasonable reactivity protocols (such as the Kuopio Ischemic Heart Disease Risk Factor Study) may be far enough out to begin testing for event prediction and mortality. Reactivity's "plausibility" as a mechanism in the etiology of atherosclerotic CVD requires evidence of a different kind, particularly from studies in the pathobiology of atherosclerosis and of processes surrounding the precipitation of clinical events. I am not a vascular biologist, but cognizant that speculation regarding the role of reactivity in disease pathogenesis must be wedded to an evolving understanding of arterial wall biology, which has advanced far beyond the days when we could imagine reactivity promoting atherosclerosis just via hemodynamic perturbations propagated at predilection sites for lesion development.

4. Do you have an academic crush – someone whose work fascinates or inspires you?
There are more scientists whose work I admire than I can list! Looking back, though, there is one person, in particular, whom I would cite as inspiration. When I first met Jay Kaplan, I was Head of the Division of

Medical Psychology for the Psychiatry Department at Wake Forest (then called Bowman Gray School of Medicine), though I was destined to leave soon for Pittsburgh. Jay was in the Department of Comparative Medicine, which was chaired by Tom Clarkson. Tom was (still is) one of the foremost atherosclerosis researchers in the world and perhaps the most open-minded scientist I've known. On Saturday mornings, Tom would meet for several hours with Jay and me and discuss every manner of topic relating to his passion, coronary artery disease – not just the biology, but granting activities, the NIH, recent papers and findings, or new directions in the arteriosclerosis center's research programs. He was open to any new idea, including (which was critical to our work) the novel use of monkeys for modeling individual differences analogous to behavioral risk factors in human research. And he opened my eyes to big, programmatic science in ways I had not known from my earlier years in a small-lab Psychology department. Indeed, in large measure I trace my own aspirations and approaches to science to the many lessons I learned from Tom Clarkson on those sparkling Saturday mornings that he so generously shared with us in the winter and spring of 1979.

5. Assuming for a moment that you are not always doing science, do you have any guilty pleasures?
I wish I could offer something exotic, like collecting early American botanical prints or writing haiku under tutelage of a Zen master or reading exciting new literature emerging from the old Soviet republics. But alas, my off-work hours are spent in ordinary suburban luxuries – getting the car washed, carpooling to swim meets, watching my daughter grow up. In a few years, however, Carolyn will be off to college; perhaps then I can begin tackling those young novelists from Belarus.

6. If you could pick the location of the next meeting site where would it be?
Paris, definitely.

7. Pittsburgh is famous for Primanti Brothers sandwiches (meat, fixins, and fries all *in* the sandwich) which is located a very short distance from your office. Be honest, how often do you go and do you have any recommendations?
I'm afraid Primanti Brothers is gone now*, but at last we have a wonderfully plush faculty club with café, bar, and restaurants located just a block from the lab. That's my new haunt.

**Note: the editor was quite disturbed by this news. However, in follow-up, Dr. Manuck reported that only the Oakland location closed and other locations have opened.*

> *"… lasting contributions are probably to be found in the students whom one helped launch and whose own contributions will extend further into the future. And in this regard, I have been blessed…"*

8. At the end of the day, what contribution do you hope you've made to the field?
I hope the "end of the day" is still pretty distant, and like many, any previous contribution always seems inconsequential next to the importance I fantasize for whatever I'm working on presently. Coincidentally, I was discussing this question with a friend, the geneticist Robert Ferrell, just the other day. Bob pointed out that few scientists are much remembered a decade out, and this is probably as it should be for any vigorously advancing field. That said, lasting contributions are probably to be found in the students whom one helped launch and whose own contributions will extend further into the future. And in this regard, I have been blessed with a number of terrific students who also claim an intellectual home in APS and psychosomatic research, including Kevin Larkin, Shari Waldstein, Anna Marsland, Liz Bachen, and Jeanne McCaffery, and most recently, Maria Bleil and Karen Petersen.

9. Any advice to the young professionals out there?
Advice is best brief, as in Kurt Vonnegut's apocryphal one-word commencement address: "sunscreen." Because scholarship is only recognized by its communication, here's my abbreviated advice: "write (often and well)."

John Burns, PhD

Dr. John Burns is a longtime devotee of APS. He completed his doctorate in clinical psychology at the State University of New York, Stony Brook in 1992 and is currently professor in the Department of Behavioral Sciences at Rush University Medical Center in Chicago.

1. Tell us a little about your current research interests.
My interests are broadly focused on medical patients with chronic painful conditions. In one line of research, we are examining therapeutic mechanisms in psychosocial chronic pain treatments to address the question: do different treatments produce the same overall effects via distinct mechanisms, or do different treatments achieve benefits through common underlying mechanisms? Our results so far suggest that while mechanisms thought to be specific to certain approaches (e.g., CBT and pain catastrophizing changes) may work in those approaches to bring about clinical change, they may also "work" in other treatments. In a second line of research, we focus on interpersonal processes and the impact on chronic pain patient emotional and physical functioning. We propose that chronic pain symptoms elicit negative spouse responses, and that such responses then worsen and maintain symptoms; a vicious spiral, which leads to poor adjustment.

However, my primary line of research examines the mechanisms by which the regulation of anger impacts acute and chronic pain. In particular, Phil Quartana (my student at the time) and I hypothesized that suppression of anger during anger provocation will paradoxically increase the accessibility of anger and irritability during subsequent pain induction, thus magnifying perceived pain intensity. Borrowing the "white bear suppression" paradigm from Wegner, we demonstrated this to be the case, including results showing that observed pain behaviors during a natural movement task were greater among low back pain patients who suppressed anger than among those who did not. Also, greater reactivity of the lower paraspinal muscles during anger provocation among those who suppressed partly

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explained differences in frequency of later pain behaviors. In collaboration with Mark Lumley, we are extending this model in a current study to determine whether expression of anger following suppression can "undo" the ironic effects of anger suppression on later pain and pain behavior among low back pain patients. This line of research also includes the effects of anger expression. We (Steve Bruehl at Vanderbilt University) hypothesized that regulating anger through overt physical and verbal expression may affect pain sensitivity as much as anger suppression, but through different mechanisms. Using an opioid-blockade paradigm (naloxone vs placebo), we discovered that people with a predominant orientation toward expressing anger (high anger-outs) may experience increased pain sensitivity because of a dysfunction in their endogenous opioid systems. Additional findings have led us to our current work testing the hypothesis that difficulties modulating strong negative emotions (high anger-out), exaggerated cardiovascular reactivity and sensitivity to pain rest on a common substrate of dysfunction in an endogenous inhibitory system (endogenous opioids).

"… I sat in on a Health Psychology course offered by the psychology department, and one day the professor said in reference to stress, social support, Type A Behavior and health, 'to make real progress and achieve legitimacy, this field will have to put physiological teeth in our psychosocial gums.'"

2. How did you get started in this field?
In the early 80's, I was a graduate student pursuing a PhD in political science, and was faced with the choice of working for a government agency as a public policy analyst or being an itinerant lecturer/gypsy following adjunct teaching positions across the country. Despairing of this dilemma and in search of something a bit more imaginative and rewarding, I sat in on a Health Psychology course offered by the psychology department, and one day the professor said in reference to stress, social support, Type A Behavior and health, "to make real progress and achieve legitimacy, this field will have to put physiological teeth in our psychosocial gums." A watershed moment for me, and on the spot I switched allegiance to people pursuing these kinds of questions.

3. Where do you see your research area going in the next 10 years?
I fear the areas in which I conduct research may be guided to an uncomfortable extent by the winds of scarce funding. I hope they will head in directions guided in equal measure by theory and intriguing new findings. Regarding anger regulation and pain, work is already exploring physiological mechanisms from the point of view of brain imaging. I don't have to quote my colleagues in saying that opening up the brain to scrutiny is vital for our fields to progress, and I'm hoping to be included in the journey (I would like to manipulate anger suppression and pain induction in an fMRI environment, for example). I hope we continue to notice each other's work in seemingly disparate areas, and forge links such as those beginning to build between people studying depression, heart disease and immune function. Although the importance of social support and relationships has certainly been viewed as important in the pain literature, I hope that additional attention will be directed toward how actual relationship processes affect adjustment to chronic pain (I know it is difficult to study couples in action, but…), and how maladaptive (dyadic, family) processes may be curtailed through intervention. I must mention genes. Underlying many of our moderate or weak associations among emotions, cognition and pain may be genotype "moderators" that help localize the most toxic effects. Finally, I am really excited about systematically investigating therapeutic mechanisms in psychosocial interventions not only for chronic pain, but for other conditions impacting physical health. In recent years, it has become difficult to read a report on an RCT and NOT see at least some mention of mechanisms. This I anticipate will increase both in frequency and in the methodological sophistication in which questions of mechanism will be evaluated.

4. Describe a perfect day for you.
I refer here to a perfect day at work. No patients. Did I say that out loud? Snow, rain, heat, or gloom of night notwithstanding, a perfect day is when results of analyses of hard won data either somewhat support hypotheses or lead in new but reasonably coherent directions. That is, when we discover something.

5. What would be your biggest splurge if you suddenly won the lottery?
Let's assume we're talking in the $100 million range. After stuff like setting up a trust fund for my children, paying off mortgage, gifts for family and friends, I would…. well, truth be told, I would "retain" the services of an fMRI research group to teach me about, and help me study connections among anger regulation, pain and brain activity.

"… expecting a Cub team that shows promise all year to actually excel in the playoffs but then blows it … well, this simply cannot be good for you."

6. What was the first APS meeting you attended and what do you remember?
It was the 1991 meeting in Santa Fe, New Mexico, and I was still a graduate student. I remember jamming 5 people on low budgets in a cab to get from Albuquerque to Santa Fe, free buffets at all poster sessions (including beer), wandering through a big snow storm with friends and colleagues, the natural beauty of northern New Mexico, and me nervously trying to explain my overly complex poster to senior investigators who'd been drinking free beer.

7. As one who appreciates good science, do you have a favorite paper that you wish you wrote/Study you wish you had done?
Actually, I'm not dead yet. So, I have time left to write a wonderful paper and/or to conduct a fabulous study. Let's hope.

8. What one piece from your career should people read?
I really like, Burns JW, Quartana P, Gilliam W, Gray E, Matsuura J, Nappi C, Wolfe B, Lofland K (2008). Effects of anger suppres-

sion on pain severity and pain behaviors among chronic pain patients: evaluation of an ironic process model. *Health Psychology, 27*, 645-52, and its compendium piece, Burns JW, Quartana PJ, Gilliam W, Matsuura J, Nappi C, Wolfe B. (in press). Suppression of anger and subsequent pain intensity and behavior among chronic low back pain patients: the role of symptom-specific physiological reactivity. *Journal of Behavioral Medicine*. Together, these capture my favorite study methods: a blend of strict laboratory control of conditions with tests of emotional/physiological mediators.

9. If they made an APS movie, who would you like to see play you?

I cannot recall whether Jack Nicholson has played a role as a mad scientist, but I'd like to see him get the nod.

10. Two Chicago questions for you. First, any evidence that being a Cubs fan is a risk factor for health?

One answer is "it must be so." Cheering for a perennial loser, expecting a Cub team that shows promise all year to actually excel in the playoffs but then blows it, or chokes, or falls prey to some random mishap, having your heart shattered and the pieces ground into the turf at Wrigley Field every 5 years or so… well, this simply cannot be good for you. However, there is another kind of Cub loyalty that I believe confers health benefits. This kind of loyalty is shown by people who enjoy the game for its own sake, who praise the strengths of players and coaches but forgive their weaknesses, who do not expect the Cubs to win the series (although remaining guardedly hopeful) but experience each strikeout, homerun, fielding error as crucial parts of the rich tapestry of our national pastime. These mindful Cub fans – at latest count, there were exactly 5 of them — will live forever.

Second, where would a real Chicagoan eat pizza?

According to folklore, a real Chicagoan should eat only thick crust pizza. According to many years of observation by this humble pizza fan, however, it seems that MOST pizza consumed even here is of the thin-crust variety (please note that even thin crust in Chicago is at least 3 times thicker than New York style pizza). That said, there are enough establishments serving thick crust pizza and heralding their own unique differences and embellishments that sectarian strife has in fact arisen among Chicago pizza eaters. At the risk of alienating fellow citizens, I must confess that my favorite thick crust pizza is served at Lou Malnati's (now a chain, but when I grew up it was a single restaurant in the "border-suburb" of Lincolnwood). It is best known for spreading a ½ inch thick LAYER of sausage on pizzas, as opposed to the more typical and less brave practice of dropping little sausage nuggets.

***Practical Science**, continued from page 5*

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Awards: 2011 Herbert Weiner Early Career

Naomi I. Eisenberger, PhD, Assistant Professor of Psychology, University of California, Los Angeles

It was truly an honor to receive the 2011 Herbert Weiner Award and especially rewarding to receive this honor from the American Psychosomatic Society. Although my work tends to straddle several different fields, my very first research interest centered around trying to understand how psychological experience could impact physiology and health—a question that is central to many of the members of this society. And while my current work focuses on exploring the neural substrates underlying social rejection and connection, this line of research was largely motivated by trying to understand why social relationships have such a profound effect on health and well-being. Here, I reflect on the unexpected and fortuitous path that led me to this line of research and what we have learned, scientifically, along the way.

When I first learned about the research showing strong links between social relationships and health, I wanted to understand why. For many researchers, this very same question led to an interest in exploring the downstream health consequences of having or not having strong social relationships; however, for me, it led to an interest in the upstream correlates of social relationships, which may ultimately provide insight about how social relationships link to health. Indeed, my first question in this domain was a rather simple one: Why does rejection hurt? (Although at the time, the question was not stated so succinctly; it was more along the lines of "why will we do almost anything to avoid social rejection and when we are rejected, why does it make us feel bad?")

To explore this, we decided to investigate what was going on the brain in response to social exclusion. Fortunately, we had a paradigm that could be used to socially exclude individuals within the fMRI scanner—a non-trivial challenge, as participants are not able to speak, move, or be in the presence of others while in the fMRI scanner. In our study, participants played a virtual ball-tossing game, supposedly with two other participants. Here, they viewed a computer screen where they saw cartoon representations of the two other players and their own computerized hand, and they had the ability to toss an animated ball amongst themselves by pressing one of two buttons. Of course, the task was rigged; in one round of the game, participants were included by the other players (who were actually computer-controlled) and in another round of the game, participants were excluded when the other players stopped throwing them the ball.

Although we had no definitive hypotheses at the time about what we would see neurally in response to social exclusion, the pattern of data revealed something very interesting. In response to social exclusion, our participants looked, neurally, like they were experiencing physical pain.[1] Specifically, they showed increased activity in the dorsal anterior cingulate cortex (dACC) and the anterior insula—the two regions that show up most frequently in physical pain studies and whose neural activity correlates with how distressing or unpleasant a painful stimulus is reported to be. In fact, there was such a striking similarity between what we observed in response to social exclusion and what others have found in response to physical pain, that neither my fellow graduate student—who was analyzing a neuroimaging study of pain in irritable bowel syndrome patients—nor I could easily distinguish between the two studies by simply looking at the patterns of neural activity; they looked so similar.

It was this finding that propelled a line of research looking at the similarities between physical pain and 'social pain'—the painful feelings following social rejection or loss. Indeed, this initial finding led to the hypothesis that physical and social pain may rely, in part, on some of the same underlying neural circuitry.[2,3] The idea here is that because humans are so dependent on others (caregivers) for so long, the social attachment system, which ensures proximity to a caregiver, may have 'borrowed' the pain signal to ensure social closeness. In other words, if being separated from a caregiver is such a threat to survival, what better way of reducing the likelihood of social separation than by making it painful? This basic premise led to a series of studies that tested whether experiences of social pain rely on pain-related neural regions as well as what the consequences of such an overlap might be.

In the work that ensued, we have shown that how rejected a person feels in response to socially painful experience correlates directly with activity in these pain-related neural regions.[3] We have also observed that the kinds of socially painful events that can activate these pain-related neural regions spans a wide range of experiences: from viewing disapproving faces[4] or receiving negative social feedback[5] to reliving a relationship break-up[6] or remembering a lost loved one.[7]

We have also examined some of the expected consequences of this physical-social pain overlap. For example, we have shown that individuals who are naturally more sensitive to physical pain are also more sensitive to social rejection (both behaviorally and neurally).[8,9] We have also demonstrated that factors that increase or decrease one kind of pain affect the other in a similar manner. Along these lines, we have shown: 1) that inflammatory activity, known to increase physical pain sensitivity, can also increase social pain sensitivity,[10,11] 2) that social support, in addition to reducing social pain, can also reduce physical pain,[12,13] and 3) that Tylenol, a common physical painkiller, can double as a social painkiller.[14]

> *"And while my current work focuses on exploring the neural substrates underlying social rejection and connection, this line of research was largely motivated by trying to understand why social relationships have such a profound effect on health and well-being."*

In our current work, we are attempting to reconnect this line of work with physiological and health-related responding by examining whether this pain-related neural circuitry contributes to the relationship be-

tween social ties and health. For example, we are in the process of examining whether greater pain-related neural activity in response to negative social experiences (social rejection, evaluation, stress) is associated with greater cortisol and inflammatory responses,[15] which may have health implications. Identifying the specific neural mechanisms that link social relationships with physiological or health-related outcomes may provide us with better leverage in understanding the specific array of social factors that can influence health.

I would like to note that this work would not have been possible without the training, mentorship, and guidance of a whole array of smart and generous individuals, including Matthew Lieberman, Shelley Taylor, Margaret Kemeny, Christine Dunkel Schetter, Shelly Gable, and Michael Irwin. I have learned a great deal from each of these individuals and greatly value the role that they have played in shaping my thinking as a researcher and scientist. I would also like to thank all of the impressive members of APS, who have inspired a generation of scientists, like myself, to try to unravel and understand the mechanisms that link social relationships with health.

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SAVE THE DATE

UK Society for Behavioural Medicine
7th Annual Scientific Meeting
December 13-14, 2011

STIRLING , SCOTLAND

The UK Society for Behavioural Medicine (UKSBM) 7th Annual Scientific Meeting in association with the National Prevention Research Initiative (NPRI) will take place on December 13th and 14th, 2011 at the University of Stirling, Scotland. The theme of the meeting is *Motivating, Enabling, and Prompting Behaviour Change for Health.*

Keynote Speakers include:

Professor Theresa Marteau, University of Cambridge, King's College London.
The Automaticity of Being: Implications for Changing Behaviour to Improve Population Health

Professor Kavita Vedhara (Psychology), University of Nottingham.
What has stress got to do with it…?

Professor David Blanchflower (Economics), Dartmouth College & National Bureau of Economic Research, University of Stirling, University of Munich, University of Bonn.
Happiness, bio-markers and health

Professor Christopher Butler (Primary Care), Cardiff University.
Promoting behaviour change in primary care: clinical and research challenges

The deadline for receiving abstracts for oral presentations (including Discussions and Workshops) is Friday, August 12th, 2011. The deadline for receiving abstracts for poster presentations is Friday, October 7th, 2011.

Further information about the conference is available on the web at http://uksbm.org.uk/asm2011/. Information about UKSBM is available at http://uksbm.org.uk.

Update from the APS Liaison Committee

Benjamin Natelson, MD & Urs Nater, PhD, Co-Chairs

The Liaison Committee serves a liaison function by which the society fosters closer relationships with other general or specialist medical societies, on one hand, and with other psychological or behavioral medicine societies on the other. Specifically, the Committee functions by encouraging APS members who are active in other general or specialized medical or psychological societies to work on developing bridges between APS and those societies. Operationally, this translates into APS supporting one or several APS members to develop and then participate in workshops and symposia at those non-APS societies. The Committee reaches out to those APS members who are involved with other organizations and are encouraging them to suggest activities in other societies in which APS could become involved. To date, our society has close connections to many societies, such as the International Society for Behavioral Medicine and the Society of Psychophysiological Research, thanks to the collaborative effort of the Liaison Committee and individual APS members.

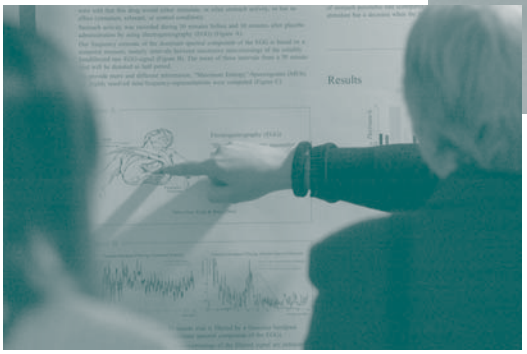
In the future, we will reach out and plan to work more closely together with the Sleep Research Society, the American Heart Association, the International Society of Psychoneuroendocrinology, the European Association for Consultation-Liaison Psychiatry and Psychosomatics, and the European Psychosomatic Research Network, to name just a few. Of course, if you have ideas of which societies could benefit from APS' message (and the other way round!), please contact us.

> *"Specifically, the Committee functions by encouraging APS members who are active in other general or specialized medical or psychological societies to work on developing bridges between APS and those societies."*

Since earlier this year, we also have given thought to other ways of carrying the message of APS to researchers who are either unaware of our society or not yet members. Council approved our moving into a new direction of liaising activities, i.e. actively focusing on attracting non-APS members to our meetings rather than providing funds to APS members "spreading the word" to other societies. Going into this new direction will allow Liaison Committee members to work together with both the Membership Committee and the Program Committee. This approach could result in having prominent non-APS researchers attend our meeting and interact with our membership, and we will encourage them to join APS itself.

Going together with this approach, it seemed critical to identify areas of research to specifically focus our efforts. These areas should be under-represented in our society and they should be of national and international relevance. After extensive discussion within the Committee and the Council, we reached consensus to focus on the following three areas: Oncology, Diabetes & Obesity, Sleep & Fatigue. Toward attaining that goal, we have used the latter focus area to forward the goals of the Liaison Committee. We thought that researchers with interests in sleep and fatigue clearly had interests that dovetailed with those of APS. So, we have worked with members of the Program Committee to organize a symposium on Sleep & Fatigue to be presented by non-APS members. We are also working on identifying individuals spearheading efforts in Oncology and Diabetes & Obesity. Again, your input on these efforts will be most welcome!

In closing, we wish to ask APS members who are either involved in other societies whose interests overlap with those of APS or who are working in one of the three focus areas that are currently underrepresented in our society to please contact either of us in order to help APS move its mission ahead.



2012 Athens Update!

Suzanne Segerstrom, PhD on behalf of the Program Committee

Greetings from your Program Committee! We are hard at work to make sure that the scientific program in Athens is of the very highest quality, which is of course what we all have come to expect from the APS meeting. The theme of the meeting is **Symptoms and Patient-Reported Outcomes**. This theme cuts across our disease and disciplinary "silos" and challenges us to understand subjective experience from biological, psychological, and social perspectives. It also invites us to consider diseases, disorders, and conditions that have patient-reported outcomes such as pain, fatigue, cognitive dysfunction, and distress at their core.

> *"The theme of the meeting is **Symptoms and Patient-Reported Outcomes.** This theme cuts across our disease and disciplinary "silos" and challenges us to understand subjective experience from biological, psychological, and social perspectives."*

To give you a preview of some of the scientific program, we are looking forward to addresses by **Simon Wessely, MD,** of King's College London and **Yael Benjamini, PhD,** of Tel Aviv University. Dr. Wessely is an international expert on chronic fatigue syndrome, Gulf War Syndrome, and trauma, particularly trauma arising from combat. He will be giving a talk entitled "20 years in and out of chronic fatigue". If you have ever written about self-rated or subjective health, you have almost certainly cited Dr. Benjamini's seminal 1997 review of studies in which self-rated health predicted mortality. She will be providing the most current view on how subjective feelings of health affect future health outcomes in her talk, entitled "What can we learn from what people say about their health: Self-rated health, morbidity, and mortality." Our own Past President **Joel Dimsdale, MD,** is chair of the DSM-V Somatic Symptom Disorders workgroup, and he and colleagues will be presenting a symposium with an update on how these complex disorders of subjective ill health should be assessed and diagnosed.

But the meeting can't be successful without you, because submitted programming is the essence of how we share science with each other. Please consider submitting an abstract or symposium in response to the Call for Abstracts. Degnon Associates has been hard at work streamlining our abstract submission process (particularly the CME portion). When you're thinking about what to submit, keep in mind not only our meeting theme, but also the Society's core targets, such as cardiovascular disease, psycho-oncology, and sleep. We look forward to submissions in traditional APS domains such as psychoneuroimmunology and social relationships as well as in newer domains such as pregnancy. Is there something you've always wanted to see on the program? Submit it! Or encourage someone who is doing that work to submit an abstract for the meeting. Encourage your colleagues and students to plan to come to the meeting and to submit abstracts. The more, the merrier.

In the next few months, while you are working on your abstracts, the committee will be developing potential topics for workshops and round tables. If you have a good idea or a request for a workshop or round table topic, please feel free to email me at segerstrom@uky.edu or contact any of the program committee members (go to http://www.psychosomatic.org/about/index.cfm and click on Program Committee to see a complete list.)

> *"But the meeting can't be successful without you, because submitted programming is the essence of how we share science with each other."*

The Program Committee can't take credit for the meeting site, but I'll try anyway. The meeting will be held at the Hilton Athens, which is both convenient (25 minutes from the airport and accessible by subway) and beautiful. Go to their website (http://www1.hilton.com/en_US/hi/hotel/ATHHITW-Hilton-Athens/index.do) and check out the photos of the indoor pool, the Pilates studio, and the Acropolis views from the restaurant and bar (and some of the guest rooms!) Conference rates will be available for guest rooms, but maybe you want to splurge on the King Presidential Suite, with more square footage than my first house, a large living room, desk, dining table for ten, bar, home cinema, Jacuzzi, sauna, king-size bed, and kitchen? Maybe you would like to invite the hard-working members of your Program Committee up to the suite for drinks? Well, we won't hold it against you if you don't. Or at least I won't.

If you can tear yourself away from the science/bar/sauna or if you're enjoying a few extra days in Athens, be sure to see the sights, including the Acropolis and the New Acropolis Museum (2 miles from the hotel), the ancient marketplace (agora) and adjacent Roman forum (1 mile), and the National Gallery (across the street).

I look forward to seeing you all in Athens!

Update from the Professional Education Committee: Your Help is Needed!

Serina A. Neumann, PhD and Daichi Shimbo, MD

Two of the **American Psychosomatic Society's Strategic Goals** this year are to (1) focus on developing formal mentoring, training and educational programs throughout membership "lifespan"—formal and informal, and (2) foster dissemination of psychosomatic research. As co-chairs of the Professional Education Committee, we are leading the initiative to improve the understanding of mind-body interactions in health and disease in medical education by developing and disseminating various types of educational materials on our APS website for both members and nonmembers of APS.

We are requesting the following types of materials from you if you are willing to share them with others interacting with our website. All materials will be reviewed by our committee (for content and quality) before being posted on the website. Additionally, you will need to read and approve of the APS website copyright guidelines for these posted materials: http://www.psychosomatic.org/Edres/index.cfm

Types of Educational Material*

- Slides including those from the annual APS conference
- Handouts from annual APS conference
- Lecturecasts of PMIG talks and also selected talks from the annual APS conference (symposium talks, special lectures, etc)
- Reading lists/Reference lists
- Teaching syllabi
- Links to other relevant teaching/educational sites
- Contact list of "master teachers" who would consent to being educational consultants.
- Teaching cases in psychosomatic medicine
- Assessment tool
- Up-to-date material about statistics: state of the art methods & programming related to specific software

**The educational material could have a clinical focus, educational focus, and/or research focus.*

Expected Format of Files

- Powerpoint slides
- Lecture casts (video/audio)
- MP3s (audio only)
- Word documents
- Adobe PDF files
- Other videos

Priority Content Areas

- Mental illness and physical health (e.g. depression & diabetes, anxiety & CVD)
- Psychoneuroimmunology
- Neuroscience, social neuroscience, brain imaging
- Cardiovascular health/psychology (includes depression, anxiety, hostility, social support, etc)
- Stress physiology (ANS, HPA; includes measurement and research design issues)

Please contact us directly if you have any

> *"We are requesting ...materials from you if you are willing to share them with others interacting with our website."*

questions or you have material that you would like to send us.

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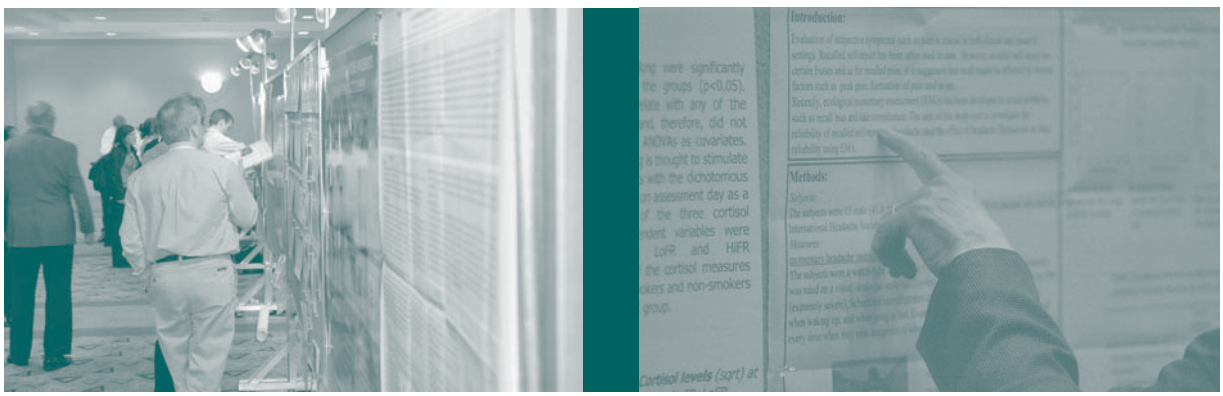

***President**, continued from page 1*

the highest quality science and maintaining its impact factor at nearly 4.

Second, with the administrative guidance of George Degnon and Victoria White, the website for *Psychosomatic Medicine* has been updated to increase the visibility of all journal content, and to facilitate immediate access for downloading. In addition, all members should now be able to receive electronic distribution of the journal contents with active links to all published articles (if you have not yet activated this feature, contact Christine Lusk at Degnon Associates, christine@degnon.org).

Third, the Professional Education Committee, co-chaired by Serina Neumann, PhD and Daichi Shimbo, MD are actively developing, providing, and disseminating educational materials (i.e., lectures from APS or other venues) related to mind-body interactions in health and disease.

Furthermore, this committee in concert with Dr. Kop and Mr. Degnon, is developing a format to link the activities of the Society with the publication of *Psychosomatic Medicine* by using podcasts to highlight respective research. Finally, the the textbook *Principles of Biopsychosocial Medicine*, is moving forward with a shared sponsorship between American Psychosomatic Society and the Editors (Shari Waldstein, PhD, Dennis Novack, MD, Jason Satterfield, PhD, and Matthew Muldoon, MD); the publication of this important volume that will surely guide the field for years to come.

"Increasingly, it is recognized that the cross-talk between disciplines is critical in advancing our understanding of the biobehavioral mechanisms that promote health and prevent disease; in contrast, artificial silos that separate investigators are an impediment to such progress."

Finally, in an effort to enable the American Psychosomatic Society to deliver programs and value more efficiently to the membership, the volunteer efforts of Mary Coussons-Read, PhD and the professional guidance of Degnon Associates is working to bridge the technology gap so that communication technologies are an integral component of the organization's functioning. As noted above, we are quickly moving toward electronic delivery mechanisms to streamline the dissemination of information and education materials to our membership (e.g., podcasts posted on the APS website, *Psychosomatic Medicine* content, APS newsletter, and annual meeting materials). These efforts will take the Society to individual members, wherever they happen to be. Additional efforts are underway to exploit social media (e.g., see APS on Facebook) to enable personalized delivery of information and facilitate networking and collaborative exchanges throughout the year amongst our membership.

In summary, this an extremely exciting time for the American Psychosomatic Society as the volunteer efforts of so many members build a framework for the future to balance programmatic priorities with an organizational business model that continues to secure the Society's stature as a vibrant organization at the cutting edge of biobehavioral medicine.

APS Strategic Goals

The mission of the American Psychosomatic Society is to promote and advance the scientific understanding and multidisciplinary integration of biological, psychological, behavioral and social factors in human health and disease, and to foster the dissemination and application of this understanding in education and health care.

The APS has set forth the following goals in order to achieve its mission:

- To increase Society membership and its diversity with respect to training and field of study, as well as demography
- To develop formal mentoring, training and educational programs throughout membership "lifespan"
- To integrate basic biological and behavioral sciences within APS
- To foster dissemination of psychosomatic research
- To build collaborative and interactive bridges to other societies
- To develop mechanisms for leadership growth
- To establish a 5-year business model to maintain financial health

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APS 70th Annual Scientific Meeting

March 14 - 17, 2012
Athens, Greece

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The APS Newsletter is published 3 times a year by the American Psychosomatic Society with the cooperation of Degnon Associates.

Comments and Suggestions are invited. Remember, this is YOUR Newsletter.

The deadline for submission for our next Newsletter is November 1.

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