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INSTRUCTIONS FOR AUTHORS

Position Paper for the APA Presidential Task Force on IRB Issues

Submitted by the Society for a Science of Clinical Psychology
(Division 12, Section III)

The myriad challenges and difficulties associated with the review and approval of research involving human subjects are being actively debated in a number of venues. A recent survey of our members revealed a number of concerns. Some are related to issues that have been discussed at greater length elsewhere (e.g., Gunsalus et al., 2005); others, to our knowledge, have not been raised before. In this document, we briefly underscore the major issues that have been raised in other settings, and discuss the novel concerns at slightly greater length.

First, we would like to reinforce a number of concerns about IRBs and the human subjects review process that have been raised elsewhere. These include:

- IRB oversight (i.e., the level of scrutiny) could be considerably reduced for many kinds of low-risk human subjects research.
- IRBs often appear to overstep their roles and go beyond their expertise when they attempt to evaluate the scientific merit and validity of proposals. Specifically, the IRB's role should be to protect individuals from harm and not also to decide whether the research value is "worth" the individual's involvement.
- It is important to include a psychologist on IRBs that review psychological science protocols, and a clinical psychologist on IRBs that review clinical science protocols.

Second, our members raised a set of concerns that we have not seen discussed elsewhere, but which is central to the missions of both SSCP and APA. Many of the most critical challenges facing APA involve the integration of science and practice and the tension between scientists and clinicians. APA represents both groups, and therefore works to bridge the differences in their cultures, needs, and agendas. In addition, the growth of the field depends in large part on promoting research that can inform clinical practice, disseminating this work, and encouraging its incorporation into clinical practice. One important means of addressing these issues is by bringing practicing clinicians into the research process by encouraging them to conduct research in their own clinical settings. This could involve analyzing data that was collected for clinical purposes, asking patients to complete additional assessments for research or joint clinical and research purposes, and modifying established intervention approaches for research or joint research and clinical purposes. This could have the dual benefits of enriching the scientific literature and strengthening the links between science and practice.

However, a significant obstacle in accomplishing this goal is that it is not clear how clinicians can or should go about incorporating research into their practices. One issue involves clarifying the circumstances in which external review is advisable or necessary in order to ensure the protection of human subjects. A second issue is that most practitioners do not have access to IRBs or other oversight mechanisms. APA is probably the only organization that can address these issues and provide guidelines and mechanisms for clinicians to conduct research within their practices.

Conversely, current IRB regulations can make it difficult for academicians to collaborate with independent practitioners on research. For example, some IRBs require that the person who "consents" the participants have a position within the University, which effectively precludes a faculty member from working with a private practitioner to use data that s/he might collect in the normal course of his/her practice for research purposes.

We strongly encourage the Task Force to recognize these problems and recommend that APA begin to consider ways to address them.

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Gunsalus, C.K., Bruner, E.M., Burbules, N.C., Dash, L., Finken, M., Goldberg, J.P., Greenough, W.T., Miller, G.A., Pratt, M.G., Iriye, M., & Aronson, D. (2005). The Illinois White Paper – Improving the system for protecting human subjects: Counteracting IRB "Mission Creep". In C.K., Gunsalus (Ed.), Human subject protection regulations and research outside the biomedical sphere. Urbana, IL: University of Illinois Center for Advanced Study.

Feature Article:

What is the Role of Sex Hormones in Risk for PTSD?

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Although men are exposed to more traumatic events than women, women are approximately twice as likely to have a diagnosis of PTSD. This sex difference in prevalence of PTSD may be related to vulnerability for developing the disorder. Lifetime prevalence rates indicating a two to one ratio of women to men with the disorder supports this notion (Breslau et al., 1991). However, sex differences in PTSD *onset* are not the only possible explanation for the two-fold greater prevalence of PTSD in women. These sex differences may also be related to the greater chronicity of PTSD found in women; PTSD has been shown to persist four times longer in women than in men (Breslau et al., 1998).

In the literature on sex differences in PTSD, there has been much attention paid to differences in the types of trauma commonly experienced by men versus women. For example, women are much more likely than men to experience a sexual assault, and this type of trauma is associated with the highest risk of PTSD. However, differences in trauma type or severity do not fully account for these sex differences (Norris et al., 2002). In addition to psychosocial factors, biological factors may contribute.

A number of different biological factors have been explored as they relate to sex differences in PTSD. The presence or depletion of sex-related hormones such as estrogen and progesterone may partially account for the sex difference (Wider & Resnick, 2005). These hormones, which vary between the sexes and across women's menstrual cycle, influence physiological and neurobiological responses to stress (Rasmusson & Friedman, 2002). Circumstantial evidence for a role of sex-related hormones in PTSD development and maintenance is derived from data on the magnitude of sex differences in PTSD across the lifespan. In 18 to 55 year olds, the prevalence of PTSD in women is significantly higher than in men (13% vs. 8%). However, in individuals over 55, rates of PTSD in women (3%) do not differ from men (6%) (Norris et al., 2002). Thus, the limited data available suggest that PTSD risk in women may decrease with menopause.

Very few studies have assessed the neurobiology of PTSD in women, and even fewer studies have examined sex differences in the neurobiology of PTSD. One possible reason for this dearth of research is that women's reproductive status and menstrual cycle makes this type of research challenging to conduct. However, women's fluctuations in sex-related hormones across the menstrual cycle, their increased levels of estrogen and progesterone during pregnancy, and their decreased levels of these hormones during menopause may contribute to sex differences in the development and maintenance of PTSD. Women in different reproductive states appear to have different neurobiological responses to stressors in general (Rasmusson & Friedman, 2002). Levels of estrogen, progesterone, and testosterone that vary with different phases of the menstrual cycle, pregnancy, and menopause affect several stress-responsive systems that have been linked to the pathophysiology of PTSD. Implicated systems include those involving gamma-amino-butyric acid (GABA) the hypothalamic-pituitary-adrenal (HPA) axis-related steroids such as allopregnanolone/ pregnanolone (ALLO) and dehydroepiandrosterone (DHEA), neuropeptide Y (NPY), serotonin, and catecholamines. Despite these compelling observations, there have been no studies to date comparing the neurobiology of PTSD across the menstrual cycle, during pregnancy, and after menopause in women.

In an effort to highlight the promise and increase the prospect of such research, we review below the influence of gender- and reproductive state-related hormones on neurobiological factors of relevance to the risk for PTSD. While an exhaustive review is not possible given limits on the scope of the article, some of the most compelling observations are presented.

GABA. GABA is the brain's primary inhibitory neurotransmitter, allowing the brain to filter out irrelevant information (Krystal et al., 1995). GABA and its neuromodulators have been shown to be associated with PTSD (Vaiva et al., 2004). Because progesterone has important effects on GABA (Epperson et al., 2002), GABA and neuromodulators of GABA may affect menstrual phase and sex differences in PTSD. GABA levels are decreased in healthy women during the luteal phase of the menstrual cycle when progesterone

levels are at their highest, compared with the follicular phase, when they are low (Epperson et al., 2002). Supporting the suspected role of GABA in PTSD, women with mood disorders have been shown to have increased anxiety and reduced sensitivity to benzodiazepines when they are in the luteal compared with the follicular phase (Sundstrom et al., 1997; Wang et al., 1996).

There are also data linking GABA to performance on psycho-physiological findings associated with PTSD. It is well documented that healthy women in the luteal phase of the menstrual cycle, when at least occipital brain GABA levels are low (e.g., Jovanovic et al., 2004), have difficulty filtering irrelevant stimuli. In addition, $GABA_A$ antagonists are related to difficulties filtering irrelevant information (Kodsi & Swerdlow, 1995) and $GABA_A$ agonists are associated with impaired fear conditioning in animals (Wilensky et al., 1999). These data are relevant to PTSD because difficulty filtering irrelevant information may be related to the reexperiencing symptoms and fear conditioning is relevant to the development and maintenance of PTSD symptoms.

Other progesterone-influenced neuroactive steroids that modulate effects of GABA may also be relevant to the pathophysiology of PTSD. DHEA, for example, is an androgen with negative modulatory effects at brain $GABA_A$ receptors. ALLO is the most potent and selective positive modulator of the effects of GABA at brain $GABA_A$ receptors (Rasmusson et al., 2006). Women with PTSD have increased plasma DHEA reactivity and decreased cerebrospinal ALLO levels (Rasmusson et al., 2006, 2004).

HPA axis. Cortisol, as a measure of HPA axis activity, has received much attention in PTSD. However, in individuals with PTSD, the pattern of basal cortisol is inconsistent, with some studies showing lower (e.g., Mason et al., 1986; Yehuda et al., 1995), the same (e.g., Kosten et al., 1990; Mason et al., 2002) or higher (e.g., Rasmusson et al., 2001) cortisol levels in individuals with PTSD as compared with healthy controls. Although there are mixed results regarding the level of basal cortisol in PTSD, generally, the pattern in premenopausal women and postadrenarchal girls is more consistent, showing high cortisol levels and reactivity in those with PTSD, particularly in those with co-morbid depression (Rasmusson et al., 2001; Young & Breslau, 2004), which is believed to constitute more severe PTSD (Breslau et al., 2000). Further, the only studies that have found low or comparable basal urinary cortisol in PTSD participants, compared with controls, have been those involving men alone or post-menopausal women (Rasmusson et al., 2004).

Sex-related hormones have been shown to be related to HPA axis activity. Increased cortisol reactivity has been observed during the luteal phase, compared with the follicular phase in healthy women (Kirschbaum et al., 1999). This menstrual phase difference may be due to the effects of progesterone, estrogen, and/or testosterone, because all have been shown

to affect HPA axis activity (Roca et al., 2003). For example, progesterone enhances HPA axis activity (Roca et al., 2003). The influence of progesterone on HPA axis activity may be due to progesterone's antiglucocorticoid properties (Rasmusson & Friedman, 2002).

In contrast to the data supporting progesterone's positive relationship with HPA axis activity, there are conflicting data on the relationship between estrogen and HPA activity. Some studies support a positive relationship between estrogen and HPA activity (see Charney, 2004; Rasmusson & Friedman, 2002, for reviews). Other more recent studies have found that estrogen may suppress HPA activity (see Charney, 2004; Rasmusson & Friedman, 2002, for reviews), perhaps through effects on arousal mediated by cortical-subcortical interactions. For example, Goldstein et al. (2005) found that arousal correlated positively with central amygdala, orbito-frontal cortex and anterior cingulate activity during midcycle — when estrogen is high and progesterone is low. In contrast, during the early follicular phase, when both estrogen and progesterone are low, arousal and amygdala activity were not related, but the relationship between arousal and cortical activity remained positive.

Testosterone may also influence HPA axis reactivity in women. It inhibits HPA axis reactivity in men (e.g., Handa et al., 1994; McCormick et al., 2002). In women, testosterone levels are lower than in men and peak mid-menstrual cycle when HPA reactivity appears to be reduced. Inhibition of HPA axis activity could be mediated by testosterone indirectly increasing ALLO synthesis (Mitev et al., 2003). ALLO, in turn, provides delayed negative feedback to the HPA axis (Barbaccia et al., 2001).

Serotonin. Serotonin affects several central nervous system functions including sleep regulation, anxiety, mood, and neuroendocrine regulation. There is evidence for serotonergic dysregulation in PTSD (Southwick et al., 1999). In addition, estrogen modulates serotonin 2A ($5\text{-}HT_{2A}$) receptors. For example, ovariectomies in rats result in decreases in serotonin 2A ($5\text{-}HT_{2A}$) receptors in the frontal cortex, striatum, dorsal raphe nucleus, and frontoparietal cortex; and these effects are reversed with estrodial treatment (Cyr et al., 1998).

Charney (2004) argues that short-term, stress-induced increases in estrogen might protect against PTSD because it blunts HPA axis and noradrenergic responses, but that long-term increases in estrogen might decrease serotonin $5\text{-}HT_{1A}$ receptors. Selective serotonin reuptake inhibitors (SSRIs) are the only psychopharmacological treatments currently approved by the Federal Drug Administration for the treatment of PTSD. Their effects are modest, and there appear to be sex differences in their effectiveness. Several studies have found that women exhibit better response than men (Brady et al., 2000; Davidson et al., 2001). In fact, a recently published study by Friedman et al. (2007) showed no difference between sertraline and placebo in treating male

combat veterans with PTSD.

Interestingly, emerging research suggests that the therapeutic effects of SSRIs for PTSD and depressive symptoms may derive from their capacity to increase brain ALLO levels rather than from their direct serotonergic effects (Pinna et al., 2006). A reduction in the capacity to synthesize ALLO may account for SSRI treatment resistance (Uzunova et al., 1998). While not yet studied in men, a reduced capacity for ALLO synthesis has been found in some women with PTSD (Rasmusson et al., 2006).

Neuropeptide Y. Neuropeptide Y (NPY) is released when the peripheral sympathetic and central nervous systems are intensely activated (Wahlestedt & Reis, 1993). NPY in the amygdala restrains activation of stress-related defensive behaviors, while in the periphery enhances the efficiency of noradrenergic neurotransmission. Male veterans with combat-related PTSD have decreased baseline plasma NPY levels and decreased NPY responses to intense activation of the sympathetic nervous system by yohimbine. Low levels of NPY may contribute to comorbid conditions in PTSD including poor memory, decreased hippocampal volume, disrupted sleep, chronic pain or blunted positive emotions (see Rasmusson & Friedman, 2002, for review).

Testosterone influences both baseline levels and release of NPY (Zukowska-Grojec, 1995), and thus may contribute to menstrual cycle variability in NPY release. For example, NPY levels increase in response to maximum load exercise at mid-cycle when testosterone levels are highest, though not during the luteal phase when testosterone levels are low. Lower NPY levels during the luteal phase may contribute to the increased norepinephrine levels observed in women during this phase (Goldstein et al., 1983).

Catecholamines. Epinephrine and norepinephrine are catecholamines involved in modulation of cardiovascular, respiratory, and metabolic reactions to stress. The release of these catecholamines during the "fight-or-flight" response is associated with intense arousal, activation of the HPA axis, fear, and anger (Rasmusson & Friedman, 2002). There are many studies documenting high levels of catecholamines in men with PTSD (Southwick et al., 1999; Wider & Resnick, 2005). Similarly, Lemieux & Coe (1995) found that premenopausal women with PTSD have higher norepinephrine and epinephrine levels than a control group of trauma-exposed women without PTSD.

However, there appear to be gender differences in catecholamine adaptations to stress. In a study of PTSD related to motor vehicle accidents, high levels of catecholamines were associated with PTSD in men, but not women (Hawk et al., 2000). There are also data supporting gender differences in catecholamine levels in samples without psychopathology, with men having higher norepinephrine levels than women (Frankenhaeuser et al., 1978). Catecholamines and the noradrenergic system appear to vary across the menstrual

cycle, with women in the luteal phase of the cycle having increased norepinephrine and greater cardiovascular responses to stress than women in the follicular phase (see Rasmusson & Friedman, 2002, for a review). These menstrual phase differences suggest that higher levels of progesterone and/or estrogen are associated with greater catecholamine reactivity to stress. However, other research has found that estrogen may have an inhibitory effect on catecholamines. For example, estrogen levels within the luteal phase of the menstrual cycle have a negative association with cardiac responses to stress(Sita & Miller, 1996). In addition, estrogen replacement has been shown to reduce blood pressure and catecholamine responses to psychological stress in post-menopausal women (Komesaroff et al., 1999; Lindheim et al., 1992). Thus, it is possible that estrogen and progesterone work differently on catecholamine physiology. These opposing relationships may be similar to the relationships of these sex-related hormones to HPA axis activity.

Summary. A review of the literature suggests that sex hormones modulate many of the neurobiological factors involved in the pathophysiology of PTSD. A more rigorous understanding of sex hormone modulation of these factors thus may advance our understanding of the two-fold greater risk for PTSD in women as compared with men. For example, it is possible that women are at increased risk for PTSD if they experience a traumatic event when estrogen and progesterone levels are high, and/or when testosterone levels are relatively low. It is also possible that PTSD symptom profiles may vary across time in congruence with variations in sex-related hormone levels, either within the menstrual cycle or at different stages of life. For example, a recent study has shown that women at the midpoint of the menstrual cycle (when estrogen is high, but progesterone is low), have decreased extinction of a conditioned fear response as compared with women in the early follicular phase (when both estrogen and progesterone are low) (Milad et al., 2006).

It will also be important to rigorously study sex hormone influences on neurobiological factors associated with PTSD, regardless of the traditional relationship between specific sex hormones and gender. For example, a reduction in testosterone levels would be expected to reduce both NPY and ALLO responses to stress and contribute to PTSD risk. And indeed, reductions in testosterone have been observed in men with PTSD (Mulchahey et al., 2001). Testosterone physiology has not, however, been studied in relation to PTSD risk in women. Conversely, preliminary evaluations of progesterone and estrogen in relationship to factors that may influence PTSD risk have begun in women, but need to be undertaken in men. Down the road, future basic research on this topic may lead to novel prevention and treatment strategies for men and women, such as using drugs that influence levels of sex hormones to prevent or adjunctively treat PTSD.

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2007 SSCP Student Poster Award at APS

Twenty students presented posters at the SSCP student poster session at the 2007 annual meeting of the Association for Psychological Science on May 24-27 in Washington D.C. This was the largest number of student posters to date, and the session was a great success. The quality of research being conducted by SSCP student members and their mentors was outstanding, and the judges had great difficulty deciding which of the many deserving submissions should receive the award.

The winner of the 2007 SSCP Student Poster Award is:

Coreen Farris

Indiana University

Alcohol intoxication influences perceptual processing of women's sexual interest cues
with Teresa A. Treat, Richard J. Viken, Richard M. McFall, & James T. Townsend

Congratulations to Ms. Farris and her mentors and co-authors!

2007 American Psychological Association Convention
San Francisco, CA, August 17-20

If you are planning to attend the APA convention, please come to the following SSCP-sponsored events:

Friday, August 17
1:00-1:50 Susan Mineka, recipient of SSCP's 2007 Distinguished Scientist Award, will give her award address titled "Integrative perspective on risk for mood and anxiety disorders: Evidence from a longitudinal study of adolescents".
Moscone Center, room 2007, second floor - west building.

Saturday, August 18
8:00-8:50 (AM) Dan Klein will give the 2007 SSCP Presidential Address, titled "Classification of depressive disorders in DSM-V: The case for a two-axis system."
Moscone Center, room 2001, second floor - west building.

1:00-3:00 SSCP Business Meeting. All members are invited to attend.
APA Division 12 Hospitality Suite.

Sunday, August 19
10:00 - 11:50 SSCP Symposium "Internalizing and externalizing disorders of childhood and adolescence: Perspectives from developmental psychopathology". Moderator: Emily Durbin. Participants: Ted Beauchaine, John Abela, and Eric Youngstrom.
Moscone Center, room 2004, second floor - west building.

Division 12 Sponsored Continuing Education Workshops

Full-day Workshops offered at the Hilton San Francisco Hotel, August 16, 2007, just prior to the APA Convention. 7CE Credits

Recent Developments in MMPI-2 Interpretation: The Restructured Clinical Scales and New Restructured Form, the MMPI-2-RF
Yossef S. Ben-Porath, Ph.D.

Movies and Mental Illness: Using Films to Understand Psychopathology
Danny Wedding, Ph.D.

Mentoring Women and Ethnic Early Career Academic Psychologist (ECP)
Helen D. Pratt, Ph.D.

Dialectical Behavior Therapy for Borderline Personality Disorder
 Anthony P. DuBose, Psy.D.

Psychological Interventions for Patients with Heart Disease
Judith A. Skala, RN, Ph.D., and Kenneth E. Freedland, Ph.D.

Treating Victims of Mass Trauma and Terrorism
Larry E. Beutler, Ph.D.

Diagnosis and Treatment of Obsessive-Compulsive Disorder
Jonathan Abramowitz, Ph.D.

Advances in Evidence-based Treatment for Bipolar Disorder
Robert Reiser, Ph.D.

Improving Therapy Outcome by Monitoring Process and Outcome
Jacqueline B. Persons, Ph.D.

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