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HIGHLIGHTS FROM THE EDITION

✦ Interview with Dr. Mohit Khera
  Juan I. Martínez-Salamanca, Spain

✦ Key from Kols: Long-term results after partial
  plaque excision and grafting with collagen fleece
  in Peyronie’s disease
  Georgios Hatzichristodoulou, Greece

✦ Key from Kols: The treatment for the hypogonadal
  male: Replacement or restoration? The argument
  for the use of SERMS
  Andrew McCullough, USA

✦ Have you read ? Best of the Best:
  Clinical and Basic Research
  Nicola Mondaini, Italy; Javier Angulo, Spain
I am delighted to welcome you to this 2016 Issue of the ESSM newsletter. We have in our society great things coming soon.

We are very excited having next ESSM Meeting in Madrid, my home city, would be a big pleasure and honor to host you along with my mentor and friend Ignacio Moncada, on behalf of the Spanish Association of Andrology & Reproductive Medicine (ASESA). Our ESSM Scientific Committee, leader by Maarten Albersen, did a great job to built an outstanding program including two exciting live surgeries sessions.

We encourage you to come to Madrid, and enjoy a great meeting along with a fantastic city, food, nightlife, history and more important, amazing people.

In this issue, we have included an interesting interview with a world-class expert in Sexual Medicine, Dr. M. Khera. We cover main relevant publications both Clinical and Basic by my Associated Editors and upcoming Events (Dr. Mondaini, Angulo & Vozmediano).

Also, we add two very interesting Keys from Kols collaborations regarding Peyronie’s disease & Treatment of Hypogonadal Male, by Dr. Hatzichristodoulou and Dr. McCullough respectively.

I hope you will enjoy reading it.

Finally, I would like to thank you all for your continued support of our society and I look forward to seeing you in Madrid very soon.

My very best
Juan I. Martinez-Salamanca
Dr. Mohit Khera is an internationally known figure and world-class leader in the field of Sexual Medicine. He is an outstanding contributor to the field of sexual medicine as a researcher, patient advocate, educator, innovator and author. Dr. Khera clinical interest areas include Testosterone in Aging Men, Male Reproductive Medicine and Sexual Wellness. Mohit Khera, M.D., M.B.A., M.P.H., Associate Professor, is the Director of the Laboratory for Andrology Research at McNair Medical Institute, Baylor College of Medicine. He is also the Medical Director of the Executive Health Program at Baylor. Dr. Khera is a Board-certified urologist specializing in male infertility, male and female sexual dysfunction, and declining testosterone levels in aging men.

Having you here it is a real pleasure and honor not only for me but also for all ESSM Members.

**JIMS:** Dr. Khera, if you were not a urologist, what would you be?

That is an easy question. I would be a professional tennis player. I am passionate about tennis and play 3–4 days per week. It is an amazing sport and I commonly play doubles tennis with my 3 children.

**JIMS:** In the field of Peyronie’s Disease, what do you think are the main challenges to achieve? What is your experience with the use of Xiaflex?

The medical management of Peyronie’s can be challenging in the fact that does require a commitment from both the patient and the physician. Xiaflex is an excellent medication but realize that it improves curvature by approximately 40 percent. Xiaflex is typically a 3 month process involving 8 injections. The challenges with a penile plication is the loss of penile length. The greater the curvature, the greater the loss in penile length. Many times I will initiate Xiaflex injection in a patient and reduce their curvature and then perform a penile plication. I refer to this as “priming” for surgery as this process mitigates the overall loss in penile length size after plication and I believe it improves overall patient satisfaction. Finally, the main concern with excision or incision and grafting procedures is the development of erectile dysfunction and penile numbness. Patients have to be counselled appropriately before performing any of these procedures.

**JIMS:** Dr. Khera, what do you most often wish you could stay to patients, but didn’t?

I have always taken a straightforward approach and tend to be very honest with my opinions with patients. I believe that good communication improves patient care.

**JIMS:** Dr. Khera, what do you most often wish you could stay to patients, but didn’t?

I believe Urology and more specifically the field of sexual medicine is the most fascinating and rewarding field of medicine. There are a tre-
mendous amount of research opportunities in this field. More specifically, there are many opportunities for translational research which can be very rewarding. I am passionate about taking care of my patients as well as my basic science and clinical research and it makes coming to work extremely enjoyable.

JIMS: Dr. Khera, regarding the latest controversy about Testosterone Replacement Therapy & Cardiovascular Risk, what is your personal opinion about that?

Ten years ago the number one safety concern with testosterone was prostate cancer. Today the number one safety concern with testosterone is cardiovascular disease. For decades we have had excellent data supporting testosterone’s beneficial effects on the heart. In 2006, Shores et al demonstrated that those men with lower testosterone levels were much more likely to die at an early age. Numerous later prospective studies also found that men with lower serum testosterone values were much more likely to die. Many of these studies found that the cause of death in these hypogonadal men was primarily cardiovascular in origin.

It was in 2010 with the Basaria study, and then in 2013 with the Vigen and Xu studies, and finally in 2014 with the Finkel study that there were some concerns with testosterone and its potential role in causing cardiovascular events. I believe there are significant limitations with these studies. In fact, many studies after 2014 have demonstrated once again no increased risk of cardiovascular events in men using testosterone therapy. Currently the FDA has issued a warning on the use of testosterone and the potential risk of a cardiovascular event. I do believe that we should counsel our patients that this warning does exist and that there are inconsistencies with the literature and more studies are needed.

JIMS: What is your most important piece of advice for doctors just starting out?

The advice I received when I first started my practice was to follow the 3 “As”. Always be available, have a great attitude, and make sure you are always improving your aptitude. One should never lose focus for why they have entered the field of medicine and most importantly they should enjoy what you do.

JIMS: And last but not least, which do you consider the most important challenges for our specialty (Sexual Medicine) and for our society (ESSM) in the next 5 years?

In the next 5 years I believe there will be a shift in our treatment paradigm for erectile dysfunction. ED is progressive disease that will affect almost every man if they live long enough. Currently our treatment options take a reactive approach and do not treat the disease process itself. I believe that disease modification and preventive therapies are the future of ED treatments. Future therapies such as stem cells, PRP, and even shock wave may improve and potentially reverse the ED disease process. Other therapies such as diet and exercise, use of statins and use of daily PDE5i are other examples of modifying the ED disease process. I believe that we must think of ED as a progressive disease and develop strategies to reverse this process.

It was a great pleasure to interview you; I am convinced that your points of view, fruits of a lifetime devoted to your work, will be highly appreciated by our readers. Thanks once again.
Erectile dysfunction


The present study aims to evaluate the relationship between physical activity and sexual dysfunction amongst an ethnic South Asian population living in the United Kingdom and compare the association with that of the native Caucasian population.

Twenty-five general practitioner clinics from eight primary care trusts in the United Kingdom collaborated in the Oxford Sexual Dysfunction Study. In each practice, a sample of diabetic and non-diabetic patients of European/Europid and South Asian origin were invited for the study. Erectile dysfunction (ED) was assessed using a five-item version of the International Index of Erectile Function. Premature ejaculation (PE) was diagnosed using the premature ejaculation diagnostic tool. Libido was assessed by asking participants to grade their desire for sexual activity. Physical activity during the past week was assessed using the short version of the International Physical Activity Questionnaire (IPAQ). A binary logistic regression analysis was performed in all adults, Europids and South Asians with ‘presence of ED’ as the dichotomous dependent variable (0 = ED absent; 1 = ED present) and age, diabetes status, physical activity, ethnicity, current smoking and use of antihypertensive medications as the independent variables.

Sample size was 510, and mean age was 56.9 ± 9.7 years. There were 63.9% (n = 326) Europid males in the study population. The prevalence of ED was 64.5% and it was significantly higher in men with diabetes than in those without diabetes (84.4 vs. 49.0%, p < 0.001). The overall prevalence of PE was 28.8%, (with diabetes 32.6%, without diabetes 25.8%; p = 0.109). Reduced libido was reported by 26.9 % of study participants (with diabetes 32.8%, without diabetes 22.0%; p < 0.01). The median (IQR) total physical activity of the study population was 2373 (3612) MET-min/week. In the IPAQ categorical score, 36.8% (n = 184/434) males were ‘highly active’, and 17.8 % (n = 89/434) were ‘inactive’. In all adults, age (OR: 1.06), South Asian ethnicity (OR: 1.40), physical inactivity (OR: 1.62) and presence of diabetes (OR: 3.90) all were associated with significantly increased risk of developing ED. A similar result was observed in Europids but not in South Asians.

Erectile dysfunction was associated with physical inactivity, mainly in Europid males, irrespective of diabetes status. This association was not observed in South Asian males with or without diabetes.


Prolactin (PRL) has been long deemed as a hormone involved only in female reproduction. However, PRL is a surprising hormone and, since its identification in the 1970s, its attributed functions have greatly increased. However, its specific role in male health is still widely unknown. Recently, low PRL has been associated with reduced ejaculate and seminal vesicle volume in infertile subjects. In addition, in men consulting for sexual dysfunction, hypoprolactinemia has been associated with erectile dysfunction and premature ejaculation, findings further confirmed in the general European population and infertile men. Several metabolic derangements, recapitulating metabolic syndrome, have also been associated with low PRL both in men with sexual dysfunction and from the general European population. In men with sexual dysfunction, followed-up for more than 4 years, low PRL was identified as an independent predictor of the incidence of major adverse cardiovascular events. Finally, an association with anxiety or depressive symptoms has been found in men with sexual dysfunction and from the general European population. While a direct role for impaired PRL function in the pathogenesis of these reproductive, sexual, metabolic and psychological disorders is conceivable, the possibility that low PRL is a mirror of an increased dopaminergic or a decreased serotonergic tone cannot be ruled-out. Hyperactivity of the dopaminergic system can explain only a few of the aforementioned findings, whereas a hypo-serotonergic tone fits well with the clinical features associated with low PRL, and there is significant evidence supporting the hypothesis that PRL could be a mirror of serotonin in the brain.


The aim of this study was to assess postprostatectomy erectile function compared to preoperative status by subjective patient perception and the abbreviated International Index of Erectile Function (IIEF-5) questionnaire.

The study used data from a prospectively collected database and a cross-sectional, questionnaire-based study in patients following radical prostatectomy. Erectile function was assessed with the IIEF-5 and the question “Is your erectile function as good as before the surgery (yes/no)”. Patients were included if they were sexually active before surgery and had at least 1 year of follow-up. The main outcome measure was the proportion of patients returning to self-perceived baseline erectile function. Secondary outcome measures included the proportion of patients returning to baseline erectile function according to the IIEF-5 and predictors of return to baseline function. Questionnaires from 210 patients were available. Overall, 14 patients (6.7%) reported that their erections were as good as before surgery. Bilateral nerve-sparing was the only significant predictor of a return to baseline erectile function (p = 0.004). Forty-three patients (20.5%),

Have you read? Best of the Best: Clinical
A brief summary of the best papers and abstracts published in the main journals related to Sexual Medicine by Nicola Mondaini

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who did not report use of erectile aids, showed no decline in IIEF-5 score. When including patients who used erectile aids, 69 (32.9%) maintained their preoperative IIEF-5 score. On multivariate analysis a low preoperative IIEF-5 score was a significant predictor of return to baseline IIEF-5 score (p < 0.0001).

Return to subjective baseline erectile function following radical prostatectomy is rare. The IIEF-5 questionnaire may not adequately reflect patients’ experience. This should be considered in preoperative patient counselling.

Penile surgery


Heretofore, the published data on penile implant patients consisted generally of small series of single-surgeon, retrospective experiences rather than prospective or large, multicenter evaluations. This study establishes a baseline of data collection from PROPPER (Prospective Registry of Outcomes with Penile Prosthesis for Erectile Restoration). PROPPER is the first large, prospective, multi-center study with the aim of minimizing long-term toxicity. Infertility and gonadal dysfunction are adverse effects of antineoplastic treatments that will be more efficiently prevented and treated if addressed before treatment initiation. This review focuses on these issues in young cancer survivors of childbearing age, where methods of protecting or restoring endocrine function and fertility need to be considered.

FSD

Maseroli E et al: Bringing the body of the iceberg to the surface: The Female Sexual Dysfunction Index-6 (FSDI-6) in the screening of female sexual dysfunction. Endocrinol Invest. 2015 Sep 3.

Female Sexual Dysfunction (FSD) is still poorly studied and underdiagnosed. The aim of the study was to produce an improved version of FSFI-6 (6-Item Version of the Female Sexual Function Index), entitled Female Sexual Dysfunction Index-6 (FSDI-6), and to estimate its accuracy as a screening instrument for FSD. In the new version, an item related to the personal interest in having a satisfying sex life was added, while the item rating the entity of sexual arousal was removed. We administered FSDI-6 in a consecutive series of female adult patients not consulting for sexual problems (n = 120, Cohort 1), and in another series of patients specifically consulting for sexual problems, which were considered as the control group (n = 160, Cohort 2).

FSDI-6 score was significantly higher in patients in Cohort 2 (p < 0.0001). Cronbach’s alpha for FSDI-6 was 0.784, indicating a high level of reliability. The estimated area under the ROC curve for FSDI-6 was 0.657 (p < 0.0001, 95% CI 0.584-0.730). The proportion of subjects with a pathological FSDI-6 score (≥16.5) was 29.9 (n = 32) and 59.4% (n = 95) in Cohort 1 and 2, respectively (p < 0.0001). Among subjects with a pathological FSDI-6 (score ≥16.5), those consulting for FSD had been postmenopausal for fewer years, had a higher level of education, a lower BMI and a lower prevalence of chronic diseases than those not consulting for FSD (p < 0.05).

Although a lower educational level, overweight/obesity, menopause and chronic diseases are risk factors for FSD, they are often associated with the failure in medical consultation for FSD. We propose that FSDI-6 should be performed by health care providers in non-specialist settings to detect potential FSD, which otherwise could remain under-diagnosed.

Fertility


The significant improvement of cancer treatments entailed a longer life in cancer survivors and raised expectations for higher quality of life with minimized long-term toxicity. Infertility and gonadal dysfunction are adverse effects of anticancer therapy or may be related to specific tumors. In female cancer survivors, premature ovarian failure is common after antineoplastic treatments resulting in infertility and other morbidities related to osteoporosis. In male cancer survivors, infertility and persistent azoospermia is a more common long-term adverse effect than hypogonadism because germ cells are more sensitive to chemotherapy and radiotherapy than Leydig cells. Gonadal toxicity and compromise of reproductive functions will be more efficiently prevented and treated if addressed before treatment initiation. This review focuses on these issues in young cancer survivors of childbearing age, where methods of protecting or restoring endocrine function and fertility need to be considered.
Female sexual dysfunction – Genital blood flow impairment by pelvic nerve injury

As in men, injury of nerve structures supplying female genitalia may result in sexual dysfunction in women. This is not surprising since neurovascular control of genital structures orchestrates and integrates the sexual stimuli to yield a functional sexual response. In fact, in addition to bladder and anorectal functional derangements, radical hysterectomy is associated with sexual dysfunction in women. Despite the adoption of nerve-sparing radical hysterectomy surgical techniques, neurapraxia may damage autonomic nerves supplying the vaginal wall and clitoris. Castiglione and colleagues aimed to create a female animal model mimicking damage of pelvic nerves caused by nerve-sparing radical hysterectomy and to evaluate the impact of this damage on genital blood flow responses. They induced unilateral crush injury of only the pelvic nerve (PNC) or of the pelvic nerve, the hypogastric nerve and vesico-genital branches of the pelvic plexus (clock-nerve crush; CNC) and evaluated genital blood flow responses to pelvic nerve electrical stimulation at 3 and 10 days after PNC or CNC. Pelvic nerve injury resulted in impaired blood flow responses to electrical stimulation as shown by the ~50-60% reduction in blood flow increase in vaginal wall and clitoris after stimulation of crushed nerve when compared to preserved contralateral nerve. There were no significant differences between PNC and CNC, and between 3 and 10 days after injury. An additional experimental series consisted of an experimental group of female rats undergoing bilateral PNC and a control group of animals undergoing sham operation for evaluating expression profiles in vagina and clitoris 10 days after surgery. Histological and immunofluorescence assays revealed distal vaginal fibrosis with altered immunodetection of collagen I and III. Western blots confirmed significant increase in collagen I and III protein expression in vagina but this increase was not observed in clitoris. Epithelium, muscular and lamina propria layers as well as vasculature of distal vagina displayed substantial nNOS fluorescence that appeared lower in the distal vagina of injured rats. Reduced nNOS expression in both vagina and clitoris after PNC was confirmed by Western blot. In contrast, in the vagina, expression of eNOS was confined to the vascular structures and no alteration of its expression in vagina and clitoris after PNC was observed.

Castiglione and colleagues describe an animal model to evaluate female genital blood flow impairment resulting from injury of the pelvic nerve. They propose that pelvic nerve injury causes a loss of nitrergic nerve fibers that results in a reduction of genital blood flow responses and vaginal fibrosis. Like in the male setting, it is assumed that fibrosis results from the relatively hypoxic state derived from the loss of nitrergic regulation of blood supply caused by nerve injury. However, in this case as well as in the male counterpart, the development of hypoxia after nerve injury requires further demonstration and it is still on debate. In the same way, the impact of nerve injury on endothelium (a putative victim of hypoxia-induced changes) is controversial. This study, although lacking functional evaluation of the endothelium-mediated actions, points to no alteration of endothelial structures (eNOS expression) in coincidence with some studies analyzing endothelial function of corpus cavernosum after cavernous nerve injury in rats and radical prostatectomy in humans.

It should be considered that longer evolution periods need to be analyzed and that the evaluation of genital blood flow responses specifically gives information of female sexual arousal without providing functional testing of other female sexual function components. However, it is undeniable that the animal model described in this article represents a key step, not only for deciphering the pathophysiology of sexual dysfunction after nerve-sparing hysterectomy but also to raise and evaluate potential strategies aimed to minimize the sexual impact of this sometimes unavoidable surgical procedure.

Aging-related ED – Impact on sirtuins and NO-pathway

The increase in the number of aged people is an outstanding phenomenon of the last decades that will be aggravated in the next future. This represents a challenge for combating age-related chronic diseases. These age-related diseases include erectile dysfunction (ED) whose prevalence notably increases with age. Vascular (erectile?) aging does not merely result from the accumulation of age-related co-morbidities but also from specific process due to ageing. MicroRNAs (miRNAs) are a family of highly conserved, small (~21 – 23 nucleotides) noncoding RNAs that regulate gene expression at the post-transcriptional level. In general, miRNAs bind to complementary sites of target mRNAs leading to a negative regulation of transcript stability and translation. The ability of miRNA to
regulate many targets at the same time makes them good candidates to control multifactorial physiological processes like aging. In this sense, growing evidence supports a crucial role of miRNAs as mediators of vascular aging both in animal models and in humans.

In a previous work, Pan and colleagues identified four up-regulated miRNAs in corpus cavernosum of aged rats that could be targeted to genes involved in endothelial NO/cGMP pathway. In the present study they analyze the possible involvement of one of these miRNA, miR-200a, on pathophysiology of aging-related ED in rats. By testing erections in response to apomorphine subcutaneous injection, they segregated aged rats (18 months old) into two groups: One displaying normal erectile responses and the other constituted by rats showing impaired erectile responses to apomorphine and then considered to have ED. Aged rats with ED had indeed reduced erectile responses to cavernous nerve stimulation and decreased content of endothelium and smooth muscle in cavernosal tissues when compared to either young or aged rats without ED. A near 3 fold up-regulation of miR-200a was detected in penile tissues of aged rats with ED that was accompanied by significant reductions in sirtuin-1 (SIRT1) protein expression as well as reductions in eNOS and cGMP content. Authors aimed to strength this associative relationship with confirmation of a causal impact driven by miR-200a on endothelial cells isolated from cavernosal tissue of aged rats without ED. Transfection of these cells isolated from cavernosal tissue of aged rats without ED leads to down-regulation of SIRT1 that of miR-200a in cavernosal tissue of aged rats leads to down-regulation of SIRT1 that of miR-200a in cavernosal tissue of aged rats leads to down-regulation of SIRT1 that results in defective eNOS/NO/cGMP pathway and impaired erectile function. Aging has been related to alterations in several critical cellular homeostatic and stress resistance pathways that suppress oxidative stress and inflammation. Sirtuins are NAD-dependent deacetylases involved in cellular response to stress. In fact, they have been shown to counterbalance NF-κB inflammatory system. SIRT1 seems to be involved in orchestrating different stress response pathways and the decline in SIRT1 activity with aging is accompanied by increased inflammation and oxidative stress. The study by Pan and colleagues suggests that modulation of miR-200a up-regulation and/or of SIRT1 down-regulation could be a therapeutic target for preserving erectile function in advanced age. However, it opens novel interesting questions that are worth to be addressed before establishing this therapeutic target. For instance, since pharmacological activation of SIRT1 is possible, this approach should be tested in the relief of ED in aged animals. This combined with functional evaluation of endothelial responses would help to confirm the endothelial function as the process regulated by miR-200a/SIRT1 axis. Finally, it would be key to know why some aged rats are resistant and others prone to the miR-200a up-regulation and subsequent cavernosal alterations and ED for proposing therapeutic interventions.


As mentioned for the above commented article, preservation of NO/cGMP pathway would be key to maintain adequate erectile function in advanced age. Asymmetric dimethylarginine (ADMA) is an endogenous inhibitor of NO synthases (NOS) whose levels modulate the NO/cGMP pathway. In fact, increased circulating ADMA levels have been associated with the presence of ED in patients. ADMA is removed by enzymatic degradation by dimethylarginine-dimethylaminohydrolase (DDAH). Two DDAH isoforms exist, DDAH1 and DDAH2, while heterozygous deletion studies suggest that DDAH1 is the primary isoform responsible for ADMA degradation in vascular endothelium and seems to play a role in endothelial function in vivo.

Wang and co-workers analyze the possible involvement of DDAH/ADMA/NOS pathway in ED associated with advanced age in rats. Aged rats (18 months old) showed reduced erectile responses to intracavernosal injection of papaverine. This functional deficiency was associated with structural alterations consisting of diffuse fibrosis and damaged endothelium. Increased levels of ADMA were detected in the penises of aged rats while the cGMP content was diminished. Expressions of penile DDAH1 and DDAH2 were reduced as they were those of eNOS and nNOS.

The authors propose that alterations of DDAH/ADMA/NOS pathway associated with aging could be responsible for the impairment of erectile responses in aged rats. The results points to an involvement of DDAH/ADMA in pathophysiology of ageing-related ED but it should be consider that the evidence provided by Wang and colleagues is just associative and not causal. It is reasonable to think that down-regulation of the main enzyme responsible for ADMA degradation in aged penis would lead to increased levels of this NOS inhibitor in penile tissue and would subsequently result in reduced production of NO by eNOS and/or nNOS. NO production would be further limited by the diminished expression of eNOS and nNOS, likely compromising erectile function. However, this plausible chain of events should be confirmed, for instance, by interventions aimed to reverse this pathogenic mechanism in aged rats. Moreover, the endogenous production of ADMA has been proposed as an explanation for the “L-arginine paradox”. The observation that exogenous L-arginine in vivo or in vitro increases NO production, despite its baseline concentrations that should saturate NOS. This might be consistent with the presence of an endogenous competitive antagonist, such as ADMA, at the...
active site of NOS that would be displaced by high concentrations of the substrate, L-arginine. In this sense, increased penile levels of ADMA would explain the positive effect attained by administering large doses of L-arginine to ED patients described by Zorgniotti & Lizza more than two decades ago.

Cavernous nerve injury-induced ED – Prolonging the staying of stem cells in corpus cavernosum


Evaluation of cell therapy strategies to prevent or reverse ED in animal models, and more specifically ED secondary to nerve injury, is one of the most recurrent research topics in the journals covering Sexual Medicine field. As majority of evidences have previously shown beneficial effects of cell therapy in alleviating ED in animal models of cavernous nerve injury, actual research efforts are focused in improving efficacy by modulating functional potential of stem cells or proposing more effective and/or convenient modalities of cell therapy. The here selected article is one example.

Lin and his collaborators show a novel approach to increase the time that intracavernosally injected stem cells remain in corpus cavernosum in rats undergoing bilateral cavernous nerve crush. The procedure consisted of magnetizing adipose-derived stem cells (ADSCs) by including magnetic nanoparticles (NanoShuttle). Application of a magnet to the penis allowed for retaining ADSCs in the corpus cavernosum for up to 3 days after injection while in the absence of nanoparticles or the magnet the ADSCs were washed out from corpus cavernosum after 1 day. Functional assays performed 4 weeks after nerve injury and concomitant ADSCs injection showed that magnetized Nano-ADSCs were more effective in preventing ED and in increasing smooth muscle and endothelium markers in cavernosal tissue of the rats.

Thus, it is proposed that increasing the time the stem cells are remaining in the corpus cavernosum will result in larger beneficial effects on cavernosal tissue that will preserve erectile responses after cavernous nerve injury. However, it would have been interesting to analyze the impact of retaining magnetized stem cells on nNOS content, since loss of nitrergic innervation in cavernosal tissue is a hallmark of cavernous nerve injury-induced ED. One the other hand, it should be noted that prolonging the exposure of corpus cavernosum to ADSCs does not necessarily means that the functional improvement obtained is related to enhanced incorporation of these cells to cavernosal structures. In fact, although this is a controversial issue, substantial evidences point to the incorporation of stem cells to host tissue as not very relevant to functional improvements.
Introduction
Peyronie’s disease (PD) is an acquired benign disease of the penile tunica albuginea leading to fibrous plaques. Approximately 3–9% of males between 40 and 70 years are affected. However, PD also occurs in the younger patient population. The fibrous plaques can cause penile deviation, penile shortening, painful erections and erectile dysfunction (ED). Indications for surgical therapy of PD include treatment of stable penile deviation in PD [5]. Main symptoms of the acute disease phase are increasing plaques and deviation, in combination with pain. In the stable phase, which usually begins 12 months from onset, deviation and plaques remain stable while deviation is the main and predominant symptom [6]. When penile deviation leads to inability for sexual intercourse surgical therapy is indicated, which remains the gold standard for treatment of stable penile deviation in PD [5].

Indications for surgical therapy
Indications for surgical therapy of PD include disease duration of at least 12 months, stable disease of minimum 6 months, stable penile deviation, no penile pain, unsuccessful conservative treatment, and most important the inability to perform sexual intercourse. Surgical therapy is subdivided in three main procedures: 1. plication techniques 2. grafting techniques with partial plaque excision or incision followed by defect closure with various grafts 3. correction of deviation with simultaneous penile prosthesis implantation in patients with ED not responding to medical therapy [3, 5].

Surgical technique using a self-adhesive collagen fleece for grafting following partial plaque excision
After lateral dissection and mobilization of the neurovascular bundle, an artificial erection is performed to assess the degree of deformity and the point of maximum curvature. Partial plaque excision is performed at the concave side of deviation at the point of maximum curvature by excision of an ellipsoid part of the tunica albuginea. The tunical defect is then extended laterally in the transverse direction up to half of the circumference on both sides of the penile shaft, leaving at least 1 cm space to the urethra. Grafting of the tunical defect is then performed using a ready-to-use, self-adhesive collagen fleece coated with tissue sealant (TachoSil®, Takeda, Berlin, Germany). This provides a watertight closure of the tunical defect. Additional fixation by sutures is not necessary (figures 1 – 4) [9]. Results of correction are documented intraoperatively by repeatedly artificial erection using physiological saline.

Results of partial plaque excision and grafting with collagen fleece
During a 9-year period (2004 – 2013) a partial plaque excision and grafting with the collagen fleece was performed in n= 244 patients with dorsal penile deviation. Mean patient age was 57.0 years (range: 33 – 73 yrs). Mean operative time was 80.8 minutes (range: 60-165 min). A totally penile straightening was achieved in 238/244 patients (97.5%). Mean long-term follow-up was 36.8 months (range: 3 – 100 mon). Erectile function improved in 230/244 patients (94.3%) had normal glans sensibility in the long-term.

Summary and conclusions
Grafting of the tunical defect by the self-adhesive collagen fleece following partial plaque excision in the surgical management of PD is safe and successful. Long-term results in regard to penile straightening, erectile function, and the sensibility at the glans are encouraging. The most important advantages of the collagen fleece for grafting of the tunical defect following partial plaque excision are: Easy application, no need for exact adjustment of the graft to the tunical defect, and no need for suturing the graft into the tunical defect. Therefore this technique is less time-consuming comparing to other grafting techniques. Moreover, the costs of the collagen fleece are about 190 EUR and thus more cost-effective comparing to other grafts for PD surgery (e.g. 4-layer SIS approx. 390 EUR). Additionally, an haemostatic effect is provided by the collagen fleece. Thus, the collagen fleece represents a feasible and safe alternative to other grafts for PD reconstructive surgery.
Keys from Kols: Long-term results after partial plaque excision and grafting with collagen fleece in Peyronie’s disease

References
The only FDA treatment for the hypogonadal man, barring pituitary/hypothalamic dysfunction, is testosterone (T) replacement with short or long lasting injections, topical gels, buccal losenges, intranasal T or T pellets. Hypogonadism is an endocrine disorder that can have profound, yet not life threatening, effects on the male. The treatment of endocrine disorders, by and large, is primarily replacement therapy. Adrenal insufficiency and thyroid deficiency are good examples. Yet treatment of the most common endocrinopathy, diabetes type 2 (DT2), is not replacement. After an attempt of life style modification most DT2 patients are started first on sulfonylureas, and metformin. Life style modification has been shown to positively influence glycemic control but in 10 year study compliance after 6 months was less than 20%. Whereas sulfonylureas increase the pancreatic insulin production, metformin decreases hyperglycemia primarily by suppressing hepatic glucogenesis. Replacement therapy with insulin is recommended after life style modifications. Metformins and sulfonylureas have failed to achieve glycemic control.

Can we model treatment of the hypogonadal male after the treatment of type 2 diabetes? Is there any evidence that we can restore T without life threatening, effects on the male. The treatment of endocrine disorders, by and large, is primarily replacement therapy. Adrenal insufficiency and thyroid deficiency are good examples. Yet treatment of the most common endocrinopathy, diabetes type 2 (DT2), is not replacement. After an attempt of life style modification most DT2 patients are started first on sulfonylureas, and metformin. Life style modification has been shown to positively influence glycemic control but in 10 year study compliance after 6 months was less than 20%. Whereas sulfonylureas increase the pancreatic insulin production, metformin decreases hyperglycemia primarily by suppressing hepatic glucogenesis. Replacement therapy with insulin is recommended after life style modifications. Metformins and sulfonylureas have failed to achieve glycemic control.

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The efficacy of CC in relieving the symptoms of hypogonadism is often anecdotally reported as being inferior to exogenous therapy without the detrimental impact of exogenous T on fertility. Clomiphene citrate, a selective estrogen receptor antagonist (SERM), has been used off FDA label to treat the hypogonadal infertile man since 1964. A recent meta-analysis confirmed the positive effect of SERMS on serum concentration and percent motility in infertile men.

One of the champions for the use of SERMS for the treatment of LOH was the late Andre Guay. Guay et al. challenged 21 older men with erectile dysfunction (ED) and secondary hypogonadism with 50 mg CC bid for 7 days and normalized their T, demonstrating that at that at least in the short term, the restoration of normal T levels was possible in older hypogonadal men. He then expanded the concept with an 8 week double blind placebo controlled crossover study in older men (mean age 62) with secondary hypogonadism and ED (documented with nocturnal penile tumescence scan (NPT)). Again, normalization of serum T was seen but, disappointingly, no improvement was seen in NPT or sexual function questionnaires in the group as a whole. When the study population was split between younger and older groups (mean age 53 and 66 respectively) in a secondary analysis, not surprisingly, the differences between the treatment groups with the sexual function questionnaires and NPT testing achieved statistical significance. This was the first demonstration that a SERM could not only normalize T levels in SHGD but result in symptomatic improvement. Guay et al. then began treating men in his practice with SHGD with CC (50 mg) 3 times a week. He reported an observational series of 173 men with ED and SHGD treated for 4 months. The diagnosis of ED was based on self-report and not a validated questionnaire, and a placebo arm was lacking. The outcome was measured as “responder” to treatment (successful intercourse > 75% of the time), partial responder (successful intercourse 50%–75% of the time) and nonresponder. As in his previous studies, LH, FSH and free T levels increased. Sexual function improved in 75% and did not change in 25%. Age and vascular co-morbidities negatively affected the response rates.

Taylor and Levine in an observational study compared the biochemical efficacy of CC to exogenous gel treatment (T replacement therapy [TRT]) in 104 men (65 CC vs 39 on TRT). The groups were not strictly identical but demonstrated comparable increases in T. Prostate-specific antigen (PSA) levels and hematocrit (HCT) did not significantly change in follow-up (23 months) 37 Moskovic et al. demonstrated an excellent chemical response in a younger cohort of 29 men (mean age 44) followed for 3 years on CC 25 mg every other day. 75% of men had altered bone mineral density (BMD) at baseline (75%) which normalized at 1 year in 25%. No improvement in BMD was observed after the first year. Though estradiol increased, significantly no gynecomastia or breast tenderness occurred. No side-effects were reported.

The only FDA treatment for the hypogonadal man, barring pituitary/hypothalamic dysfunction, is testosterone (T) replacement with short or long lasting injections, topical gels, buccal losenges, intranasal T or T pellets. Hypogonadism is an endocrine disorder that can have profound, yet not life threatening, effects on the male. The treatment of endocrine disorders, by and large, is primarily replacement therapy. Adrenal insufficiency and thyroid deficiency are good examples. Yet treatment of the most common endocrinopathy, diabetes type 2 (DT2), is not replacement. After an attempt of life style modification most DT2 patients are started first on sulfonylureas, and metformin. Life style modification has been shown to positively influence glycemic control but in 10 year study compliance after 6 months was less than 20%. Whereas sulfonylureas increase the pancreatic insulin production, metformin decreases hyperglycemia primarily by suppressing hepatic glucogenesis. Replacement therapy with insulin is recommended after life style modifications, metformins and sulfonylureas have failed to achieve glycemic control.

The efficacy of CC in relieving the symptoms of hypogonadism is often anecdotally reported as being inferior to exogenous therapy without the support of randomized double-blind studies. Katz
et al. retrospectively looked at symptom relief with CC (25 mg every other day) in 86 young (mean age 29) hypogonadal men, most of whom were presenting for infertility (57%) over a 4 years period at a Sloan Kettering andrology practice. The men were followed for a mean of 19 months. Surprisingly, the median number of positive baseline responses on the androgen deficiency in aging males (ADAM) questionnaire was 5 that dropped to 2. These “generally very healthy” young men started at a mean T level of 192 ng/dl and increased their T to 485 (despite a target treatment level of 550 ng/dl). The symptoms that showed significant increases included “decreased libido, lack of energy, decreased life enjoyment, sad/grumpy, decreased sports performance.” The lack of a placebo arm weakens the strength of the study. Further support of the efficacy of CC in relieving hypogonadal symptoms comes from a retrospectively gathered observational comparative study from Baylor by Ramasamy et al. In examining the effect of CC versus replacement therapy on hypogonadal symptoms, no significant differences were seen in between T injections, T gels or CC. T levels were highest with injections (1104 ng/dl) versus CC (504 ng/dl) or the gels (412 ng/dl). The lack of a difference in symptom support suggests the concept that symptom relief may be tied to a threshold level that is achieved with T restoration with SERMS.

Clomiphene citrate is a 60/40 mixture of two steri- somers, enclomiphene and zuclophene. Recently, there has been interest in the trans isomer of CC (EC). Distinct differential pharmacokinetics of the two isomers have been demonstrated. Though the Cmax and Tmax were comparable, the area under the curve for the isomers is dramati- cally different, after a single dose administration of 50 mg of CC in women with polycystic ovaries. At 456 h, 2C was detected in 9/9 patients versus 1/9 for EC. The half-life of EC is 7–8 h. EC was evaluated in a randomized, open label, fixed dose, active control (7 EC and 5 exogenous gel), two center phase IIb study in 12 men with secondary hypogonadism treated previously with topical T. After T discontinuation of exogenous T, T levels in both groups averaged 165 ng/dl. After treat- ment T levels increased in both groups to over 540 ng/dl but decreased to baseline after cessation of treatment suggesting that the hypothalamic testicular axis reverts to its pretreatment state and continued therapy is necessary. Baseline sperm counts were severely depressed at baseline. With treatment sperm counts increased in all men on EC at 6 months only 2 of 5 of gel patients increased their sperm concentrations to over 20 million/ml. Gonadotropins increased only in the EC arm. In follow-up clinical trials, safety and clinical efficacy were comparable to a gel preparation while preserving sperm counts. Sperm counts were decreased in the men treated with gels. Side effects were comparable to CC. The most significant adverse events were hot flushes (10%), visual disturbances headaches, nausea and vomiting. Aside from the hot flushes, all events occurred in < 5% of the study population.

The reader should be aware that SERMS are not FDA approved for the treatment of hypogonadism and are “off label” use. So how do I treat hypogonadal men? Clearly the hypogonadal infertile male should not be treated with exogenous T. Peer reviewed data support the use of SERMS in these men. How do we treat these men after the successful pregnancy? Should they be converted to exogenous therapy or continued on SERMS? There is no reason to believe that their T is going to magically return to normal. They will most likely require lifelong treatment, just as the hypothyroid or DT2 patient. What are the dangers of long term clomid therapy?

The safety of long term SERM use has been evaluated in the breast cancer trials and is nicely summarized by Goldstein et al. The most common side effect are menopausal symptoms unique to women (hot flushes and atrophic vaginitis). SERMS appear to be protective of bone mineral density and some studies have shown an increase in BMD while on therapy. There appears to be no increased risk of cardiovascular events but a mild increase in venous thromboembolism and pulmonary embolism, with a relative risk of 1.6 and 3.01, respectively. In the Stockholm Breast Cancer Study, there was no increased rate of hospitalization for thromboembolic events. An increase in the risk of cataracts (RR 1.14) was also described. In a 3 month randomized prospective, placebo controlled, comparator trial of EC and Androgel 1.62%, 21 % of the men described adverse events, though there was no difference between the treatment groups. There were no ophthalmologic changes and no significant one death from an ischemic stroke was reported in the EC group in a patient at high risk for embolic disease (unmedicated atrial fibrillation, obesity, DT2). It was felt that the patient’s underlying disease was more likely the cause of the event vs the medication. As with T replacement, caution should be advised in treatment men at high risk for thromboembolic events. Long term prospective safety studies are lacking in men, particularly with respect to prostate health. To date none of the studies suggest and increase in PSA over what would be expected for T replacement.

With proven biochemical and therapeutic efficacy and tolerability, should symptomatic men with secondary hypogonadism (<LH< 9.4 and T< 300) be treated primarily with SERMS, reserv- ing replacement for men who do not respond? Why not? In my practice, I obtain two morning total free and total T and E-2 levels, LH, FSH, prolactin, SHBG, CBC and PSA. After a complete history, physical and a thorough informed consent discussion men are started on generic clomiphene citrate 25 mgs or tamoxifen 10 mgs daily. The choice of medication is based on insurance reim- bursement. Repeat labs are performed in 4 weeks. Responders should have a significant in- crease in their T with symptomatic improvement. Unless the etiology of the HGD is clearly pituitary insufficiency or in cases of combined primary and secondary hypogonadism. Compliance and efficacy of medication is easily monitored by ex- amining LH, FSH and estradiol levels. A lack of T normalization without increase in gonadotropins and estradiol are consistent with noncompliance whereas an increase in LH, FSH and estradiol without a concomitant increase in T is indica- tive of primary testicular failure. In the marginally responsive man, sometimes doubling the dose will provide the desired result. In a retrospective
In summary, it is possible for us to model therapy after the treatment of DT2 treatment, stimulate endogenous production (in men who still have testicular reserve, 85% of men) and reserve exogenous replacement for those who do not respond.

References

Keys from Kols: The treatment for the hypogonadal male: Replacement or restoration? The argument for the use of SERMS
## MEETINGS AND EVENTS CALENDAR 2016

### FEBRUARY

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<tr>
<th>Event</th>
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<td>18th Congress of the European Society for Sexual Medicine</td>
<td>February 4 – 6, 2016</td>
<td>Madrid, Spain</td>
<td><a href="http://www.essm-congress.org">www.essm-congress.org</a></td>
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### MARCH

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<td>64th Annual Meeting of the Pacific Coast Reproductive Society</td>
<td>March 9 – 3, 2016</td>
<td>Rancho Mirage, California, USA</td>
<td><a href="http://www.pcrsonline.org">www.pcrsonline.org</a></td>
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<td>2nd International Workshop on Klinefelter Syndrome</td>
<td>March 10 – 13, 2016</td>
<td>Münster, Germany</td>
<td><a href="http://www.klinefelter2016.de">www.klinefelter2016.de</a></td>
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<td>ENDO 2016 – 98th Annual Meeting and Expo of the Endocrine Society</td>
<td>April 1 – 4, 2016</td>
<td>Boston, Massachusetts, USA</td>
<td><a href="http://www.endo-society.org">www.endo-society.org</a></td>
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<td>ASA 41st Annual Conference</td>
<td>April 2 – 5, 2016</td>
<td>New Orleans, Louisiana, USA</td>
<td><a href="http://andrologysociety.org/meetings/asa-annual-meeting/future-meetings/general-meeting-information.aspx">http://andrologysociety.org/meetings/asa-annual-meeting/future-meetings/general-meeting-information.aspx</a></td>
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<td>XIII Congreso Sociedad Española de Contracepción</td>
<td>April 6 – 8, 2016</td>
<td>Malaga, Spain</td>
<td><a href="http://sec2016.sec.es">http://sec2016.sec.es</a></td>
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### MAY

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<tr>
<td>XXXII Congresso Nazionale SIA - 2016</td>
<td>May 28 – 31, 2016</td>
<td>Stresa, Italy</td>
<td><a href="http://www.andrologiaitaliana.it">www.andrologiaitaliana.it</a></td>
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### JUNE

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<td>The 9th INYRMF Meeting</td>
<td>June 9 – 11, 2016</td>
<td>Rennes, Brittany, France</td>
<td><a href="http://andrologysociety.org/getattachment(%E2%80%A6)">http://andrologysociety.org/getattachment(…)</a></td>
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**MEETINGS AND EVENTS CALENDAR 2016**

Dr. Raul Vozmediano-Chicarro  
Associate Editor  
Section of Andrology  
Department of Urology  
Carlos Haya University Hospital  
Malaga, Spain  
vozme@msn.com

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<td>WS-02</td>
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<td>How to take the pain out of the sex?</td>
<td>Male sexual dysfunction – medical</td>
<td>Sex in the disabled</td>
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<td>RT-03</td>
<td>ESSM-01</td>
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<td>Advocating sexual health - ...</td>
<td>Sex and conception after gynecological malignancy</td>
<td>New opportunities for research funding from ESSM &amp; interim...</td>
<td>10:00–10:30 in the Poster Area HP-01 Prostate cancer treatment and Peyronie’s</td>
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<td>SA-01</td>
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<td>The &quot;Absent-Penis&quot;...</td>
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<td>LV-01</td>
<td>ESSM-02</td>
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<td>Sex steroids: the fuel or the catalysator of sexual function?</td>
<td>Industry sponsored Live Surgery Broadcasted from Puerta de Hierro Hospital in Madrid</td>
<td>ESSM resident’s corner and presentations of case studies</td>
<td>PS-02 Psychological research</td>
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<td></td>
<td>ISSM lecture</td>
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<td>Endometriosis and its sexual consequences</td>
<td>Management of the fibrotic penis</td>
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<td>19:00 – 20:00 Networking Reception in the exhibition area</td>
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**ABBREVIATIONS**

- **RT** – Round Table
- **ML** – Master Lecture
- **WS** – Workshop
- **VS** – Video Surgery Course
- **SA** – Industry sponsored Satellite Symposium
- **PS** – Podium Session
- **ESSM** – ESSM Session
- **LV** – Live Surgery Session
- **NA** – National Session Symposia
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<td>NA-02</td>
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<td></td>
<td>Mysteries of the female orgasm</td>
<td>Intersex challenges – caring for families and individuals with dsd</td>
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<td>Highlighted Posters 10:00–10:30 in the Poster Area HP-03 Body and mind, gender and sexual orientation</td>
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<td>How to deal with sexual consequences of menopause?</td>
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<td>T use and abuse focus on body composition and metabolic profile</td>
<td>Industry sponsored Video Surgery Course</td>
<td>Basic and translational research</td>
<td>English language</td>
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<td>ML-06</td>
<td>PS-05</td>
<td>PS-04</td>
<td>Male sexual dysfunction – mixed</td>
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<td>Do we smell sexy?…</td>
<td>Female sexual function &amp; dysfunction</td>
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<td>RT-10</td>
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<td>Sexual health is cardiovascular health? A 2016 update</td>
<td>Joint session with EPATH</td>
<td>AIUS – Association Interdisciplinaire post Universitaire de Sexologie</td>
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<td>ML-07</td>
<td>PS-06</td>
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<td>Mood, and its role…</td>
<td>MSD surgical/Peyronie/penile disorders</td>
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**Program Schedule I**  Saturday, 6 February 2016

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<td>RT-12</td>
<td>Dermatological changes in the vulvovaginal area...</td>
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<td>RT-14</td>
<td>Where does normal stop and perverse start?</td>
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<td>ML-08</td>
<td>State of the art in male contraception</td>
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<td>RT-16</td>
<td>Shocking penile therapies: ESWT from bench to bedside</td>
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<td>Penile controversies: the foreskin</td>
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<td>Industry sponsored Session: cases that matter</td>
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<td>15:00</td>
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<td>16:00</td>
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<td>Urethral surgery and its effects on sex life</td>
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<td>Closing Ceremony</td>
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**Registration Counter**  
07:30 – 17:00

**Exhibition**  
08:00 – 17:00

**Poster Exhibition**  
08:00 – 17:00

**Highlighted Posters**  
10:00 – 10:30 in the Poster Area HP-07

**Basic Science HP-08**  
ED and prostate

**Hormones HP-09**  

**Difficult cases in psychogenic ED: from diagnosis to treatment**  
WS-06

**Sexual consequences of sexual transmitted infections (STI)**  
WS-07

**Difficult cases of ejaculatory disorders: practical tips**  
WS-08

**Infertility and sexuality: why and how to pay attention**  
WS-09

**Difficult cases in psychogenic ED: from diagnosis to treatment**  
WS-06

**Sexual consequences of sexual transmitted infections (STI)**  
WS-07

**Difficult cases of ejaculatory disorders: practical tips**  
WS-08

**Infertility and sexuality: why and how to pay attention**  
WS-09
PAYMENT OF THE ESSM MEMBERSHIP FEE 2016

To be sent back to:
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Via Ripamonti 129 – 20141 Milano, Italy
www.essm.org

Membership goes from January to December
❑ New Member
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PAYMENT DETAILS

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Membership type
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❑ Combined ESSM + ISSM EUR 160,00

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Scientific work (two most important – peer reviewed – publications) – for new members only
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❑ Herewith confirms the payment of EUR 50,00 for the ESSM membership cost for the year 2016 by:
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* A letter of the Chairman of the Department is necessary

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In order to process your membership of the European Society for Sexual Medicine (ESSM) we will store your details in an electronic database. This information will be used to process your application only and will not be used for any other communications. The information will not be sold, lent or otherwise divulged to third parties, other than where it is necessary to process your application.

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Announcement for the next Congress

18th Congress of the European Society for Sexual Medicine
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