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In the Spotlight

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Looking Into Adhesion Formation and Its Relationship With Endometriosis

Endometriosis is an estrogen-dependent inflammatory disease characterized by the presence of endometrium-like tissue growth in sites outside the uterine cavity. It is often associated with chronic pelvic pain, pain during intercourse, and infertility and is believed to afflict 5% to 10% of women of reproductive age in the United States alone.[1] Although the etiology for the multiple variations of the disease presentation is far from being completely understood, it is generally accepted that it has both an endocrine and an immune component. The first somehow leads to the endometrial growth, which once established, progresses and promotes the development of adhesive disease powered by chronic inflammation in the peritoneal cavity.[2]

The lack of knowledge regarding the basis of endometriosis and its relationship with adhesion formation, in combination with the burden that these 2 conditions convey to the individuals affected and the society in general, has led to several studies, many of which previously published in *Reproductive Sciences*.[3-8] Once again, in the current issue, Stocks et al revisit the topic to determine the role of the inflammatory pathway activation in the development of adhesions in an endometriosis background.[9]

Given that adhesions are commonly associated with an endometriosis diagnosis and are especially prone to occur following surgery to alleviate the disease, Stocks and colleagues used endometriosis chimeric mouse models for their study, which were ovariectomized and placed on estradiol-releasing silastic capsules. Sixteen hours following surgery, the mice received peritoneal injections of human endometrial tissue fragments or endometrial tissue conditioned media (CM) from women with or without endometriosis. A subset of mice receiving CM was also treated with interleukin-1 receptor antagonist (IL-1ra).

The results obtained by Stocks and colleagues show that peritoneal injection of endometrial tissue fragments close to the time of surgery results in extensive adhesion formation, independent of the origin of the tissue received. Importantly, however, adhesion formation was significantly higher in mice receiving CM from patients with endometriosis as compared to the ones receiving CM from the controls. Interestingly, when mice were co-treated with IL-1ra, adhesion formation was dramatically decreased. In addition, cytokine bead array analysis of endometrial CM showed increased expression of IL-1β in patients with endometriosis as compared to the controls.

Altogether, the data presented by Stocks and colleagues suggest that the enhanced expression of IL-1β in women with endometriosis could be at the basis of their increased susceptibility to develop postsurgical adhesions. These findings suggest that inflammatory activation mediators, in particular IL-1β, could prove to be an appropriate target to design new therapeutic strategies to prevent postsurgical adhesion formation in the peritoneal cavity of patients with endometriosis.

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