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Treatment with a serotonin reuptake inhibitor increases reproductive hormone secretion in stress sensitive monkeys.

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When exposed to stresses some individuals develop reproductive dysfunction (i.e., are stress-sensitive, SS), while others show a high level of stress resilience (HSR). We have previously reported that there are stable physiological characteristics that differentiate SS individuals, including lower peak estradiol levels in the follicular phase and lower peak progesterone levels in the luteal phase of the menstrual cycle. SS animals also have lower central serotonergic activity, as shown by lower gene expression for SERT and MAO-A, and a trend towards lower gene expression for TPH-1, 5HT-1A, and MAO-B. In this study we questioned whether chronic treatment with a serotonin reuptake inhibitor, citalopram, would increase reproductive hormone secretion in SS animals. Twenty five monkeys were initially exposed to a combination of mild social and metabolic stress (mild diet + moderate exercise + relocation) and were categorized as SS (stress rapidly induced anovulation; n=9), medium stress resilient (MSR; stress slowly induced anovulation; n=7) or HSR (maintained ovulatory menstrual cycles throughout stress; n=9). Later, half of the monkeys in each group were treated for 15-17 weeks with 1.2-4.8 mg/kg S-citalopram (bid), to elevate blood levels of citalopram into the therapeutic range for treating depression. S-citalopram treatment caused a significant increase in peak estradiol levels (pre-treatment: 360 ± 67 pg/ml, post-treatment: 544 ± 82 pg/ml, p=0.05), and peak progesterone levels (pre-treatment: 6.7 ± 1.4 ng/ml, post-treatment: 11.3 ± 1.8 ng/ml, p=0.05), with the greatest increases occurring in the most stress-sensitive animals. We conclude that treatment of stress-sensitive individuals with a drug that increases brain serotonin levels stimulates activity of the reproductive axis.

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