

Age and social position moderate the effect of stress on fertility

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Abstract

There is now compelling evidence that psychosocial stress is a cause of reproductive suppression in humans. However, women continue to conceive in the harshest conditions of war, poverty, or famine, suggesting that suppression can be bypassed. The reproductive suppression model (RSM) proposes that natural selection should favor factors that reliably predict conditions for reproduction. In this study, we examine two such factors, age and social position, in women undergoing fertility treatment. We hypothesized that stress-related reproductive suppression would be more likely in younger compared to older women and in women in lower compared to higher social positions. The final sample consisted of 818 women undergoing fertility treatment. Psychosocial stress and sociodemographic data were collected prior to the start of treatment (Time 1), whereas fertility, as indexed by pregnancy or live birth, was assessed 12 months later (Time 2). The results showed that younger women were four times more likely to suppress than older women, and that unskilled and manual workers were more likely to suppress than those in middle social positions (e.g., white collar workers). However, significant associations between stress and fertility were also observed for women in higher social positions (e.g., professionals and executives). The findings provide support for the RSM. © 2006 Elsevier Inc. All rights reserved.

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1. Introduction

One of the most important life history trade-offs is that between current and future reproduction (Chisholm, 1999; Stearns, 1992). Reproduction, particularly for humans, is costly, not only because of the requirement for extra nutrients to support pregnancy and lactation, but also in terms of time and the risk to survival because of childbirth. Thus, investment in current reproduction may incur costs that decrease the opportunity for, or success of, future reproductive efforts. But under what environmental conditions is it better for the individual's lifetime reproductive success to defer reproduction? One key assumption of the reproductive suppression model (RSM) (Wasser & Barash, 1983) is that not all environmental conditions are equally favorable for investment in reproduction. Similarly, the developmental model of Ellison (1990) proposes that ovarian function is responsive to the quality of ecological conditions, for example, availability of food resources. These, and other (e.g., flexible response model; Vitzthum, 2001), evolutionary models propose that under conditions of environmental stress, reproductive functioning is temporarily suppressed because harsh conditions mean that the animal is either unlikely to be able to provide adequately for itself or the offspring, and/or the offspring is unlikely to survive.

The link between stress and reproductive suppression is well established in nonhuman mammals. In these groups, stress may be indicated by poor resource availability, low social status, competition, and persistent receipt of aggression, and all have been associated with decrements in reproductive potential (e.g., Cameron, 1997; deCatanzaro & MacNiven, 1992; Wasser & Barash, 1983). Physiological and demographic studies in humans also demonstrate variations in ovarian function according to the intensity of ecological stressors (for review, see Ellison, 1990, 1995).

In addition to ecological factors, Wasser and Barash (1983) highlight the importance of the psychosocial environment to human survival and reproductive success. Wasser and Barash (1983), Wasser and Isenberg (1986), and Wasser and Place (2001) argue that psychosocial stressors, particularly those that make it difficult to provide adequate care for the child (e.g., lack of social support, anxiety), should also activate the physiological mechanisms that suppress reproductive function. Effects of psychosocial stress on human reproductive function have been shown: negative affect (e.g., anxiety, depression) has been associated with longer cycle lengths (Hjollund et al., 1999) and reduced conception in healthy women trying to conceive (Sanders & Bruce, 1997). Further evidence comes from fertility treatment studies showing that stress indicators are associated with a poorer ovarian functional response to treatment (Lancastle & Boivin, 2005) and reduced fertilization, implantation, and live birth rates (e.g., Facchinetti, Matteo, Artini, Volpe, & Genazzani, 1997; Gallinelli et al., 2001; Klonoff-Cohen, Chu, Natarajan, & Sieber, 2001; Sanders & Bruce, 1999a; Smeenk et al., 2001). These associations remain after controlling for obvious confounders, for example, lifestyle (e.g., smoking) and health (e.g., weight) factors (Klonoff-Cohen et al., 2001; Sanders & Bruce, 1999a).

Given the harsh realities of life, however, reproductive suppression whenever stress is experienced would not, in practice, be efficient. Indeed, women continue to conceive in the harshest conditions of war, poverty, or famine. Moreover, despite the converging evidence in

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