ORIGINAL RESEARCH—EPIDEMIOLOGY

Metabolic and Cardiovascular Outcomes of Fatherhood: Results from a Cohort of Study in Subjects with Sexual Dysfunction

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ABSTRACT

Introduction. Previous cross-sectional and longitudinal studies reported a negative correlation between fatherhood and testosterone (T) levels, likely due to a centrally mediated downregulation of the hypothalamic–pituitary–gonadal axis. Moreover, epidemiological data indicate that fatherhood might affect metabolic and cardiovascular outcomes, although different results have been reported. Up to now, no studies have evaluated these associations in a population of men seeking treatment for sexual dysfunction (SD).

Aim. To explore biological and clinical correlates of number of children (NoC) and its possible associations with forthcoming major cardiovascular events (MACE) in a sample of men with SD.

Methods. A consecutive series of 4,045 subjects (mean age 52 ± 13.1 years old) attending the Outpatient Clinic for SD was retrospectively studied. A subset of the previous sample (N = 1,687) was enrolled in a longitudinal study.

Main Outcome Measures. Information on MACE was obtained through the City of Florence Registry Office.

Results. Among patients studied, 31.6% had no children, while 26.3% reported having one child, 33.4% two, and 8.8% three or more children. Although fatherhood was negatively related with follicle-stimulating hormone levels and positively with testis volume, we found a NoC-dependent, stepwise decrease in T plasma levels, not compensated by a concomitant increase in luteinizing hormone. NoC was associated with a worse metabolic and cardiovascular profile, as well as worse penile blood flows and a higher prevalence of metabolic syndrome (MetS). In the longitudinal study, after adjusting for confounders, NoC was independently associated with a higher incidence of MACE. However, when the presence of MetS was introduced as a further covariate, the association was no longer significant.

Conclusions. This study supports the hypothesis that bond maintenance contexts and fatherhood are associated with an adaptive downregulation of the gonadotropin-gonadal axis, even in a sample of men with SD. Moreover, our data suggest that NoC predicts MACE, most likely because of an unfavorable, lifestyle-dependent, parenthood-associated behavior. **Fisher AD, Rastrelli G, Bandini E, Corona G, Balzi D, Melani C, Monami M, Matta V, Mannucci E, and Maggi M. Metabolic and cardiovascular outcomes of fatherhood: Results from a cohort of study in subjects with sexual dysfunction. J Sex Med 2012;9:2785–2794.**

Key Words. Fatherhood; Number of Children; Testosterone; Major Cardiovascular Events

Introduction

Reproductive behaviors of vertebrates are often underpinned by temporal patterns of hormone secretion [1]. In fact, vertebrates are constrained by finite energy and time, and consequently, in these species, males often face trade-offs between conflicting behaviors related to mating and parenting [2,3]. Specifically, variations in testosterone (T) levels have been proposed as a physiological mechanism underlying this trade-off [3], as they may reflect and affect differential

behavioral allocations to mating and parenting efforts [4]. In fact, in some mammalian species, such as humans, higher T levels seem to be associated with dominance and competitiveness (even in sexual situations) and with encouraging mate-seeking behavior; conversely, lower levels are connected to bond maintenance contexts [4–7]. In particular, different studies [4,7–11] have reported that polyamorous men, more often involved in competitive relationship statuses, have higher T levels than monoamoursly partnered subjects. In line with this "low T-pair bonding" link in men, fatherhood and paternal care were reported to be associated with lower T levels [12–16]. A longitudinal study confirmed this evidence, reporting that high T predicts subsequent mating success, but then declines rapidly after men become fathers [3]. In addition, it was recently observed that this T reduction is not linked to a peripheral decrease in Leydig cell sensitivity or hormone binding by carrier globulins, but is instead likely secondary to a centrally mediated downregulation of the hypothalamic–pituitary–gonadal function [12]. Taken together, these results support the hypothesis that bond maintenance contexts and parenthood are associated with an adaptive down-regulation of the gonadotropin-gonadal axis, possibly aimed at minimizing any potential diversion from nest care and protection.

Epidemiological data indicate that fatherhood might affect metabolic and cardiovascular outcomes, although different results have been reported [17–25].

However, up to now, no studies have evaluated the association between number of offspring and sexual attitudes and hormones, as well as its correlation with lifestyle and metabolic parameters and related risk of major cardiovascular events (MACE) in samples of men seeking treatment for sexual dysfunction (SD).

Aims

The aim of our study is to explore biological (including T levels) and clinical correlates of number of children (NoC) in a sample of men consulting for SD. Moreover, we have evaluated here, in a subset of our sample, the possible associations between parenthood and forthcoming MACE.

Materials and Methods

Cross-Sectional Study

A consecutive series of 4,045 heterosexual male patients attending the Outpatient Clinic for SD for the first time were retrospectively studied. All patients enrolled underwent the usual diagnostic protocol applied to newly referred subjects at an andrology outpatient clinic. All the data provided were collected as part of the routine clinical procedure. An informed consent for the study was obtained from all subjects. Patients were interviewed prior to the beginning of any treatment, and before any specific diagnostic procedures, using the Structured Interview on Erectile Dysfunction (SIEDY, [26]). This is a previously validated [26,27] 13-item structured interview made up of three scales, which identify and quantify organic, intraspychic, and relational components concurrent to erectile dysfunction (ED, [26,28]).

Information on offspring number was assessed using the following question: "How many sons/daughters do you have?" Because a low number of subjects had a very high number of offspring, we collapsed those with three or more children into the same category.

Relational factors (such as relationship span, conflicts within the couple and the family, privacy during sexual intercourse, and partner hypoactive sexual desire, HSD) were assessed only in the subsample of subjects engaged in a stable relationship (N = 2,450). Partner's HSD, as perceived by the patient, was investigated using question #8 of SIEDY ("Does your partner have more or less desire to make love than in the past?") as a dummy variable, rating 0 = unmodified or mildly reduced desire, 1 = moderately reduced or never-present desire [26]. The characteristics of this subset were not significantly different from those of the whole sample (data not shown).

Alcohol consumption was evaluated using a standard question, formulated according to the Ministry of Health/Italian Society of Human Nutrition (Ministero della Sanita' / Societa' Italiana di Nutrizione Umana) criteria, considering alcohol abuse as more than four drinks per day [29].

Patients were asked to report any kind of drugs used. Chronic Disease Score (CDS), an index of concomitant morbidities, was calculated as previously described [30]. This is an aggregate comorbidity measure based on current medication used and originally validated for use as a predictor of physician-rated disease status, self-rated health status, hospitalization, and mortality [21]. The assessment of cardiovascular risk was evaluated using risk engine, derived from the Prospective Cardiovascular Münster (PROCAM) study [31].