

Special Article – Male Fertility

Infertility: What is our Direction?

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Editorial

In the last few decades, the number of patients with infertility, mostly male, has extremely increased [1-4]. The infertile population, however, spread equally between men and women [5]. What are the reasons for the decline of the reproductive function and what can we expect in the future? One of the influences affecting the reproductive functions of man today could be external factors, which in the past did not exist, or exist only partially. These factors might be: food consumption (fish from contaminated water or meat containing xenobiotics [6], pesticides [7] and insecticides [8]), vegetables and fruits (the source and quality of watering and irrigation which are not always known [9,10]), air pollution (the amount of exhaust gases, industrial emissions [11], the number of which increases from year to year, smoking [12,13]), or organic solvents [14]. All of the above factors are known today as endocrine disrupting compounds (EDC) affecting reproductive functions [15]. Another important factor is the radiation emanating from mobile phones [16], computers, satellite dishes etc. The risk of infertility depends on how many chemicals or radiation the man is exposed to, as well as the duration and frequency of exposure. However, it could be a stronger factor that affects reproductive functions.

Such a factor is the absence of a natural selection in human society (survival of the fittest). In the animal world, there is a strict selection - the strongest survives. The strongest, in turn, gives stronger more viable offspring; but in the case of a natural selection, it is not limited to this. In the newborn, only a few survive, since the majority die at different stages of development; only those individuals who excel in health, strength and adapt to the environment reach puberty. At this stage of selection, natural selection does not stop. Having reached puberty, the strongest males struggle amongst themselves, and only the strongest conquers and fertilizes the females. The defeated are eliminated, stay away from the females and therefore, do not have offspring. In nature, everything is cyclical and repeats itself. Therefore, the next generation will preserve the genotype of the strongest and healthiest individuals.

In the human society, this natural selection does not appear. From the man's side, there is no struggle between strong males for attaining a female. The "physiological weakness of the male body" created families, from which children were born with a "physiological weakness". This "weakness" tag is encoded in genetic material, and is retained and transmitted into the next generation.

Medicine has always treated diseases that were fatal in the past.

Today, diseases that previously shortened the duration of life are cured and, as a result the human life period is expanded.

In the past centuries, due to the absence of contraceptives, a high quantity of children were born; however not all of them survived due to high infant mortality. During the last few decades, child birth was regulated by contraceptives and, therefore, infant mortality was low due to progress, new knowledge and various technologies in medicine. The number of children in families in developed countries began to decline sharply. Children, who in past generations did not survive as weaker individuals, today are happily nurtured and developed and, at some point, become adults bearing offspring.

The reproductive system is the internal barometer of any organism and could respond to its weakness. Reproduction works in a strong and healthy body and malfunctions in a weak one [17,18]. The reproductive system is also the first of all systems in our body to stop functioning: menopause in women [19,20] and andropause in men [21].

Scientific data of recent decades show that the quantity and quality of spermatozoa in men is much lower than in other mammals. Sociologists have explained this by the fact that man creates marriages (family) for a long-term period and, therefore, there is no need for a high number (or a high concentration) of spermatozoa, since men (in contrast to wild nature) do not have to compete and struggle for women. Even a low number of spermatozoa are enough to fertilize an egg, whereby a normal embryo could be developed. A biological explanation may be less elegant than a sociological one. Reduced number and quality of spermatozoa in human population could be explained by the negative effect of monogamy and the progress in medicine. For thousands of years, with the creation of a monogamous system humans, as biological individuals, have enabled almost every woman to have her own man (and it may well turn out that he is not the strongest or healthiest) with whom she conceives a child. In such a situation, in the absence of natural selection, an unhealthy woman or an unhealthy man has the opportunity to give birth to a child who is biologically and physiologically a weaker individual. This child may have small deviations in the function of reproductive capacity after puberty, which are not demonstrated in this generation. However, in the future, this individual creates a family and gives the next generation a weaker reproductive function (internal barometer will switch on). In the future generations we will see lower semen parameters affecting modern man.

It is known that over the past 50-60 years there has been a sharp decrease in semen parameters, continuing to decline from year to year. During this time, the successes of medicine have grown dramatically; mortality, as a consequence of illness, has decreased and therefore, the level and duration of life have increased. Strengthening the viability of mankind, through medicine and technological progress (in the absence of natural selection), increased the likelihood of survival of a number of weak individuals. This would at best, give a weaker generation minor reproductive harm and, at worst suffer infertility.

References

1. Carlsen E, Giwercman A, Keiding N, Skakkebaek NE. Evidence for decreasing quality of semen during past 50 years. *BMJ*. 1992; 305: 609-613.
2. Lackner J, Schatzl G, Waldhor T, Resch K, Kratzik C, Marberger M. Constant decline in sperm concentration in infertile males in an urban population: experience over 18 years. *Fertil Steril*. 2005; 84: 1657-1661.
3. Spingart C, Frapsause C, Veau S, Barteley C, Royere D, Guerif F. Semen variation in a population of fertile donors: evaluation in a French centre over a 34-year period. *Int J Androl*. 2012; 35: 467-474.
4. Levine H, Jorgensen N, Martino-Andrade A, Mendiola J, Weksler-Derri D, Mindlis I, et al. Temporal trends in sperm count: a systematic review and meta-regression analysis. *Hum Reprod Update*. 2017; 23: 646-659.
5. CDC. Reproductive Health. 2006.
6. Swan SH, Liu F, Overstreet JW, Brazil C, Skakkebaek NE. Semen quality of fertile US males in relation to their mothers' beef consumption during pregnancy. *Hum Reprod*. 2007; 22: 1497-1502.
7. Sanchez-Pena LC, Reyes BE, Lopez-Carrillo L, Recio R, Moran-Martinez J, Cebrian ME, et al. Organophosphorous pesticide exposure alters sperm chromatin structure in Mexican agricultural workers. *Toxicol Appl Pharmacol*. 2004; 196: 108-113.
8. Chen M, Tao L, McLean J, Lu C. Quantitative analysis of neonicotinoid insecticide residues in foods: implication for dietary exposures. *J Agric Food Chem*. 2014; 62: 6082-6090.
9. Swan SH. Environmental phthalate exposure in relation to reproductive outcomes and other health endpoints in humans. *Environ Res*. 2008; 108: 177-184.
10. Vrooman LA, Oatley JM, Griswold JE, Hassold TJ, Hunt PA. Estrogenic exposure alters the spermatogonial stem cells in the developing testis, permanently reducing crossover levels in the adult. *PLoS Genet*. 2015; 11: e1004949.
11. Vighi M, Yunesian M, Shariat M, Niroomanesh S, Ramezanzadeh F. Environmental carbon monoxide related to pregnancy hypertension. *Women Health*. 2011; 51: 724-738.
12. Ramlau-Hansen CH, Thulstrup AM, Aqgerholm AS, Jensen MS, Toft G, Bonde JP. Is smoking a risk factor for decreased semen quality? A cross-sectional analysis. *Hum Reprod*. 2007; 22: 188-196.
13. Jensen TK, Jørgensen N, Punab M, Haugen TB, Suominen J, Zilaitiene B, et al. Association of in utero exposure to maternal smoking with reduced semen quality and testis size in adulthood: a cross-sectional study of 1,770 young men from the general population in five European countries. *Amer J Epidemiology*. 2004; 159: 49-58.
14. Cherry N, Moore H, McNamee R, Pacey A, Burgess G, Clyma JA, et al. Occupation and male infertility: glycol ethers and other exposures. *Occup Environ Med*. 2008; 65: 708-714.
15. Delgado Filho VS, Lopes PF, Podratz PL, Graceli JB. Triorganotin as a compound with potential reproductive toxicity in mammals. *Braz J Med Biol Res*. 2011; 44: 958-965.
16. Makker K, Varghese A, Desai NR, Mouradi R, Agarwal A. Cell phones: modern man's nemesis? *RBM Online*. 2009; 18: 148-157.
17. ESHRE Capri Workshop Group. Nutrition and reproduction in women. *Hum Reprod Update*. 2006; 12: 193-207.
18. Sarais V, Pagliardini L, Rebonato G, Papaleo G, Candiani M, Viganò P. A comprehensive analysis of body mass index effect on in vitro fertilization outcomes. *Nutrients*. 2016; 8: 109.
19. Scheffer GJ, Broekmans FJ, Looman CW, Blankenstein M, Fauser BC, teJong FH, et al. The number of antral follicles in normal woman with proven fertility is the best reflection of reproductive age. *Hum Reprod*. 2003; 18: 700-706.
20. Leridon H. Can assisted reproduction technology compensate for the natural decline in fertility with age? A model assessment. *Hum Reprod*. 2004; 19: 1548-1553.
21. Girsh E, Katz N, Genkin L, Girtler O, Bocker J, Bezdin S, et al. Male age influences oocyte-donor program results. *J Assist Reprod Genet*. 2008; 25: 137-143.