insulin/glucose ratios, allows us to suppose a diminished response of beta-cells to sugar stimulus. Since exogenous serotonin inhibits glucose-induced insulin secretion[10,11], we think that raised levels of the amine, registered during captivity, could explain the impaired tolerance to glucose.

We found that captivity induced similar effects to those obtained after sulpiride pre-treatment, whereas liberation- and haloperidol-effects were the same[4]. Hence we speculate that low doses of the latter drug provoke catecholamine release from catecholaminergic terminals by blocking presynaptic receptors, which induces hyperactivity of this system[12-14]. On the contrary, sulpiride would block postsynaptic receptors only, inducing dopaminergic system hypoactivity[15,16]. According to the well established antagonism (central and peripheral) between catecholaminergic and serotonergic systems[17], hypo- and hyper-activity of the serotonergic system would correspond, respectively, to the 1st and 2nd of these situations.

The demonstration of dopaminergic[18] and serotonergic[19] fluorescences in beta-cells, in addition to the ability of methysergide maleate (a serotonin antagonist) to enhance insulin secretion[20], reinforces the peripheral antagonism hypothesis, whereas the facts showing that both hypoglycemic and hyperglycemic effects can be induced by brain catecholaminergic mechanisms[21,22] support the central antagonism hypothesis. In addition, it has been shown that the activity of catecholaminergic neurons in the brain and in the peripheral sympathetic nervous system is increased during acute exposure to stress[23]. Stress induces significant depletion of catecholamine stores[24] and predominance of serotonergic neurons[25].

In 2 out of the 6 dogs investigated, captivity was prolonged. Normalization of glucose tolerance tests was registered in 2 experiments performed 3 months after their freedom II tests. Serotonin levels were found normal, also. The fact that animals chronically exposed to stress become resistant to the catecholaminergic depletion[26], is in line with these latter findings.

The 'diabetogenic effect' induced by captivity stress is a complex phenomenon in which other hormones (GH, ACTH, cortisol, catecholamines, glucagon, etc.) and factors like physical inactivity should be considered; however, our results strongly suggest that serotonin plays a role in producing that effect. Since serum prolactin levels were found raised following restraint stress in the rat[27], this hormone should be investigated, also.

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2 This work was supported by a grant of the Consejo de Desarrollo Científico y Humanístico de la Universidad Central de Venezuela.
3 Correspondence should be addressed to: Dr F. Lechin, Apartado 80.983, Caracas 108, Venezuela.
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Semen copper in normal and infertile subjects

K.P. Skandhan[1] and B.N. Mazumdar*

Department of Physiology, B.J. Medical College, Ahmedabad 380016 (India), 13 October 1978

Summary. Concentration of copper in seminal plasma was found to be less than that of normal in cases of oligospermia and azoospermia. It was more in oligoasthenospermia and asthenospermia when compared with that of normal. Chances of initiation of sperm motility by copper is discussed alongwith the inhibitory role it plays.

The role of electrolytes in semen is not well known. The possible role of sodium, potassium, calcium, magnesium[2] and zinc[3] in semen has been discussed earlier. Association of copper deficiency in diet and infertility is well established[4-8]. Also, the toxicity of copper on uterus[9] and spermatozoa[10] is reported. Keeping all these reports of experimental studies in mind, we estimated the copper in seminal plasma in normal and in infertile cases to know if this element has a part to play in the viability of spermatozoa.

Material and methods. 1 sample each from 55 subjects was collected, between 09.00 and 11.00 h, onto clean and dry glass bottles after an abstinence of at least 5 days. The age of the subjects varied from 23 to 35 years. Samples were classified into 5 groups according to its sperm count and motility, as shown in table 1.

Estimation of copper was done colorimetrically using sodium diethyl dithiocarbamate method[12]. All necessary precautionary measures were taken to avoid the infiltration of copper from any source.

Results. Motility of sperm of different study groups is shown in table 2.

The results are given in table 3. Standard error is calculated and values are compared with each other.

Discussion. Literature on copper level in whole semen or seminal plasma is scanty[13,14]. Our values for normal (table 3) are above that of the same in whole semen[13]. Interestingly, the reverse was the pattern for zinc[3]. It is known that copper and zinc are antagonistic in action in biological systems[14].

Stankovic and Davic[13] ruled out the importance of copper in semen as the levels of it in normal and in subfertile groups were found to be the same. Our results (table 3) showed a different picture. Though the range of it was wider than that in normal both in oligospermia and azoospermia, the mean values were lower than in normal. Statistically it was highly significant when values of azoospermia was compared to that of normal.

In the other 2 subfertile groups namely, oligoasthenospermia and asthenospermia, the concentration of copper in seminal plasma was found to be more than normal. Zinc concentration was least in asthenospermic sample[3]. Do these 2 trace elements interplay in initiation of sperm motility? X-ray microanalysis of semen studies revealed the positive role copper has to play in it[16]. Also they found addition of copper in the medium replaces zin from the head[17]. Fujii et al.[18] reported the release of zinc from the cell for its motility. Does the copper enter first to release zinc? This is possible, when we look into the results. Zinc is essential for motility and its level was found to be minimum in asthenospermia[3]. Copper level was found as maximum in the same group. Possibly in normal semen copper enters the cells followed by the release of zinc from inside to make them motile. If this were the true pattern in normal, in the case of oligoasthenospermia and asthenospermia the entry of copper into sperm cells may be closed by some mechanism and thus the motility is partially or completely closed.

Addition of extra copper to the surrounding medium of sperm made it less motile or even immotile[17,19,20]. Can copper be an inhibitory factor to sperm motility? It is likely to be when it is above a physiological limit. This is one of the probable mechanism responsible for its contraceptive action in intra-uterine copper contraceptive device[21-24]. Thus, the increased level of copper in seminal fluid may be 1 of the factors directly responsible for oligoasthenospermia and asthenospermia.

Table 1. Sperm count and percentage of motility of different groups included in the study

	Sperm count in millions	Motility (%)
Normal[11]	> 40	> 60
Oligospermia	< 40	> 60
Oligoasthenospermia	< 40	< 60
Asthenospermia	> 40	< 60
Azoospermia	Sperm was not present in deposit even after centrifugation (10,000 rpm × 10)	

Table 2. Observations of motility in different groups

	Sperm motility (%)	Degree of sperm motility
Normal	> 60	Excellent, fair
Oligospermia	> 60	Excellent, fair
Oligoasthenospermia	0-30	Nil, sluggish fair
Asthenospermia	0-30	Nil, sluggish fair
Azoospermia	-	-

Table 3. Copper level in fertile and sub-fertile groups and their comparison with each other

Sample	Number of samples	Copper level in µg % Mean ± SE	Range
Normal	15	153 ± 13	100-200
Oligospermia	9	122 ± 25	50-300
Azoospermia	17	110 ± 08	50-300
Oligoasthenospermia	8	200 ± 43	100-400
Asthenospermia	6	380 ± 117	100-700

Comparison	p-value	Remarks
Normal v/s azoospermia	< 0.01	**
Normal v/s oligospermia	> 0.1	-
Normal v/s oligoasthenospermia	0.5 > p > 0.2	-
Normal v/s asthenospermia	0.02 < p < 0.01	*
Oligoasthenospermia v/s asthenospermia	> 0.1	-
Azoospermia v/s oligospermia	0.8 > p > 0.5	-
Azoospermia v/s asthenospermia	< 0.002	**
Azoospermia v/s oligoasthenospermia	0.02 > p > 0.01	*
Oligospermia v/s oligoasthenospermia	> 0.1	-
Oligospermia v/s asthenospermia	0.02 < p < 0.05	*

-, Non significant; *, significant; **, highly significant.

* Present address: Deputy Director, Directorate of Medical Education and Research, Gujarat State, Ahmedabad 380016 (India).

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