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D-Aspartate-Induced Fructose Elevation and Its Impact on Sperm Motility: A Systematic Review

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ABSTRACT

Male infertility, particularly due to reduced sperm motility, remains a significant challenge in reproductive medicine. This review evaluates the role of D-aspartate (D-Asp), a naturally occurring amino acid, in enhancing sperm motility through fructose elevation in seminal plasma.[11]

D-Aspartate is an endogenous amino acid involved in luteinizing hormone (LH) and testosterone release in humans. This study evaluated the effect of oral sodium D-aspartate supplementation on sperm quality in sub- fertile men and the resulting pregnancy rates in their partners. It to be a promising natural agent for improving sperm motility through its ability to elevate seminal fructose concentrations. However, more standardized clinical trials are needed to confirm optimal dosing, duration, and long-term effects on male fertility outcomes.

Dose: 10 ml of 2.0 M sodium D-aspartate solution (equivalent to 2.66 g of D-aspartic acid neutralized with 0.46 g NaOH)

Frequency: Once daily, dissolved in half a glass of water or fruit juice, taken at lunch or dinner

Duration: 90 days (3 months) continuous treatment.

Sixty sub-fertile patients were enrolled—30 with oligo-asthenozoospermia and 30 with asthenozoospermia. Each received a daily oral dose of sodium D-aspartate (10 ml of 2.0 M solution) for 90 days. Sperm concentration, motility, and pregnancy outcomes were assessed before and after treatment.

Fructose, a key energy substrate in seminal plasma, supports ATP production and flagellar activity of spermatozoa[26]. Elevated fructose levels are associated with enhanced progressive motility and fertilizing potential, whereas reduced fructose correlates with poor motility and subfertility[25]

Sodium D-aspartate supplement improves fructose level in semen. It also improves sperm concentration and motility in sub-fertile men, leading to increased pregnancy rates in their partners, without harmful side effects.

Keywords: D-Aspartate; Oligo-Asthenozoospermia; Pregnancies ;Human spermatozoa, reproduction, acrosome reaction, fructose,in vitro fertilization, motility,

I. INTRODUCTION

Reproductive disturbance has been a growing problem in developed countries during the last decades. According to the World Health Organization, more than 10% of men worldwide are infertile, contributing to the increasing demand for assisted reproduction techniques such as in vitro fertilization (IVF) and intracytoplasmic sperm injection (ICSI), where sperm quality is crucial. Male infertility is therefore a serious and increasing phenomenon.

Several factors are responsible for male infertility, including sociological causes, over-production of reactive oxygen species (ROS)[1], and spermatozoa DNA degradation[2]. Reduction of sperm concentration and motility are considered the main causes of male infertility. Various nutritional supplements have been tested to improve semen quality in infertile men. A wide number of antioxidants have been proposed, such as vitamin E, vitamin C, coenzyme Q10, folic acid, glutathione, selenium, and zinc[3].

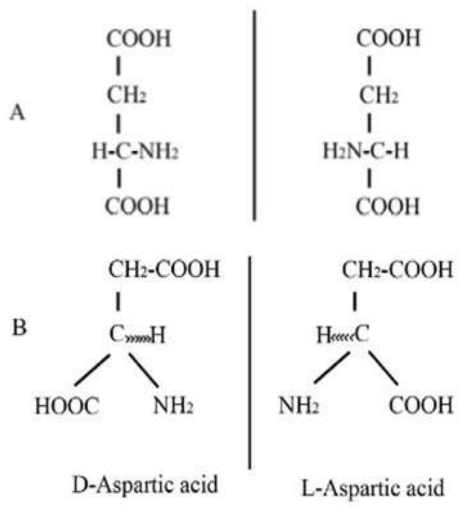
Coenzyme Q10 plays a general role in electron transport within mitochondria and protects cell membranes from oxidative stress[4]. Folic acid contributes to DNA synthesis and acts as a free radical scavenger[5][6]. Lycopene and vitamins A, C, and E have also been used as supplements, but evidence for their effect on semen quality is lacking[7]. Vitamin E supplementation has been reported to reduce sperm DNA fragmentation and improve quality[8]. Zinc and selenium are important trace elements in seminal plasma with antioxidant roles[9]. Other amino acids such as L-carnitine, acetyl-L-carnitine[10], and L-arginine[11] have also been studied as nutritional approaches to enhance sperm quality.

D-aspartic acid (D-Asp), an endogenous amino acid present in nervous and endocrine tissues including testes, has gained attention. It is involved in hormone regulation at the hypothalamic–pituitary–gonadal axis[12][13]. D-Asp has been found in seminal plasma and spermatozoa, with levels correlating positively with semen quality[15]. Moreover, it stimulates testosterone synthesis in rat Leydig cells[16], improves semen quality in rabbits[17], and increases LH and testosterone levels in humans[18]. Based on this evidence, the present study was conducted to evaluate whether oral sodium D-aspartate supplementation can improve sperm quality in sub-fertile men and enhance pregnancy rates in their partners.

Fructose, secreted mainly by the seminal vesicles, is the primary energy source for spermatozoa[27]. It provides the ATP needed for flagellar motion and progressive motility, and reduced fructose levels are associated with poor sperm motility and male subfertility[10].

STRUCTURE

Linear and Stereochemical formula of D-aspartic and L-aspartic acid. Chemical structure of D-aspartic acid and L-aspartic acid by using a linear formula (panel A) and a carbonium presentation based on the tetrahedral configuration (panel B).



AIM

To evaluate the effect of d aspartate induced fructose elevation and its impact on sperm motility and to determine its therapeutic agent for improving male fertility.

OBJECTIVE

- To investigate the relationship between D-aspartate-induced fructose elevation in seminal plasma and its role in energy metabolism and sperm motility.
- To determine the impact of fructose supplementation.
- To evaluate the effect of oral sodium D-aspartate supplementation in sub-fertile men.
- To assess changes in sperm concentration after treatment and To measure improvement in sperm motility following therapy.
- To determine the impact on pregnancy rates in partners of treated patients.
- To evaluate safety and tolerability of sodium D-aspartate supplementation.

NEED FOR THE STUDY

- Male infertility is a growing global issue, affecting >10% of men worldwide[1].
- Fructose, the main seminal energy source, is directly linked to sperm motility, and its deficiency is associated with subfertility.
- D-Aspartic acid (D-Asp) is a natural amino acid found in testes, seminal plasma, and spermatozoa, known to regulate LH and testosterone release.
- The study reported that oral sodium D-aspartate (10 ml/day for 90 days) in sub-fertile men significantly: Increased sperm concentration ,Improved motility , Raised pregnancy rates in partners .
- Adverse effects shows No harmful clinical or biochemical changes were observed; only a physiological increase in LH and testosterone was detected, confirming treatment safety.

METHODOLOGY

STUDY DESIGN - A systematic review

STUDY STRATEGY

The strategy combines both literature synthesis and experimental observations to explore and understand the effects of D- aspartate-induced fructose elevation on sperm motility.

Databases searched

PubMed, Springer, web of science, google scholar, Elsevier.

Search terms/keywords used

D-aspartate, Oligoasthenozoospermia , acrosome reaction, fructose, pregnancies, antioxidants, human spermatozoa, in vitro fertilization, seminal plasma.

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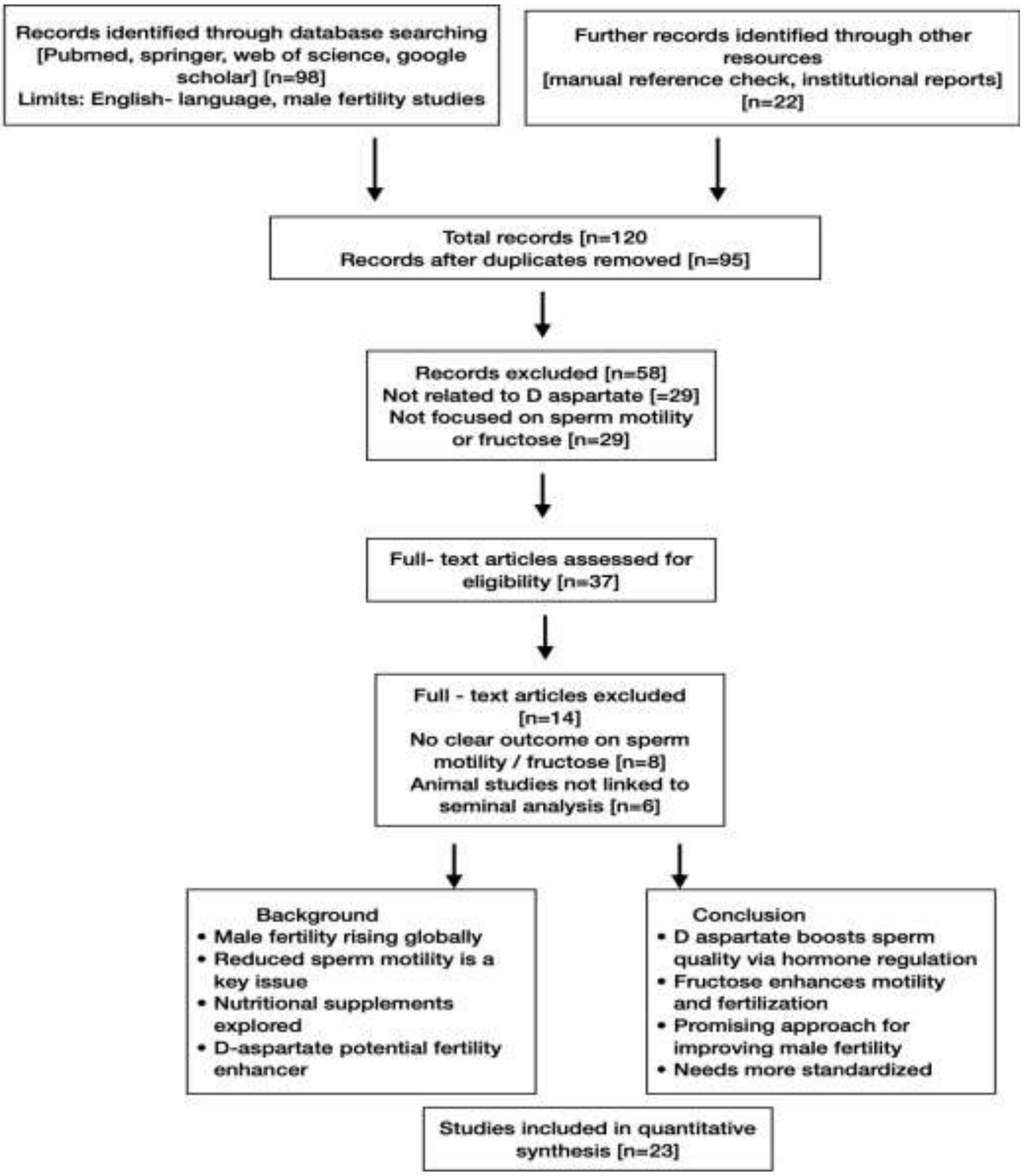
Inclusion criteria

- Articles published in English
- Studies evaluating D-aspartate effects on sperm parameters, hormone levels, or fructose metabolism.
- Peer reviewed journals
- Male participants with subfertility (oligozoospermia, asthenozoospermia, or both).
- Original research articles (clinical or preclinical).

Exclusion criteria

- Review articles without original data.
- Reports lacking pre- and post-treatment semen analysis.
- Non English publications.
- Studies with short treatment duration (<30 days) not adequate to assess sperm quality changes.
- Articles with incomplete or unclear methodology.
- Studies not focused on male fertility, sperm motility, or fructose metabolism.
- Studies unrelated to reproductive outcomes.

STUDY SEARCH DIAGRAM



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II. DISCUSSION

D-Aspartic acid (D-Asp) is an endogenous amino acid synthesized in endocrine glands, particularly in the testis, by the enzymatic conversion of L-aspartate through D-aspartate racemase[18]. Previous research has shown its involvement in endocrine regulation and the synthesis and release of sexual hormones in both animals and humans [12][16][18][20][24]. Importantly, D-Asp has been detected in human seminal plasma and spermatozoa, with a direct correlation between its concentration and semen quality[15].

Based on these findings, it was hypothesized that supplementation with D-Asp in sub-fertile men (oligo- asthenozoospermic and asthenozoospermic) could improve sperm concentration and motility, ultimately enhancing pregnancy rates in their partners. In this study, 60 patients who had been unable to achieve conception for at least two years were treated with oral sodium D-aspartate for three months. Results demonstrated a significant improvement in both sperm count and motility across both patient groups, which translated into a higher rate of natural pregnancies among their partners.

Specifically, 20% of partners of oligo-asthenozoospermic patients and 33% of partners of asthenozoospermic patients conceived during the treatment period. Remarkably, some pregnancies occurred as early as the first month of supplementation. This clinical evidence highlights the potential of D-Asp in enhancing male fertility outcomes.

Another key observation was the absence of adverse effects. Clinical and biochemical evaluations confirmed that all measured parameters remained within normal ranges after treatment. The only notable changes were physiological increases in LH and testosterone, consistent with earlier studies[18]. This suggests that sodium D- aspartate is a safe therapy for sub-fertile men when administered over a period of 90 days.

In conclusion, the data support that daily supplementation of sodium D-aspartate in sub-fertile men improves sperm quality by increasing sperm number and motility, which consequently enhances natural conception rates. Although the exact molecular mechanism remains unclear, it is strongly hypothesized that D-Asp acts by elevating testosterone levels in the testes, thereby promoting spermatogenesis and sperm maturation.

Serum Clinical Analysis

To rule out any possible adverse effects of sodium D-aspartate supplementation, all participants underwent detailed medical examinations and blood tests before and after the 90-day treatment. The laboratory evaluations included:

1. **Hematology:** Complete blood count and platelet count.
2. **Serum biochemistry:** Levels of glucose, urea, ammonia, cholesterol, triglycerides, creatinine, uric acid, and essential electrolytes (sodium, potassium, calcium, magnesium, iron, copper).
3. **Enzymatic profile:** Activities of aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase, acid phosphatase, prostatic phosphatase, cholinesterase, and lactate dehydrogenase (LDH).
4. **Hormonal profile:** Concentrations of testosterone, luteinizing hormone (LH), follicle-stimulating hormone (FSH), thyroid-stimulating hormone (TSH), triiodothyronine (T3), thyroxine (T4), free T3 (FT3), and free T4 (FT4). [18][19]
5. **Serum protein electrophoresis** to evaluate protein distribution.

The results demonstrated that all measured clinical and biochemical parameters remained within physiological ranges after treatment. The only notable findings were modest increases in LH and testosterone levels, which were consistent with previously reported effects of D-aspartate on hormonal regulation. These increases were not considered harmful, confirming that the 90-day supplementation was safe and well tolerated.

FUNCTION OF FRUCTOSE ON SPERM MOTILITY

From the functional point of view,motility is strongly related to the spermatozoa’s ability to manage its energy status and a high percentageof spermatozoa energy consumption is centered on the contraction of the flagellar membrane. Modification of energy status may therefore result in notable modifications to motility patterns.[18] In actuality, the striking alterations seen in spermatozoa as they pass through the female reproductive system may be caused by this connection between energy metabolism and motility patterns.[19][20]

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They can get energy by consuming glycolyzable carbohydrates like glucose, fructose, mannose, and maltose, as well as by mitochondrial oxidative phosphorylation . Fructose is present in seminal plasma in many mammalian species and is believed to be a key energy source for ejaculated spermatozoa. [21][20]The positive effects of fructose range between species, and it has been studied for its various impacts on gametes in terms of metabolizable energy and reproductive potential in numerous species. Twelve Boar spermatozoa's AR and capacitation are marginally impacted when glucose serves as the sole energy source in the culture medium [22]

Even while 1.67 mmol fructose14 is a spermatozoa nutrient in fresh swine semen, it is eliminated when semen samples are cleaned for use in IVF procedures or preserved for use in artificial insemination programs.

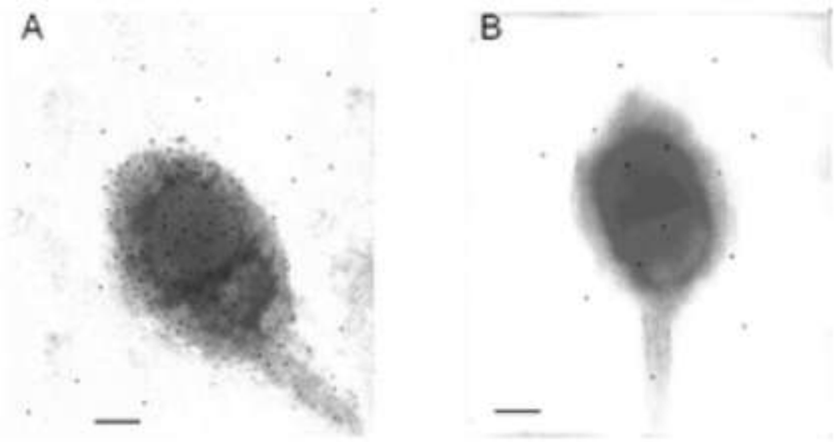
Therefore, we believed that in order to prepare the spermatozoa for boar IVF programs, fructose must be added to basic medium at an optimal quantity. However, no research has been done on how fructose affects swine spermatozoa's ability to fertilize. The goal of this study was to find out how fructose affected the boar spermatozoa's motility, acrosome response, and IVF capacity [23]

Determination of D-Aspartic Acid in Seminal Plasma

The concentration of D-aspartic acid in seminal plasma was determined by a high-performance liquid chromatography (HPLC) method combined with the use of D-aspartate oxidase (D-AspO), as previously described[12]. In brief, 0.1 ml of seminal plasma was mixed with 0.4 ml of 0.2 M trichloroacetic acid and centrifuged. The supernatant was neutralized with 1.0 M NaOH, and 50 µl of this sample was used for HPLC analysis.

FIGURE 2

Immuno-electron localization of D-Asp in human spermatozoa from normospermic donors. This is a typical example of human spermatozoa from normospermic donors, treated with (**A**) an antibody against D-aspartate as primary antibody, and as secondary antibody, the antirabbit IgG coupled to gold particles (in this case, the gold particles are well established as being localized in both acrosome and nucleus, demonstrating that D-Asp occurs in both acrosome and nucleus); or (**B**) omission of antibody against D-aspartate (control). Magnification, ×60,000. Scale bar = 0.66 µm.



D'Aniello. D-aspartic acid in human spermatozoa. Fertil Steril 2005.

The presence of D-Asp was confirmed by comparing the HPLC peak before and after enzymatic oxidation with D-AspO, where complete disappearance of the peak indicated its specificity for D-aspartic acid[12]. Analyses were performed both at the Zoological Station "A. Dohrn" (Naples, Italy) and at Barry University (Miami, USA), and mean values were obtained by comparing results from the two laboratories. This dual-site evaluation ensured reproducibility and accuracy of the data.

SEMEN ANALYSES

Semen samples were obtained from patients by masturbation under hygienic conditions and collected in sterile containers. Each sample was taken after a minimum of three days of sexual abstinence. Semen collected prior to treatment served as the control. After liquefaction at 37 °C, ejaculate volume was measured, and part of the sample was stored for future analysis of D-aspartate content, while the remainder was used for assessing sperm concentration and motility[14]

Sperm concentration was measured using a Neubauer haemocytometer chamber maintained at 37 °C. To immobilize spermatozoa and ensure uniform distribution, samples were mixed with a formalin–sodium bicarbonate solution. At least 200 spermatozoa were counted for each analysis. If the semen contained very high sperm counts, appropriate dilution was performed before analysis.

Sperm motility was evaluated using a Computer-Assisted Semen Analysis (CASA) system, which provides a quantitative assessment of sperm movement. Motility was classified into:

1. **Rapid progressive motility** (spermatozoa moving actively in a straight line or large circles with speed
>25 µm/sec at 37 °C),
2. **Moderate motility** (spermatozoa with limited progression or moving in small circles), and
3. **Slow or non-progressive motility** (spermatozoa showing slow linear, zigzag, or in-place vibration movements).

Each analysis was performed simultaneously by two biologists, and if results were inconsistent, the test was repeated. To allow accurate comparisons before and after treatment, corrections were made for differences in ejaculate volume. Samples with moderate to severe agglutination of spermatozoa were excluded from the study. Morphology and vitality assessments were carried out according to WHO guidelines.

LINK BETWEEN D ASPARTATE AND FRUCTOSE ELEVATION

D-aspartate plays a pivotal role in regulating male reproductive function through its influence on the hypothalamic–pituitary–gonadal axis.By stimulating the release of gonadotropin-releasing hormone (GnRH) in the hypothalamus, D-aspartate enhances luteinizing hormone (LH) secretion from the pituitary gland. Increased LH stimulates Leydig cells in the testes to produce testosterone.

Testosterone is a key regulator of seminal vesicle activity, promoting the synthesis and secretion of fructose into seminal plasma. Since fructose serves as the principal energy substrate for spermatozoa, its elevation directly supports flagellar motion and progressive motility.

Several studies have demonstrated that oral or in vitro administration of D-aspartate not only raises circulating testosterone levels but also significantly increases seminal fructose concentration. This dual effect—hormonal stimulation and metabolic enhancement—explains the improvement in sperm motility and fertilizing capacity observed in treated subjects.

III. RESULTS

The findings of this investigation showed that sodium D-aspartate administration markedly increased spermatozoa concentration and motility in both oligo-asthenozoospermic and asthenozoospermic patients. Regarding the concentration of sperm, we discovered that the mean number of spermatozoa in oligo-asthenozoospermic patients increased by 2.0 times ($P < 0.001$) from a baseline of 8.2 ± 4.5 million/ml of seminal plasma to 16.5 ± 5.5 million/ml. A 1.52-fold increase ($P < 0.001$) in the overall concentration of spermatozoa per ejaculate was also noted, rising from 30.1 ± 14.7 million/ejaculate to 45.9 ± 24.4 million/ ejaculate.The asthenozoospermic patient group showed a similar enhanced phenomena, with a significant rise in spermatozoa numbers from a basal value of 29.9 ± 5.7 million/ml of seminal fluid to 48.7 ± 12.6 million/ml, a 1.63-fold increase ($P < 0.001$). Additionally, there was a substantial 1.57-fold increase in the total number of spermatozoa/ejaculate, from 97.1 ± 36.1 million/ejaculate to 153.0 ± 47.0 million/ejaculate ($P < 0.001$). (Table 1).

Table 1. Sperm concentration in oligo-asthenozoospermic and asthenozoospermic patients treated with sodium D-aspartate.

1st group: 30-Oligo-Asthenozoospermic patients			
Concentration of spermatozoa/ ml of seminal plasma (n. × 10^6)		Total concentration of* spermatozoa/ejaculate (n.×10^6)	
Before Treatment	After Treatment	Before Treatment	After Treatment
8.2 ± 4.5	16.5 ± 5.5	30.1 ± 14.7	45.9 ± 24.4
P < 0.001		P < 0.001	
2nd group: 30-Asthenozoospermic patients			
Concentration of spermatozoa/ ml of seminal plasma (n. × 10^6)		Total concentration of* spermatozoa/ejaculate (n.×10^6)	
Before Treatment	After Treatment	Before Treatment	After Treatment
29.9 ± 5.7	48.7 ± 12.6	97.1 ± 36.1	153.0 ± 47.0
P < 0.001		P < 0.001	

*Refers to the total number of spermatozoa obtained by multiplying the spermatozoa concentration/ml of ejaculate by total volume of the ejaculate. The mean and SD were obtained by one-way ANOVA with repeated measurements (Stat-View program). The P value was obtained by comparison of the data of each group of subjects between before and after D-aspartate treatment.

Both oligo-asthenozoospermic and asthenozoospermic patients had a markedly increased spermatozoa motility, according to our findings. The rapid progressive sperm motility in the oligoasthenozoospermic subjects group increased from 15.5% ± 4.4% to 23.1% ± 4.7% (1.49 fold increase, P < 0.001), and the total sperm motility increased from 30.6% ± 9.3% to 54.4% ± 9.7% (1.77-fold increase, P < 0.001) (Table 2).

Both the rapid progressive motility and the total spermatozoa motility significantly increased in the group of asthenozoospermic participants after receiving sodium D-aspartate treatment. The treatment actually caused a 1.86-fold increase in rapid progressive motility (P < 0.001) in this instance, from a baseline of 11.6% ± 3.9% prior to treatment to 21.6% ± 7.5% following treatment, and a 1.81-fold increase in total sperm motility from 25.2% ± 8.1% to 45.7% ± 10.9% (Table 2).

Table 2. Sperm motility in oligo-asthenozoospermic and asthenozoospermic patients treated with sodium D-aspartate.

1st group: 30-Oligo-Asthenozoospermic patients			
Rapid progressive* spermatozoa motility (% of total)		Total spermatozoa** motility (% of total)	
Before Treatment	After Treatment	Before Treatment	After Treatment
15.5 ± 4.4	23.1 ± 4.7	30.6 ± 9.3	54.4 ± 9.7
P < 0.001		P < 0.001	
2nd group: 30-Asthenozoospermic patients			
Rapid progressive* spermatozoa motility (% of total)		Total spermatozoa** motility (% of total)	
Before Treatment	After Treatment	Before Treatment	After Treatment
11.6 ± 3.9	21.6 ± 7.5	25.2 ± 8.1	45.7 ± 10.9
P < 0.001		P < 0.001	

The patients in this study were the same patients reported in table 1. *Rapid progressive spermatozoa motility indicated those spermatozoa with a forward motility >25 µm/s measured at 37°C, expressed as the percent of rapid progressive spermatozoa motility on total spermatozoa. **Total spermatozoa motility indicate the sum of rapid progressive moving spermatozoa, the slow progressive moving spermatozoa, the zigzag moving spermatozoa and the vibrating in situ moving spermatozoa and are expressed as total motile spermatozoa on the total. The mean and SD were obtained by one-way ANOVA with repeated measurements (Stat-View program). The P value was obtained by comparison of the data of each group f patients between before and after D-aspartate.

The highly substantial increase in pregnancies among the partners of the treated patients is an intriguing feature of this study. In actuality, 16 of the 60 partners of participants receiving sodium D-aspartate became pregnant, representing a 26.6% pregnancy rate.

As an example, of the 30 partners of the oligoasthenozoospermic patients, two became pregnant during the second month of their partners' D-aspartate treatment, and four became pregnant during the third month of their partners' D-aspartate treatment. Of the 30 partners of the asthenozoospermic patients, however, two became pregnant after one month of treatment, four after two months, and four after three months. This represents a total of 10 pregnancies (Table 3).

Table 3. Number of pregnancies occurring to the partners of oligo-asthenozoospermic patients and asthenozoospermic patients before and after treatment with sodium D-aspartate.

Numbers of pregnancies occurring to the partners of the 30 oligo-asthenozoospermic patients				
Before D-aspartate Treatment*	After D-Aspartate treatment			
	After 1 month	After 2 months	After 3 months	% of total*
0%	0%	2%	4%	20%

Numbers of pregnancies occurring to the partners of the 30 asthenozoospermic patients				
Before D-aspartate Treatment*	After D-Aspartate treatment			
	After 1 month	After 2 months	After 3 months	% of total*
0%	2%	4%	33%	20%

*Before treatment indicate the number of pregnancies occurred during the last two years before starting the D-aspartate treatment. After treatment indicate the number of pregnancies occurred to the partners of subjects after the first, the second and the third month of an daily oral dose of D-aspartate.

The study's final intriguing finding relates to the D-Asp concentrations in the seminal fluid both before and after therapy. Prior to and following oral administration of sodium D-aspartate at the dosage previously recommended, we assessed each patient's seminal plasma's D-Asp concentration,The findings showed that after receiving D-Asp therapy, the concentration of this amino acid increased noticeably. The mean D-Asp concentration in oligoasthenzoo-spermic patients was actually 15.0 ± 4.0 nmol/ml of seminal plasma prior to treatment, but it rose to 29.5 ± 7.2 nmol/ml (1.96-fold increased, $P < 0.001$) following treatment. Prior to treatment, the D-aspartate concentration in asthenozoospermic individuals was 16.3 ± 4.5 nmol/ml of seminal plasma; following treatment, this value rose to 32.6 ± 7.5 nmol/ml (a 2.0-fold increase, $P < 0.001$) (Table 4).

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Table 4. D-Aspartic acid concentration in seminal plasma of patients before and after treatment with sodium D-Aspartate (nmole/ml).

	Before treatment	After treatment
1st group		
30 Oligo-Asthenozoospermic patients		
Mean ± SD	15.0 ± 4.0	29.5 ± 7.2
Student's t-test	P < 0.001	
2nd group:		
30 Asthenozoospermic patient		
Mean ± SD	16.3 ± 4.5	32.6 ± 7.5
Student's t-test	P < 0.001	

The determination of D-aspartic acid was carried out by HPLC by using the OPA-NAC method [19]. The statistical data (mean, SD and P value) were obtained by using the one-way ANOVA, with repeated measurements (Stat-View program).

concentrations that were found to be increased between 1.3 - 1.6 fold compared to their basal levels in the D-aspartate group. However, the increased levels of LH and testosterone observed in this study were in agreement with the previously reported results [18] demonstrating that D-aspartate has the capacity to increase LH and testosterone blood levels. Therefore, D-aspartate treatment for a prolonged time (90 days) is in no way harmful to health.

As previously mentioned in the methodology section, every patient who had received D-aspartate treatment had a complete blood clinical study to confirm whether the medication could cause undesirable side effects. In addition to the total blood and platelet count, none of the blood metabolites were below the healthy range.

The sole change was in the LH and testosterone concentrations, which were found to be 1.3–1.6 times higher than their baseline values in the D-aspartate group. D-aspartate has the ability to raise blood levels of LH and testosterone, as seen by the higher levels of both hormones seen in this investigation, which were consistent with previously published findings [18]. Therefore, long-term (90 days) D-aspartate medication is not in any way damaging to health.

Figure 1 illustrates the impact of supplementing mTALP medium with fructose (1.0 mmol) and glucose (5.0 mmol) on progressive motility. Fructose-treated spermatozoa were shown to have a considerably ($P < 0.05$) larger percentage of spermatozoa exhibiting increasing motility and hyperactivation than simply glucose-treated spermatozoa.

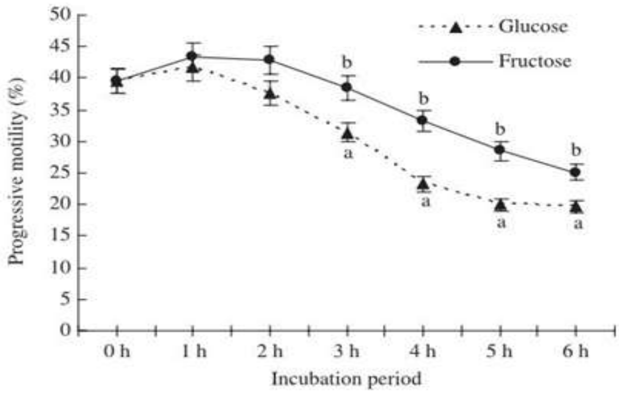

Figure 1 Effect of fructose (1.0 mmol) and glucose (5.0 mmol) supplementation in mTALP medium on progressive motility of boar spermatozoa incubated for 0 – 6 h. Values are expressed as means ± SEM values from five replicates. Different letters indicate significant differences ($P < 0.05$) in the same incubation period.

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These sugars may have distinct functions in controlling in vitro spermatozoa function, according on the progressive motility seen following incubation with either glucose alone or glucose and fructose. Notably, glucose plays a special role in spermatozoa hyperactivation. Our findings indicate that the combination of fructose and glucose improves progressive motility more than glucose alone.

Only after losing their acrosomal caps may actively motile spermatozoa fertilize eggs. An acrosome reaction might be interpreted as a sign that the spermatozoa have finished capacitation and gained the capacity to fertilize eggs.

An increased sperm acrosome reaction in the presence of sugars may promote a better penetration rate through a higher collision frequency, or it may influence the eggs rather than the sperm if glucose or other sugars are present during the fertilization step.

For boar sperm to successfully fertilize, glucose is necessary. 30 Additionally, the current investigation discovered that the combination of fructose and glucose significantly affected the ability to fertilize.

IV. CONCLUSION

This study demonstrated that supplementation with sodium D-aspartate in sub-fertile men significantly improved sperm quality, as reflected by increased sperm concentration and motility, and consequently led to higher pregnancy rates in their partners[18] The effect is likely mediated through stimulation of testosterone production in the testes, which enhances spermatogenesis and maturation of spermatozoa[16][21][22].

Glucose and fructose together improve spermatozoa motility, acrosome response, and penetration rate. Consequently, it is proposed that the TALP medium can be supplemented with a combination of glucose and fructose to enhance the boarspermatozoa's capacity for fertilization, acrosome response, and progressive motility [35]

In addition, several micronutrients and antioxidants—such as vitamins A, C, D3, E, B-complex, folic acid, selenium, zinc, manganese, copper, L-carnitine, L-arginine, coenzyme Q10, lycopene, grape seed extract, N- acetyl cysteine, astaxanthin, and ginseng extract—have been reported to improve semen quality by reducing oxidative stress and DNA fragmentation, supporting mitochondrial function, and enhancing sperm motility[3] [11]. The synergistic action of these agents may provide additional benefits in male infertility management.

Regarding safety, treatment with sodium D-aspartate for 90 days showed no adverse clinical or biochemical effects, with only a physiological rise in LH and testosterone observed[18]. Similarly, antioxidant vitamins and amino acids used in fertility supplements are generally considered safe at recommended doses, although excessive intake may occasionally cause gastrointestinal discomfort or metabolic imbalance[7][9]. Sodium D- aspartate supplement improves fructose level in semen and also sodium D-aspartate in combination with antioxidant vitamins and amino acids—represents a safe and effective therapeutic approach to improve sperm quality and fertility potential in sub-fertile men and to improve fructose level.

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